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Prolapse of gastric mucosa

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PROLAPSE OF GASTRIC MUCOSA

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INTRODUCTION.

The purpose of this paper is to present a survey and a resume of the condition described as Prolapse of Redundant Gastric Mucosa. Increasing attention has been directed toward this subject in the past few years with the result that an increasing number of such cases have appeared in the literature. It is felt quite certain that an awareness on the part of the medical profession will lead to the conclusion that this disease entity is certainly more common than is generally thought.

In the following pages an attempt will be made to briefly review the historical aspects of this subject. Statistics of this entity and its statistical relationship to other gastro-intestinal diseases will be discussed under incidence. The different theories as to the etiology will be dealt with as thoroughly as possible for in this way greater understanding of the pathology as well as of the symptomatology result. Physical findings will be discussed though the subject is somewhat limited. Of the laboratory and other diagnostic procedure findings, the roentgenological findings are of a high order of importance and will be dealt with accordingly. The several conditions closely resembling the roentgenological as well as the other aspects of this condition will be mentioned. The whole will be summarized and a number of conclusions drawn.

HISTORICAL.

Von Schmieden (1) first reported a case in 1911 but the report apparently aroused little interest among the medical profession in general and the roentgenologists and gastroenterologists in particular. The entity was apparently not recognized in this country until 1925 and 1926 when Eliason and Pendergrass and Wright (2) were the first group to call attention to it. In fact, up until 1943 there were only 19 cases found to be reported in the literature after an exhaustive search by Melamed and Miller (3). Table I tabulates chronologically the early reports of this condition.

Author	Year	Cases
Von Schmeiden	1911	1
Eliason and Wright	1925	1
Eilason, Pendergrass and Wright	1926	2
Shiflett	1932	1
K. A. Meyer and Singer	1935	1
A. H. Meyer	1935	1
Pendergrass and Andrews	1935	3
Rees	1937	4
Bohrer and Copleman	1938	1
Archer and Cooper	1939	4
Rubin	1942	1
Malamed and Miller	1943	1

TABLE I.

Following the impetus supplied by the publication of 14 by Scott (4) in 1946, the number of cases reported in the literature has markedly increased. Manning and Highsmith (5) in a review of the literature in 1947 found 37 cases reported.

Artz and Gants (6) in a survey of the literature up to June, 1950, found more than 100 cases reported in the literature. This author knows of one group which has an article in preparation for publication. Rees (7) states that the number of cases reported are increasing in proportion to the number of patients suffering from gastric complaints who are subjected to roentgenological examination by means of an opaque meal. Manning and Highsmith (5) point out that as the syndrome has become more widely recognized and the criteria for diagnosis more clearly defined, it appears that this condition is not at all rare. Undoubtedly the recognition of the possibility of this lesion will lead to the diagnosis of more cases. Archer and Cooper (8) relate that one of the examiners of the American Board of Radiology had films of such a case and only one person of the entire group examined at one session of the Board diagnosed this condition correctly. They further state that it is probably overlooked more frequently than is generally recognized.

There are those who do not believe in the identity of this entity and their status in the field of gastroenterology is to be certainly clearly recognized. Bockus (9) still believes that only severe prolapse associated with gastritis can cause symptoms, and then only after producing partial pyloric obstruction. Alvarez (10) states that it usually has no practical significance. It has been the experience of one investigator (4) in past few years that the characteristic filling defects seen on

roentgenological examination of this condition are often misinterpreted and confused with other gastrointestinal tract pathology. There are others who share this opinion and their opinions and findings will be shown subsequently.

INCIDENCE.

Statistics regarding the incidence of prolapse of gastric mucosa vary with the author and report. Archer and Cooper (8) state that the condition is not rare but do not give figures. Feldman (11) states that the condition occurs in approximately 0.1% of the patients with gastrointestinal tract disease. Morgore and Shuler (12) state that at the United States Naval Hospital, Seattle, Washington, several patients who had previously been labeled as malingerers were found to have extrusion of the gastric mucosa into the pylorus as a basis for their complaints. In a recent article Artz and Gants (6) presented the following statistics as shown in Table II Page 8

Rees (7) divides lesions of this character into two groups. In the first group he includes tumors of the stomach in which a polypoid mass is engaged by peristaltic action and carried through to the duodenum. In the second group are those in which the gastric mucosa of the antrum without tumor is loosened and carried through the pylorus. In breaking down the figures of Scott (4) in which he considers only lesions classified as being in the second group above, we note the following as is shown in Table III.

Author	Clinic	X-ray Upper G.I.	Prolapsed Mucosa	Incidence
Rees	San Diego Co. Hosp. Rees Steely Clinic	5550	6	0.19 %
Scott	U.S.N.	1346	14	1.04 %
Ferguson	Grady Memorial Emory Uni. Hosp.	297	23	7.7 %
Van Noate, et.al.	Walter Reed	960	10	1.04 %
Cove and Murphey	Murphey Clinic Stockton, Calif.	650	22	3.38 %

Table II.

TOTAL HOSPITAL ADMISSIONS.	19,228	
Upper G.I. Series	1,346	
Gastric ulcer	13	(0.96%)
Duodenal ulcer	325	(24.1%)
Duodenitis	17	(1.3%)
Prolapse	14	(1.03%)
Gastric tumors	0	(0.0%)

TABLE III.

It should be pointed out that the above group includes only healthy young males who had previously passed Service physical examinations. Attention is directed to the incidence of Gastric Ulcer compared with Prolapse. The incidence of 1.04% is higher than that reported by Rees (7) who found prolapse in 4 out of 300 consecutive upper gastrointestinal tract roentgenologic examinations. Van Noate, Arnold, and Palmer (13) point out that prolapse is occasionally found on roentgenologic examination of patients in whom no other explanation of upper gastrointestinal symptomatology is demonstrable. They (13) found 10 cases of prolapse of gastric mucosa in 960 patients studied by means of upper gastrointestinal tract roentgenologic examinations, excluding three of these cases because of the coexistence of duodenal ulcer, carcinoma of the pancreas and cholelithiasis. Appleby (14) found 7 cases of prolapse in 594 cases of gastric resection. An analysis of his findings is shown in Table IV.

GASTRIC RESECTIONS

Ulcer	406
Malignancy	106
Prolapsing Mucosa	7
Others	19
Total	594

TABLE IV.

In this connection, it is his opinion that this condition is not infrequently overlooked at surgery due to the fact that under anesthesia, the prolapses are nearly all reduced and the stomach to all gross appearances is normal.

ETIOLOGY.

Just as the statistics on the incidence of the condition vary, variation as to the etiology is also evident. Suffice it to say that there are several possible eticlogical factors and for convenience they can be classified into the following three groups.

A. Irritation, hypertrophy and mechanical prolapse.

B. Narrowing of pylorus, hyperperistalsis, loosened mucosa and prolapse.

C. Neuorgenic stimulation, abnormal peristalsis and forced prolapse.

Eliason and Wright (15) originated the theory of chronic irritation and believed that physical, nutritional, functional, chemical or bacterial agents, singly or in combination could act as chronic irritants and so bring about this condition. These agents produce a low grade inflammation of the antral mucosa which leads to the production of hypertrophic folds which enlarge and elongate through the pyloric ring on mechanical stimulation from hyperactive peristaltic waves and the pressure of the gastric contents as they are forced on the way to the pylorus. This results in a redundancy of the antral gastric mucosa which prolapses through the pyloric canal into the duodenum and in some cases in the formation of single or multiple polyps of the prolapsed gastric mucosa. Bralow and Spellberg (16) believe that the irritation is a be-

nign peptic ulceration located at the base of the duodenal bulb or in the prepyloric area. The observations of Golden (17) and of Forrsell (18) on the gastric mucous membrane and the studies of Golden (17) on antral systole throw further light on the possible etiology of this condition. The observation (17) that the gastric mucosa is freely moveable over the muscle wall is readily seen at the necropsy table. It has been noted in some cases that the mucosal folds in the antrum run irregularly transverse to the long axis of the stomach, and during antral systole can be observed to change direction and run parallel with the long axis, apparently as the result of a cephalad movement with tightening of the mucosa beneath the muscular contraction. This mucosal tautening is due, in part, at least, to contraction of the muscularis mucosa. Should this tautening and cephalad movement of the mucosa fail to occur in antral systole, the transverse folds are exaggerated and tend to be forced by the pressure of the gastric contents and the muscular contraction of the stomach toward the pylorus, the extent of the mucosal involvment depending upon the degree of redundancy of the mucosa. It is possible that inflammatory infiltration of the muscularis mucosa, interfering with its normal contractility, as well as the mechanical effects of thickening and hypertrophy play a role in the etiology of prolapse of the gastric mucosa. Irritability and spasm of the antrum may further increase the mucosal redundancy. Golden (17) and Vaughan (19) have noted the frequency of antral gastritis. Janning and Highsmith (5) believe

that antral gastritis is an important etiologic factor in the development of prolapse of gastric mucosa into the duodenum in some cases, and that in the presence of a hypertrophic antral gastritis, the subsequent development of prolapse of the gastric mucosa can often be predicted. On the basis of gastroscopic studies, other investigators, (13) believe that the theories of etiology which presuppose chronic inflammation of the antral mucosa are not correct and are not acceptable to them. Manning and Highsmith (5) are lead to the opinion that chronic hypertrophic gastritis from any cause involving the antral mucosa, with interference with the normal mechanism of antral systole as described by Golden (17) resulted in the development of prolapsing redundant mucosa in a considerable number of the 16 cases which they report. Zacho (20) regards the abnormal laxness and redundance of the gastric mucosa as the pathologico-anatomic basis for this disease. He likes the term "relaxation" of the gastric mucosa, and feels that the condition is far more frequently due to the presence of polypoid tumors and points out that the chronic inflammation and hypertrophy which he noted in some of his cases could be regarded as a consequence of frequent incarcerations into the pylorus. Zacho (20) feels that the attending hypertrophy of the pylorus could be due to the increased work of drawing the gastric mucosa down into the duodenum and as the mucous membrane became loosened still more, it would be more easily caught by peristalsis and in this manner a vicious cycle develops which

rise to attacks increasing in frequency and severity.

Rees (7), a proponent of the factor of narrowing of the pylorus, has observed that narrowing of the pyloric lumen has resulted in stimulation of hyperperistalsis producing loosening of the muscularis mucosa on the muscularis with resultant trauma, hypertrophy, and prolapse, and still later, polypoid degeneration. The development of gastritis in these cases is considered secondary.

MacKenzie, Macleod, and Bouchard (21) find little to support either of these theories of mechanism and suggest that the role of emotional factors as emphasized by Scott (4) seemed of possible importance in the production of redundant mucosa. The latter feels that the necessary structural conditions are inherent in normal stomach and prolapse occurs only after fibers in the flexible submucosa have been stretched and loosened by gastric peristalsis. The increased motility is initiated by neurogenic or chemical stimuli produced by worry, excitement, irregular daily schedule or excessive use of coffee, tobacco, or alcohol. He believes that such psychosomatic stimulation contributes more to prolapse than does pre-existing disease.

Scott (4) found that 4 of his cases were 20 to 29 years of age, 7 cases were 50 to 59, and 3 cases were 40 to 49 years of age. Bralow and Spellberg (16) in reporting 4 cases noted that all were over 40 years of age. Cove and Murphey (22) found that the average in a series of 20 patients was 49 years of age with a range from 26 to 64 years of age.

In the series reported by Cove and Murphey (22) it is noted that 14 of the patients were males and 6 were females. In the cases reported by Melamed and Hiller (3), Bralow and Spellberg (16) and Ferguson (23), a majority of the patients were males.

Cove and Murphey (22) have noted that in their series of 20 patients, 12 were obese and 6 were overweight. They noted over-indulgence in alcohol in 9 cases and in several of the cases reported by Ferguson (23) exacerbations of symptoms were noted with over-indulgence in alcohol.

PATHOLOGICAL FINDINGS.

At operation Rees (7) noted that the prolapsed portion of the gastric mucosa appeared as a loose collar of redundant hypertrophic mucosa that had invaginated into the duodenum and could be forced back into the stomach to protrude as a long rosette. The prolapsed folds appeared as normal mucosa, soft and pliable and not fixed, thick, or indurated. As has been previously stated narrowing of the pylorus was noted (7). Artz and Gants (6) have called attention to redundancy and increased mobility of the antral mucosa with varying degrees of hypertrophy and chronic inflammation. Appleby (14) noted that anatomically the gastric mucosa was loosely attached by the areolar submucous tissue and that relaxation of this layer permitted the mucosa to stretch. The duodenal mucosa on the other hand was intimately attached so that a fulcrum was created at the junction of the fixed duodenal with the loosely attached gastric mucosa. Appleby points out that, " Over this fixed circumference the mucosal cuff cascades." He further points out that surgeons who perform the familiar Ramstedt operation for pyloric stenosis in infants are aware that accidental perforation of the mucosa invariably occurs at this fixed junction toward the duodenal end of the incision. Ferguson (23) points out that the mobility of the mucosa on the muscularis is greater than normal and that there is a redundancy of the mucosa of the pyloric

end of the stomach with greatly hypertrophied rugae. Observations at autopsy (23) have shown that the mucosa of the normal stomach is moveable on the muscularis, but in no case sufficiently mobile to allow it to be drawn down into the duodenum. In one case reported by this investigator (23) there was such marked redundancy and mobility of the prepyloric mucosa on the muscularis that the mucosa could be drawn down into the duodenum 6 to 7 centimeters beyond the pylorus. In this case the pyloric muscle was greatly hypertrophied, but the lumen of the pylorus was apparently normal, and the mucosa could be passed in and out of it without difficulty. In this case the prolapsed mucosa could be felt through the duodenal wall, but this finding is quite infrequently seen according to Ferguson (23).

Microscopically a variety of findings have been reported. Artz and Gants (6) called attention to redundancy and increased mobility of the antral mucosa with varying degrees of hypertrophy and chronic inflammation. Also noted was some evidence of chronic gastritis with congestion of the vascular channels and lymphocytic infiltration of the lamina propria. Norgore and Shuler (12) have observed round cell infiltration of the mucosa with germinal center formation. Melamed and Hiller (3), found, in addition, submucosal involvement with masses of acute and chronic inflammatory cells. MacKenzie, McLeod, and Bourchard (21) described simple mucosal inflammation plus some small hemorrhages. Some authors might prefer to describe this picture as one of gastritis. Schindler,

Necheles and Gold (24) have shown that such histological pictures may be produced by the mere use of clamps and ligatures in gastric surgery. Scott (4) noted that in the 5 of his cases which were operated on that microscopic examination revealed a slight increase in lymphocytes, plasma cells and eosinophiles throughout the mucosa, but he did not feel the findings conclusive enough to warrant a diagnosis of gastritis. The presence of ulcers, polyps, and carcinoma has been reported. Rubin (25) reports a case of definite malignant change in a case of prolapsed gastric mucosa.

SYMPTOMS.

Due to the wide variance in the extent of the prolapsed mucosa, the patients, in general, do not present a characteristic group of complaints. As with most complaints related to the gastrointestinal tract, these complaints are rather difficult to evaluate. Bockus (9) believes that though redundancy of pyloric mucosa is not uncommon, that unless it is associated with severe gastritis or actual prolapse of gastric mucosa into the duodenum in sufficient quantity to interfere with evacuation of stomach contents, symptoms are not produced. Artz and Gants (6) consider the most consistent symptom to be that of intermittent upper abdominal discomfort which may or may not be relieved by the ingestion of food. Scott (4) found that in 10 of 14 patients, the epigastric distress was relieved by food but not by alkalies. Artz and Gants (6) further point out that at the stage in which the elongated fold of gastric mucosa is forcibly herniated into the duodenum there may be pain, epigastric distress, fullness and nausea and vomiting. Rees (7) states that periodic distress and an upper abdominal sensation of excessive fullness are frequently noted. He points out that cramp-like pain in the right upper quadrant may be severe after the ingestion of rough foods. He further states that vomiting may be a symptom with or without demonstrable obstruction. In every atypical ulcer history, variability and intermittency of the symptoms should provoke suspicior of prolapsed gastric mucosa (6), (22). Pendergrass and Andrews (26) agree

with the other investigators, (22), (13), (5) that the symptoms are never characteristic enough to permit a clinical diagnosis.

A list of the most frequently appearing symptoms would be as follows: (6), (22), (5).

1. Intermittent, epigastric distress with cramping pains.
2. Varying relationship to food.
3. Sense of fullness.
4. Bloating.
5. Heart burn.
6. Nausea and vomiting.
7. Gastro-intestinal hemorrhage.

Norgore and Shuler (12) have evolved the following classification of complaints:-

1. Periodic abdominal distress with a sensation of fullness in this region.
2. Cramp-like pains after eating, more pronounced on taking solid foods.
3. Vomiting, last sympton to occur.
4. Loss of weight, which is the logical result of the first three.

Zacho (20) reports a case in which epigastric distress and pain were brought on by bodily exertion and states that vomiting lessens or entirely eliminates the symptoms. Some patients complain of an oppressive sensation in the abdomen after large meals, (20). In a series of 7 cases, Appleby (14) found that all

of the patients followed a roughly uniform pattern and the outstanding point was that all complained of intermittent cramping pain aggravated rather than relieved by food. Appleby (14) concurs with Zacho in that while emesis was not a prominent factor, it was present at intervals in all of his cases and always brought about a measure of prompt relief. Two of his patients routinely induced vomiting to provide relief. Ferguson (23) states that the pain may be of an aching type, felt in the epigastrium and frequently radiating either under the costal margin or to the back, at times becoming prostrating. Nausea and vomiting occurred in 5 of the 6 cases reported in this series (23). In this same series, 4 of 6 had sour belches. Four of 6 also had hematemesis and melena. Pendergrass (27) first called attention to the anemia caused by ulceration and oozing. Anorexia and anemia (which Archer and Cooper (8) believe has not been adequately stressed) is a fairly common symptom. Loss of weight may be a dominant symptom (22). Melamed and Hiller (3) report a case of profuse intestinal bleeding and the resultant secondary anemia and point out that though this has been reported in other cases it is not a necessary accompaniment of the disease, Archer and Cooper (8) in reporting 4 cases state that the presenting symptom in 2 of their cases was hemorrhage, in one case occurring from the bowel and in the other case from the mouth.

Van Noate, Arnold and Palmer (1) state that the duration of symptoms prior to detection ranged from 1 to 6 years

with an average of 3 years. Cove and Murphey (22) state that the duration of symptoms in their cases was 2 to 8 years. Appleby (14) called attention to the fact that the symptoms increase in severity from year to year and in his series the average duration of symptoms before recognition was 7 years.

In relating the symptoms to the degree of prolapse, Cove and Murphey(22) noted that 6 cases with symptoms had mild prolapse, 7 cases with symptoms had moderate prolapse, and 7 cases with symptoms had marked prolapse. Four of those patients with marked prolapse were controlled poorly by diet, 2 with moderate prolapse were controlled poorly with diet, while only one with mild prolapse was controlled poorly with diet. Therefore, with severe prolapse it can be seen that it will be difficult to control the patient's symptoms by dietary management. Of 6 patients with mild prolapse, 5 had vague complaints. Of 7 patients with moderate prolapse, 5 had bothersome complaints, while of the 7 with marked prolapse, 6 had severe complaints. So from this limited number of cases it appears that the degree of prolapse correlates fairly well with the severity of the symptoms and in a rough way dictates the type of therapy to which the condition is amenable.

PHYSICAL FINDINGS.

The objective physical findings in this condition are few and they are equivocable. A palpable tumor mass in the epigastrium in the region of the pylorus has been reported by Cove and Murphey (22) in several of their cases. According to these investigators, such a mass, if present, is of a doughy consistency, associated with free mobility and with a very characteristic feeling to the examiner's fingers when the prolapse is reduced. They point out the similarity to inguinal hernia. There are no other reports in the literature of the finding of a palpable tumor mass. Mild epigastric tenderness has been noted in some of the cases, (13), (22). In three of the cases reported by Van Noate, Arnold and Palmer (13), the degree and localization would have been consistent with the findings of active duodenal ulcer. All appeared well nourished and not seriourly ill and psychiatric examination revealed no functional basis for their complaints in this series. (13).

LABORATORY FINDINGS.

Cove and Murphey (22) state that the laboratory findings are negative except for a secondary anemia. Pendergrass (27) called attention to the severe secondary anemia resulting from this condition. Blood has been noted in the stools of a number of patients, (8), (22). Manning and Highsmith (5) did not note anemia in any of the 16 cases in their series. Hyperchlorhydria has been reported in one-third of the cases composing the series of Scott (4). In the following examinations, complete blood count including differential, sedimentation rate, urine analysis, flocculation for syphilis and stool examination, Van Noate, Arnold and Palmer (13) noted the findings to be within the normal limits or negative except for a mild hypochromic anemia in a patient with a history of gastro-intestinal tract hemorrhage. They found that gastric analysis revealed hyperacidity in 1 case after histamine stimulation.

GASTROSCOPY.

Van Noate, Arnold, and Palmer (13) gastroscoped the 7 patients making up their series. Five of the patients were gastroscoped on 2 occasions, 1 on 5 occasions and 1 was gastroscoped once. The pre-examination medication included atropine, gram 0.0008, subcutaneously. All examinations included visualization of the pylorus and in each patient prolonged study of the antrum and pylorus was possible during at least one examination. In none of the cases was the diagnosis of prolapse made, nor was gastritus, tumor or other lesion of the antral mucosa found. It did not appear to these observers that the pyloric action had exerted any important traumatic effect on the prolapsed segment. In one patient chronic hypertrophic gastritis involving the proximal posterior wall and greater curvature was followed gastroscopically for 10 months, but the process did not involve the distal portion of the stomach. No features of peristaltic activity were recognized which might help to explain the mechanism of prolapse, but they noted mucosal redundancy at times. In one patient, hyperactive, but coordinate antral peristalsis with a gathering of the mucosa was found, and as successive waves passed through the antrum, it was seen that there was some redundancy of the mucosa. When the waves reached the mid-antrum, the outline of the contraction ring, rather than being fine and sharp, was encroached upon by compressed folds. These folds were

not seen to obliterate the lumen of the ring and they disappeared during diastole. As each wave approached the pylorus, it was noted that the redundancy cleared and the pylorus itself was normal in action and appearance. They go on to state that they do not believe that prolapse of gastric mucosa falls into the category of gastroscopically diagnosable diseases. Distention of the antrum by gastroscopy allows the prolapsed mucosa to return to its normal position. But gastroscopy retains its value because it will exclude antral tumor and it is important to know of the presence of secondary superficial traumatic gastritis in the prolapsed segment, especially when blood is found in the stools. If antral resection is contemplated, they (18) point out that a knowledge of the state of the proximal mucosa becomes an important consideration and finally gastroscopy may leas to an understanding of the mobility features involved.

Among the other investigators who have employed the gastroscope in this condition are Cove and Murphey (22) who state that no uniform results are obtained by gastroscopy. Manning and Highsmith (5) noted on the gastroscopic examination of one patient, antral gastritis with superficial ulceration of the prepyloric mucosa. They state that it is not possible to visualize the prolapsed fold of redundant mucosa gastroscopically, but the finding by a hypertrophic gastritis particularly in the antrum with the flexible gastroscope should be considered valuable supportive evidence for the diagnosis of prolapse. In summary it may be

said that the principal value of gastroscopy is in the ruling out of other lesions.

ROENTGENOLOGICAL APPEARANCE.

Most of the investigators agree that the appearance at fluoroscopy and on roentgen film is quite characteristic and constitutes a valuable diagnostic aid. Melamed and Miller (3) state that the roentgenologic demonstration is practically the only reliable means of diagnosis. Norgore and Shuler (12) state that many roentgenologist are not aware of the condition and, therefore, fail to diagnose this condition. Rubin (25) reported a case with proved malignant change in prolapsed polypoid mucosa so it is quite important to make a diagnosis. Pendergrass and Andrews (26) are of the opinion that the errors in diagnosis are not made by calling these lesions other diseases, but by calling other diseases these lesions.

As first described by Eliason, Pendergrass and Wright (28), the roentgenologic picture consists of a central, circular, or irregularly circular filling defect in the duodenum, usually at the base of the duodenal bulb with often elongation and narrowing of the pyloric channel. The extent of the defect appears to be determined by the amount of prolapsed mucosa. Not uncommonly they found heavy mucosal folds, especially in the antrum, often with antral spasm.

According to Rees (7) the important finding with a barium meal is that of a negative shadow containing a central opaque streak, in the duodenum. They feel that this streak differentiates it from tumor. Rockus (9) states that when

complete prolapse into the duodenum occurs, the differentiation between pedunculated tumor and prolapsing gastric mucosa will often be impossible. He points out that if there is a large defect in the pyloric canal and a negative shadow in the duodenal bulb, the condition can usually not be distinguished from that of pyloric ulcer, hypertrophic gastritis or carcinoma. Pendergrass and Andrews (26) feel that one cannot make a definite diagnosis between polyp or any other type of tumor protruding through the pylorus and a redundant mucosal fold. Therefore, they advocate the use of the term "prolapsing lesion". Melamed and Hiller (3) feel that the roentgenological differentiation between a prolapsing gastric polyp and prolapsing hypertrophic gastric mucosa is impossible. They add that in either instance one in unable to determine whether the lesion is benign or malignant. Rees (7) was able to make a roentgenological diagnosis in all four of his cases, though in one case, prolapse of the mucosa of the antrum of the stomach was observed roentgenologically prior to the first surgical procedure, but its importance was not appreciated. Gastroenterostomy relieved the gastric retention but did not permanently relieve the symptoms or halt the progress of the disease. In this case the increase in the size of the prolapsing lesion led to a later mistaken roentgenological diagnosis of a duodenal polyp which in turn resulted in an incorrect approach to the true pathological condition and a consequent operative procedure which did not relieve the symptoms or remedy the pathology.

According to Cove and Murphey (22) prolapse of redundant gastric mucosa into the duodenal bulb is represented by a central filling defect near the base of the bulb, most often lobulated and to which they apply the term of "mushroom" or "cauliflower-like". They noted that the defect is traversed by lines of increased density which can be followed into the stomach and which are continuous with the gastric rugae. Van Noate, Arnold and Palmer (13) point out that on X-ray one may note an "umbrella-like" defect at the base of the duodenal bulb. They feel that it is necessary to exclude by gastroscopy other antral lesions which might present a similar roentgenological appearance, before the roentgenological diagnosis of prolapse can be considered valid. In their series (13) they noted that gastric ulcer, gastric tumor and gastric polyp had been diagnosed previously on the basis of X-ray findings alone in 5 cases. Manning and Highsmith (5) state that the characteristic roentgenologic findings in their cases were of a circular "mushroom" or "umbrella" filling defect in the base of the duodenal bulb.

Mention was made earlier of the rather frequently occurring elongation and narrowing of the pyloric channel by one group of investigators (2). Manning and Highsmith (5), in their series, often noted elongation and narrowing of the pyloric channel and an increase in the size of the rugae of the pylorus and antrum. Heavy mucosal folds were noted in the body and fundus in some of these cases. MacKenzie, Macleod and Bouchard (21) have

stated that the redundant mucosa is responsible for an abnormal appearance of the gastric rugae in the prepyloric area. These rugae were described as more prominent than usual and lacking a usual normal pattern being oriented in an irregular arrangement instead of running horizontally and vertically. They describe a second roentgenological deviation of prolapsed gastric mucosa as that of pyloric narrowing. This abnormality occurs much less frequently than the deformity described above and occurs as a constant, persistent narrowing at the level of the pyloric antrum. In these cases they described the pylorus as being longer and perhaps broader than usual and the base of the cap showed a concave outline with or without definite filling defect. Manning and Highsmith (5) were unable to recognize this latter deviation in their series of cases. Cove and Murphey (22) noted narrowing of the pylorus in several of their cases and thought that this indicated or suggested hypertrophy.

Pendergrass and Andrews (26) do not feel that there is any disturbance in the passing of peristaltic waves. Cove and Murphey (22) found that hyperperistalsis was not constantly associated. Still other investigators believe that gastric peristalsis is more active and vigorous than is seen in the average patient (6).

Some investigators advise examination in the prone position, but the lesion may also be observed in the upright position. MacKenzie, Macleod and Bouchard (21) advise the right

decubitus position. Welamed and Miller (3) feel that the examination must include the patient in both the erect and recumbent positions. The condition is usually proved in the recumbent positions according to their investigations. Scott (4) feels that fluoroscopy and serial "spot" film technique is the examination of choice.

To summarize the observations of some of these investigators, (3), (4), (5), (7), (13), (20), (22) and (26) we may say that the filling defects in the duodenal bulb due to prolapse of gastric mucosa are invariably located in the base, immediately around the pyloric opening. The redundant folds produce a central mushroom or cauliflower-like shadow. The filling defects vary in size, shape and appearance during a single examination as well as in repeated examination, and may even become temporarily reduced. Usually the redundant rugae can be traced from the antral canal through the pyloric opening in the base of the bulb. Visualization may be enhanced by exerting moderate compression. A singly prominent gastric rugae is normal and is frequently seen extending across the pylorus into the bulb. The bulb is not quick or irritable in contra-distinction to duodenal ulcer or duodenitis. Gastric peristalsis may or may not be more active and fiborus than in the average patient. No ulcer crater, niches, or incisurae are seen, except in the instances noted in the discussion above. Fluoroscopy and serial spot film technique is the examination of choice.

The condition is most easily observed in the prone and right decubitus positions but may be seen in the erect position.

Though the differential roentgenological diagnosis is beyond the scope of this paper, brief mention should be made of some of the conditions which might lead to confusion in the roentgenographic picture. As mentioned before, prolapsing polypoid tumors of the pyloric end of the stomach may pose a difficult differential diagnosis, though the tracing of the rugal folds may be helpful. Hypertrophy of the pyloric spincter may be present. In this condition there is a curved pressure defect on the base of the duodenal bulb. Duodenitis may cause confusion, but the lack of spasm and irritability of the bulb should establish the diagnosis. In case of duodenal ulcer, the margins of the bulb are not smooth as they are in prolapse. Hypertrophic gastritis may be ruled out by gastroscopy. In the case of a polyp of the duodenum, the gastric rugae are not traced into the bulb. Kirklin and Harris (28) and Cunha (29), have listed other pyloric conditions which must be differentiated from carcinoma and which may be confusing in this condition. They are secondary hypertrophic pyloritis associated with pyloric stenosis, gastric syphilis, and cirular myoma.

TREATMENT.

Opinion is divided in the literature as to the proper mode of treatment of prolapse of the gastric mucosa. There are those who believe that the vast majority of these patients will respond to medical treatment while others believe that surgical measures are the only definitive method of treatment. According to Bralow and Melamed (30) slight and moderate prolapses respond well to frequent small feedings and a bland diet. Artz and Gants (6) concur but further state that for slight or moderate prolapse, a thorough trial on a medical regimen is indicated. They feel that unless complicated by hemorrhage or obstruction every patient should receive an adequate trial on medical therapy. Cove and Murphey (22) feel also that the vast majority will respond to medical therapy. They concur with Bralow and Melamed (30) and with Artz and Gantz (6) that frequent small feedings of a bland diet, rest in bed, freedom from tension and strain, and the elimination of all condiments such as tobacco, alcohol and caffeine are essential parts of the therapy. Other factors such as chronic upper respiratory infections and carious teeth are included by Manning and Highsmith (5). Van Noate, Arnold and Palmer do not feel that antispasmodics are helpful, though Ferguson (23) and several others feel that they are. Van Noate, Arnold and Palmer (12) point out that the patient's own observations regarding control of symptoms are helpful. They also add that for immediate relief a large glass of water may be helpful. Suffice it

to say that the medical regimen should be directed not only toward alleviating the symptoms, but also toward preventing a degree of prolapse which will require surgery.

Rees (7) states that surgical measures are indicated in case of a large prolapse since they always produce symptoms and are usually resistant to medical therapy. Another indication is the fact that they may develop large or small hemorrhages and may produce partial obstruction. MacKenzie, Macleod and Bouchard (21) took as their indications for surgery, equivocable roentgenological findings with difficulty in differentiating from prolapse, continued bleeding and evidence of pyloric obstruction. Artz and Gantz (6) state that the indications for surgical treatment are as follows: obstruction, hemorrhage, persistent symptoms after adequate medical therapy and equivocable roentgenological findings. They feel that wherever there is doubt about the presence of a pedunculated tumor, surgical exploration is required. Cove and Murphey (22) feel that when the pathology is definite enough for one to conclude that the lesion is on a mechanical basis and once established will in all probability recieve no relief short of mechanical correction, then surgery is indicated. Factors concerning surgery according to Cove and Murphey (22) are persistence, severity, intractability of symptoms, pyloric obstruction, and repeated gross gastro-intestinal hemorrhages. Norgore and Shuler (12) feel that the surgical treatment for this condition depends on the age of the patient, gastric acidity and the

skill of the surgeon. In the cases reported by Manning and Highsmith (5) surgical intervention was not considered necessary. They believe that surgery should be reserved for those patients with considerable degrees of pyloric obstruction, polyp formation with suspected malignant degeneration ulceration with recurrent severe hemorrhages not prevented by adequate medical therapy, severe anemia due to chronic blood loss or suspected malignancy. Ferguson (23) states that the curative treatment of this entity is "unquestionably" surgical and directed toward removing the prolapsing mucosa, short circuiting the diseased area, or enlarging the gastric outlet so that prolapsed mucosa can move back and forth at will without causing obstruction, or becoming incarcerated. Melamed and Miller (3) feel that, if the lesion is of sufficient size to produce symptoms or if it is the origin of gastro-intestinal bleeding, the treatment is surgical. Bralow and Spellberg (16) believe that surgery should be restricted to cases with intractable pyloric obstruction. Pendergrass and Andrews (26) believe that the treatment is almost entirely surgical.

There is also variation in the technique and methods of surgical correction, but in general the following procedures are the ones more frequently done: gastrojejunostomy, partial gastrectomy, pyloroplasty and simple excision of redundant mucosa.

Rees (7) believes that antral gastrostomy with excision of the redundant mucosa and anchorage of the remaining mucosa to the muscularis in the line of incision followed by sectioning of the

pyloric muscle is the method of choice. Manning and Highsmith (5) believe that the procedure of choice is excision of the prolapsed redundant mucosal folds with pylorosplasty. Zacho (20) believes in the simple excision of the projecting fold of mucous membrane. Appleby (14) is of the opinion that the best therapeutic results are obtained by partial resection rather than by less radical procedures. Since in Scott's (4) cases it was necessary to return the patients to active military duty as soon as possible, 56% of the patients in his series were operated on with complete relief of the symptoms and no recurrence on a full diet. Others have reported almost total alleviation of symptoms following surgical procedures. (25).

SUMMARY

That this condition may well be the underlying factor in many of the undiagnosed digestive tract disturbances is indicated by the statistics which show that prolapse of gastric mucosa is not rare. In one series prolapse occurred as frequently as gastric ulcer.

There are three schools of thought concerning the etiology of this condition which are as follows: irritation, hypertrophy and mechanical prolapse, narrowing of pylorus, hyperperistalsis, loosened mucosa and prolapse, neurogenic stimulation, abnormal peristalsis and forced prolapse.

Pathologically the condition is an abnormal mobility and redundancy of the prepyloric mucosa with prolapse through the pylorus. The microscopic findings are variable.

Though a distinctive syndrome is not recognized, prolapse of redundant gastric mucosa produces symptoms referable to the upper gastrointestinal tract that may be suggestive but are not characteristic enough to permit a clinical diagnosis. The condition should be suspected in duodenal ulcer patients with atypical histories, in patients that are refractory to an ulcer regime, and in those who have recurrences when placed on solid foods. Mild degrees of prolapse of the gastric mucosa seldom produces symptoms, while moderate or marked degrees are often associated with complaints of sufficient severity to cause the patient some degree of disability.

The roentgen examination of these patients, because there is no characteristic clinical syndrome, occupies a place of prime importance in the diagnosis of these obscure lesions. The roentgenologist should look for these cases, especially when there is a clinical picture of ulcer and no definite roentgenographic evidence. The filling defects are fairly characteristic and should not be confused with those produced by duodenal ulcers, duodenitis, or other disorders of the duodenum and pylorus. The typical filling defect in prolapse of the gastric mucosa is a negative "cauliflower-like' or "umbrella" shaped defect in the base of the duodenal bulb opposite the pylorus, varying in size and shape during a single examination and on repeated examinations. These defects can be overlooked at fluoroscopy and films should always be made. Fluoroscopy supplemented by "spot" films in the prone or right decubitus position is the most desirable method of examination. In the case of small prolapses, repeated roentgenologic studies should be made, as the mucosal pattern varies from time to time, and frequently the prolapse becomes temporarily reduced. Since large prolapse of the gastric mucosa are known to produce symptoms, are the cause of gastric hemorrhage, and may result in partial pyloric obstruction, the roentgenologist should be aware of the condition, should be able to distinguish them from other duodenal defects, and report them to the referring physician for evaluation.

The laboratory findings are inconclusive and of little help in making the diagnosis. Secondary anemia has been noted by some of the investigators of this condition but is not necessarily an accompaniment

of the disease. The principal value of gastroscopy lies in the ruling out of other disease processes, for the precedure in itself will not allow the making of the diagnosis of prolapse of gastric mucosa.

The most common complication is hemorrhage. Pyloric obstruction and severe anemia have been reported by several investigators. There is one case of proved malignant degeneration reported in the literature.

The treatment in the case of mild and moderate prolapse of gastric mucosa is medical until such time as repeated and severe attacks or complications occur. In the case of large prolapse of gastric mucosa or in case of persistent pain, hemorrhage, and obstruction, surgical measures are indicated. Surgical procedures include partial gastrectomy, pyloroplasty, gastrojejunostomy, and simple excision of redundant mucosa.

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CONCLUSIONS

1. Prolapse of gastric mucosa is more common than is generally suspected.

2. The exact etiology is unknown.

3. The pathology is an abnormal mobility and redundancy of the prepyloric mucosa with prolapse through the pylorus.

4. Mild degrees rarely produce symptoms. Moderate or marked degrees are often associated with complaints of sufficient severity to cause the patient some degree of disability.

5. The diagnosis is generally by roentgenographic methods as the clinical picture is extremely variable.

6. Complications are intractability, hemorrhage and obstruction.

7. Mild and moderate prolapse respond to medical therapy. Failure to respond or the presence of the complications enumerated above are indications for surgery.

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