PEPTIC ULCER OF THE ESOPHAGUS*

CHEVALIER JACKSON, M.D.

PHILADELPHIA

Of the various ulcerative diseases of the esophagus one kind has been termed "peptic" because of the close similarity of its postmortem aspect to that of peptic ulcer of the stomach. All of the recorded cases are based on a diagnosis made only at autopsy. The development of esophagoscopy to the degree of perfection that permits of examination of the esophagus in any patient in a few minutes, without any anesthetic, general or local, has rendered practical the study and the diagnosis of this disease in the living and has rendered its local treatment quite simple. Therefore, it may be said that a new era has begun in the study of this disease.

INCIDENCE

Pepitic ulcer has been diagnosed in eighty-eight out of more than 4,000 cases of esophageal disease in forty-two years of my experience. Of these eighty-eight cases, twenty-one were active ulcers and sixty-seven were scars probably due to preceding peptic ulcers. This, however, is not a sound basis on which to estimate the infrequency of the disease, because until recent years few opportunities have been afforded for esophagoscopy, and when done at all it has been only in the presence of symptoms of esophageal obstruction. Peptic ulcer of the esophagus is for a long time not obstructive and the symptoms are erroneously considered gastric. When the time shall have come when every patient with the slightest discomfort or abnormality in swallowing, every patient with pain or discomfort back of the sternum, every patient with gastric hematemesis, every patient with regurgitation, "heartburn" or "waterbrash" is examined esophagoscopically, peptic ulcer of the esophagus will be found less rare than it is now thought to be, though it is probably not a common disease.

ETIOLOGY

The word "peptic" applied to esophageal ulcer would seem to drag in all the etiologic factors, and, it might be added, all the etiologic controversies of peptic ulcer of the stomach. Doubtless most of the factors, accepted and disputed, are worthy of consideration in a study of peptic ulcer of the esophagus; but they are too well known to require even enumeration here. Coexistent gastric ulcer in some of the cases would suggest the possibility of a cause common to the two. As has been so clearly pointed out by Rehfuss,¹ it is necessary to consider two classes of causes: (1) those that produce the acute ulcer, and (2) those that perpetuate the acute ulcer into the chronic form in which it is usually encountered. Doubtless the same is true of esophageal ulcer.

The Causes of Acute Ulcer of the Esophagus.—The trend of opinion of gastro-enterologists toward infection as a cause of gastric ulcer led me to review the histories in our cases of esophageal ulcer in a search for data bearing on infective foci. Fortunately our routine of fully recording the condition of the mouth, teeth, nose and throat of every patient enabled me to obtain interesting and almost complete data. Of the twenty-one patients with active peptic esophageal ulcers, nineteen (90 per cent) had evidence of a chronic infective focus. In the two others, no infective foci were found; the patients both had very unclean mouths, however, with large accumulations of septic materials about the teeth, though the roentgen examination did not reveal any peridental focus. Of the nineteen patients there was more than one focus in seventeen (89 + per cent). The tonsils were diseased in fifteen (71 + per cent). There was disease of the nasal accessory sinuses in seven (33 + per cent). In six (28 + per cent) there were infective foci at the roots of the teeth. In the sixty-seven cases in which the diagnosis of healed peptic ulcer had been made, the results of the search of our clinical records for data bearing on focal infections revealed similar results. Of the sixty-seven patients, fifty-eight (88 + per cent) had the disease or had had a tonsillectomy; three of the remainder had carious teeth or foul mouths. This made sixty-two (92 + per cent) out of sixty-seven who had evidence of focal infection. When the two classes of cases are combined, there is evidence that eighty-one out of eighty-eight (92 + per cent) patients with peptic ulcer of the esophagus had signs of focal infections in the tonsils, teeth or nasal sinuses. These observations in the records were so striking that it seemed advisable to obtain data for control. For this purpose our cases of suppulsive disease of the lung would not serve because these patients almost all have or have had focal infections in the tonsils, teeth or sinuses. Cases of foreign body, on the other hand, being mere accidents, would give a fair average incidence of focal infections unrelated to the condition for which the patient was admitted.

Of the eighty-eight patients over 18 years of age with foreign bodies in the air and food passages, eleven (12 + per cent) had tonsillar disease or had had tonsillectomy; three (3 + per cent) were recorded as having sinus disease without tonsillar disease. This made fourteen instances of evidence of focal infection in eighty-eight persons practically selected at random (15 + per cent). Due allowance being made for the fact that possibly the patients with foreign body were not studied as closely for focal infections as were those with peptic ulcer of the esophagus, nevertheless the contrast between the incidence of focal infection in 92 + per cent of patients with peptic ulcer of the esophagus, as compared to evidence of similar foci in 12 + per cent of persons practically selected at random, is overwhelmingly in favor of focal infection as the chief cause of peptic ulcer of the esophagus.

Perpetuation of Acute Ulcer.—In the case of gastric ulcer the perpetuating factor resulting in chronicity is commonly accepted as the gastric juice. Peristalsis and other factors are considered supplementary.

Retrograde Flow of Gastric Juice.—When one comes to apply the chief perpetuating factor in gastric ulcer to the etiology of peptic ulcer of the esophagus, one is at once confronted by the fact that food and the gastric juices as composed during active digestive processes do not constantly and normally bathe the thoracic esophagus, even at its lowermost end. That a retrograde flow may occur in normal persons is undoubted, and that it does occasionally occur in every one seems probable. It certainly is the active cause of the characteristic pain of peptic ulcer of the esophagus whether it has originally caused the ulceration or not. The disproved theory that peristalsis is the cause of the pain of gastric ulcer seems illogical as well as unproved, as applied to the esophagus. Malfunction of the stomach, hour-glass deformity, pyloric stenosis, hepatic disease,
and duodenal and other intestinal disorders are conceivable causes of retrograde flow, but our records of cases of peptic ulcer of the esophagus do not afford corroborative evidence of them as important etiologic factors.

Preventriculosis (so-called cardiospasm).—This furnishes strong evidence that something other than irritation and stasis is necessary for the causation of peptic ulcer of the esophagus. When one considers the prolonged irritation and maceration of the mucosa of the esophagus from the retention, for days and even weeks, of fermenting foods, in so-called cardiospasm, it is remarkable that in 287 very severe cases of this disease that we have studied esophagogoscopically not one case of peptic ulcer was found. Erosions were common. There was one case, in an early stage, with slight peptic esophageal ulcer, but it seemed probable that the ulcer had antedated the incoordination of the diaphragmatic pinchcock, and that the two diseases were unrelated.

Insufficiency of the Cardia.—Given as a cause in the older textbooks, this condition is at once excluded from consideration here because there is no sphincteric action, sufficient or insufficient, at the anatomic cardia. Kinking of the abdominal esophagus is slightly obstructive of the retrograde flow of the gastric contents into the esophagus; but the true proximal sphincter of the stomach is the diaphragmatic pinchcock.

Insufficiency of the Diaphragmatic Pinchcock.—It is curious to note in all of the cases hitherto reported, chiefly autopsic, that insufficiency of the cardiac sphincter was stated as a cause apparently on purely theoretical considerations, notwithstanding the fact that there was no anatomic basis demonstrable in the way of the existence of a cardiac sphincter, sufficient or insufficient. As the sphincteric action prevents regurgitation of the stomach contents is at the diaphragm and not at the cardia, the theory of insufficiency, if applied at all, must apply to the diaphragmatic pinchcock. None of our patients with peptic ulcer of the esophagus were found to have any abnormal patulousness at either the diaphragmatic pinchcock or the cardia. On the other hand, in patients with abnormal patulousness and in some instances hernia of the stomach through a large deficiency in the hiatus esophageus, no ulceration or even irritation of the mucosa was noted.

Islands of Gastric Mucosa.—The presence of islands of gastric mucosa in the lower end of the esophagus is probably a factor, but that it is an essential factor is unproved. Even if no islets were to be found at autopsy it could not be proved that one did not originally exist at the site of the ulcer which had completely destroyed it. If the islands are present in all cases, it still remains undecided whether the aberrant gastric mucosa is passively etiologic by reason of supplying vulnerable tissue or actively by the peptic and especially the oxyntic secretions of its glandular elements. That the pain in peptic ulcer of the esophagus is due to regurgitated acid secretions from the stomach seems certain; but this does not imply that the ulcer was caused by such regurgitations. The ulcer, whatever its cause, would be sensitive to acid contact. Moreover, it seems probable that slight retrograde flow of gastric contents occurs unnoticed occasionally in every human being that the acid could be produced in the esophagus itself is not impossible in view of the fact that islands with villous glandular structure, columnar and hydrochloric acid-producing cubical cells have been found to exist in the esophagus. This has been true in seven of our twenty-one cases of peptic ulcer at the bronchoscopic clinics. Islands of gastric mucosa have also been found involved in the lesion in ten of our cases of esophageal cancer. Peptic ulcer of Meckel's diverticulum may be associated with the presence, in the diverticulum, of gastric columnar and cubical cells; but ulcer here could not be attributed to a retrograde flow of acid gastric contents. The now clearly proved selective gastrototoxic action of bacteria, toxins and even chemicals and drugs (Reffuss), when introduced in various ways, renders of utmost importance the presence of islets of gastric mucosa in the esophagus in the etiology of peptic ulcer of the esophagus.

Symptomatology

Pain back of the lower half of the sternum and extending through to the back between or under the shoulder blades is the most constant and significant symptom. It greatly exceeds in severity the pain of cancer in the same location. Slight tenderness to the passage of food may be present, and persistent pain may start on swallowing; but in almost all our cases the pain came on a half hour or longer after eating, in some cases only after the heartiest meal of the day, dinner in the evening. In these cases the pain would last all night unless stopped by the taking of alkali. There seems little reason to doubt that the pain is due to contact with acid secretions, and it seems probable that, in most cases at least, the acid secretions have been regurgitated from the stomach, though this does not imply that the ulcer itself has resulted from hiatal insufficiency. The acid may possibly have been produced from the cells in esophageal islets of gastric mucosa, though this seems unlikely. The prompt relief following the taking of alkalis is so marked and so constant as to justify the conclusion that pain is due to acid. The onset time in relation to the intake of food is significant. Peristalsis does not seem a factor in esophageal pain.

"Heartburn," "waterbrash" and similar subjective sensations were present in about three fourths of our cases.

Hematemesis and melena were noted in a number of our cases. They appeared in the recent as well as in the long standing cases. "Coffee-ground" and livelike particles in the vomitus were present in a few instances. Collapse and death from loss of blood occurred in one case.

Odynophagia is a usual but not a constant symptom, nor is it constantly present in any case. About half of our patients could feel the bolus of food pass a tender point and in some the pain continued until it was stopped by the taking of an alkali. It was the slight sense of pain due to the passage of food that led to the patient being sent to the clinic for the detection of the supposed foreign body. The ulcer in these cases was old and peptic in character, evidently having originated long before the subjective sensation of pain.

Dysphagia is not present in the early stages of peptic ulcer of the esophagus. In the cases we have seen in an early stage of the ulcer, the esophagogoscopy was not done because of difficulty in swallowing. Half of our patients with peptic esophageal ulcer had no symptoms referable to the esophagus, and four of the twenty-one had no symptoms of any kind until a short time before, notwithstanding the ulcers were quite evidently of long duration.

Vomiting at times is usually present. It was noted in eleven of our cases and was especially frequent in those in which the ulcer involved the hiatal constriction.
The vomitus was mixed with blood in ten of the cases but there was only an occasional streak in most instances. Regurgitation was present in a few cases.

**ESOPHAGOSCOPIC APPEARANCES**

The chief characteristics, broadly stated, are the flatness of the lesion, the absence of annular infiltration of the esophageal wall, and the absence of exuberant fungations.

In the early stages, the flatness of the lesion and the absence of infiltration are well marked (1, in the accompanying illustration). Later the edges become somewhat raised, and, still later, slightly undercut (2, in the illustration). In only two cases out of twenty-one did I get that "roll-rim" effect that the endoscopist gets in gastric ulcer. In no instance were there found the funguations usually seen in cancerous ulceration. In three cases a fungating lesion appeared on the edge of a scar and in all of these histologic examination of the esophagoscopically removed fungations showed them to be cancerous implantations. The esophagoscopic view in one of these patients is shown at 6.

There is usually a zone of contrasting intense hyperemia around the rim of the ulcer. The bed may be covered with yellowish exudate (7). If this is absent or wiped away, a bleeding granular bed may be seen. The bleeding, if present, comes from this ulcer bed, not from exuberant fungations as in cancer.

In three out of our twenty-one cases, the lesion was multiple. The size varied from about 14 mm in greatest diameter to about 3 mm.

The ulcer in most cases was elongated, but five were round. In determining form, care was taken to eliminate error from the foreshortening of the perspective.

**DIAGNOSIS**

Deductive methods of diagnosis, unreliable anywhere, have no place whatever in the diagnosis of disease of a viscus so easily and safely inspected as the esophagus. The bougie, an inferential means, is useless and dangerous. The roentgen examination, of fundamental importance in all other esophageal diseases, is usually negative in peptic ulcer. In a number of our cases this has obviously been due to the flatness of the ulcer as revealed by esophagoscopy. Possibly also the overlapping of diaphragmatic and hepatic shadows may add to the technical difficulties. An ulcer of long standing may show a filling defect but the niche of gastric ulcer was absent in all of our cases, notwithstanding close scrutiny of the shadows at the level of the ulcer as demonstrated esophagoscopically. The cicatrization sequel of a peptic ulcer may show roentgen evidence of stenosis. Not only for this reason but for other obvious reasons roentgen examination should precede diagnostic esophagoscopy in every case. The mention of the fact that ulcer of the esophagus often does not show on roentgen examination is intended only as a warning against excluding peptic ulcer on the basis of a negative roentgen report. The esophagoscopic appearances and biopsy are the important diagnostic means.

In every patient with symptoms of gastric ulcer in whom the diagnosis is not confirmed by the roentgen ray and by the other diagnostic means familiar to the gastro-enterologist, esophagoscopy to exclude esophageal ulcer is indicated to determine the presence or absence of peptic ulcer of the esophagus.

**Typhoid.—**The esophageal ulcers of typhoid are at a higher level (postcricoidal) and usually do not persist long after the convalescent period.

**Cancer.—**The characteristic esophagoscopic appearances of cancer are infiltration and fungation. Bleeding fungations appear sooner or later; polypoid masses are common.

**Syphilis and Tuberculosis.—**As with ulcerative lesions everywhere in the body, syphilis and tuberculosis should be excluded by the usual means in every case of ulcer of the esophagus. Primary tuberculosis in the esophagus is quite superficial, looks whitish or grayish, and is not surrounded by a red zone. Tuber-

---

culous invasion by suppurating glands looks more like peptic ulcer but usually it is located at a much higher level (near the bifurcation of the trachea) than peptic ulcer.

*Actinomycosis.*—As a cause of an ulcerative lesion of the esophagus, actinomycosis is rare, but its character can be readily demonstrated by biopsy.

*Blastomycosis.*—Blastomycosis of the esophagus presents a granular, an eroded or a whitish appearance that is quite different from the clearly cut loss of substance seen in peptic ulcer. Biopsy is necessary for diagnosis.

*Cicatrices.*—In making a diagnosis as to the cause of scars found, one is justified in attributing them to peptic ulcer when they are located close to the hiatus of the esophagus. Provided there is no history of a corrosive substance having been swallowed.

*Biopsy.*—This is the final arbiter. The specimen should be taken from the edge of the ulcer; this will show the transition from the pathologic to the relatively normal and cancer can be thus excluded with certainty. Moreover, the taking of a specimen at the edge is safe because in the inflammatory rim no vessels are likely to be encountered. The autopsies reported in the literature show that perforations into vessels have not occurred in the superficial edge of the rim. A specimen taken in this way under the guidance of the eye, and failing to show evidence of a malignant process, is conclusive as to the primary process, it would not preclude the possibility of cancer developing as a secondary, but only one case of cancer secondary to peptic ulcer has been recorded at the bronchoscopic clinics. In this case the cancerous process developed in the scar left after healing of the ulcer.

**PROGNOSIS**

Of the twenty-one patients in my experience, one died of spontaneous hemorrhage, five were untraceable, twelve recovered and two are still under treatment. The longest duration after the diagnosis was made was five years; at the end of this time the ulcer had healed. One of the patients has now been under treatment three years and the other four years. In both these patients the ulcers are healing. In addition to the twenty-one patients just mentioned, in sixty-seven other patients scars at the lower end of the esophagus were attributed to peptic ulcers that had healed. It would seem justifiable to say that healing usually takes place ultimately but not promptly under esophagoscopic treatment, combined with dietetic management by the gastro-enterologist. A scar always remains, but stenosis does not occur unless the scar involves most of the circumference of the esophagus.

That the disease is a rather serious one cannot be questioned; but it must be remembered that the cases reported in the literature are almost all based on a diagnosis made only at autopsy, and in most of them death was due to perforation of the ulcer. Until the advent of the esophagoscope there was no method of making a diagnosis during life. No one knows how many patients had the disease without dying of it. The era of esophagscopy on which physicians are just entering will doubtless alter the statistics by recording other than fatal cases, a class previously unrecorded. In the literature the recorded autopic cases clearly include some instances of fatal perforation of the ulcer bed by a blindly passed bougie, and in two instances there was probably no ulcer but rather a granulating wound following a slowly fatal perforation by the bougie. Most of the deaths in the recorded cases were due to spontaneous perforation into the pleura or into a large vessel.

**TREATMENT**

The most important treatment is the elimination of foci of infection. Next in importance are direct endoscopic applications to the ulcer. Silver nitrate in solution not stronger than 10 per cent on an esophagoscopy swab, once weekly, with interim applications of bismuth subnitrate by esophagoscopy insufflation is the best form of local treatment in our experience. Crystalline bismuth subnitrate in small doses, dry on the tongue, should be also given at frequent intervals. Dietetic treatment as in gastric and duodenal ulcer is probably indicated, and it is important to avoid all mechanically irritating rough foods. All food should be well masticated and should be taken in small bolus. Granular foods, cereals and rough vegetables should be avoided for mechanical reasons. All liquids should be taken in sips rather than gulps. As malfunction of the stomach is possibly one of the factors in regurgitation, general and dietary management of the patient by the gastro-enterologist is absolutely essential for cure.

Prophylaxis consists in the discovery and elimination of foci of infection wherever found.

**CONCLUSIONS**

1. Deductions from eighty-eight cases of peptic ulcer of the esophagus suggest focal infection as the chief etiologic factor, with the tonsil as the most frequent site of the focus. Islands of gastric mucosa are accessory causes. Retrograde flow of gastric juice may or may not be a perpetuating etiologic factor, but it is certainly a cause of the pain.

2. The most characteristic symptom of peptic ulcer of the esophagus is retrosternal pain or discomfort extending through to the back. Chronic esophagitis is accompanied by the same symptoms, but of less severity. Ulcer may be symptomless.

3. The diagnosis of the peptic ulcer can be certainly made only with the esophagoscope.

4. Esophagscopy for diagnosis is indicated in every patient complaining of the slightest abnormality in swallowing or the slightest degree of retrosternal pain or discomfort.

5. The best treatment of peptic ulcer is by eradication of focal infection, plus the local esophagoscopy application of argentc nitrate, or bismuth subnitrate. Palliative control of the symptom, pain or discomfort is afforded by alkalis, especially sodium bicarbonate. Opiates are unnecessary and are contraindicated.

235 South Fifteenth Street.

**Plants and Eczema.**—The external causes of eczema are immemorial. They include vegetable, animal and other chemical as well as many mechanical irritants. One must always be on the lookout for vegetable irritants as the cause of eczema. The number of plants and their derivatives that will produce eczema in susceptible individuals is surprisingly large. The Bureau of Plant Industry of the United States Department of Agriculture has furnished a list of 113 of these irritant plants, and this does not include all that are known. They range from poison ivy and crownfoot to such attractive, succulent, useful and apparently harmless things as primroses, fox-eye daisies, tomatoes, asparagus, and Chinese lacquer. We are constantly learning of new plants that produce eczema at times. So, in such cases, it is necessary to be suspicious of practically all sorts of plants, and, in uncertain cases, to warn patients against contact with all plants.—Pusey, W. A.: Our Changing Knowledge of Eczema, *Pennsylvania M. J.*, December, 1928.