terference with respiration and circulation may be produced by pressure upon the lungs or diaphragm or by pleural or peritoneal dropsies. In general it may be said that the prognosis in œdema is serious because of the important pathological conditions underlying its appearance.

TREATMENT.—In general this is directed to the condition which gives rise to the œdema. Extreme distention of œdematous skin may be relieved by puncture and continuous drainage. Collections of fluid within the body cavities may also be removed by aspiration (see also *Ascites*).

Aldred Scott Warthin.

ŒDEMA NEONATORUM, or œdema of the new-born, was long confounded with "scleroma neonatorum"—a distinct affection. Œdema may occur in infants prematurely born or in those born at term but of poor vitality; it differs in these subjects in no respect from œdema in older patients, and can hardly be considered more than a symptom, associated as it is with many different conditions of the body. As with œdema in general the parts are soft, waxy white, pit on pressure, and in the more dependent areas the swelling is greatest. Bad feeding, defective hygiene, exposure to severe cold soon after birth, feeble heart action, and atelectasis of the lungs are all causes tending toward the production of the symptom of œdema in new-born infants. The treatment is that of the general condition and should be directed toward the underlying causes. Artificial heat to maintain the body temperature is an important adjunct.

Charles Townshend Dade.

ŒNANTHE. See *Poisonous Plants*.

ŒSOPHAGUS, PATHOLOGY OF.—The wall of the œsophagus consists of a mucosa, submucosa, inner circular and outer longitudinal muscular coats, and an external fibrous tunic. The mucosa is covered with stratified squamous epithelium, and contains sparsely scattered mucous glands and few lymph follicles. In the upper portion striped muscle is also present in the wall, in the lower portion only unstriped. The poor blood supply, the lack of mucous glands and lymph follicles, and the thick covering of stratified squamous epithelium render the œsophagus less liable to disease than the closely associated structures, the pharynx and the stomach. The independent part played by the œsophagus in affections of these organs is often strikingly shown in the sharply limited borders of inflammatory processes in the pharynx, the inflammation ceasing abruptly at the beginning of the œsophagus. Though œsophageal disease is relatively rare, it is nevertheless of very great clinical importance, not only from the fact that disease of the œsophagus may interfere with the proper passage of food into the stomach, and thus give rise to general impairment of nutrition, but also because of the proximity of this organ to such important structures as the trachea, lungs, and aorta. Further, the examination of the œsophagus is relatively difficult, and possible only through the use of special instruments or apparatus. (For methods of examination see *Stomach, Surgery of the.*)

CONGENITAL MALFORMATIONS.—Though relatively rare, these conditions are of practical interest, inasmuch as children so affected may live for some time after birth, or even reach adult age. The malformations may exist alone or in connection with other defects. The following forms have been described:

1. *Œsophago-tracheal Fistula.*—Abnormal communications between œsophagus and trachea may occur. The most common form is that in which the œsophagus at the upper third ends in a blind tube, while the lower portion opens at its upper end into the trachea or bronchus. The upper and lower ends of the obliterated œsophagus may be connected by a muscular band or a firm fibrous cord. This malformation may be due to primary disturbances of development, or may be acquired during intra-uterine life as a result of suppurative processes in the glands lying between the œsophagus and the trachea. The upper part of the œsophagus may be closed in this way by cicatricial contraction, while the lower part may be connected with the lumen of the trachea, or the reverse condition may occur. Children showing this malformation may be otherwise well developed; they die shortly after birth from inanition or aspiration pneumonia. In this connection should be mentioned also the rare occurrence of cysts lined with ciliated columnar epithelium, lying between the œsophagus and the bifurcation of the trachea. These cysts represent remains of the communicating canal between œsophagus and trachea. They may reach such a size as to cause compression of the œsophagus and secondary dilatations of the same, and are therefore of clinical importance. In other cases they may be discovered only accidentally, having given rise to no symptoms.

2. *Stenosis.*—Partial obliterations or narrowing of the lumen may occur as congenital malformations of either the upper or the lower portion of the œsophagus. The lower end of the upper portion may open into the trachea, or may form a blind sac. In other cases the œsophagus may be open as far as the level of the bifurcation of the trachea, at which point complete obliteration of the lumen may be found. In a few cases a membranous stenosis or obliteration has been observed, or the lumen has been closed by a ring-like fold of mucosa. Those cases of obliteration in which the continuity of the œsophagus has been completely broken have been explained by the development of the trachea and bronchi at the cost of the œsophagus; while those cases in which the continuity is not wholly lost, but in which the two portions of the œsophagus are connected by a muscular band, have been explained as the result of a fœtal pressure-atrophy. The conditions are rare; only the partial stenoses have clinical significance.

3. *Total absence* of the œsophagus is without clinical significance and is found only in acardiac monsters.

4. *A reduplication* of the œsophagus (*diœsophagus*) has also been very rarely observed. The reduplication may be complete or partial, and occurs in different degrees of double monsters. It is likewise without practical significance.

5. *Congenital Dilatations.* In very rare cases there has been observed just above the cardiac orifice a peripheral dilatation of limited extent, the so-called "fore-stomach" or "antrum cardiacum." Children presenting this anomaly usually show the clinical symptom of rumination.

CIRCULATORY DISTURBANCES.—*Active hyperæmia* occurs in the early stages of inflammatory conditions. It may be due also to the irritation of certain foods or drinks. *Passive congestion* occurs in all cases of general passive congestion, particularly in chronic heart and lung diseases, cirrhosis of the liver, etc. In chronic passive hyperæmia the mucosa of the œsophagus is dark bluish-red; the epithelium often shows plaques of thickening (leukoplakia). Local dilatations of the veins occur, the so-called *œsophageal hemorrhoids* or *varices* (see Fig. 3615). In the upper part of the œsophagus they are relatively frequent, forming small blue nodules or saccular elevations; these possess no clinical significance. In the lower portion of the œsophagus they occur even more frequently, particularly in the region of the cardiac orifice, or a few centimetres above this. The enlarged veins project above the level of the mucosa, and may form saccular papillomatous masses resembling rectal hemorrhoids. The dilated tortuous veins may be as thick as a lead pencil or even larger. They represent vicarious enlargements of the collateral branches connecting the portal circulation (through the vena coronaria ventriculi) with the vena azygos. They occur particularly in portal obstruction (cirrhosis, syphilitic hepatitis, pressure atrophy of the liver, obstruction or thrombosis of the portal vein), as well as in chronic passive congestion due to cardiac insufficiency. They are found in the majority of cases in connection with rectal hemorrhoids. Their presence is revealed clinically by hemorrhage, which may be fatal. As an early diagnostic symptom in cirrhosis of the liver bleeding from œsophageal hemorrhoids is of great importance. Preceding the hemorrhage there may be observed pain in the stomach, swelling of the spleen, and distention of the abdomen, severe pain radiating from the stomach region to the shoulders or extremities—these symptoms are followed by sudden hemorrhage from the œsophagus. Similar symptoms are often seen preceding hemorrhages from piles. The cause of the œsophageal hemorrhage may be due to increase of venous pressure or to ulcerative changes in the mucosa over the varices. Rupture of the varix has followed coughing, severe muscular exertion, dyspnœa, etc. In other cases no direct cause for the hemorrhage can be found. Repeated hemorrhages of small size may lead to severe anæmia. The relative frequency of hemorrhage from œsophageal varices makes the condition a dangerous one.

Œsophageal hemorrhages may be caused also by injury, ulceration, new growths, etc. The hemorrhages very often arise from the large vessels in the neighborhood of the œsophagus, rather than from its own vessels. Bleeding from the latter occurs particularly in cancer of the mucosa. An aortic aneurism may erode the wall of the œsophagus and rupture into the lumen; or, on the other hand, the œsophagus as a result of ulcerative or carcinomatous changes may break into the aorta, carotid, left auricle, etc. Such an event is most likely to happen in cases of œsophageal carcinoma. In whatever way produced, the entrance of arterial blood into the œsophagus makes itself known by the appearance of the blood vomited up, providing the hemorrhage is of sufficient volume to cause immediate vomiting. There may, however, be a continuous slight oozing from an eroded artery or arterial aneurism, and the blood passing into the stomach may be so changed that its arterial character is lost before vomiting takes place. In some cases the blood may be digested and passed on into the intestines.

RETROGRADE CHANGES.—*Œsophagomalacia* is in the great majority of cases a post-mortem digestion of the mucosa of the œsophagus by stomach fluids which have passed, after or during death, through the cardia into the œsophagus. The epithelium is macerated, desquamated, or liquefied; the musculature may also be liquefied and perforated, the stomach juices passing into the pleural cavity. In the lightest grades, which are present in the majority of cadavers, the mucosa of the organ shows longitudinal stripes of desquamation corresponding to the longitudinal folds of the contracted mucosa. An intravital œsophagomalacia is of very rare occurrence, but has been described as the *round* or *peptic ulcer* of the œsophagus, which corresponds in all particulars to the round ulcer of the stomach. An *agonal* œsophagomalacia has been observed in severe cases of cerebral disease.

Atrophy of the œsophagus wall occurs in cachexia. *Degenerations* of the œsophagus wall are of very rare occurrence and have been studied but little. *Necrosis* is the most important retrograde process found in this organ. The most common cause is pressure, either from foreign bodies lodged within the lumen or from the pressure of an aneurism or tumor from without. As a result of the local anæmia caused by the pressure there occur necrosis and ulceration. The *peptic ulcer* has been mentioned. *Decubital* ulcers may be found in advanced stages of severe cachexias. *Noma* of the cheek or pharynx, gangrenous tonsillitis, or gangrene of the lung may be associated with *gangrene* of the œsophagus. Corrosive poisons may cause more or less extensive necrosis of the œsophageal mucosa associated with inflammatory changes. Gangrenous œsophagitis occurs also in severe infections as a rare complication.

INFLAMMATION.—*Acute catarrhal œsophagitis* is the most common form. It is caused chiefly by irritating foods or drinks, through extension of inflammation from the pharynx or stomach, or as a secondary phenomenon in some of the acute infections (measles, scarlatina, typhoid fever, variola). As a result of the small number of glands in the mucosa there is often but little secretion. The epithelium may be desquamated, and the mucosa beneath hyperæmic, or cloudy white, or yellowish. Small ulcers may be formed over the surface of the folds, and these may heal with the formation of small longitudinal scars. In the case of foreign bodies deeper ulcers may be produced. The symptoms of acute catarrhal œsophagitis are, pain in swallowing, regurgitation, thirst, raising of

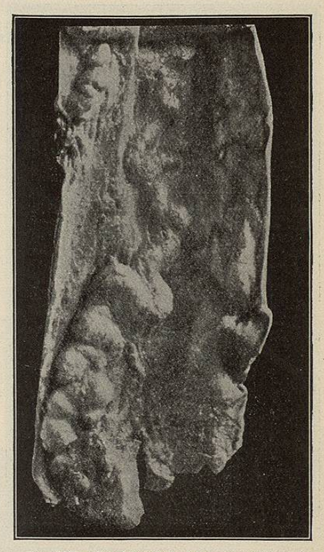

FIG. 3615.—Œsophageal Varices. (After Kraus.)

secretion, pain on moving the neck, tenderness on pressure in the deep cervical region. Spastic contractions of the œsophagus may also occur.

Chronic catarrhal œsophagitis occurs chiefly in smokers and drinkers, as well as in individuals suffering from chronic pharyngitis or, more rarely, gastritis. The condition is often secondary to the chronic passive congestion caused by cardiac or pulmonary disease. It is found also in the portion of the œsophagus above a stenosis. It may be associated with diverticula. In cases of chronic gastritis characterized by frequent eructations of irritating substances or by frequent vomiting of the same, there may be produced a chronic œsophagitis, which may be of a purulent or ulcerative character. This condition is not infrequently found in old men.

Chronic œsophagitis may not always give rise to symptoms. In severe cases there may be pain and discomfort in swallowing. The pain has usually the character of pressure, more rarely it is stinging or burning; when it is severe, spastic contractions and regurgitation may occur. Moderate quantities of mucus may be expectorated; this may occasionally contain streaks of blood. The differential diagnosis from the accompanying affections of pharynx or stomach is often very difficult. The symptom of dysphagia, less marked in the case of fluids than in the swallowing of solid substances, is the most constant and characteristic symptom. Examination with œsophagoscope or sound is usually difficult, but is necessary for the exact determination of the condition. This is of great importance, inasmuch as the symptoms of chronic œsophagitis and beginning carcinoma of the œsophagus are the same; and the differential diagnosis can be made only by means of the œsophagoscope.

In chronic œsophagitis the mucosa is deep reddish-blue in color, and is thickened, often showing polypoid or papillomatous hyperplasias, or flattened plaque-like areas of epithelial hyperplasia (leukoplakia) (see Fig. 3616). The muscular coats are often hypertrophic. Over

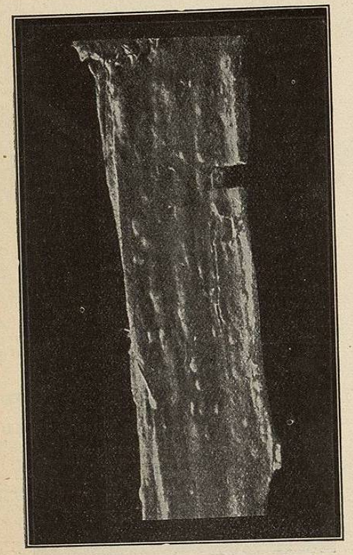

FIG. 3616.—Leukoplakia of the Œsophagus. (After Kraus.)

the mucosa lies a layer of thin or thick, often very tenacious mucus, which at times is muco-purulent in character. Small or large erosions or ulcerations may also be present. The lumen is usually dilated, but may be narrowed either symmetrically or irregularly.

Abscesses may form in either acute or chronic œsophagitis, but are rare. In these cases the local symptoms are severe, chills and fever occur, blood or bloody purulent material may be expectorated. The course is usually prolonged; dysphagia may exist for many months. In very severe cases death may occur; but milder cases usually end in recovery after several weeks or months.

Follicular œsophagitis occurs when the glands of the mucosa become involved. The gland ducts are obstructed, there is extensive mucus formation, the gland lumen is dilated, and cysts filled with mucus are in this way produced. These rarely reach the size of a pea. About the cystic glands there is a small-celled infiltration; this may lead to suppuration and formation of ulcers. Occasionally a phlegmonous œsophagitis may be associated with the follicular form.

Phlegmonous œsophagitis is of rare occurrence. It may be caused by the presence of foreign bodies or corrosive poisons, but the most frequent cause is an extension from pericœsophageal abscesses (purulent lymphadenitis, perichondritis cricoidea, spiral abscesses) or from phlegmonous processes in the pharynx, or more rarely in the stomach. It may also follow a follicular œsophagitis; through the confluence of the small follicular abscesses a diffuse purulent process may be produced. In advanced cases of pulmonary tuberculosis diffuse purulent œsophagitis or abscess formation in the œsophagus wall may occur without other evident cause.

Phlegmonous œosphagitis begins as a purulent infiltration of the submucosa, followed by a liquefaction of the tissue and the formation of circumscribed or extensive collections of pus. The mucosa may be extensively undermined; at the same time it may be reddened, and may present throughout its reddened surface numerous sieve-like yellowish apertures through which pus exudes. Large circumscribed collections of pus may cause bulgings of the mucosa into the lumen. The tissues about the œsophagus may be involved (periœsophagitis phlegmonosa), and the abscesses may rupture into the larynx and trachea, or more rarely into the mediastinum and pleura.

The symptoms of phlegmonous œsophagitis are usully obscure and offer few characteristics sufficiently striking to make the diagnosis from other conditions certain. Fever, chills, and dysphagia are the most constant symptoms. Pain may be felt behind the sternum or larynx; pressure upon the epigastrium may occasionally give rise to violent pain. Dyspnœa may be caused by pressure of an abscess upon the trachea or upon the bronchi. The dysphagia may increase to such an extent that fluids can no longer be swallowed. If the rupture of a large abscess into the œsophagus be followed by expectoration or regurgitation of pus, the diagnosis of phlegmonous œsophagitis is rendered more probable; but even in the event of such regurgitation the pus may come from an abscess in the neighborhood of the œsophagus, which has ruptured into its lumen. Only when foreign bodies are known to be lodged in the œsophagus can the diagnosis of phlegmonous œsophagitis be made with certainty. In the event of fistulous communication between the œsophagus and respiratory tract purulent pneumonia or gangrene of the lung usually results quickly. The prognosis is doubtful. Recovery may take place, the formation of cicatricial tissue in the submucosa and mucosa may give rise to stenosis, or in other cases intraparietal diverticula may be formed, the abscess cavities beneath the mucosa healing at the base but remaining open and communicating with the lumen of the œsophagus by wide openings in the mucosa having sharply cut undermined edges.

In connection with phlegmonous œsophagitis may be considered also the *periœsophageal abscess*. This has its origin most frequently in tuberculous lymph glands situated in the neighborhood of the œsophagus or in tuberculous caries of the vertebral column, or it arises in the course of a pyæmia. Further, purulent processes of the parotid or submaxillary may extend to the connective tissue about the œsophagus. Involvement of the œsophagus wall or rupture into the lumen of this organ may set up a phlegmonous œsophagitis. Rupture into the respiratory tract, pericardium, or pleura may occur. The periœsophageal abscess may reach a very large size, though often it is small. It is usually found between the fourth and seventh cervical vertebræ. The abscess arising in tuberculous lymph glands or tuberculous vertebræ is found most often in children, and its location is ordinarily between the vertebræ named. The symptoms are those of phlegmonous œsophagitis: fever, chills, dysphagia, pain on turning the neck, dyspnœa, etc. Children frequently become comatose; convulsions may occur; and finally the diagnosis may be made clear by the appearance of a swelling in the neck. In the case of tuberculous caries of the vertebræ the abscess may develop very slowly with few or no symptoms. The sound may be passed into the stomach without difficulty, but usually with more or less pain.

Œsophagitis pustulosa is the designation given to the changes in the œsophageal mucosa which occur in smallpox. Papules develop throughout the mucosa, these become cloudy and purulent, the epithelium over them is cloudy, thickened, and finally undergoes desquamation, leaving small ulcers.

Membranous Œsophagitis (Œsophagitis Fibrinosa or Diphtheritica).—True diphtheria of the œsophagus is rare; only in exceptional cases is there an extension from the pharynx into the œsophagus. Cases have been observed in which the œsophagus remained free when both pharynx and stomach were attacked; the œsophagus may, therefore, be said to possess a certain immunity in respect to diphtheria. A membranous or fibrinous œsophagitis accompanied by diphtheritic necrosis is of relatively frequent occurrence as a secondary condition in severe cases of smallpox, scarlet fever, measles, pyæmia, cholera, typhus fever, typhoid, chronic Bright's disease, pneumonia, tuberculosis, and in children as a frequent complication in intestinal catarrh. The process is rarely diffuse, but is circumscribed, and usually localized on the highest parts of the folds of the mucosa. Small ulcers may be formed at these places. Usually the symptoms of diphtheritic œsophagitis cannot be separated from the accompanying disease, but in certain cases the disease may manifest itself through hemorrhage or discharge of pseudomembranes from the œsophagus. The prognosis is very grave. When recovery takes place cicatrization of the diphtheritic ulcers may lead to stenosis of the lumen.

Œsophagitis corrosiva is produced by the action of corrosive agents, acids, or the caustic alkalies, most commonly by concentrated lye, which have been swallowed either purposely or accidentally. (See Fig. 3617.) The change produced by the corrosive agent is of the nature of a necrotic inflammation; its severity depends upon the strength or concentration of the poison. In mild cases the superficial epithelium is necrosed, and is desquamated in grayish shreds resembling a croupous membrane. Alkalies may cause the cells to swell and form a soap-like mass. A more severe action of a corrosive agent may convert the entire mucosa into a dirty gray or black eschar; the vessels are injected; the submucosa contains numerous ecchymoses, and there is a line of demarcation separating the dead tissue from the inflamed tissues of the submucosa. Active suppuration occurs and the necrotic mucosa is desquamated. If healing results, the lumen may become greatly narrowed or gradually completely occluded. In the most severe cases the deeper layers of the œsophagus may be affected. The symptoms are severe burning pain beneath the sternum, dysphagia, intense thirst, hemorrhage, and collapse. Portions of the eschar, or masses of bloody mucus may be expectorated. In the mild cases the pain ceases after twenty-four hours, the dysphagia becomes less from day to day, until the symptoms finally disappear altogether. If much scar tissue is formed, difficulty in swallowing may be experienced again after several weeks. Severe cases may be immediately fatal from shock or from hemorrhage, or from perforation; in rare cases the covrse is prolonged, abscess formation, mediastinitis, pyopneumothorax, etc., occurring as complications. Patients recovering from severe corrosive poisoning are sure to suffer from cicatricial contraction of the lumen.

The diagnosis in the majority of instances is revealed by the history of the case and the evidences of corrosive action in the mouth and pharynx. It is of importance to discover the nature of the poison in those cases which are seen shortly after the poisoning has occurred. Litmus paper may be applied to the mucosa of the mouth or pharynx as a rough method of diagnosis as to whether acid or alkali had been used, in case the facts cannot otherwise be ascertained. The majority of such cases are caused by concentrated lye or sulphuric acid. According to von Hacker one-fourth of the cases of poisoning with concentrated lye are fatal as the direct result of the poisoning; in the case of sulphuric acid, about one-half. Of those living after poisoning with concentrated lye about one-half acquire severe stricture of the œsophagus; of those living after sulphuric-acid poisoning about a third show contraction of the lumen.

Œsophagitis gangrœnosa is of rare occurrence. It may follow the action of corrosive poisons, pressure of foreign bodies within the œsophagus or of tumors, aortic aneurism, etc., from without, or it may occur as an extension from gangrenous tonsillitis, pulmonary gangrene, etc. Decubital ulcers may occur in the upper part of the œsophagus or in the lowest portion of the pharynx. (See Fig. 3618.) Two small ulcers, one on the anterior wall, the other on the posterior wall, corresponding in position, shape, and size to each other, occur coincidently, so that one ulcer appears as the impress of the other. The condition occurs only in extremely cachectic individuals who have been confined to bed for a long time; it is due to the pressure of the larynx upon the œsophagus. The ulcers appear shortly before the death of the affected individual and cause difficulty in swallowing, in this way hastening the end.

Œsophagitis exfoliativa (Œsophagitis Desiccans Superficialis).—A number of cases of acute inflammation of the œsophagus, characterized by a rapid desquamation of the entire mucosa, have been described. The desquamated epithelium may form a hollow tube, 15-25 cm. long. The desquamation may be caused by subepithelial inflammation, action of chemicals, etc., but the exact nature of the disease is unknown. The majority of the patients were neurotic or hysterical.

Acute œsophagitis of young children occurs in nurslings

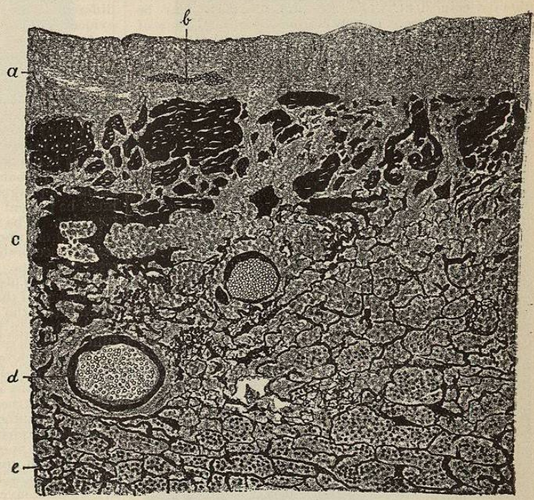

FIG. 3617.—Œsophagitis Corrosiva (after swallowing concentrated lye). *a*, The necrotic mucosa; *b*, bacteria embedded in *a*; *c*, upper portion of submucosa, infiltrated with homogeneous deeply staining masses (fibrin?); *d*, blood-vessel with necrotic wall; *e*, deeper portion of submucosa infiltrated with fibrinous network and leucocytes. (After Weichselbaum.)

as a result of a poor quality of mother's milk, soreness of the nipple, etc. It may be associated with gastro-intestinal irritation. Attempts at nursing are interrupted after a few seconds, the child cries, is restless, and regurgitates the greater part of the milk taken. Pressure upon

the lower part of the sternum is apparently painful, the child often crying out. The disease is important in that it may pass over into an ulcerative or gangrenous inflammation.

Ulcus Pepticum Œsophagi.—Ulcers in all respects similar to the round ulcer of the stomach may occur in the lower portion of the œsophagus, and are regarded as due to the same causes: viz., a digestion by the gastric juice. In cases in which the resistance of the œsophageal mucosa is lowered, the repeated entrance of the stomach fluids into the œsophagus may be followed by the development of one or more round or oval ulcers. These vary in size and depth, their edges are smooth; and the bottom of the ulcer is covered with stringy brownish-black masses. Occasionally the ulcers are very large and circular, extending ring-like about the lumen. They all show a marked tendency to cicatrization, and lead to strictures, stenosis, or the formation of diverticula. Ulcers extending deeply into the œsophagus wall may perforate, and, following this, pyopneumothorax, mediastinitis, pulmonary gangrene, pericarditis, erosion of the aorta or other large vessels, liver abscess, etc., may result. Carcinoma may develop in the scar, as is frequently the case in the stomach. Not infrequently the round ulcer of the stomach and that of the œsophagus are coincident.

The symptoms caused by round ulcer of the œsophagus are chiefly burning pain and a sensation of pressure behind the lower part of the sternum during the act of swallowing. As a rule, the pain is more severe when solid food is taken than when fluid diet is given. At other times severe cardialgia may be present; also nausea, acid regurgitations, vomiting, and hemorrhage, bloody stools, etc. These symptoms cannot be separated from those of round ulcer of the stomach, which is frequently coincident. The absolute diagnosis may be made only by means of the œsophagoscope. Ulcers of slight extent may heal without complication; after cicatrization new symptoms may appear as the result of the narrowing of the lumen. Severe cases are very danger-

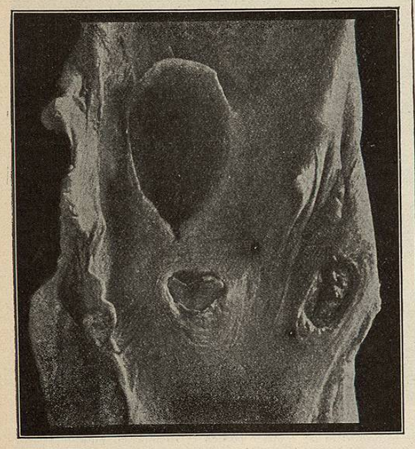

Fig. 3618.—Decubital Ulcers in the Upper Part of the Œsophagus (lower part of pharynx). (After Kraus.)

ous because of the important complications which may follow, and on account of the accompanying rapid emaciation and anæmia.

Specific Infectious Diseases.—*Tuberculosis* occurs as a secondary process in severe tuberculosis of other organs, particularly of the larynx and lungs and the neighboring lymph glands. Primary œsophageal tuberculosis has not yet been reported. The infection of the œsophagus may take place by direct extension from neighboring organs, most frequently from the rupture, into its lumen, of caseating lymph glands; or from the swallowing of sputum in cases of pulmonary tuberculosis, and very rarely from the metastasis of tubercle bacilli in acute miliary tuberculosis. It has been noted that infection does not occur in case of rupture of vertebral abscess into the œsophagus. Infection is made more likely by preceding affections of the œsophageal mucosa, ulcerations, etc. The tuberculous ulcers are usually superficial, but may lead to perforation. The edges are covered by hyperplastic epithelium, scattered through which small yellowish or gray nodules are seen; the bottom of the ulcer is either smooth or papillomatous. Tuberculosis of the œsophagus may exist without any symptoms; when symptoms referable to the œsophagus occur, they consist in dysphagia and pain behind the sternum during the act of deglutition. Thrush may exist coincidently with tuberculosis of the œsophagus, but it is to be remembered that in cases of pulmonary tuberculosis thrush or carcinoma may be found in the œsophagus as an independent affection. The use of the œsophagoscope is necessary for the absolute diagnosis.

Syphilis.—Ulcerations of the mucosa of the œsophagus may be present during either the secondary or the tertiary stage, but on the whole they are rarely observed. They have been seen also in cases of congenital syphilis. The most frequent syphilitic lesion of the œsophagus is the gumma. This may lead to ulceration, perforation, cicatricial stenosis, hypertrophy of the œsophagus wall, etc. Difficulty in swallowing is the chief symptom. The differential diagnosis rests chiefly upon the anamnesis, evidences of syphilis elsewhere, therapeutic test, etc.

Actinomycosis.—A number of cases of primary actinomycosis of the œsophagus have been reported. Inasmuch as the entrance of infection in this disease is usually through the air passages, the œsophagus may be involved by extension from the peribronchial lymph glands or from the lungs, mouth, pharynx, etc. In a given case it may be difficult to decide whether the œsophageal condition is primary or secondary; but in the majority of cases it is likely that the primary seat is in the mouth. It is also probable that, for the infection of the mucosa of the œsophagus, some other lesion (erosion, ulcer, etc.) must be present as a factor favoring the entrance of the organism. The diagnosis of œsophageal actinomycosis rests entirely upon the finding of the parasite in the material expectorated or removed by means of the sound or œsophagoscope.

Thrush.—This is the most common and important parasitic disease of the œsophagus. It may be primary or may extend from the mouth and pharynx. It is most commonly found in poorly nourished children, and in adults suffering from continued fevers, particularly typhoid and sepsis, from chronic cachexias, and from chronic tuberculosis, nephritis, and diabetes, in their last stages. (See Fig. 3619.) Healthy children and adults may be occasionally affected; but the disease is by far most common in cachectic children suffering from chronic digestive disturbances. As a rule, the pharynx and mouth are affected at the same time. The appearance of the parasite upon the mucosa of the mouth, tongue, or pharynx is very characteristic and the diagnosis is easily made. The parasite develops in the upper layer of the mucosa, its filaments forming a dense feltwork among the epithelial cells. Slightly elevated whitish or grayish patches, which can be easily scraped off, leaving a bleeding or ulcerated surface, are seen over the mucosa. In the œsophagus small white, flattened, or nodular patches may be scattered over the mucosa, or the patches may be arranged longitudinally, corresponding to the folds of the mucosa. In other cases a more diffuse growth may be seen, the œsophageal lumen to a large extent or throughout being lined by the growth. In very severe cases the growth may be so extensive as to form thick-walled casts of the lumen or even

solid cylinders. The growth may penetrate into the submucosa or even reach the muscle coats. Penetration into the blood-vessels may occur and metastasis of the parasite result. The reactive inflammation of the œso-

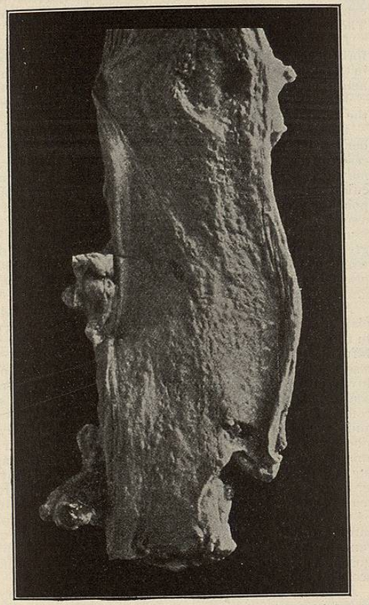

Fig. 3619.—Lower Portion of Œsophagus Showing the Presence of Both Thrush and Tuberculosis. (After Kraus.)

phageal wall is usually in proportion to the amount of infiltration, by the parasite, of the mucosa and underlying structures.

Thrush of the œsophagus rarely gives rise to independent symptoms, except when the growth is extensive. Dysphagia followed by aphagia is the chief symptom, especially in the case of children. Occasionally hollow or solid cylindrical casts of the œsophagus may be regurgitated. In those cases in which the masses of the growth are firmly adherent to the mucosa death may result from the complete obstruction of the lumen of the œsophagus.

Animal Parasites.—In general trichinosis the encysted worms may be found in the striped muscle of the œsophagus wall. Their presence may be regarded as explaining the painful deglutition occurring in the course of trichina infection. Round worms (ascaris lumbricoides) may wander into the œsophagus from the stomach; and may be found occasionally in œsophageal diverticula. From the œsophagus they may reach the respiratory passages, and entering the larynx may cause severe or even fatal obstruction to respiration. Occasionally other forms of animal life may gain accidental entrance to the œsophagus (flies, bees, wasps, leeches, hairworms, etc.); in the majority of cases no symptoms are produced, the intruder acting as a simple foreign body, except in the case of leeches and stinging insects. In the case of the former hemorrhage may be produced; and the sting of the latter may give rise to a severe œsophagitis.

Progressive Changes.—*Hypertrophy* of the wall of the œsophagus occurs above a stenosis of a portion of the lumen or of the cardiac opening. In the latter case the lumen throughout its entire length may be greatly dilated.

Tumors.—New growths of the œsophagus are on the whole not frequent. Metastatic growths are of rare occurrence. Of the primary tumors carcinoma is by far the most common. Benign tumors causing symptoms during life are very rare. The great majority of benign growths are small and clinically unimportant. Of these the most common are small *papillomatous warts* (œsophageal warts, verrucæ) (see Fig. 3620). These are usually about the size of a pinhead or bean, rarely larger, projecting above the mucosa, and are often multiple or confluent. They are found especially in old individuals. In structure these little growths consist of hyperplastic papillæ, covered with thickened epithelium resembling the condylomata. They rarely ulcerate, and only exceptionally may they offer slight obstruction to the passage of food. They are usually discovered only at autopsy, but their presence in the œsophagus could be revealed during life by means of the œsophagoscope, should they reach such a size as to cause symptoms.

Next in frequency occur *fibromata*, which usually take their rise in the connective tissue outside of the œsophageal wall, but, through pressure, are finally forced to occupy a position in the mucosa of this organ. The prevertebral fascia, the periosteum of the vertebræ, the peri-

Fig. 3620.—Papillomata of the Œsophagus. (After Kraus.)

chondrium, the pericesophageal connective tissue, and less frequently the connective tissue of the muscle and submucosa of the œsophagus wall form the points of origin