usually complete, though repeated attacks may lead to subacute gastritis or to the establishment of chronic dyspepsia.

Phlegmonous Gastritis; Acute Suppurative Gastritis.—This is an excessively rare disease, characterized by the occurrence of suppurative processes in the submucosa. The affection is more common in men than in women. The cause is seldom obvious. It has been met with as an idiopathic affection, but it has occurred also in puerperal fever and other septic processes, and has occasionally followed trauma. Anatomically there appear to be two forms, a diffuse purulent infiltration and a localized abscess formation, in which case the tumor may reach the size of an egg, and may burst into the stomach or into the peritoneal cavity.

The *symptoms* are variable. There are usually pain in the abdomen, fever, dry tongue, and symptoms of a severe infective process, delirium and coma preceding death. Jaundice has been met with in some instances. Occasionally, when the abscess tumor is large, it has been felt externally, in one case forming a mass as large as two fists. There are instances which run a more chronic course, with pains in the abdomen, fever, and chills.

The *diagnosis* is rarely possible, even when with abscess rupture occurs, and the pus is vomited, as it is not possible to differentiate this condition from an abscess perforating into the stomach from without. It is stated, however, that Chvostek made the diagnosis in one of his cases.

Toxic Gastritis.—This most intense form of inflammation of the stomach is excited by the swallowing of concentrated mineral acids or strong alkalies, or by such poisons as phosphorus, corrosive sublimate, ammonia, arsenic, etc. In the non-corrosive poisons, such as phosphorus, arsenic, and antimony, the process consists of an acute degeneration of the glandular elements, and hæmorrhage. In the powerful concentrated poisons the mucous membrane is extensively destroyed, and may be converted into a brownish-black eschar. In the less severe grades there may be areas of necrosis surrounded by inflammatory reaction, while the submucosa is hæmorrhagic and infiltrated. The process is of course more intense at the fundus, but the active peristalsis may drive the poison through the pylorus into the intestine.

The *symptoms* are intense pain in the mouth, throat, and stomach, salivation, great difficulty in swallowing, and constant vomiting, the vomited materials being bloody and sometimes containing portions of the mucous membrane. The abdomen is tender, distended, and painful on pressure. In the most acute cases symptoms of collapse supervene; the pulse is weak, the skin pale and covered with sweat; there is restlessness, and sometimes convulsions. There may be albumen or blood in the urine, and petechiæ may develop on the skin. When the poison is less intense, the sloughs may separate, leaving ulcers, which too often lead, in the œsophagus, to stricture, and in the stomach to chronic atrophy, and finally to death from exhaustion.

The *diagnosis* of toxic gastritis is usually easy, as inspection of the mouth and pharynx shows, in many instances, corrosive effects, while the examination of the vomit may indicate the nature of the poison.

In poisoning by acids, magnesia should be administered in milk or with egg albumen. When strong alkalies have been taken, the dilute acids should be administered. For the severe inflammation which follows the swallowing of the stronger poisons palliative treatment is alone available, and morphia may be freely employed to allay the pain.

Diphtheritic or Membranous Gastritis.—This condition is met with occasionally in diphtheria, but more commonly as a secondary process in typhus or typhoid fever, pneumonia, pyæmia, small-pox, and occasionally in debilitated children. An instance of it came under my notice in pneumonia. The exudation may be extensive and uniform or in patches. The condition is not recognizable during life.

Mycotic and Parasitic Gastritis.—It occasionally happens that fungi develop in the stomach and excite inflammation. One of the most remarkable cases of the kind is that reported by Kundrat, in which the favus fungus developed in the stomach and intestine.

In cancer and in dilatation of the stomach the sarcinæ and yeast fungi probably aid in maintaining the chronic gastritis. As a rule, the gastric juice is capable of killing the ordinary bacteria. Orth states that the anthrax bacilli, in certain cases, produce swelling of the mucosa and ulceration. Klebs has described a *bacillus gastricus* which develops in the tubules and produces numerous spores, and Eug. Fraenkel has reported a case of acute emphysematous gastritis probably of mycotic origin. The larvæ of certain insects may excite gastritis, as in the cases reported by Gerhardt, Meschede, and others. In rare instances tuberculosis and syphilis attack the gastric mucosa.

III. CHRONIC GASTRITIS

(*Chronic Catarrh of the Stomach; Chronic Dyspepsia*).

Definition.—A condition of disturbed digestion associated with increased mucus formation, qualitative or quantitative changes in the gastric juice, enfeeblement of the muscular coats, so that the food is retained for an abnormal time in the stomach; and, finally, with alterations in the structure of the mucosa.

Etiology.—The causes of chronic gastritis may be classified as follows: (1) Dietetic. The use of unsuitable or improperly prepared food. The persistent use of certain articles of diet, such as very fat substances or foods containing too much of the carbohydrates. The use in excess of tea or coffee, and, above all, alcohol in its various forms. Under this heading, too, may be mentioned the habits of eating at irregular hours or too rapidly and imperfectly chewing the food. A common cause of chronic

catarrh is drinking too freely of ice-water during meals, a practice which plays no small part in the prevalence of dyspepsia in America. Another frequent cause is the abuse of tobacco. (2) Constitutional causes. Anæmia, chlorosis, chronic tuberculosis, gout, diabetes, and Bright's disease are often associated with chronic gastric catarrh. (3) Local conditions: (*a*) of the stomach, as in cancer, ulcer and dilatation, which are invariably accompanied by catarrh; (*b*) conditions of the portal circulation, causing chronic engorgement of the mucous membrane, as in cirrhosis, chronic heart disease, and certain chronic lung affections.

Morbid Anatomy.—Anatomically two forms of chronic gastritis may be recognized, the simple and the sclerotic.

(*a*) **Simple Chronic Gastritis.**—The organ is usually enlarged, the mucous membrane pale gray in color, and covered with closely adherent, tenacious mucus. The veins are large, patches of ecchymosis are not infrequently seen, and in the chronic catarrh of portal obstruction and of chronic heart disease small hæmorrhagic erosions. Toward the pylorus the mucosa is not infrequently irregularly pigmented, and presents a rough, wrinkled, mammillated surface, the *état mammeloné* of the French, a condition which may sometimes be so prominent that writers have described it as *gastritis polyposa*. The membrane may be thinner than normal, and much firmer, tearing less readily with the finger-nail. Ewald thus describes the histological changes: The minute anatomy shows the picture of a parenchymatous and an interstitial inflammation. The gland cells are in part eroded or show cloudy granular swelling or atrophy. The distinction between the "haupt" and "beleg" cells cannot be recognized, and in many places, particularly in the pyloric region, the tubes have lost their regular form and show in many places an atypical branching, like the fingers of a glove. Individual glands are cut off toward the fundus, but appear at the border of the submucosa as cysts, partly empty, with a smooth membrane, partly filled with remnants of hyaline and refractile epithelium. An abundant small-celled infiltration presses apart the tubules and is particularly marked toward the surface of the mucosa, and from the submucosa extensions of the connective tissue may be seen passing between the glands. The mucoid transformation of the cells of the tubules is a striking feature in the process and may extend to the very fundus of the glands.

(*b*) **Sclerotic Gastritis.**—As a final result of the parenchymatous and interstitial changes the mucous membrane may undergo complete atrophy, so that but few traces of secreting substance remain. There appear to be two forms of this sclerotic atrophy—one with thinning of the coats of the stomach, *phthisis ventriculi*, and a retention or even increase of the size of the organ; the other with enormous thickening of the coats and great reduction in the volume of the organ, the condition which is usually described as *cirrhosis ventriculi*. Extreme atrophy of the mucous membrane of the stomach has been carefully studied by Fenwick,

Ewald, and others, and we now recognize the fact that there may be such destruction and degeneration of the glandular elements by a progressive development of interstitial tissue that ultimately scarcely a trace of secreting tissue remains. In a characteristic case, studied by Henry and myself,* the greater portion of the lining membrane of the stomach was converted into a perfectly smooth, cuticular structure, showing no trace whatever of glandular elements, with enormous hypertrophy of the muscularis mucosæ, and here and there formation of cysts. In the other form, with identical atrophy and cyst formation, there is enormous increase in the connective tissue, and the stomach may be so contracted that it does not hold more than a couple of ounces. The walls may measure from two to three centimetres; the greatest increase in thickness is in the submucosa, but the hypertrophy also extends to the muscular layers. While one is not justified in saying that all cases of cirrhosis of the stomach represent a final stage in the history of a chronic catarrh, it is true that in most cases the process is associated with atrophy of the gastric mucosa, while the history indicates the existence of chronic dyspepsia.

Symptoms.—The affection persists for an indefinite period, and, as is the case with most chronic diseases, changes from time to time. The appetite is variable, sometimes greatly impaired, at others very good. Among early symptoms are feelings of distress or oppression after eating, which may become aggravated and amount to actual pain. When the stomach is empty there may also be a painful feeling. The pain differs in different cases, and may be trifling or of extreme severity. When localized and felt beneath the sternum or in the præcordial region it is known as heart-burn or sometimes cardialgia. There is pain on pressure over the stomach, usually diffuse and not severe. The tongue is coated, and the patient complains of a bad taste in the mouth. The tip and margin of the tongue are very often red. Associated with this catarrhal stomatitis there may be an increase in the salivary and pharyngeal secretions. Nausea is an early symptom, and is particularly apt to occur in the morning hours. It is not, however, nearly so constant a symptom in chronic gastritis as in cancer of the stomach, and in mild grades of the affection it may not occur at all. Eructation of gas, which may continue for some hours after taking food, is a very prominent feature in cases of so-called flatulent dyspepsia, and there may be marked distension of the intestines. With the gas, bitter fluids may be brought up. In other instances a clear watery fluid is ejected (pyrosis or water-brash). The vomiting does not often occur when the stomach is empty, but either immediately after eating or an hour or two later. The vomitus consists of food in various stages of digestion and slimy mucus, and the chemical examination shows the presence of abnormal acids, such as butyric, or even acetic, in addition to lactic acid, while the hydrochloric acid, if indeed it is present,

* American Journal of the Medical Sciences, 1886.