mediastinitis and pleuritis. The cases operated upon for foreign bodies have already been discussed. Except in the case of Forgue, in which the operation was interrupted, the pleura was almost always injured, either during resection of the ribs, during its separation, or subsequently by the sharp edges of the ribs. Of the 5 cases operated upon with the object of sufficiently exposing the œsophagus (Rehn, 2; Henle, Llobet, and Enderlen, 1 each), all except the last died as a result of the operation.

Till further experience has been gained, this operation, which is in itself serious, and which is almost sure to be followed by pyopneumothorax, is principally justifiable in abscess of the posterior pleural cavity following perforation of the pharynx and œsophagus, as it may certainly be expected that in some cases this operation may prevent an otherwise certain fatal termination. In the presence of foreign bodies the latter could be removed from the œsophagus or from the peri-œsophageal tissues by incision, or in case of perforation into the pleura they could be removed from the latter. At the same time any existing purulent ichorous exudation into the pleural cavity could be removed at once by incision.

CHAPTER IV.

DISEASES OF THE ŒSOPHAGUS.

INFLAMMATORY PROCESSES OF THE ŒSOPHAGUS.

Acute catarrhal inflammation of the œsophagus, which may be caused by irritation of the mucous membrane by foreign bodies, scalding, or irritating substances, is characterized by moderate mucoid secretion. Where inflammation is more severe there may be loss of epithelium, resulting in superficial erosions and ulcers, which usually heal without causing any complications.

Chronic catarrhal inflammation of the œsophagus occurs in alcoholics, also as a result of irritation caused by food accumulating in the dilated portions above the stenosis, in cases of diverticulum and carcinoma, also in the form of a congestive catarrh in chronic cardiac diseases. It is characterized occasionally by hyperæmia, leading to ectases, and marked thickening of epithelium. On œsophagoscopic examination there are seen a whitish cloudiness and a loosening of the mucous membrane, which secretes a mucoid, sticky substance; occasionally also there is slight dilatation of the œsophagus. In certain cases there occur circumscribed papillary proliferations as well as flat ulcerations; more rarely there are deeper ulcers. Where the inflammation continues for some time there may be thickening of the mucosa and the muscular coat (seldom leading to actual constriction); in other cases there are relaxation of the muscle and diffuse dilatation of the œsophagus. (Zenker-Ziemssen.)

Catarrhal œsophagitis may occasionally be followed by swelling of the follicles and result in a *follicular œsophagitis;* or if followed by dilatation of the follicles, small abscesses may occur, and by confluence of the latter *phlegmonous œsophagitis* may develop.

Croupous, necrotic, and **diphtheritic inflammation of the œsophagus** possesses surgical interest in so far as deposits of fibrinous pseudomembrane or dense infiltrations of the mucous membrane have been observed in the œsophagus; also in rare cases true diphtheritic ulcers, with subsequent cicatricial formation, occurring after extension from the pharynx or associated with acute infectious diseases (typhoid, measles, scarlatina, smallpox, sepsis, diphtheria, etc.). Such stricture formations have been recently observed, particularly after so-called diphtheria. (Leube-Panzaldt, v. Eiselsberg, Ehrlich, v. Hacker.) In the three last-named cases the stricture was always situated near the level of the bifurcation. In the author's case and that of Ehrlich it was particularly difficult to dilate the stricture.

The occurrence of thrush should not be confused with diphtheria; it occurs most frequently in children, but occasionally is met with in adults who have become exhausted by disease. In these cases it usually extends from the mouth and forms a yellowish or grayish-white deposit. In rare cases this may extend beyond the deeper layers of epithelium and invade the bloodvessels (E. Wagner), and may even produce metastases in the brain (Zenker). Where the disease is absent from the mouth the diagnosis may be determined by microscopical examination of particles adhering to the fenestrum of the stomach-tube. According to the case, mechanical cleansing of the œsophagus, or in case of stricture irrigation of the œsophagus with a 3 per cent. solution of borax, is effective in curing the disease. (Aufrecht.)

Phlegmonous inflammation of the œsophagus, first fully described by Zenker and Ziemssen, involves the submucous tissues, and like phlegmonous gastritis is very rare. This purulent inflammation may be circumscribed or diffuse, extending over large areas. If the collection of pus ruptures into the mucosa, complete recovery may take place where the abscess is small. Large abscesses may cause bulging of the mucous membrane over extensive areas, and then produce a sieve-like perforation of the latter, resulting in the formation of a phlegmonous ulcer. The abscess cavities may in part remain and be covered over with epithelium from the edges of rupture.

The disease occurs after injury by penetrating foreign bodies, fish-bones (Belfrage and Hedluins), and injuries caused by caustics (sulphuric acid), extension of abscess of the stomach, most frequently, however, by rupture of peri-œsophageal abscesses (glandular, vertebral abscesses, perichondritis of cricoid cartilage, etc.). If in addition these abscesses rupture into the larynx or trachea, there may be produced communication with these organs, and after the process has subsided permanent fistulæ may remain. The occurrence of phlegmonous œsophagitis was also observed in a case of laceration of the mucous membrane following violent vomiting (Voigt), and in one case without evident cause. It is of interest that in this disease there is no tendency for pus to rupture into the mediastinum with the production of progressive ichorous necrosis, or rupture into the pleural cavity, as so frequently occurs after perforation of the œsophagus.

Symptoms.—The symptoms vary: fever, chills, difficulty in swallowing, pain over the course of the œsophagus, particularly behind the sternum and radiating to the back, nausea, depression, cough, occasionally regurgitation of pus where rupture has taken place. These symptoms will render diagnosis possible only in case a foreign body has become impacted.

Treatment.—While ordinarily the treatment can only be symptomatic, under the last-mentioned circumstances œsophagoscopy may be of therapeutic importance. By the aid of this method a bulging abscess can be punctured and incised or the mucous membrane divided where it has become separated. In evidence of this fact is the author's experience. By means of œsophagoscopy an embedded piece of bone

was removed after the surrounding mucous membrane had become separated by an accumulation of pus. Immediately after removal of the foreign body pus escaped from the opening, followed by gradual recovery of the patient.

Toxic Œsophagitis.—Toxic or corrosive œsophagitis occurs after the ingestion of caustic chemical substances, and from a surgical point of view is the most important inflammation of the œsophagus on account of its complications, particularly the formation of strictures. At the same time there is usually erosion of the mouth and pharynx, frequently also of the stomach. Scalding fluids may act in the same way. Where the effect of the caustic substance is only superficial, the epithelial layer is thrown off either in patches or circular portions without further consequences. Where the formation of a slough involves the entire thickness of the mucosa and part of the muscular coat, the border of necrosis is marked by a line of purulent inflammation which may occasionally develop beyond the region of the œsophagus, setting up peri-œsophageal abscesses extending into the mediastinum with or without perforation, or leading to pleuritis, pericarditis, etc. After rupture into the respiratory passages œsophagobronchial fistula or tracheal fistula may develop. The ulcer undergoes cicatrization after the slough has been thrown off. As a result of contraction of the scar stricture is produced, which varies in length and degree according to the extent and depth of the lesion.

Swallowing concentrated solutions of the alkalies or sulphuric or nitric acid is as a rule fatal, principally on account of the extensive sloughing of the stomach, which in sulphuric acid poisoning leads directly to perforation. Cases of toxic œsophagitis applying for treatment are therefore usually such as have swallowed weaker solutions, particularly those of the alkalies used for household purposes. In most instances poisoning is accidental; occasionally the alkalies are taken with the intention of committing suicide. In children the mortality is usually high if the œsophagus and entrance of the larynx have been severely burned even where only small quantities have been swallowed, the cause of death being a subsequent inflammation of the respiratory passages.

Treatment.—The treatment of toxic œsophagitis is principally symptomatic at first: ice, fluid diet, feeding by the rectum, narcotics, stimulation. In very severe cases, which frequently die within twelve to twenty-four hours as a rule of perforation, which injury has extended over the surface of the stomach or œsophagus, little can be hoped from operation (laparatomy in perforation of the stomach, duodenostomy where there is severe injury of the stomach). Maydl's proposal to perform gastrostomy immediately in case of recent injury by caustics is worth considering. In the author's opinion this operation should not be performed in very severe or in very mild cases. In those cases which run a more chronic course, in which in the beginning the degree of injury is still doubtful, operation is indicated as soon as dead tissue has been thrown off from the œsophagus.

This process always leads to ulceration and the formation of extensive contractile scars. If as a result of the action of caustics or ingested food upon the ulcerated areas there are acute swelling and contraction of the muscular coat, and as a result of the latter more or less complete closure of the œsophagus, gastrostomy is urgently indicated. This applies also to cases in which there is perforation of a peri-œsophageal abscess, which can be recognized by the repeated regurgitation of pus mixed with blood. The œsophagus should be prevented from sustaining further injury by feeding through the gastric fistula.

Examination with bougies should certainly not be undertaken as long as there are manifestations of recent ulceration (fever, traces of blood in the saliva or in the vomitus). Such ulceration usually continues for from three to four weeks after the injury according to the degree of the latter. After this period, as a prophylactic measure, sounds may be passed into the œsophagus and left in place for a time; bougies may be passed through the mouth or soft drainage-tubes inserted by means of a piece of silk passed from the mouth to the gastric fistula. This procedure should not be continued for more than a few hours, having the portion of the œsophagus behind the larynx free in order to prevent pressure-necrosis. Even here great care should be taken.

Œsophagotomy is seldom permissible during the inflammatory stage following injuries from caustics. This operation would be indicated only in the presence of peri-œsophageal abscess; but even then it would be desirable, as Tietze has pointed out, to establish a gastric fistula.

ULCERS OF THE ŒSOPHAGUS.

Besides the catarrhal, diphtheritic, phlegmonous, and corrosive ulceration, discussed above, must be mentioned gangrenous ulcers caused by pressure, syphilitic and tuberculous ulcers, as well as peptic or round ulcers of the œsophagus. Ulcers caused by foreign bodies or carcinoma are discussed elsewhere.

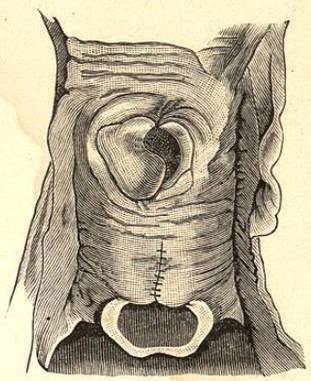
Gangrenous ulcers may be caused by pressure from within or without. Struma or some other tumor may press the cricoid or one of the tracheal cartilages against the œsophagus so that one or both walls of the latter may become ulcerated. The same effect may be produced by aneurism of the descending aorta, the latter rupturing after the slough has been thrown off. Foreign bodies act in the same way from within, particularly tubes left in place for some time. Most frequently the latter cause ulceration situated on the anterior wall behind the cricoid cartilage, similar to the so-called decubital ulcer. Perichondritis and necrosis of a portion of the cricoid cartilage may ensue, and after the latter has been thrown off, the mucous membrane may be drawn into the defect, causing the formation of a small diverticular recess usually situated at a typical site behind the cricoid cartilage. (Fig. 17.) More rarely there is produced ulceration of the posterior wall at a point opposite the cricoid cartilage or at both

sites. In exceptional cases (if stomach-tubes, etc., are left in place for a long time) ulceration may also occur at the two other sites of constriction of the œsophagus (bifurcation, hiatus, Kermanner).

In case of decubital ulcer, *κατ'εξοχην*, there is produced on opposite points of the anterior and posterior walls of the pharynx an ulceration the size of a dime; at the bottom of the anterior ulcer the cricoid cartilage may be exposed; at the bottom of the posterior ulcer the body of the vertebra. Usually this form of ulcer occurs shortly before death in patients very much run down by disease, who constantly assume the dorsal position, and is caused by pressure of the plate of the cricoid cartilage, upon the folds of mucous membrane.

Syphilitic ulcers may occur in the œsophagus (Virchow, West, Klob, and others); they are very rare, and as a rule affect the upper portions of the œsophagus and the lower portions of the pharynx (Hermann). Exceptionally they affect the lower portion of the œsophagus. Frequently, cicatricial contraction in the pharynx obstructs the entrance of the œsophagus. This may also be produced by the formation of folds. The author made an œsophagoscopic examination in such a case at Prof. Neumann's clinic in Vienna. Mraczek has described such a case. In the majority of cases the lesion is a gumma bringing about constriction either through fibrous contraction or ulceration and formation of scars. In a number of cases the diagnosis was only made by noting the effect of antisyphilitic treatment. In a case of gumma of the entrance of the œsophagus, reported from Mikulicz's clinic (Gottstein), the diagnosis was made by the aid of œsophagoscopy.

FIG. 17.



Diverticular recess following gangrenous ulcer and necrosis of the cricoid cartilage. (Author's observation.)

Tuberculous Ulcer.—The occurrence of tuberculous ulcers of the œsophagus has recently been positively confirmed. These ulcers, associated with extensive tuberculosis of other organs, do not always produce symptoms (Mazotti); occasionally they run their course accompanied by pain and difficulty in swallowing. As a rule they are superficial, and as such rarely lead to constriction. Beck described a case, however, in which a diffuse ulceration, advancing from the pharyngolaryngeal portion of the gullet, was diagnosticated during life as carcinoma, and resulted in marked constriction; a similar case was described by Zenker. Most frequently, according to Zemann, stenosis results from rupture of cheesy glands into the œsophagus. (Weichselbaum, Beck, E. Frerichs, and others.) In such cases stenosis is caused more by the formation of scars and diverticula than by the occurrence

of tuberculous ulcers. More recently cases associated with carcinoma have been observed. (Lubarsch, Pepper, and Edsall.)

Actinomycosis.—It is only recently that cases of actinomycosis of the œsophagus have been described. In this disease infection takes place at a site where the epithelial covering has been destroyed by wounds or ulcers. The disease is certainly rare, and where the neighboring structures (soft parts of the neck, lungs, etc.,) are involved, it is difficult to determine the point of entrance. The involvement of neighboring organs, particularly the formation of abscesses and fistulæ in the neck, as well as the characteristic bodies in the pus, will render the diagnosis positive. In Mikulicz's clinic this was determined by examination of sections obtained by the aid of œsophagoscopy, in a case of stenosis with ulcerating tumor situated 26 cm. from the teeth, which had been mistaken for carcinoma. According to the conditions present, the treatment consists of incision, scraping, and administration of potassium iodide, sodium iodide, or of internal medication alone.

Peptic or Round Ulcer of the Œsophagus.—The occurrence of these ulcers, analogous to round ulcers of the stomach, and frequently observed at the same time as the latter form of ulcer, has been confirmed by a number of recent careful investigations. (Quincke, Chiari, Zahn, Debove, Lindemann, Ewald, Huwald, A. Fränkel, and others.) They occur only in the lowest portion of the œsophagus, being caused by the repeated action of the acid gastric juice upon portions of the œsophageal wall whose circulation has been impaired. The presence of Schaffer's heterotopically developed gastric glands was confirmed in Fränkel's case, but they possess little etiological significance and are of rare occurrence. Either they produce no symptoms or the latter are similar to those of gastric ulcer. They may give rise to profuse hemorrhage or to perforation. Perforation rarely takes place suddenly, but usually as a result of infiltration of the connective tissue caused by chronic inflammation, with formation of cavities in the mediastinum, from which site it may lead to inflammation of the neighboring organs, pleuritis, pericarditis, or even rupture into the pleura (pyopneumothorax), into the lungs (gangrene), into the trachea or the bronchi (bronchitis putrida), and possibly the formation of a fistula, or into the aorta with fatal hemorrhage.

Finally, cicatrization of round ulcer may lead to constriction, which may easily be mistaken for carcinoma (Quincke, Reher, Debove), or to the formation of pockets or valves; on the other hand, however, complete recovery may follow. Clinical diagnosis can only be made by means of œsophagoscopy.

It may be remarked that in rare cases simple round ulcer of the stomach when situated close to the cardia may extend to the œsophageal portion of the latter and bring about severe hemorrhage (K. Zaleski); also that as a result of cicatrization stricture may result extending to the œsophagus (Zenker and Ziemssen).

STRICTURE OF THE ŒSOPHAGUS.

Etiology.—The most important constrictions of the œsophagus are those caused by diseases of the wall of the œsophagus, and which are strictures in the true sense of the term.

From the point of view of differential diagnosis, those forms of narrowing of the œsophagus are important which are caused by penetrating bodies (foreign bodies, thrush, neoplasms, polyps, etc.), so-called *obstruction stenosis;* also that form of narrowing must be considered which results from pressure of enlarged or displaced neighboring structures, so-called *compression stenosis.* The latter may be caused by neighboring lymph-glands, usually those that have undergone cheesy degeneration, particularly those of the bronchi and the mediastinum, also tumors of the thyroid glands, aneurisms, enlargement of the cartilages, curvature of the spine, pleuritic and pericardiac exudate, mediastinal tumors and abscesses, etc.

Obstruction stenosis is considered under foreign bodies and neoplasms; compression stenosis, in the differential diagnosis of strictures.

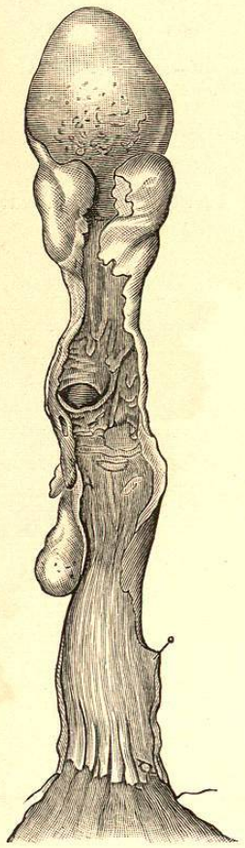
Of actual strictures of the œsophagus, those caused by carcinoma are the most frequent, and next in frequency those due to contact of caustics. All other causes producing stricture, including especially other inflammatory and ulcerative processes, are comparatively rare. Strictures caused by the swelling which is present during the acute stage of these inflammatory processes are called inflammatory, while those arising from contraction and subsequent constriction during the process of repair are called cicatricial strictures. Of 270 patients suffering from diseases of the œsophagus treated in the outpatient department of Billroth's clinic, from the year 1877 to 1886, 48.5 per cent. were cases of carcinoma, and at least 17.7 per cent. were strictures caused by caustics. Of the cases with œsophageal diseases treated in the hospital during the same time, 55.2 per cent. were cases of carcinoma, and 27.6 per cent. cases of stricture caused by caustics, and both diseases were associated (v. Hacker) in 82.8 per cent. of the patients.

Pathological and Anatomical Relations.—In discussing the pathological and anatomical relations of cicatricial strictures the author will consider strictures caused by caustics as the type, the remaining forms of cicatricial stricture being discussed under differential diagnosis.

According to the depth and extent of destruction, the strictures vary in form. Where the action has resulted only in a superficial destruction of epithelium the mucous membrane remains intact after the latter has become regenerated. But even in the milder cases there is usually necrosis of the mucosa, and occasionally of the submucosa, followed by subsequent scar formation, the latter either in itself or by retraction of the neighboring mucous membrane producing trabeculated (linear), crescentic, or valvular (semilunar) and annular strictures. These strictures, caused by superficial scar formation, are called "superficial strictures". Examples are shown in Figs. 18 and 19.

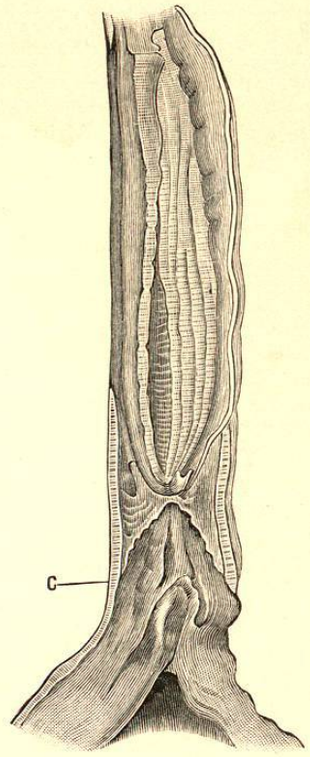
More frequently necrosis extends to the deeper tissues involving the muscular coat, the inner or both muscular coats being traversed by scars. There may even be indurated swelling extending beyond the œsophagus into the peri-œsophageal tissues, or a knuckling or displacement of the œsophagus from its proper course.

FIG. 18.



Valvular stricture above the bifurcation. (v. Hacker.)

FIG. 19.



Annular cutaneous stricture 1 cm. above the cardia (C), following cauterization with alkali. (v. Hacker.)

Through such scars affecting the deeper tissues callous strictures which involve the entire circumference are produced. According to whether they are more than 2 or 3 cm. long or extend over more than 5 to 10 cm. of the œsophagus, they are called annular or tubular strictures. (Figs. 20 and 21.)

Above annular or tubular strictures which cause considerable obstruction muscular insufficiency may result in dilatation, particularly if, as is frequently the case, the walls have undergone cicatricial

transformation. (Figs. 20 and 21.) Occasionally this change is only superficial. Frequently dilatation is only relative, the lumen not being abnormally increased.

Hypertrophy of the muscular coat, particularly of the circular fibres, occurring as a result of stenosis, is most marked in cases of long

FIG. 20.

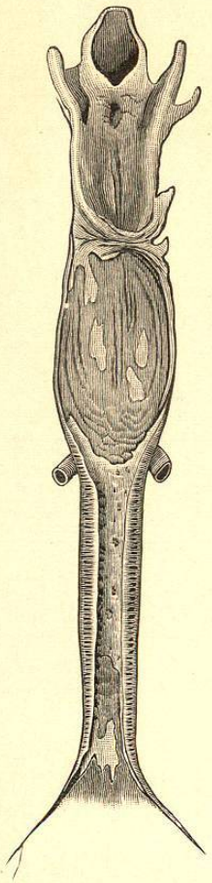


FIG. 21.

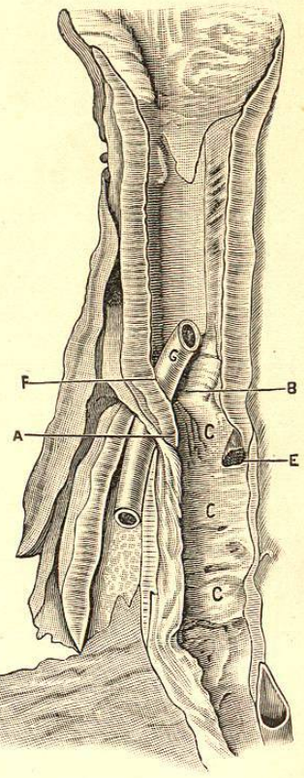


FIG. 20.—Typical tubular stricture in an adult, extending from the region beneath the bifurcation to the cardia. (v. Hacker.)

FIG. 21.—Typical false passage (C, C, C) situated in the right wall of a tubular stricture, arising in the thoracic portion and at the same time forming the prolongation of the œsophagus. G, tube showing the course of the œsophagus twisted at F. E, incision of internal œsophagotomy in the false passage, separated from the œsophagus by the spur A.

tubular strictures immediately above the narrowest point of the latter, and gradually diminishes above and below as a rule. As a result of this hypertrophy of the muscular coat and the continuous contraction of the latter the stricture may be increased in length above and below, so that at those sites where the mucous membrane is normal or super-

ficially altered the œsophagus appears as much constricted as at the site of the real cicatricial stricture. Such contractions certainly occur in recent cases as a result of inflammatory irritation, being accompanied at the same time by swelling of the scar. This occasionally happens after too energetic use of sounds. These strictures respond most readily to treatment by dilatation. Hypertrophy of the muscular coat is most marked in cases of typical tubular stricture of the portions of the œsophagus situated below the bifurcation. These strictures frequently extend from the level of the bifurcation to the diaphragm; in other words, they occur in that portion of the œsophagus in which the circular fibres are most highly developed under normal conditions. (Figs. 20 and 21.)

The most marked and the most extensive narrowing is always produced at that portion of the œsophagus where destruction was most severe. Although strictures can occur at any part of the œsophagus, they are most liable to occur at the site of the three natural constrictions of the œsophagus; at the entrance of the latter, at the level of the bifurcation of the trachea, and in the region of the hiatus œsophageus. Short annular strictures are located at these sites, as are also the centres of longer tubular strictures, from which points the latter extend upward and downward. (Figs. 20 and 21.) Accordingly as they are situated at the hiatus, the beginning, or the level of the bifurcation, there may be a combination of superficial and callous stricture. In this way there may be produced multiple strictures, or by a confluence of a number of extensive strictures total stricture of the œsophagus.

The accumulation of fluids from the region of the hiatus upward as a result of the closure of the œsophagus toward the stomach during swallowing, and the intermittent entrance of swallowed food into the stomach several seconds after the act of swallowing, is of great etiological importance. Large quantities of caustic substances remain in this situation a short time till they are propelled onward by peristalsis. In this way may be explained the occurrence of annular or tubular strictures, the latter extending upward for a variable distance above the cardia (v. Hacker), as has been typically observed especially in adults. (Figs. 20 and 21.)

In tubular strictures of the lower thoracic portion the narrowest part of the constriction is usually situated at the lower end. In tubular strictures extending along the superior thoracic portion or up to this portion the narrowest portion may be situated at or above the middle of the œsophagus. The dilatation which in the course of time is developed above the stricture, and which is occasionally formed at the expense of the length of the constriction, takes place from above and extends to the narrowest portion of the constriction, but does not extend beyond this point. In this way it happens occasionally that the line of transition between the dilatation and the tubular constricted portion, forming a projecting cicatricial ring, appears hypertrophied, being sharply marked above, and gradually passing into the deeper

portion by a number of radiating processes. In such cases sounding through the stomach may occasionally be easier than from above.

Practically it is of importance that the dilated portion, as well as the site of transition into the stricture, is frequently found in an inflamed and suppurating condition as a result of accumulating food, and that the rough, sacculated depressions, clefts, and trabeculæ of the inner wall may lead to the formation of ulcers, diverticula, inflammatory softening, peri-œsophageal abscess, or external perforation. Spontaneous perforation and perforation by bougies are frequently predisposed by sacculations of the wall above the stricture, or by the same condition in tho course of the stricture.

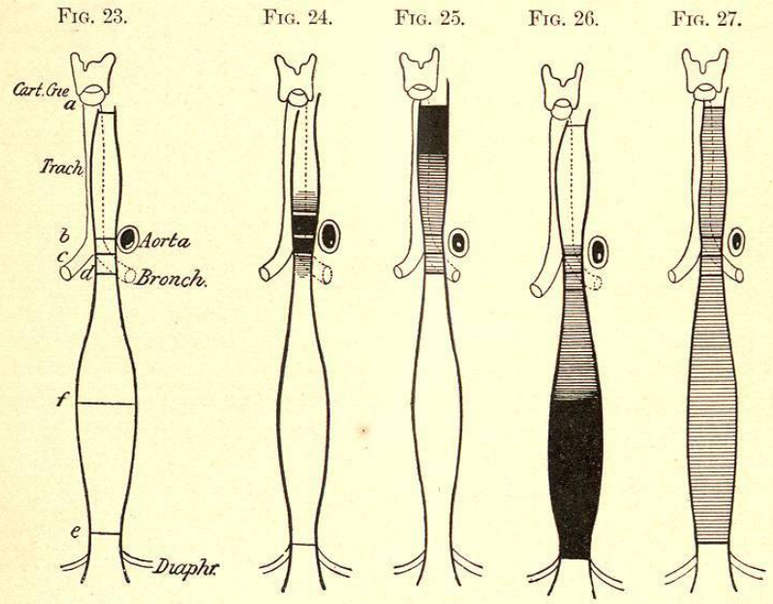
Fig. 22.

Stricture of the œsophagus. (Park.)

In all forms of stricture in which cicatricial tissue extends into the peri-œsophageal connective tissue, particularly in tubular strictures of the lower thoracic portion, the œsophagus may become adherent, fixed, twisted, and displaced from its proper course. This may occur at the site of transition from the dilated portion to the stricture, so that the lumen of the entrance of the stricture appears laterally displaced, and not in the direct prolongation of the canal. Displacement may occur at any point in the course of a long tubular stricture. This condition predisposes to the impaction of foreign bodies, the occurrence of false passages, or perforation by sounds.

If dilated, the false passage may form the direct prolongation of the œsophagus, or be mistaken for the latter when passing sounds, or, as in the case operated upon by Billroth, illustrated in Fig. 21, when performing internal œsophagotomy. According to the author's observations, there are two sites where in the course of tubular strictures false passages frequently arise: in the upper thoracic portion, on the left wall of the œsophagus, at the point where it turns from the left to the right side of the thorax, at the level of the bifurcation; in the

lower thoracic portion, on the right wall where the œsophagus turns from the right to the left side above the diaphragm. False passages frequently extend downward as far as the diaphragm. In general they rise more frequently from the stricture itself than from the dilatation above the latter. This is perfectly conceivable, as before entering the stricture the sound can still be more carefully guided. If the sound is firmly held by a cicatricial ring, it is frequently difficult to recognize when it is advancing through a false passage. Blood adhering to the sound may come from injured granulations. Pain may be present in both instances.



Schematic representation of the typical forms of stricture following ingestion of corrosive substances.

Fig. 23.—Sketch of normal œsophagus with the usual sites of circular stricture marked by lines *a*, *b*, *c*, *d*, *f*, and *e*: *a*, end of cervical portion; *b*, region of aortic arch; *c*, bifurcation of trachea; *d*, crossing of left bronchus; *e*, lower narrowing (at or somewhat above diaphragm); *f*, unusual stricture at a place between diaphragm and left bronchus.

Fig. 24.—Scheme of circular stricture of upper thoracic portion extending above and below bifurcation.

Fig. 25.—Scheme of tubular stricture of the cervical portion.

Fig. 26.—Scheme of tubular stricture of lower thoracic portion. The extent of the stricture is here, as in Figs. 24, 25, and 27, indicated by shading.

Fig. 27.—Scheme of stricture of whole œsophagus.

Symptoms.—Difficulty in swallowing is present in all constrictions of the œsophagus. In general it corresponds to the degree of constriction. Where constrictions develop slowly it gradually increases; but where stenosis occurs suddenly, as in case of obstruction, it sets in at once, so that if situated high up fluids swallowed instantly flow back out of the mouth and nose. If situated lower down, they will be vomited or regurgitated after a short time. Where narrowing develops slowly, it is a characteristic symptom that patients are careful

to take only a small amount of food at a time, that they swallow slowly, gag, and at times carry out certain motions with the head. If, however, a dilatation has developed above the stricture, the symptoms may be like those in case of diverticulum, food remaining some time in this portion being regurgitated later. Frequently the patients do not know whether food or even fluids have reached the stomach. Where the changes are marked, mucus and swallowed saliva accumulate, so that the patients are annoyed by a constant regurgitation of mucous fluid, and frequently also of tough, stringy mucus, particularly after attempts at eating. Generally fluids and soft food are more readily swallowed, but this is often not the case. Patients therefore thoroughly moisten the food with saliva or swallow fluids after taking solid food, etc.

As a rule, in cases of constriction there are marked temporary variations in the degree of difficulty in swallowing. These occur after inflammatory processes accompanied by ulceration, especially after injuries caused by caustics. During the acute stage of these processes, which are frequently manifested by regurgitation of blood, bloody mucus, pus, etc., there is usually dysphagia, which is generally accompanied by severe pain. After the ulcers have healed the difficulty in swallowing disappears for a time, recurring later when cicatricial contraction takes place and the stricture develops. Frequently this happens months afterward, and may be more severe than during the previous attacks, progressively increasing in the course of time.

Frequently patients locate the stricture incorrectly; they may refer it to the region of the sternum and cricoid cartilage when it is located in the lower thoracic portion. Marked cicatricial stricture caused by caustics may produce a condition of extreme emaciation and inanition.

Diagnosis.—The diagnosis of the presence and seat of a stricture can be determined by examination with bougies.

As a rule, a solid English flexible bougie is employed for this purpose, occasionally also a whalebone staff or a stomach-tube. It is advisable in most cases to pass first a thick cylindrical bougie, and then successively thinner ones. In this way it is possible to determine at what distance from the teeth a sound which ought to pass through a normal œsophagus is stopped, and what size bougie can pass the stricture, or that the latter is not passable for the smallest sound or even the thinnest gut-string. If a number of strictures are present, the second or third, situated lower down, may be recognized at once or frequently only after the upper stricture has been dilated. Valves, folds, and pockets may offer no obstruction to larger bougies which dilate the canal, while smaller bougies are caught by the former. It may be determined also that the obstruction is caused by stricture, and not by some other condition, and it is frequently possible to recognize that the sound is held by a circular pressure, or that a sound can only pass if a certain degree of force is exercised. In case of compression stenosis, on the other hand, one frequently receives the impression that as soon as resistance is overcome by continuous pressure no hindrance is