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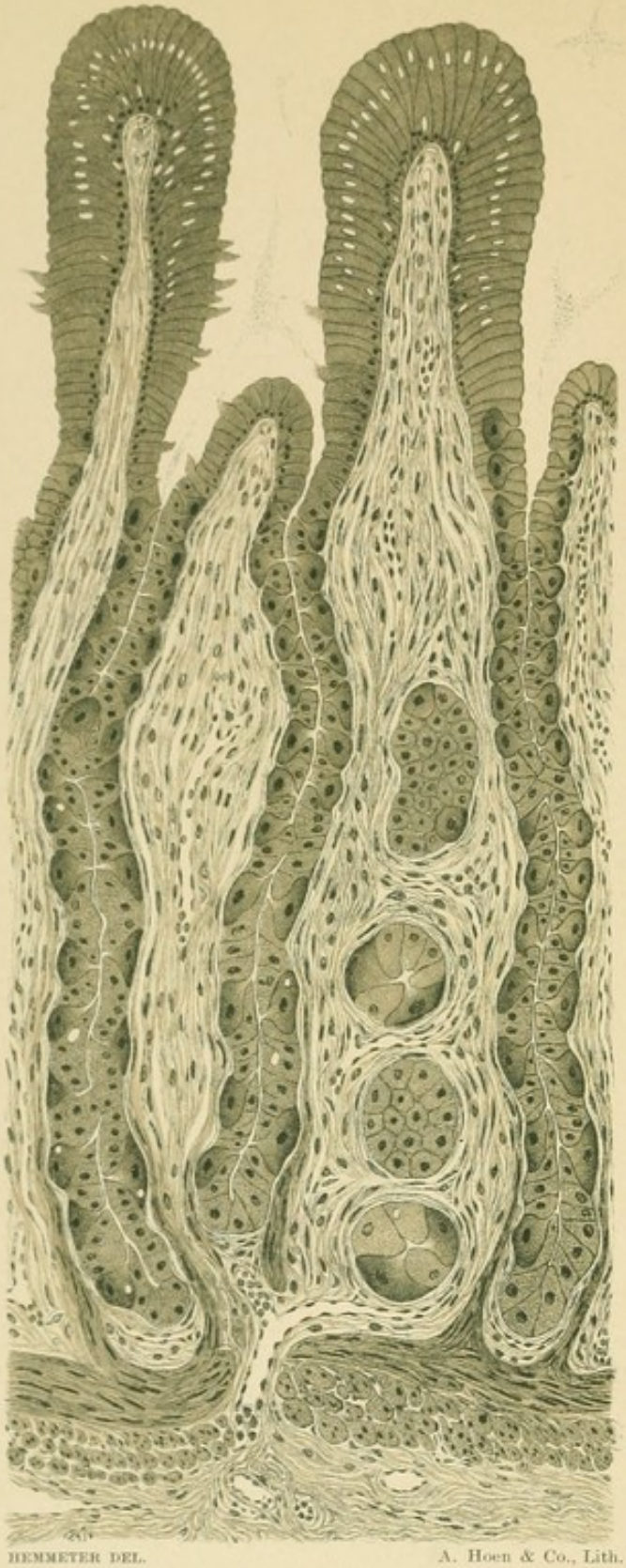

VERTICAL SECTION THROUGH NORMAL HUMAN GASTRIC MUCOSA.

DISEASES

OF THE

STOMACH

THEIR SPECIAL PATHOLOGY, DIAGNOSIS, AND TREATMENT, WITH SECTIONS ON ANATOMY, PHYSIOLOGY, CHEMICAL AND MICROSCOPICAL EXAMINATION OF STOMACH CONTENTS, DIETETICS, SURGERY OF THE STOMACH, ETC.

IN THREE PARTS

BY

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WITH MANY ORIGINAL ILLUSTRATIONS

A NUMBER OF WHICH ARE IN COLORS

AND A LITHOGRAPH FRONTISPIECE

PHILADELPHIA

P. BLAKISTON, SON & CO.

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THE BELLY AND THE MEMBERS

The members of the body rebelled against the Belly, and said, "Why should we be perpetually engaged in administering to your wants, while you do nothing but take your rest and enjoy yourself in luxury and self-indulgence?" The members carried out their resolve, and refused their assistance to the Belly. The whole body quickly became debilitated, and the hands, feet, mouth, and eyes, when too late, repented of their folly.—*Æsop*.

TO

PROFESSOR WILLIAM OSLER, M.D.

OF BALTIMORE.

Il maestro di color che sanno.—*Dante.*

THIS VOLUME IS RESPECTFULLY

DEDICATED.

PREFACE.

The tendency of modern science—not only of medical science—is toward specialization.

Diseases of the stomach alone is not a field sufficiently large to constitute a genuine specialty. It is generally associated with the study of the diseases of all digestive organs, particularly of the intestines, liver, and pancreas. The diseases of metabolism constitute a legitimate field naturally falling into the domain of the digestive clinical pathologist.

In an address before the Medical and Chirurgical State Faculty of Maryland in April, 1896, Professor Da Costa, in speaking of the manner in which medical libraries build up and increase, said that "books attract books, and, as a rule, any new work in any particular class has a striking family resemblance to those already published."

If this new contribution to the pathology and treatment of organic diseases of the stomach does not conform to Da Costa's generalization, it is not because of any premeditated plan to make it different from other works on the same subject, but because a number of entirely new methods of diagnosis have entered into it, and because an attempt has been made to do justice to the work of American clinicians in this special department. My chief effort has been to furnish the general practitioner with a work from which he can readily acquaint himself with all that has been done in this important branch of medicine, to fit himself to make examinations, to take advantage of new methods of diagnosis, and to treat this very difficult class of diseases rationally and successfully.

With this end in view I have endeavored to treat the subject systematically and concisely, giving first the special anatomy and physiology of the digestive organs, methods of diagnosis and general therapy, including dietetics, following this by a methodical discussion of the various diseases affecting the stomach, with their

symptomatology, diagnosis, prognosis, pathology, and treatment. The illustrations, of which many are from original drawings, have been selected because of their practical bearing upon the matter in hand.

Aside from the fact that the pathology, diagnosis, and therapy of diseases of the human organs have become so extensive that it is absolutely impossible for one mind to master them all, genuine advances in any particular department have hitherto been made only by such scholars as could concentrate and focus their mental energy upon a limited subject.

Experience of the last twenty-five years has demonstrated that the general, fundamental stock of medical knowledge has not been injured, but, on the contrary, it has been wonderfully enlarged and strengthened by the progress in strictly special fields of work.

To read the history of the development of medical sciences, the frequently astonishing results and indefatigable perseverance of "the grand old men of medicine," is not only a healthy training for prospective investigators, but can not fail to polish down the pride of the overambitious.

Speaking purely from a therapeutic standpoint, however, our medical ancestors of the beginning of this century were for the greater part divided into two extreme classes: First, the polypharmacists; second, the skeptics, the therapeutic nihilists. It is largely the credit of the specialties that Asclepiads have evolved from this confused opposition. It was an unspeakable comfort to be reassured by Virchow and others that, after all, the end-object, the fundamental purpose, of all medical progress, must be the relief of suffering and the cure of disease, not simply the development of abstract science. A further step in the evolution of therapy was the realization that the object of medical study and treatment must not be the "disease," but the diseased patient. Specialties can not make the adept one-sided, nor obscure his view of the general body of medical knowledge; on the contrary, the detailed development of the intellect which results from concentration of energy upon one subject will enlarge his powers of observation and analysis and insure a more comprehensive understanding of the totality of general medicine. Boerhaave could claim to be master of all applied medical branches; Langenbeck and Frerichs were credited with absolute mastership in three or four heterogeneous branches of medicine. Medicine has been enormously de-

veloped since those days. Who will claim such mastery at the present time? Bach, Mozart, and Haydn were acknowledged virtuosi on five or six instruments. Where is such a phenomenal genius of the present day in music? The enlargement of any branch of human knowledge or art brings specialization with it as a natural sequence; that this tendency is a blessing for the central fundamental stock of knowledge, science, or art has been proven in many branches. Perhaps as good an evidence of this fact as any is the advantage which general medicine is just beginning to reap from the brilliant results of bacteriology.

When the printing of this book was begun, there was no work of American origin on this subject. Since then the volume by Einhorn has appeared, being a compilation of the monographs by this author in the "Twentieth Century Practice of Medicine."

We already have a large number of eminently qualified and versatile clinicians, men with acute observing powers and analytical minds, who have worked in this interesting field. The names of Austin Flint, Pepper, Osler, and Delafield are as well known in this department in our country as those of Kussmaul, Senator, Nothnagel, Leube, Ewald, and Boas in Germany, or Hayem, Bouveret, Debove, and Mathieu in France.

Among those who have made contributions of note to this special line of work are, in addition to Einhorn, George Dock, W. D. Booker, Charles G. Stockton, Allen Jones, D. D. Stewart, Julius Friedenwald, Francis P. Kinnicut, Charles E. Simon, and other gifted experimenters and observers.

The anatomy of the stomach has received a lasting benefit through the intellect of F. Mall, of Baltimore.

The surgery of the alimentary tract has many very creditable representatives in our country, among whom may be mentioned Robert F. Weir, N. Senn, John B. Deaver, McBurney, Roswell Park, F. Lang, R. Abbe, W. Meyer, Murphy, Bull, Maurice H. Richardson, Gerster, and John M. T. Finney. The literary and practical contributions of a number of these men have reached a classic standard and compelled foreign admiration.

The physiological chemistry of digestion and internal secretion has received the benefit of the work of Bowditch, Chittenden, Howell, Vaughn, Adami, Able, and others, and dietetics has its versatile representative in Gilman Thompson.

To Messrs. Blakiston, Son & Co., the publishers, the author feels

sincerely grateful. It would be a neglect to omit an expression of this feeling. The manner in which they have executed their part of the work speaks for itself. It is a great pleasure for an author to be able to work with such intelligent and enthusiastic publishers.

To Dr. Edward L. Whitney, my associate, it becomes my pleasant duty to express thanks for the able manner in which he has written the chemical section of part first, and also for much kind assistance throughout the work.

Pathology has its men now universally acknowledged for the integrity and dignity of their work in our esteemed teachers, Welch and Councilman. Already an American School of Pathology is forming, with these men and Prudden, Flexner, and others. But in the special pathology of the digestive organs the workers are few; a very creditable beginning, however, has been made; the foundation is an honor to the prospective builders, but the land to be explored is exceedingly large in its extent, and "*the harvest is plenteous, but the laborers are few.*"

JOHN C. HEMMETER.

BALTIMORE, 1897.

"Heard are the voices,
Heard are the sages,
The worlds and the ages;
Choose well, your choice is
Brief and yet endless.

"Here eyes do regard you
In eternity's stillness,
Here is all fullness,
Ye brave, to reward you;
Work and despair not."

—*Goethe.*

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DISEASES OF THE STOMACH.

PART FIRST.

ANATOMY AND PHYSIOLOGY OF THE DIGESTIVE ORGANS.—METHODS AND TECHNICS OF DIAGNOSIS.

CHAPTER I.

ANATOMY OF THE STOMACH.

Many valuable contributions to the subject of the macroscopical and microscopical anatomy of the stomach have been made during recent years. In the subjoined brief synopsis we have availed ourselves of the valuable researches of F. Mall, and of the works quoted by him in the bibliography given in his article in the "Johns Hopkins Hospital Reports," volume 1. The comprehensive works of Oppel, Spalteholz, and others which have appeared during 1896–97, have also been consulted.

The stomach is the dilated, sac-like portion of the digestive tract, between the esophagus and the small intestine. One can distinguish a lower convex arch, the greater curvature, which is directed toward the left and downward; and an upper concave arch, the lesser curvature, which is directed toward the right and upward. The broad left end of the greater curvature is called the fundus, the size of which varies according to age. Between the fundus and the lesser curvature is situated the cardia, being the continuation and funnel-shaped expansion of the esophagus. While it is not marked on the outside of the organ, there is a distinct limiting line internally on the mucous membrane, which is caused by a change in the structure of the epithelial lining. This zigzag line separates the cardia from the esophagus. It is now

well known that at this point the arrangement of the muscular fibers and veins is also different from that in the esophagus.

The location of the cardia in the adult is at the twelfth dorsal vertebra. At about the height of the bifurcation of the bronchi, the spiral curving of the esophagus around the aorta begins. By executing this curve, the convexity of which is toward the right, the esophagus gets to the left side of the aorta, and passes through the diaphragm in the foramen œsophageum, near the spinal column.

The stomach becomes narrower from the fundus toward the pylorus. Near the pylorus there is a constriction, caused by a ring-like formation of muscular tissue, which corresponds to the pyloric valve. The muscular tissue is covered internally by the gastric mucous membrane, the latter forming the pyloric valve; the opening of this valve is of varying diameter. The part of the stomach in advance of the pylorus is called the antrum pyloricum, and is frequently separated from the greater curvature by an indentation or depression. This antrum may be elongated so as to assume resemblance to the intestine; which is frequently the case in the female.

On the anterior and posterior walls of the stomach, running along between the muscular and serous coats of the organ, are two band-like stripes, consisting of elastic, smooth, muscular fibers. These are the pyloric ligaments.

The size of the stomach depends upon age, sex, and individuality, and upon the degree of its distention. The long axis extends from 25 to 35 cm. The greatest vertical measurement, at the cardia, is 15 cm., and the greatest straight diameter is from 11 to 12 cm.; the smallest, at the antrum pyloricum, is from three to four cm. In the female it is generally smaller and more slender.

The capacity varies considerably; Ewald considers that from 1600 to 1700 c.c. is the normal limit. Three-fourths of the stomach belong to the left half of the body and one-fourth belongs to the right half. The cardia is located behind the median edges of the fifth and sixth ribs. The fundus, the largest part of the body of the organ, is in the left hypochondrium; the rest, with the pyloric part, is in the epigastrium. The pylorus lies in the right half of the body, but occasionally changes to the middle line at the level of the seventh and eighth ribs, in a line with the ensiform cartilage. The lesser curvature runs along to the left, and near the spinal column. The

vaulting dome of the fundus, which applies itself to the concavity of the diaphragm, is the highest point. The deepest point of the stomach is in the greater curvature, in the inferior half of an imaginary straight line connecting the ensiform cartilage with the umbilicus. Both the highest and lowest parts of the stomach are moved about according to the level of the diaphragm and the distention of the stomach. In an empty condition, the stomach is withdrawn into the upper portion of the abdomen; but when filled it extends in all directions, but mostly in the direction of its long axis, from the left above downward to the right. In a state of moderate distention about 40 cm. of its anterior wall comes in contact with the inner surface of the anterior abdominal wall.

The diaphragm covers the fundus and the largest part of the left segment, while the left lobe of the liver, up to the sulcus interlobularis, covers the smallest part; that is, the lesser curvature and the pyloric portion. From this fact arises the difficulty in palpating tumors in the latter place, which is impossible, except when gastroptosis, or descent of the stomach, moves it away from the liver. In the state of expansion or dilation, the stomach moves out from behind the liver; but the lesser curvature can not change its location to any considerable extent, and the change of location of the whole stomach caused by filling is produced almost exclusively by an extension of the greater curvature.

The pancreas extends along the posterior wall of the stomach. At the upper edge of the pancreas are the splenic artery and vein. The transverse colon runs along the greater curvature, and its left flexure fills the remaining space in the left hypochondrium. The location of the stomach is fixed by a ligamentous attachment of the cardia, by the pylorus, and also by a number of suspensory ligaments, which are all formations of the peritoneum. Some authors say that the stomach is supported in this position by intra-abdominal pressure. The experiments of Moritz (of Munich) and the author have proved that intra-abdominal pressure adds nothing to the support of the stomach. The gastrophrenic ligament, which toward the right passes into the lesser omentum, and toward the left extends into the phrenosplenic ligament, surrounds and embraces the cardia, which is lower than the fundus, its situation corresponding to the upper end of the sixth and seventh costal cartilages, or to the level of the ninth thoracic vertebra. This part of the stomach is therefore moved to the left of the middle line,

and next to the spinal column, at about the level of the twelfth thoracic and first lumbar vertebræ; here it is fixed to the lumbar part of the diaphragm.

The greater omentum arises from the large curvature. The posterior fold of this omentum forms the mesocolon transversum. This is the reason why changes of location in the greater omentum (hernia and inflammatory adhesions) can produce traction upon the stomach. As the stomach is really attached only at the cardia, and the pylorus adheres to the posterior abdominal wall by the descending portion of the duodenum, the organ is capable of being moved about, not so much in its entirety as in its parts (the great curvature, for instance). The stomach has a complete peritoneal covering, which consists of an anterior and posterior layer, uniting at the two curvatures of the stomach to form the lesser and greater omentum; between these two layers a space is left for the blood- and lymph-vessels of the stomach.

Muscular Layer.—The muscular stratum contains three kinds of fibers,—longitudinal, transverse, and oblique. The longitudinal layer of muscular fibers, a continuation of those of the esophagus, presents a denser arrangement at the lesser curvature than at the greater, and forms the ligamenta pylorica at the pyloric part, which are bands of muscular fibers expanded and broadened out,—not ligaments in the real sense of the word.

The circular layer of muscular fibers is placed internally to the longitudinal layer, the fibers of which it crosses at right angles. The fibers of this circular layer are placed around the stomach in a ring or belt-like manner; at the pylorus it shows a local thickening of the muscle rings—the sphincter pylori; a fold of the mucosa to the innermost side of this sphincter constitutes the pyloric valve. The longitudinal fibers also have a part in the formation of the sphincter, for while the superficial layer of longitudinal fiber passes on over the pyloric sphincter into the duodenum, the deeper longitudinal fibers enter the pyloric valve encircling and grasping the circular fibers in a loop-like manner (dilator pylori—Rüdinger). The cardia has no special sphincter, but the oblique fibers cross and decussate at the periphery of this portion. The sphincter pylori is contracted during digestion, but gas and liquids can readily escape through the cardia. The oblique fibers are limited chiefly to the cardiac end of the stomach, where they are disposed as a thick, uniform layer, some passing obliquely from left to right,

others from right to left, around the cardiac orifice. The submucosa, or cellular coat of the stomach, consists of a loose, filamentous, areolar tissue, and loosely binds the mucosa to the muscular layers.

The most important and interesting layer is the mucosa, or mucous membrane proper, of the stomach; it is a thick layer with a smooth, soft, velvety surface. During infancy, and immediately after death, it is of a pinkish tinge, but in adult life and in old age it becomes of a pale straw or ash-gray color; at the pylorus it is much thicker than at the cardia. During the contracted state of the organ it is thrown into numerous plaits or rugæ, which, for the most part, have a longitudinal direction, and are most marked toward the lesser end of the stomach and along the greater curvature; these folds are entirely obliterated when the organ becomes distended.

Structure of the Mucous Membrane.—When examined with a lens, the inner surface of the mucous membrane presents a peculiar honeycomb appearance, from being covered with small, shallow depressions, or alveoli, of a polygonal or hexagonal form, which vary from $\frac{1}{100}$ to $\frac{1}{350}$ of an inch in diameter, and are separated by slightly elevated ridges. In the bottom of the alveoli are seen the orifices of minute tubes, the gastric follicles, which are situated perpendicularly side by side in the entire substance of the mucous membrane; they are short, and of a simple tubular character toward the cardia, but at the pyloric end they are longer, more thickly set, convoluted, and terminate in dilated saccular extremities, or are subdivided into from two to sixteen tubular branches.

Watney has pointed out that these convoluted, or coiled, tubes form the transition from the simple tubular follicles to the convoluted glands of Brunner, which lie immediately below the pylorus. The gastric follicles are composed of a homogeneous basement membrane, formed by the connective tissue framework, lined upon its free surface by a layer of cells, which differ in their character in different parts of the stomach. Toward the pylorus these tubes are lined throughout by columnar or cuboidal epithelium; they are termed the mucous glands, and are supposed to secrete the gastric mucus. In other parts of the organ the deep part of each tube is filled with nucleated cells, the upper fourth of the tube

being lined by columnar epithelium: These are called the peptic glands, and are the supposed source of the gastric juice.

Simple follicles are found in greater or less numbers over the entire surface of the mucous membrane; they are most numerous near the pyloric end of the stomach, and are especially distinct in early life. The epithelium lining—the mucous membrane of the stomach and its alveoli—is of the columnar variety.

Usually four to sixteen gland openings are found at the base of each follicle. According to Sappey there are 5,000,000 of these glands in the organ, for which reason the gastric mucosa may justly be considered a continuous gland spread out into a flat surface (Hyrtl and Luschka). The gland tubules are as long as the entire thickness of the mucosa, and their sac-like and branched bases extend into the muscularis mucosa, the action of these muscular fibers assisting in the evacuation of the tubules during digestion. The ends of the tubules extending into the muscular layer are usually branched.

The cylindrical epithelium of the surface of the stomach, which is separated from the pavement epithelial lining of the esophagus by a distinct but irregular line at the beginning of the cardia, is found also in the beginning of the tubules, but only extends downward to one-quarter or one-third their length; from this point downward they are no longer observed, but two different kinds of cells are now seen to line the glandular tubule. One variety of lining cells is located toward the axis of the tube, and is known as the adelomorphous cells of Rollet, because they show no cell contours or outline in the fresh state, called by Heidenhain chief cells; they have a cubical, cylindrical form and indistinct nuclei. The second variety shows larger, round cells, the delomorphous cells of Rollet, also known as parietal cells, which in the fresh state exhibit a finely granular contour, becoming sharply outlined on addition of water or salt-solution; they show coarsely grained contents and a distinct nucleus. The first form, the cubical or cylindrical, chief, central, or adelomorphous cells, are supposed to secrete the ferments, pepsin and rennin; the second form, the larger, round, delomorphous, oxyntic, or parietal cells, are supposed to secrete the HCl.

The peptic glands consist of a duct, a neck, and a fundus; the latter is the deepest portion, and is often divided. These tubular glands apparently have a distinct membrana propria separating

them from the loose, areolar, connective tissue, in which they lie. Mall has not been able to establish the existence of a distinct basement membrane.

Three Kinds of Cells of the Peptic Glands.—First, the cylindrical cells of the gland duct and pit, lining one-fourth to one-third of the distance from the surface of the mucous membrane downward. They are a continuation of the cylindrical epithelium of the general internal surface of the gastric mucous membrane; these cells seem to secrete mucus only. Secondly, the lightly colored, pyramidal or cuboidal cells, with a granular protoplasm and spherical nucleus. These cells do not stain with anilin, and were termed adelomorphous cells by Rollet because they show no cell contours in the fresh state. Rosenheim states that they are almost clear and transparent during fasting, and become cloudy and granular during digestion. Heidenhain designated them as the chief, or central, cells, and they were held by him to be the sources of the ferments, —pepsinogen and rennin zymogen. These chief, or central, cells touch the lumen of the duct more extensively than the next following variety. The third kind of peptic cells are known as the border, parietal, or oxyntic cells, because they rest upon the membrana propria with much broader bases than the chief, or central, cells. For this very reason they participate to a less degree in the limitation of the lumen of the duct. They are generally round or triangular, finely granular, and stain intensely with anilin, and were designated by Rollet as delomorphous cells. Heidenhain supposes them to be the sources of hydrochloric acid. If we assume, for the sake of locating these various cells, a division of the tubule into four sections, beginning at the portion nearest the submucosa, we shall have (*a*) the fundus of the gland tubule; then (*b*) the outer secretory portion; (*c*) the inner secretory portion; and, opening on the inner surface of the mucosa, (*d*) the alveolus ("Vorraum"). Then, one finds the border, parietal, oxyntic, delomorphous, or anilin, cells most numerous in the outer secreting portion, and becoming scarce in the fundus or end portion. A fourth kind of cell occurring at rare intervals is known as Nussbaum's cell—its significance is unknown.

Heidenhain asserted that there were no border cells in the fundus at all; but this has been denied by Stöhr, Kupffer, and Boas. The size of border, or acid, cells depends upon the stage of digestion; as this function proceeds, the border cells increase, and

diminish again at the end of digestion. The chief, central, or ferment, cells enlarge also, and become darker during digestion. In a fasting state, the chief cells are largely in excess. Heidenhain's conclusions, that the chief, or central, cells are producers of the digestive ferments, and that the border, or anilin-staining, cells produce the hydrochloric acid, have been confirmed by a number of other observers (Grützner, von Swiezicki, and, recently, Sehrwald and Mall).

Their method of experimentation was mainly the following: It is known that the glandular tubules of the pyloric region contain only chief, or central, cells (producing ferments only, and no acid), while the gland tubules of the fundus contain both central cells and also border, or acid, cells. Now, Heidenhain succeeded, in a number of dogs, in removing the pyloric portion of the stomach entirely, and uniting the organ with the external abdominal wall. In other dogs he removed the fundus entirely, leaving the pyloric portion intact, and succeeded in making this altered stomach without a fundus unite with the external abdominal wall.

He therefore had two kinds of operated animals with stomachs opening on the abdomen. After this, it was found that animals in which the pyloric region was excised furnished a juice that contained both acid and pepsin; these are therefore produced by the glands of the fundus which contain both varieties of secretory cells. In the animals, however, that had been deprived of the fundus by excision, the only secretory surface that was left being the pyloric region, it was found that an alkaline juice was secreted containing only ferments. That this juice did contain pepsin was proved by its power of digesting fibrin when hydrochloric acid was added to it.

Now, as the gland tubules of the pylorus contain only chief, or central, cells, which do not stain with anilin, the conclusion is justifiable, that the chief cells secrete only ferments, and that therefore the border, or anilin-staining, cells must secrete the hydrochloric acid.

It has been found that the border, or acid, cells,—called also the oxyntic cells,—are in communication with the central canal of the gland tubule by a tiny canaliculus,—an extension from the central lumen of the gland to, or into, the oxyntic, or acid, cells. These canaliculi were brought out with the silver stain by Golgi.

CHAPTER II.

HISTOLOGY OF THE STOMACH.

R. R. Bensley, B.A., M.B., has published a very interesting paper on the "Histology and Physiology of the Gastric Glands," in the "Proceedings of the Canadian Institute, 1896." The work was done in the biological laboratory of the University of Toronto. Mr. Bensley was kind enough to present us with four sketches illustrating the various phases of secretion in the gland cells of the deep ends of the fundus glands of the cat's stomach. We consider his results a valuable addition to the work of Heidenhain, Ebstein, Langley, Sewall, and others. With staining as used by him, it is possible to recognize the precursory stages of the ferments within the structure of the cells. We submit Mr. Bensley's drawings, with explanatory text. The following are his conclusions:

"1. During digestion, a substance similar in chemical properties to the chromatin of the nucleus makes its appearance in the outer clear zone of the chief cells of the fundus glands. This substance, which may be called prozymogen, stains deeply and readily in hematoxylin, and presents a characteristic fibrillated appearance. During rest this prozymogen is used up in some way, giving rise to zymogen granules.

"2. The chief cells of the neck of the gland do not contain at any period of digestion either zymogen or prozymogen, but are engaged in the formation of a mucinoid secretion, which has a powerful elective affinity for indulin and Bordeaux red, and stains metachromatically in thionin.

"3. The pyloric gland cells, likewise, form neither zymogen nor prozymogen, and are similar in structure, in staining properties, and in the nature of their secretion, to the cells of the neck of the fundus gland.

"4. The cells of the pyloric glands and of the neck of the fundus gland pass, by gradual transition, into the mucous cells of the surface, to which they are obviously closely allied."

From Mall's article on the anatomy of the stomach ("Johns Hop-

kins Hospital Reports," vol. 1) we have inserted the following graphic description:

"**Mucosa.**—That more than one kind of gland is present in the

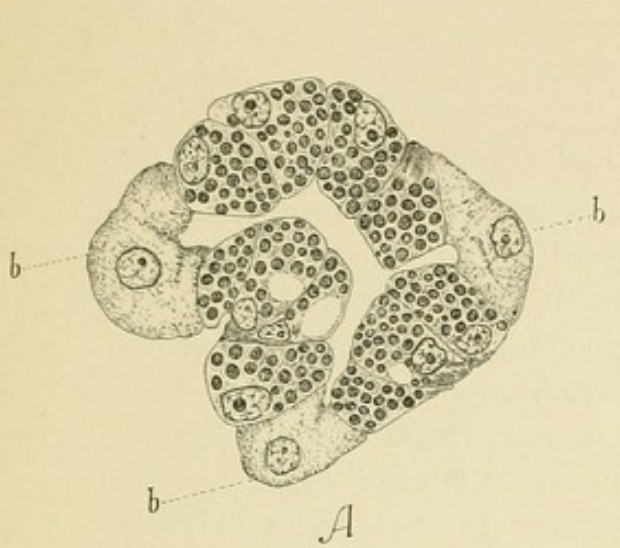


FIG. 1.

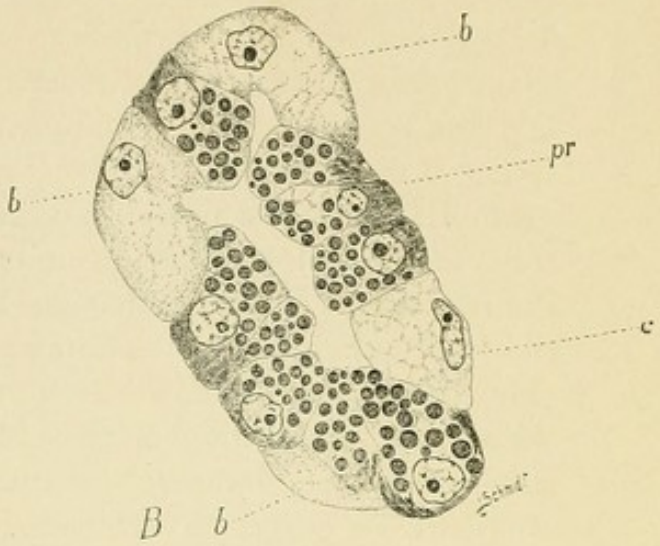


FIG. 2.

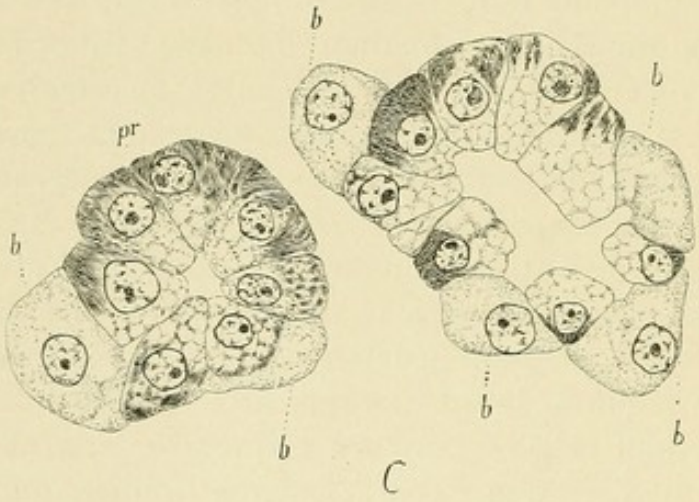


FIG. 3.

SECTIONS OF DEEP ENDS OF FUNDUS GLANDS OF THE CAT IN DIFFERENT SECRETIVE PHASES. × 1000.—(*Bensley.*)

FIG. 1.—From a fasting stomach. The chief cells are filled with large zymogen granules; nuclei near the outer ends of cells. Gentian-violet preparation. *b*. Border cells.

FIG. 2.—Six hours after an abundant meal of raw flesh. The chief cells exhibit two zones, the inner occupied by large zymogen granules, the outer by a deeply staining, obscurely fibrillar element, *prozymogen;* the nuclei lie at the junction of the two zones. *b*. Border cells. *pr*. Prozymogen. *c*. Mucin secreting cell, similar to those found in the neck of the gland. Gentian-violet preparation.

FIG. 3.—Twelve hours after feeding with sponge soaked in fat. Preparation stained in hematoxylin exhibits a deeply stained outer zone filled with prozymogen, and a clear inner zone from which the granules have disappeared in course of preparation. The nuclei are now much nearer to the lumen. *b*. Border cells. *pr*. Prozymogen.

stomach has been repeatedly noticed (Wassman, Frerichs, Brinton, Leydig, Kölliker), but a more careful study of them was delayed until 1870 (Heidenhain, Rollet).

"In general there are two kinds of glands present in the dog's stomach,—the pyloric and the peptic. The peptic, in turn, are formed in great part of two kinds of cells,—the border and the central.

"The section through the entire length of the stomach shows that in the pyloric region the necks of the glands are the longest (.68 mm.), and that they diminish in length throughout the middle zone (.25 mm.), until the cardiac portion is reached. In the pyloric portion, where the necks of the glands are the longest, many gland tubes empty into one outlet; in the middle zone there are less, in rough about nine, into each gland mouth; while in the cardiac portion each gland has a special opening—in other words, there are no gland necks. In the pyloric portion the glands are composed wholly of central cells. In the central zone there are many border cells, the proportion to the central cells being as described by Heidenhain and his pupils. Throughout the fundus are but few border cells, while around the esophagus there is a small zone in which there are many border cells.

"In the pyloric portion about 1600 gland tubes open within each square centimeter of mucous membrane, in the middle zone 2500, and in the fundus 4900. For an average stomach there are about 28 square cm. area in the pylorus, 108 in the middle zone, and 120 in the cardiac portion, or these surfaces are to each other as 7 : 27 : 30. The estimation carried farther gives somewhat over 1,000,000 gland openings in the stomach. On the other hand, if the blind tubes opposite the muscularis mucosa are estimated, the number exceeds 16,500,000. In other words, each gland neck subdivides 16 times, on an average, before the muscularis mucosa is reached. It may be interesting to note that for each gland opening in the stomach we have one villus in the intestine, and for each subdivision there is one Lieberkühn's crypt."

Mall's observation, as well as that of others, does not confirm Heidenhain's statement, *i. e.*, that "wherever we have central cells we have pepsin." Yet, it seems true, that the degree of acidity is in proportion to the number of border cells present in any portion of the stomach, and that there are portions of the stomach which do not contain border cells, but yield pepsin. In general, the formation of pepsin is most marked in those portions of the stomach which produce most acid; and this ought to be the case, for acid favors the formation of pepsin from pepsinogen (Podwyssozki,

Langley, and Edkins), and the pepsin seems more or less combined with acid (Schiff, Richet). We must, therefore, conclude with Heidenhain, that the border cells play a most important part in the formation of acid. Between the glands lie the blood-vessels, lymphatics, some round cells, and the reticulum. In those portions of the stomach in which there is a 'neck zone,' there is a distinct layer of reticulum fibrils. In this layer peculiar spindle cells are frequently seen which surround the gland openings and appear much like the subepithelial cells in the villi of the intestine. Under no condition could a basement membrane be isolated, nor does Mall believe it exists, but instead there is a most beautiful network of the reticulum.

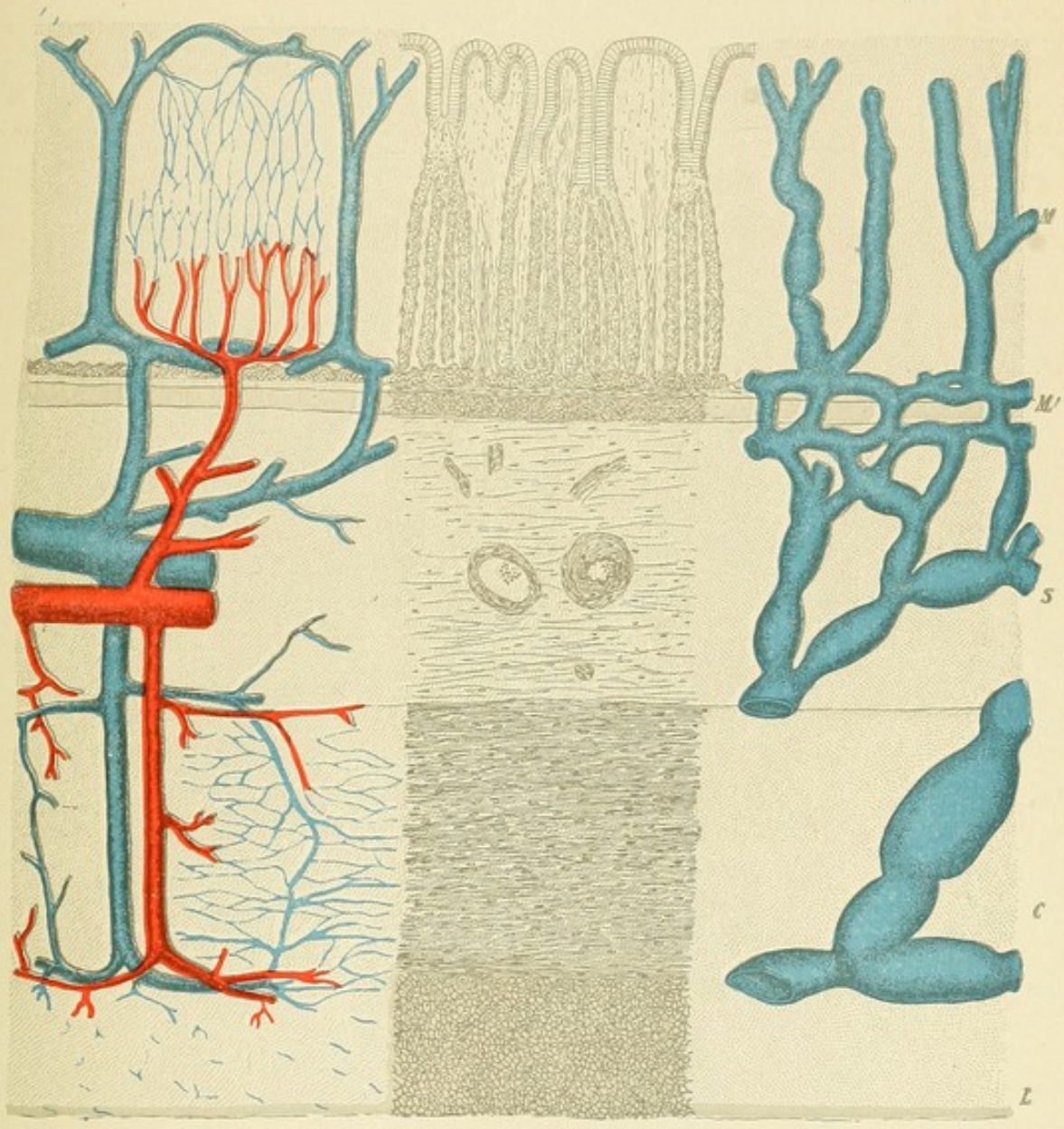
"CONCLUSIONS.—From a histological standpoint the mucous membrane of the stomach may be divided into three zones—the pyloric, with no border cells; the middle, with many border cells; and the fundus, with but few border cells.

"Digestion of the different portions of the mucous membrane with weak HCl shows that the middle zone digests most easily, the fundus less quickly, and the pyloric, as a rule, not at all. Assuming that the rapidity of digestion of the different portions is in proportion to the quantity of pepsin present, it makes it probable that most pepsin is formed in the middle zone. Although it has been proved that pepsin is formed in glands which do not contain border cells, in general it may be stated that the amount of pepsin formed by the different glands is in proportion to the number of border cells.

"The degree of acidity of the mucous membrane is in proportion to the number of border cells present. It is reasonable to suppose that the formation of acid in any portion of the stomach aids materially in the formation of pepsin in the same part. This is very essential, because acid favors the formation of pepsin from pepsinogen. Since border cells are only with the greatest difficulty digested in acid, we can not ascribe to them the power to secrete pepsin; and since the morphology of the central cells varies during digestion and rest, and they are also easily digested upon the addition of acid, we must conclude with Heidenhain that the former are probably concerned in the production of acid and the latter in the production of pepsin.

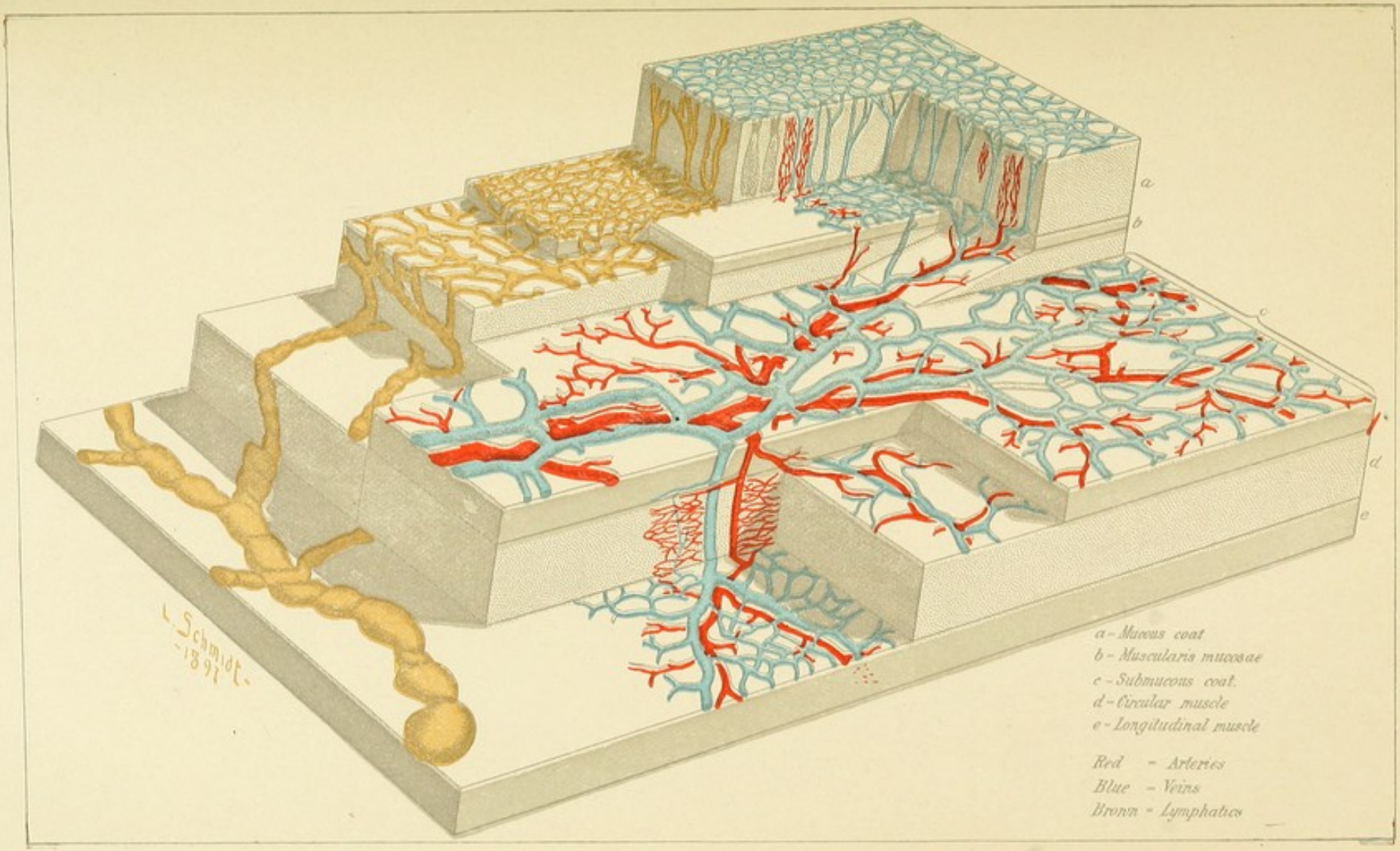
"When the stomach is forcibly distended it is found that the dilatation is mostly at the expense of the fundus. This seems also to

PLATE I.



THREE SECTIONS OF STOMACH-WALLS PLACED SIDE BY SIDE TO SHOW THE POSITIONS OF BLOOD-VESSELS AND LYMPHATICS TO THE DIFFERENT LAYERS.

M, Mucosa. *M'*, Muscularis mucosæ. *S*, Submucosa. *C* and *L*, Circular and longitudinal muscles enlarged 70 times.



RECONSTRUCTION OF A SMALL PORTION OF THE MIDDLE ZONE OF THE STOMACH.

Illustrating the distribution of the arteries, veins, and lymphatics to the various gastric layers. The arteries are in red, the veins in blue, the lymphatics in brown. The long diameter of the drawing is in the direction of the longitudinal muscle-fibers. It was built up from 36 drawings, and each drawing is an exact representation of a specimen. Enlarged 20 times.

be the case when the stomach is naturally filled with food. Although the middle zone is practically not stretched when the stomach is filled, distention seems to favor circulation through this part because the blood-vessels are more easily injected in a moderately distended, than in an empty, stomach.

"In the intestine it is found that the longitudinal and circular muscle-fibers are antagonistic. In the stomach the pyloric valve is closed, after the muscle-cells are dead, by a fold of mucous membrane being thrown into the lumen. This may take place in a living stomach. A contraction of the circular muscle tends to strengthen this valve, while a contraction of the longitudinal muscle tends to weaken it, because with the contraction of the longitudinal muscle there is always an accompanying relaxation of the circular muscle. Under ordinary circumstances it seems as though the stomach reduced its lumen by simultaneous contraction of both longitudinal and circular muscle-fibers. What complex motions take place during peristalsis are absolutely unknown. It is, however, a remarkable fact, that a bundle of the circular fibers (oblique fibers) are parallel with the longitudinal fibers, which are increased in number in the middle zone. A solution of this problem seems within the range of experimentation.

"The celiac axis supplies, besides the stomach, also the spleen and the liver. With a given pressure within the aorta, variation in the resistance in the capillaries of the spleen and the liver will have a marked effect upon the circulation through the stomach. The portion of the stomach (middle zone) supplied by the gastric artery is to a less extent under the control of these side influences than is that which is supplied by arteries arising from the main branches to the spleen and to the liver. It must be again stated, that there are, in all probability, many other influences which play most important parts in the distribution of blood.

"7. Around the two curvatures of the stomach there is a complete circle of anastomosis, which has a tendency to equalize the pressure in the arteries penetrating the muscle-walls. But the anastomoses arising therefrom have only a tendency to make gradual gradations, and not an equal pressure throughout. The additional set of anastomoses within the submucosa are, again, not sufficient to equalize the flow throughout the whole mucosa. After ligating arteries, as well as by examining the mucous mem-

brane during digestion and rest, it is found that no sharp lines can be drawn.

"8. The blood-vessels are arranged in such a manner that from any portion of the submucosa about one-fourth of the blood may go to the muscle-coats and three-fourths to the mucosa. It is therefore probable that when the flow is poured to one side it is diminished to the other, and vice versâ. There is, however, a tendency to equalize this by the submucous anastomoses.

"9. Since there is but one set of arteries to the mucosa, there must be but one sort of circulation, which may vary in degree only. Within the mucosa the arrangement is such that the portion of the gland which is deepest receives the blood richest in O. The mucous membrane, omitting the muscularis mucosæ, lies between two venous plexuses. Contraction of the muscle-fibers between the glands and those of the muscularis mucosæ should diminish the volume of the mucosa. This would have a tendency to empty the glands, as well as to press blood from the two venous plexuses, especially the lower. Whether or not there is a force within the mucosa which can augment the circulation, seems at present impossible to determine by experiment. The arrangement of the parts is very suggestive.

"10. The rich venous plexus of veins within the submucosa is sufficiently large to hold a considerable quantity of blood. This must be the case when the valves within the veins coming from the stomach are temporarily closed. When the valves are closed a contraction of the circular muscle is sufficient to drive all the blood from the underlying veins. It is therefore possible that a rhythmic contraction in any part of the stomach may favor the circulation through its walls.

"11. The arrangement of the lymphatics is much the same as that of the veins, and the above consideration (10) applies equally well to them. When we consider the resistance to be overcome while the lymph passes through so many networks before the cisterna chyli is reached, it makes it plausible to state that the circulation is favored by muscular contraction.

"12. Since the blood which leaves the stomach must pass through the capillaries of the liver, it is necessary that it be constantly under a comparatively high pressure. This pressure is also dependent upon the spleen and the intestine. If the pressure is

high, a regurgitation into the stomach is impossible on account of the presence of valves.

"13. In a stomach in which the vessels are all equally distended the rapidity of circulation in the celiac axis would be 263 times that in the capillaries. The area of the section of the celiac axis is .0592 square cm.; the immediate branches to the stomach, .0348 square cm.; to the spleen and liver, .0244 square cm. All the capillaries of the stomach: mucosa, 6.4524 square cm.; muscle-coats, 2.7214 square cm.; total, 9.1738 square cm. $9.1738 \div .0348 = 263$.

"A like estimation shows that the rapidity of circulation in all the capillaries is $\frac{1}{63}$ of that in the arteries penetrating the muscle-walls; while if the capillaries of the muscle-walls are excluded, the rapidity in the capillaries of the mucosa rises to $\frac{1}{44}$.

"Considering the glands on an average .05 cm. long and .003⅓ cm. in diameter, excluding the necks, the area of all the glands would be 8671 square cm., or 38 times the area of mucous membrane. A like estimation of the capillaries, considering each capillary .04 cm. long, gives for them a total area of 1718 square cm., or 7½ times the mucous surface. The secreting surface is five times that of the blood-supply."

Vessels and Nerves.—The arteries supplying the stomach are: the coronaria ventriculi; the pyloric and right gastro-epiploic branches of the hepatic; the left gastro-epiploic and vasa brevia from the splenic. They supply the muscular coat, ramify in the submucous coat, and are finally distributed to the mucous membrane. The arrangement of the vessels in the mucous membrane is somewhat peculiar. The arteries break up at the base of the gastric tubules into a plexus of fine capillaries, which run upward between the tubules, anastomosing with each other and ending in a plexus of large capillaries, which surround the mouths of the tubes, and also form hexagonal meshes around the alveoli. See Plate II. The veins arise from the latter, and pursue a straight course back to the submucous tissue, between the tubules, to terminate in the splenic and portal veins.

The lymphatics are abundant, and may be divided into a superficial and a deep set, which pass through the lymphatic glands found along the two curvatures. The nerves are supplied from the right and left pneumogastric, and numerous branches from the abdominal sympathetics. (Solar plexus.)

CHAPTER III.

THE SMALL INTESTINE.

The small intestine commences at the pylorus, and, after many convolutions, terminates in the large intestine. It measures, on an average, about 22 feet in length in an adult, and becomes gradually narrower from its upper to its lower end. Its convolutions occupy the middle and lower parts of the abdomen, frequently descending into the pelvis.

The small intestine is divided into three portions, which have received different names. The first 10 to 12 inches immediately succeeding the stomach, and comprising the widest and most fixed part of the tube, are called the duodenum. This part is further distinguished by its close relation to the head of the pancreas, and by the absence of a mesentery. The remainder, which is arbitrarily divided into an upper two-fifths, called the jejunum, and a lower three-fifths, called the ileum, is very convoluted and movable, being connected with the posterior abdominal wall by a long and extensive fold of peritoneum called the mesentery, and by numerous blood-vessels and nerves. Although there is no distinct line of demarcation between the jejunum and the ileum, yet that portion of the small intestine included under these two names gradually undergoes certain changes in structure and appearance from above downward, so that the upper end of the jejunum can readily be distinguished from the lower end of the ileum.

Structure of the Small Intestine.—The small intestine, like the stomach, is composed of four coats, viz.: the serous or peritoneal, the muscular, the areolar, and the mucous.

The external, or serous, coat almost entirely surrounds the intestinal tube in the whole extent of jejunum and ileum, leaving only a narrow interval behind, where it passes off and becomes continuous with the two layers of the mesentery. The line at which this takes place is named the attached or mesenteric border of the intestine. The duodenum, on the other hand, is but partially covered by the peritoneum. The muscular coat consists of two layers of fibers—an

outer longitudinal, and an inner, or circular, set. The longitudinal fibers constitute an entire but comparatively thin layer and are most obvious along the free border of the intestine. The circular layer is thicker and more distinct.

The muscular tunic becomes gradually thinner toward the lower part of the small intestine. It is pale in color, and is composed of plain muscular tissue, the cells of which are of considerable length.

The progressive contraction of these fibers, commencing at any part of the intestine, and advancing in a downward direction, produces the peculiar vermicular, or peristaltic, movement by which the contents are forced onward through the canal. In the narrowing of the tube the circular fibers are mainly concerned, the longitudinal fibers tending to produce dilatation (Exner); and those found along the free border of the intestine may have the effect of straightening or unfolding its successive convolutions. There is a gangliated plexus of nerve-fibers and a network of lymphatic vessels between the two muscular layers.

The submucous coat of the small intestine is a layer of areolar tissue of a loose texture, which is connected more firmly with the mucous than with the muscular coat. Within it the blood-vessels ramify before passing to the mucous membrane, and it contains a gangliated plexus of nerve-fibers and a network of large lymphatic vessels.

The internal coat, or mucous membrane, is characterized by the finely flocculent, or shaggy, appearance of its inner surface, resembling the pile upon velvet. This appearance is due to the surface being thickly covered with minute processes, named villi. It is one of the most vascular membranes in the body, and is naturally of a reddish color in the upper part of the small intestine, but is paler, and at the same time thinner, toward the lower end. It is lined with columnar epithelium throughout its whole extent, and, next to the submucous coat, is bounded by a layer of plain muscular tissue (muscularis mucosa); between this and the epithelium the substance of the membrane, apart from the tubular glands, which will be afterward described, consists mainly of retiform tissue, which supports the blood-vessel, nerves, lymphatics, and lacteals, and incloses in its meshes numerous lymph-corpuscles.

Valvulæ Conniventes.—The mucous membrane, in addition to small effaceable folds, or rugæ, possesses also permanent folds, which can not be obliterated, even when the tube is forcibly dis

tended. These permanent folds are the valvulæ conniventes, or valves of Kerkring. They are crescentic projections of the mucous membrane, placed transversely to the axis of the bowel, and following one another closely. The majority of the folds do not extend more than one-half or two-thirds around the interior of the tube, but it has been shown by Brooks and Kazzander that some form complete circles, and others spirals. The spiral forms may occur singly or in groups of two or three. They generally extend a little more than once around the lumen of the bowel, but in rare cases may go around two or three times. At their highest point they project inward for about ⅓ of an inch. Some of the valvulæ conniventes are bifurcated at one or both ends, and others terminate abruptly. Each consists of a fold of mucous membrane, that is, of two layers placed back to back, and united together by submucous areolar tissue. They contain no part of the circular or longitudinal muscular coats. Being extensions of the mucous membrane, they serve to increase the absorbent surface to which the food is exposed.

The valvulæ conniventes are not uniformly distributed over the various parts of the small intestine. There are none just at the commencement of the duodenum; a short distance from the pylorus they begin to appear; beyond the point at which the bile and pancreatic juice are poured into the duodenum, they are very large, regularly crescentic in form, and placed so near to each other that the intervals between them are not greater than the breadth of the valves; they continue thus through the rest of the duodenum, and along the upper half of the jejunum; below that point they begin to get smaller and farther apart, and, finally, toward the middle or lower end of the ileum, having gradually become more irregular and indistinct, sometimes even acquiring a very oblique direction, they disappear altogether.

The **villi**, peculiar to the small intestine, and giving to its internal surface the velvety appearance already spoken of, are small processes of the mucous membrane, which are closely set on every part of the inner surface over the valvulæ conniventes, as well as between them. Their length varies from 0.5 mm. to 0.7 mm., or sometimes more.

They are largest and most numerous in the duodenum and jejunum, and become gradually smaller, and fewer in number, in the ileum. According to Rauber, they are short and leaf-shaped in the

duodenum, and as the gut is followed downward they become gradually longer and thinner, so that they are tongue-shaped in the jejunum and filiform in the ileum. Occasionally two or three are connected together at their bases. In the upper part of the small intestine there are from 10 to 18 villi in a square millimeter, and in the ileum from 8 to 14 in the same space. This would give about 4,000,000 altogether (Krause).

A villus consists of a prolongation of the proper mucous membrane. It is covered by columnar epithelium, and incloses a network of blood-vessels, one or more lymphatic vessels (lacteals), and a few longitudinal, plain, muscular fiber-cells, these being all supported and held together by retiform lymphoid tissue.

Under the epithelium is a basement membrane composed of flattened cells, which, on the one hand, are connected with the branched cells of the retiform tissue, and, on the other hand, send processes between the epithelial cells. Nervous fibrils penetrate into the villi from the plexus of Meissner, and form arborizations throughout their whole substance.

Each villus receives, as a rule, one small arterial twig, which runs from the submucous coat, through the muscularis mucosæ, to the base of the villus, and then up the center to near the middle line of the villus, where it begins to break up into a number of capillaries.

These form, near the surface, a fine capillary network beneath the epithelium and limiting membrane, from which the blood is returned, for the most part, by one or two venules, which, in man, commence near the tip of the villus, and pass down to its base to join the venous plexus of the mucous membrane, whence the blood is conveyed to the large veins of the submucosa.

The **Lacteal** lies in the center of the villus, and, in the smaller villi, is usually a single vessel with a closed and somewhat expanded extremity, and of considerably larger diameter than the capillaries of the blood-vessels around. In the human subject there are never more than two intercommunicating lacteals in a single villus.

The lacteals in the villi are bounded by a delicate layer of flattened epithelial cells; these are connected with the branched cells of the tissue of the villus, and these again with the flattened cells forming the basement membrane; from the latter, prolongations extend between the epithelial cells toward the surface. Brücke first discovered the muscular tissue within the villus, consisting of unstriated, plain fiber-cells, disposed longitudinally

around the lacteal. These fibers are prolongations of the muscularis mucosæ.

When they are stimulated in animals a very evident retraction of the villus is observable.

The fiber-cells at the sides and toward the end of the villus pass from the lacteal to be attached to the basement membrane in a bifurcating manner.

Columnar epithelial cells cover not only the villi, but also the rest of the surface of the small intestine, and extend into the tubular glands. There is never any continuity between the extremity that is attached to the basement membrane and the branched corpuscles of the retiform tissue of the villus. This epithelium separates easily from the subjacent tissue. Between the cells composing it are a variable number of leucocytes, most numerous in the lower part of the intestines near the lymphoid follicles. Occasionally they are seen to be free in small lymph-spaces between the columnar epithelial cells and showing indications of karyokinesis. Hardy declares that immediately below the columnar epithelium of the villi there is frequently a well-marked layer of cells that take up the eosin stain readily. Hence he calls them eosinophilic.

Among the ordinary epithelial cells of the villus are others, the outer half of which is filled with mucigen, and at times beaker- or cup-shaped empty cells are observed from which this has been discharged as mucus, the free end being ruptured; these are sometimes called the goblet-cells. The number of cells containing mucus varies much in different animals and under different conditions in the same animal. There are comparatively few mucous cells in the glands of the small intestine.

The epithelial cells are, as far as can be ascertained, the principal agents in promoting the absorption of food materials from the interior of the gut, and the seat of the retrograde processes of metabolism which the products of digestion undergo during absorption. Peptone, when injected into the blood of an animal by whose gastric juice it has been formed, acts as a poison. It is due to these epithelial cells of the intestine that peptone is so modified during absorption that it becomes of use to the organism.

Most food particles can not be traced in microscopic specimens, but fatty or oily substances, from their property of becoming stained with osmic acid, can be, to some extent, followed out. The examination of such specimens, taken during digestion of a meal

containing fat, shows the epithelial cells turbid with oil droplets in their interior; and in some animals, at a subsequent stage, ameboid cells appear within the tissue of the villus pervaded with similar but finer fatty particles, and eventually the central lacteal becomes filled with these. It is probable that these ameboid lymph-corpuscles, appearing so abundantly within the villus and among the epithelial cells on its surface, play an important part in the transference of such particles from the epithelial cells in the lacteal; for at certain stages of fat absorption they contain abundant fatty particles. The large amount of lymphoid tissue in the lower part of the small intestine seems to be related to a greater power of absorption in that part.

In the transference of carbon particles in the lungs, from the interior of the alveoli into the lymphatics, which at least in part is due to the action of ameboid cells, we have an analogous process.

Glands.—Two kinds of true secreting glands are found in the intestine; these are: (1) the glands or crypts of Lieberkühn and (2) glands of Brunner. In addition to these, there are found also two varieties of intestinal lymph-follicles, (1) the solitary and (2) the agminate glands, the latter often designated as Peyer's patches.

Although the solitary and agminated lymph-follicles have no ducts opening upon the inner intestinal surfaces, like Brunner's and Lieberkühn's glands, they are nevertheless spoken of as glands.

The follicles, crypts, or glands of Lieberkühn are tubular pits lined by columnar epithelium, occurring between the villi and on the valvulæ conniventes. Here and there in these crypts, goblet-cells occur in the epithelium. They are present throughout the large and small intestine, and extend through the entire depth of the mucosa, their ends approaching the muscularis mucosæ.

The duodenum possesses an additional layer of true secreting structures in the glands of Brunner. They would appear to represent the direct continuations and higher specializations of the pyloric glands. In passing from the stomach into the intestines, these tubules undergo repeated division, at the same time sinking deeper into the mucosa, finally reaching below this layer to take up a position within the submucosa of the duodenum, underneath the overlying layer of the crypts of Lieberkühn, which are contained in the mucosa proper. Brunner's glands belong to the racemose type, and under the microscope they consist of a number of tubular alveoli connected by terminal ramifications of the duct

which penetrates the muscularis mucosæ, and opens either between the mouths of the Lieberkühn crypts or sometimes into their bases.

The solitary glands are isolated lymph-follicles scattered through the entire intestine, most abundant in the lower ileum. Situated in the mucosa, at times in the submucosa, the lymphoid tissue in them is denser toward the periphery, but is everywhere so closely packed that the supporting reticulum of connective tissue is masked.

The agminated glands, or Peyer's patches, are large oval aggregations of lymph-follicles held together by diffuse adenoid tissue, limited to the lower two-thirds of the small intestine. Development of these is most perfect in the ileum; appearing first within the mucosa, they later encroach upon the submucous tissue.

Where the summits of these follicles impinge against the inner layer of the mucosa, the position of the agminated glands is indicated by an elevation corresponding to them on the mucous surface. In that case the villi are frequently pushed aside.

The Blood-vessels of the Intestines.—The vessels follow the general arrangement of those in the stomach, the larger ones piercing the serous and muscular coat, giving off slender twigs to supply these tunics, and when they enter the submucosa, the vessels form a wide-meshed network. Many branches then pass through the muscularis mucosæ, to be distributed to the deeper, as well as the superficial, part of the mucosa. Around the tubular glands a network is formed by narrow capillaries, and just beneath the epithelium the capillaries become wider and encircle the mouths of the follicles. From this superficial capillary network the veins arise, and, passing down between the follicles, join the deeper venous plexus, this in turn communicating with the larger veins of the submucosa.

The villi have special additional arteries running to their bases, expanding into capillaries, and then extending beneath the epithelium and around the central lacteal as far as the ends of the villi. These capillaries terminate in venous stems which descend almost perpendicularly into the mucosa, in their course receiving the superficial capillaries encircling the gland ducts. Brunner's glands, and the solitary and agminated follicles, are supplied from the submucosa by vessels terminating in capillary networks distributed to the acini of the glands and interior of the lymph-follicles.

The blood-vessels of the intestines, taken as a whole, constitute a mighty vascular territory which is capable of taking up one-third of the total amount of blood of the body.

The arteries are all branches of the superior and inferior mesenteric arteries, which run along and approach the gut in the mesentery. The intestinal veins form the principal portion of the portal system.

Lymph-vessels.—The beginning of the lymph-vessels can be traced to the lacteals within the villi, where they begin as tiny, little blind pouches at the apex of the villus. In some broad villi there are two or three such lymph-vessels that anastomose with each other. From here they run down in the septa between the glands in the lymph-vessel meshwork over the muscularis mucosæ. Here they again anastomose with an outer lymph-vessel network in the submucosa. Here the lymphatics begin to be provided with valves.

The nerves of the intestine, like those of the stomach, originate chiefly from the mesenteric plexus, which is formed by branches from the celiac plexus, the semilunar ganglion, and vagus nerve; consisting of medullated and non-medullated fibers, that begin to form an abundant network under the peritoneum of the intestine, then penetrate the longitudinal muscular stratum, and between this and the circular layer form a peculiar plexus with numerous microscopic ganglia, constituting the plexus of Auerbach.

In the submucosa a similar network of fibers and ganglia has been termed Meissner's plexus. From Meissner's plexus very fine fibers are spun about the Lieberkühn crypts, villi, and limiting membrane.

Relations of the Duodenum.—This part of the gut in the adult is horseshoe-shaped, generally presenting well-marked angles, which divide it into four parts having four distinct directions; these are: (1) The horizontal or superior part, running backward from the pylorus, to the right, in contact with the quadrate lobe of the liver, to the under side of the neck of the gall-bladder, where it curves sharply downward to join the second part. This first or horizontal part is about two inches long when the stomach is empty. (2) The second or descending portion is about three inches long, and commences just below the neck of the gall-bladder opposite the right side of the first lumbar vertebra, and passes down to the level of the third or fourth lumbar vertebra, where it turns

sharply inward to join the third part. (3) The third or transverse portion is from two to three inches long; beginning at the right of the third or fourth lumbar vertebra, it crosses over to the left side with a slight upward inclination, and ends to the left of the aorta by curving upward to join the terminal, (4) fourth, or ascending, portion, which is about two inches long; it passes upward to the left side of the aorta, as high as the upper border of the second lumbar vertebra; here it turns abruptly forward to join the jejunum, forming the duodenojejunal flexure.

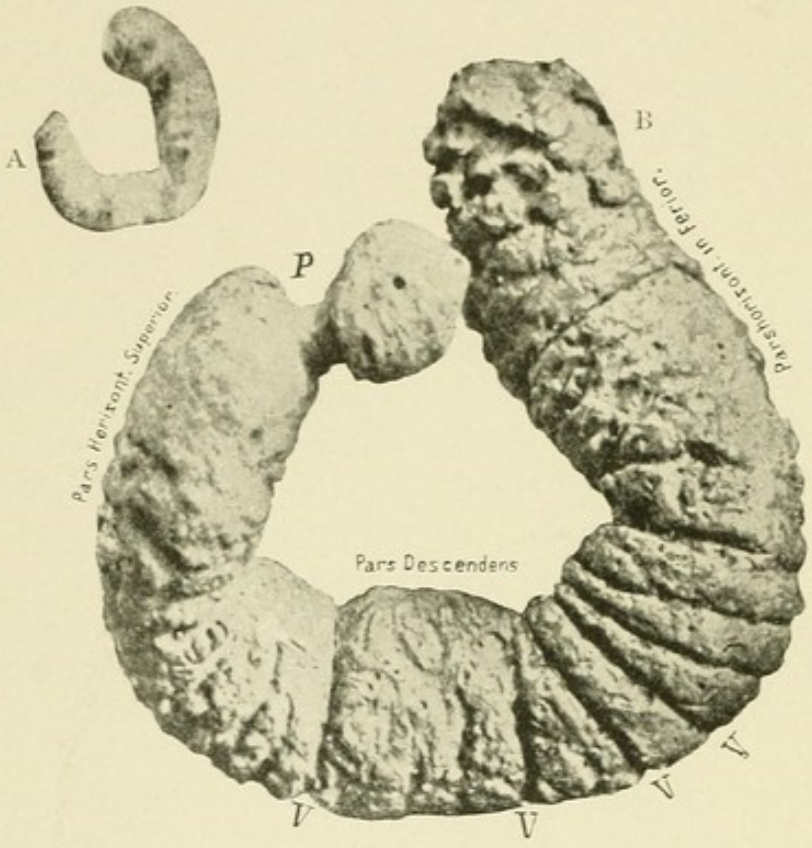


FIG. 4.—PLASTER CAST OF DUODENUM OF INFANT AND ADULT.—(From Museum of *Harvard University*).

A. Infant duodenum. B. Adult. V. Valvulæ conniventes. P. Pylorus.

Thus the end of the duodenum is brought to the same level as the beginning. It has been compared to a water-trap, its ends being always higher than its middle, which is thus fitted to retain the fluid poured into it from the liver, pancreas, and its own glands, besides that which it receives from the stomach, at the same time preventing the regurgitation of gases from the jejunum into the pyloric part of the duodenum and stomach.

Jejunum and Ileum.—The upper two-fifths of the remaining intestine immediately following the duodenum are called the jejunum, the lower three-fifths, the ileum. Both are attached to the posterior abdominal wall by an extensive fold of peritoneum, —the mesentery.

The jejunum lies above and to the left of the ileum, but the coils are so irregular that the position of any individual loop affords but little clue to the part of the intestine to which it belongs.

The large intestine consists of the cecum, the colon, and the rectum. The colon is subdivided, according to the directions it takes, into four parts, which are (1) the ascending, (2) transverse, (3) descending, and (4) sigmoid colon or flexure.

The end of the ileum, which rises out of the pelvis to the right iliac fossa, is not inserted into the beginning of the large intestine, but above the beginning and at the side of it. The part of the large intestine below this insertion is a blind pouch,—the cecum. From the inner and back part of the cecum, a little below the ileo-colic opening, a narrow, round, worm-like process, about two or three inches long, is given off,—the vermiform appendix.

The cecum continues upward into the ascending colon, which rises up in front of the right kidney to the edge of the liver; then this same large intestine passes beneath the greater curvature of the stomach, and horizontally across to the left side, as the transverse colon; here, at the lower border of the spleen, it turns downward as the descending colon.

This large gut describes two right-angled curves, the right and left colonic flexures fixed by the hepatocolic and gastrocolic ligaments respectively. The descending colon continues into the sigmoid colon or flexure, which connects it with the rectum. The rectum, following the curves of the sacro-iliac symphysis and the hollow of the sacrum, has itself two curves: an upper larger curve, concave anteriorly, and a lower smaller curve, convex anteriorly.

Only the cecum, transverse colon, and sigmoid colon have a complete peritoneal covering; the rest of the large gut is only covered anteriorly. From the third sacral vertebra on, the rectum has no peritoneum. Those parts having no complete peritoneum, therefore, have no mesentery, and are not very movable. The longitudinal fibers are contracted, or narrowed, down to three parallel bands (Fasciæ teniæ, or lig. coli). One of these bands runs along the attachment of the gastrocolic ligament on the transverse colon

(fascia omentalis), the second along the mesenteric border, and the third is free.

Running down into the rectum these bands become so broad that they occupy the entire periphery of the tube. These longitudinal bands are much shorter than the other layers of the intestinal wall, which arrangement results in the characteristic sacculation of the large intestine. In the lower part of the rectum the circular muscular layer becomes thickened to form the internal anal sphincter of involuntary fibers.

The external sphincter is composed of striated voluntary muscle-fibers. The histology of the large intestine differs from that of the small by the absence of the villi and the larger size of the crypts and follicles. Several longitudinal elevations over the anus are called the columns of Morgagni; from this point downward the cylindrical epithelium ceases and flat pavement epithelium takes its place.

CHAPTER IV.

PHYSIOLOGY OF DIGESTION.

Food Substances.—The simple chemical elements of the various food substances, namely, C, H, N, S, and P, are not assimilable as such, because the human body is not capable of constructing higher compounds from them synthetically. It is compelled to take in these compounds in the form of proteid or albuminous substances, carbohydrates, or starches and fats, together with such inorganic bodies as water and salts.

Even these food-stuffs, which are essential for the maintenance and development of the organism, are not ingested as such, but are contained, together with innutritious materials, in the various articles of diet which we derive from the animal and vegetable kingdom.

The innutritious admixtures of the food-substances are not harmful, but are important as stimulants to the intestinal mucosa and to the evacuation of feces. Among these innutritious substances are classed the connective tissue, the cartilages and tendons of meat, and the cellulose of plants.

Water plays a most important rôle in the economy of the body, for it goes to make up 60 per cent. of the total organism. We lose about 2½ liters of water in twenty-four hours, through insensible perspiration, secretion, and defecation. About 300–400 gms. of water are formed by oxidation of food-substances in twenty-four hours; so we have a deficit of 1500–1600 gms., which must be supplied by the daily consumption of a corresponding amount of water; this is done principally by the drinking of water after we have taken in part of it by our foods or in the shape of beverages (soups, milk, fruits, vegetables, potatoes, beer, wine, coffee, tea, etc.).

In mineral substances we must supply the daily loss of sodium chlorid and other salts, particularly compounds of iron; these are normally introduced in sufficient quantities in food and drink.

The chief constituents of food—albuminous bodies, fats, and carbohydrates—are of organic nature. The proteids, or albuminous

bodies, and the fats, are derived partly from the animal and partly from the vegetable kingdom. The carbohydrates are almost exclusively derived from the vegetable kingdom. The former serve for the building up of the organism, and the continuance of life processes. The latter are producers of heat by their oxidation, which finally reaches H_2CO_3 and H_2O, and are the prevailing sources of work.

In addition to these a number of other substances occur in the food that are oxidized, and might serve as sources of energy; these are the nitrogen-free vegetable acids, the amido-acids, and alcohol, for instance; quantitatively, however, they are not important.

Other organic bodies that are contained in food materials as normal constituents, such as creatin in meat, glucosides, alkaloids, and ethereal oils in vegetables and spices, pass through the body without being oxidized or assimilated; they are not food-substances, as they do not enter the metabolism of the body, nor do they develop energy by chemical transformation. However, a number of these are of importance in nutrition, as they render the food more palatable, and stimulate the secretions and the motility of the digestive tract.

It has been said that the elements S, P, Cl, K, Na, Ca, Fe, Mg, are not food-materials, but it must not be understood that they are entirely useless. They have some significance in the construction of tissue, although the organism can derive no energy from them, as they are always taken in a highly oxidized state, and leave in the same condition. Nevertheless, the body will come to grief, if any one of these elements be excluded from the food.

A certain minimum of these elements—the amount has not yet been ascertained—is absolutely necessary. Outside of the substances named above, the food contains a number of materials that are not at all absorbable or digestible, and leave the digestive tract in an unchanged form; this is the slag and dross of the food, and is taken into the body principally with vegetables.

The normal adult human organism daily loses by its metabolism 120 gm. of albuminous or proteid bodies, 80 gm. fat, 400 gm. carbohydrates, 25 gm. salts, and 2½ liters of water. Accordingly, a corresponding amount of food-stuffs must be introduced in the diet. The articles of food contain these nutritious substances in a variety of proportions. The rational combination of these substances is one of the objects of dietetics. Gilman Thompson, in his new book on "Dietetics," divides foods into six groups, as follows:

I. Water, II. Salts, III. Proteids (chiefly albuminous and allied gelatines), IV. Starches, V. Sugar, VI. Fats and oils.

It still remains extremely difficult, in the case of all foods, to trace their final uses in the body, and determine with any accuracy what proportions each furnish, respectively, of energy, repair of tissue, and heat; for there are no more complex chemical processes known than those of metabolism (Gilman Thompson). Foods have three kinds of values: (1) nutrient, (2) heat-producing, (3) force-producing.

Caloric Values.—The calculation of these different values for each kind of food has been much simplified by the introduction of the conception of calories into the doctrines of nutrition. Formerly, observers and investigators said: "A healthy man needs so many gm. proteid, so many gm. carbohydrates, so many gm. fat," etc. It was inconvenient to reckon with three magnitudes and to bring them into correct relation with the requisites of the individual organism.

Nowadays we compute the values of food-stuffs according to the physiological (kinetic) energy liberated in their oxidation. Germans call this "degree of energy," which is always expressed in terms of heat, the *Brennwerth*, *i. e.*, the value of food when it is burned in the process of metabolism, for this is nothing but a slow combustion. Now the unit for measurement of this heat energy of food is called a calorie. This capacity for heat production of foods is determined from the amount of heat which is liberated when any particular food-substance is transformed from its original composition, when it entered the body—by oxidation—into those chemical combinations in which it leaves the organism. The unit for measurement is the calorie, which signifies the amount of heat which is necessary to raise one kilogram of water 1° C.

Now,	1 gm. of albumin furnishes	4.1	calories.
	1 " carbohydrate furnishes	4.1	"
	1 " fat "	9.3	"
	1 " alcohol "	7	"

Instead of saying a man requires 100 gm. albumen, 100 gm. fat, and 400 gm. carbohydrates, one now expresses this in calories, thus: A man requires

100 gm. albumen	×	4.1, ...	410	calories.
100 gm. fats	×	9.3, ...	930	"
400 gm. carbohydrates	×	4.1, ...	1640	"
		Total, ...	2980	"

For every kilogram of body weight, an adult requires, when at rest, a food-supply of 30 to 34 calories; during light occupation, a food-supply of 34 to 40 calories; during medium occupation, a food-supply of 40 to 45 calories; during hard work, a food-supply of 45 to 60 calories.

In very adipose and obese persons the requirements for food are less than the quantities stated by one-quarter to one-third. If the above calculations of the requisite number of calories per kilogram weight of any person are correct, and the supply maintained accordingly, the individual will maintain his weight. If the supply of calories is greater, he will gain weight; if the supply is less, he will lose weight.

In a condensed statement of facts like the present, it will be expedient to pass over the physiology of hunger, appetite, and thirst, which will be considered in the clinical part of this work (bulimia, anorexia, etc.), and proceed at once to digestive actions.

Ptyalin Digestion.—Digestion really begins in the mouth, where the food is chewed into small bits and mixed with saliva, which mechanically facilitates mastication and deglutition. Chemical transformation also begins here, for the diastasic ferment of saliva, ptyalin, transforms a small portion of the starchy foods into sugar and maltose.

Ptyalin can produce this transformation of starchy foods only in an alkaline medium, accordingly the action ceases in the stomach; but not immediately, however, as the conversion of starches into sugar goes on until the degree of acidity reaches 1 : 1000. As the ptyalin ferment becomes inactive in this acidity, the question arises, whether its activity is permanently destroyed by an acidity of 1 : 1000, or only temporarily, and whether it can resume its inverting power when the acid is neutralized. Boas, who attempted a solution of this, came to the conclusion that subsequent alkalinization, or diminution of the acid, causes the ptyalin to act again, so that in later stages of stomach digestion, when the acid production ceases, the conversion of starch into grape sugar may be resumed, but it never becomes as active as before.

The existence of appetite is to a degree dependent upon the intactness of the salivary glands.

Digestion of Starches.—In order to understand the various stages of starch conversion, it is essential to study the digestion of starch by ptyalin in the clinical laboratory. There are recog-

nized four stages of starch conversion, each distinct from the other, until dextrose is reached.

1. (*a*) This is common starch, representing a glue-like, mucilaginous jelly, not a clear solution, giving a dark-blue color with iodin and iodid of potassium solution. The next stage shows the first action of ptyalin.

(*b*) *Amidulin or Amylodextrin.*—This still gives a distinctly blue color, though not so deep as No. 1 (*a*), with Lugol's solution; but amylodextrin is a soluble starch, and represents a real solution.

2. (*a*) *Erythrodextrin.*—Gradually, as the inversion progresses, the color produced by the iodin solution becomes violet-blue, violet, red violet, red, or mahogany brown; this modification is called erythrodextrin.

(*b*) *Achroodextrin.*—With continued action of the ptyalin, a substance is reached which gives no color with iodin; this is called achroodextrin. Amidulin is precipitated by tannic acid and alcohol, but erythrodextrin and achroodextrin are precipitated by alcohol and ether, not by tannic acid. These two dextrins do not reduce Fehling's solution, and do not ferment with yeast.

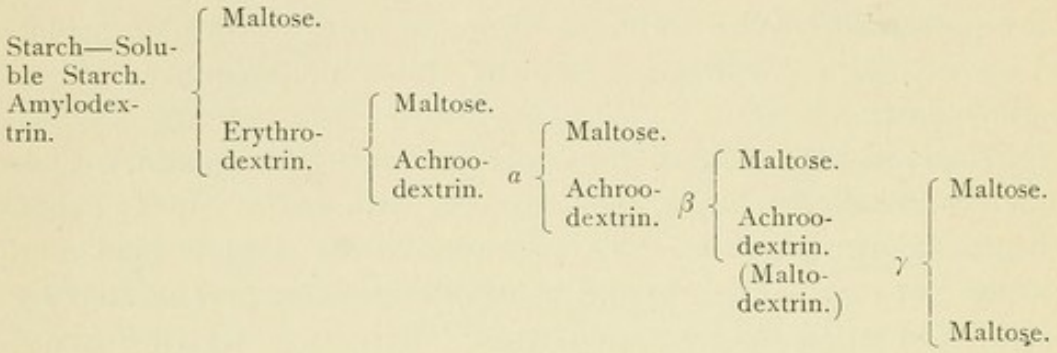
3. *Maltose.*—Soluble in alcohol, insoluble in ether; reduces Fehling's solution, but not Barfoed's reagent (a four per cent. solution of cupric acetate to which one per cent. acetic acid is added); does not ferment with yeast.

4. *Dextrose.*—Insoluble in alcohol and ether; reduces Fehling's as well as Barfoed's solution; ferments readily with yeast.

It is important to familiarize oneself with these reactions, as it often becomes necessary to determine the degree of starch conversion in cases of hyperacidity or supersecretion.

It was formerly thought that the starch was first converted to dextrin, and this in turn was converted to sugar. According to Prof. W. H. Howell ("Amer. Textbook of Physiology"), it is believed that the starch molecule, which is quite complex, consisting of some multiple of $C_6H_{10}O_5$,—possibly $(C_6H_{10}O_5)20$,—first takes up water, thereby becomes soluble (soluble starch, amylodextrin), and then splits with the formation of dextrin and maltose, and that the dextrin again undergoes the same hydrolytic process and may continue under favorable conditions until only maltose is present. The difficulty at present is in isolating the different forms of dextrin that are produced. It is usually said that at least two forms occur, one of which gives a red color with

iodin, and is known as erythrodextrin, while the other gives no color reaction with iodin, and is termed achroodextrin. It is pretty certain, however, that there are several forms of achroodextrin, and, according to some observers, erythrodextrin also is really a mixture of dextrins with maltose in varying proportions. In accordance with the general outline of the process given above, Neumeister proposes the following schema, which is useful because it gives a clear representation of one theory, but which must not be considered as satisfactorily demonstrated:



Von Mering and Ewald have shown that in the transformation of starch into sugar by ptyalin, the greater portion is converted into maltose—only a small portion into dextrose. But the maltose formed in the stomach is changed to dextrose in the intestine. If the amylaceous transformation proceeds normally in the mouth and stomach, after a time, within an hour, at least, so much starch has been changed into achroodextrin, maltose, and dextrose, that the addition of small quantities of Lugol's solution to the filtered stomach contents no longer produce any changes in color. The occurrence of a purple (erythrodextrin) or a blue color (starch) shows that the starch transformation has been incomplete. Now, this may be due either to a deficiency of ptyalin or to a rapidly increasing acidity or hyperacidity of the stomach.

Ewald states that, although he tested a large number of patients for the fermentative power of saliva, he never found a saliva that could not convert starch into sugar. This, too, when he tested the salivary secretion of patients with dental caries, angina, diphtheria, and carcinoma of the tongue.

From the above it is evident that there must be two stages of gastric digestion, (1) an amylolytic and (2) a proteolytic. Having satisfied ourselves as regards the fate of the carbohydrates, or

starches, let us proceed to study proteolytic digestion, or conversion of proteids, albumens, gelatins, fibrins, elastin, meat, etc., etc.

Gastric Juice. — *Hydrochloric Acid.* — The secretion of the stomach is a complex fluid, clear, colorless, and of acid reaction; it has only one-half per cent. of solid ingredients. The amount secreted in twenty-four hours is about 1600 gm. Its chief constituent is hydrochloric acid, which it contains in the amount of 0.1 to 0.22 per cent. (one to two per thousand). This degree of acidity is not reached at once, but gradually; at the beginning and end of stomach digestion, the percentage of HCl is considerably less. Besides the HCl, gastric juice contains two unorganized ferments, pepsin and rennin.

Hydrochloric acid acts in six different ways, all of which are of great significance for the normal progress of digestion.

1. HCl acts as an antizymotic or antiseptic, destroying pathogenic organisms and preventing abnormal fermentations. This antibacterial effect extends even into the duodenum.

2. HCl has the power to convert the pro-enzymes of the gastric glands (pepsinogen and rennin zymogen) into active ferments in a very short time (according to Langley, in one minute).

3. This gastric acid possesses a certain regulatory influence on the progress of peristalsis.

4. HCl transforms, with the aid of pepsin, albuminous bodies into peptones, gelatin into gelatin peptone, elastin into elastin peptone. But in reality the pepsin is the main or chief agent in these transformations, as the HCl can be effectively substituted by nitric, phosphoric, oxalic, sulphuric, lactic, and butyric acids.

5. By HCl cane sugar is changed to invert sugar (dextrose and levulose). This property is also ascribed to a number of bacteria that can invert cane sugar, although after a longer time.

6. HCl, finally, is instrumental in bringing into solution the soluble calcium and magnesium salts, introduced in the food.

Concerning the origin and derivation of the hydrochloric acid, we unfortunately have nothing but speculation. No free acid occurring in the blood or lymph, it is rational to conclude that it is produced in the secreting (oxyntic?) cells of the gland-ducts. It seems probable that the acid is derived from the neutral chlorids of the blood, which are in some way decomposed, the chlorin uniting with hydrogen to form HCl. The acid is secreted at the gastric mucosa, while the base remains behind, and probably passes back

into the blood. This, in a way, explains the increased alkalinity of the blood and the decrease of acidity of the urine after meals, the return of basic substances into the circulation naturally having such an effect. According to Heidenhain, a free organic acid is secreted by the cells (oxyntic), which then decomposes the chlorids. According to Maly, the HCl is the result of a reaction between the phosphates and chlorids of the blood as expressed in the following two equations:

$$NaH_2PO_4 + NaCl = Na_2HPO_4 + HCl \text{ or}$$
$$3CaCl_2 + 2Na_2HPO_4 = Ca_3(PO_4)_2 + 4NaCl + 2HCl.$$

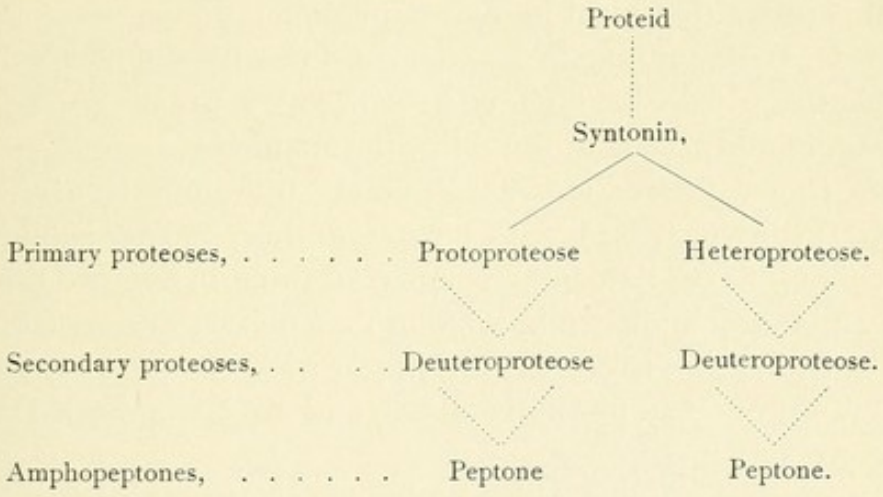
What is known thus far of the specific action of living cells, enforces the impression here, that, as in other chemical processes not yet understood, vital phenomena are difficult to express in chemical formulæ.

CHAPTER V.

PEPSINOGEN AND PEPSIN.—RENNIN ZYMOGEN AND RENNIN.—INTESTINAL DIGESTION.—DUODENAL INTUBATION.

It should not be understood that all combinations of the gastric juice with albumins are at once peptones; like the starches, the proteids reach their end stage of gastric digestion by a series of distinct intermediate stages. These are (1) proteid, (2) acid albumin or syntonin, (3) propeptone or hemi-albumose, and (4) peptone. Besides forming peptones out of albumins, pepsin deprives gelatin of its property to coagulate, or rather to gelatinize, and forms gelatin peptones out of it. Peptones are derived from egg, serum, and plant albumins, gelatin, meat, fibrin, casein, etc.

The steps in peptic digestion may be made more intelligible by the following schema, modified from that of Neumeister ("Lehrbuch d. physiol. Chemie," 1893, p. 187):



No other mineral acid gives as good results with pepsin as HCl, which can form pepsin from pepsinogen in the quickest time. It is useful to be able to test for propeptone formation. In normal digestion, one hour after the test breakfast, propeptone is present only in traces, or usually is not to be detected at all; but in abnormally slow digestion it is still abundant at that period.

The best method, up to present date, is by means of the biuret

reaction. In this reaction a dilute solution of cupric sulphate is added to stomach contents in the cold, and a few drops of potassium hydroxid added sufficient to make the solution alkaline; an intense red color results. Cupric sulphate and KOH, added to ordinary albumin and syntonin, without warming, produces a bluish violet, which must be distinguished from the purple-red of biuret.

The more marked the propeptone reactions are, the less the peptone which has been formed and eventually removed from the stomach. We can approximately estimate the amount of the peptone by the intensity of the biuret reaction if we always use the same quantities of stomach contents, caustic potash, and cupric sulphate, and compare it with the reaction given with a peptone solution of known strength. Peptone gives the same pink, purple-red color with the biuret reaction as propeptone. If we desire to estimate the rate of proteolysis in the stomach, the biuret reaction will not permit us to distinguish between these two bodies; the only differentiation possible is by precipitation of the propeptone in the following manner: The stomach filtrate is carefully neutralized, an equal quantity of common salt solution is added, and then a few drops of concentrated acetic acid. A precipitate will be propeptone, which can be filtered off and weighed; any red biuret reaction after this separation must be due to peptone.

In order to determine, in a given specimen of stomach contents, whether the pepsin or HCl is present in too great or too small a quantity, one proceeds in the following manner:

Pour 10 c.c. filtered stomach contents into four test-tubes and number them Nos. 1, 2, 3, 4. To No. 1, nothing further is added; to No. 2, enough HCl to make a solution of three to five per thousand (this can be accomplished by adding one to two drops of officinal HCl, U. S. Pharm., to 10 c.c. filtrate); to No. 3, 0.2 to 0.5 gm. (gr. iij to gr. vij) of pure pepsin is added, and to No. 4 both HCl and pepsin are supplied.

A small disc of egg albumen (which is prepared by cutting boiled egg albumen into lamellæ of uniform thickness with a microtome and punching out equal circles by a cork borer) is added to each test-tube, and they are then put in the incubator at 100°. The rate at which the albumin is dissolved will tell us whether the filtrate was perfect in the requisite amount of pepsin and HCl, whether pepsin alone, or HCl only, or, finally, whether both were necessary. In this way we can discover which factor is

at fault. In the human stomach the formation of peptone remains at a certain percentage by the removal or absorption of peptones over that amount, and also it would seem by an inhibiting influence which a certain percentage of peptone has over the proteolytic process in retarding or suspending it. As this can not be imitated in a test-tube, *i. e.*, the absorption of ready formed peptones, a seemingly delayed digestive process of egg-albumen discs in the test-tubes may in reality be due to a very active stomach filtrate.

Rennin, the second gastric ferment, produces a light, not very cohesive, coagulation of milk. This coagulation is a characteristic cake of casein floating in clear serum, more dense, not lumpy, more cohesive coagulation, than produced by acids. This ferment is a constant constituent of the stomach contents, just as pepsin and pepsinogen. With a complete absence of the rennin and its pro-enzyme, one can with certainty conclude that the case is one of atrophy of the gastric mucosa.

The demonstration of rennin ferment is carried out in the following manner; Ten c.c. of raw, unboiled milk are placed in the incubator with 2.5 drops of stomach filtrate. If rennin is present, the characteristic milk coagulation will occur in a variable time (one minute to several hours, according to the quantity of ferment).

Occasionally, rennin, the perfect ferment, is not contained in the stomach contents, while at the same time rennin zymogen is present. This is demonstrated, according to Hammarsten, by adding to the mixture just described two c.c. of a concentrated solution of calcium chlorid, $CaCl_2$.

If a rennin coagulum occurs, it follows that rennin zymogen is present, but not the perfect ferment. For these tests, raw milk only can be used, as it coagulates ten times as rapidly as boiled milk. Jaworski has pointed out that in cases where tests for rennin and rennin zymogen are both negative, it is advisable to try pouring a 0.3 per cent. to 0.6 per cent. solution of hydrochloric acid into the stomach, to see whether this HCl may not be able to awaken a secretion of rennin; this should especially be done before making the diagnosis of complete atrophy of the mucosa.

The Physiology of Intestinal Digestion.—Our knowledge of the digestive processes in the intestine is, from a physiological as well as from a pathological point of view, defective, at times contradictory. Concerning gastric digestion, we are much better in-

structed, because here the processes are simpler, and material for investigation can be more easily obtained. The stomach-tube supplies us without difficulty with gastric contents, but hitherto all intestinal contents of human beings have been obtained from rare cases of intestinal fistulæ, for the feces give no constant and reliable information of the digestive actions in the smaller intestine.

The earliest investigations of intestinal contents were made in 1662 by Regnier de Graaf, who made experimental fistulæ into the

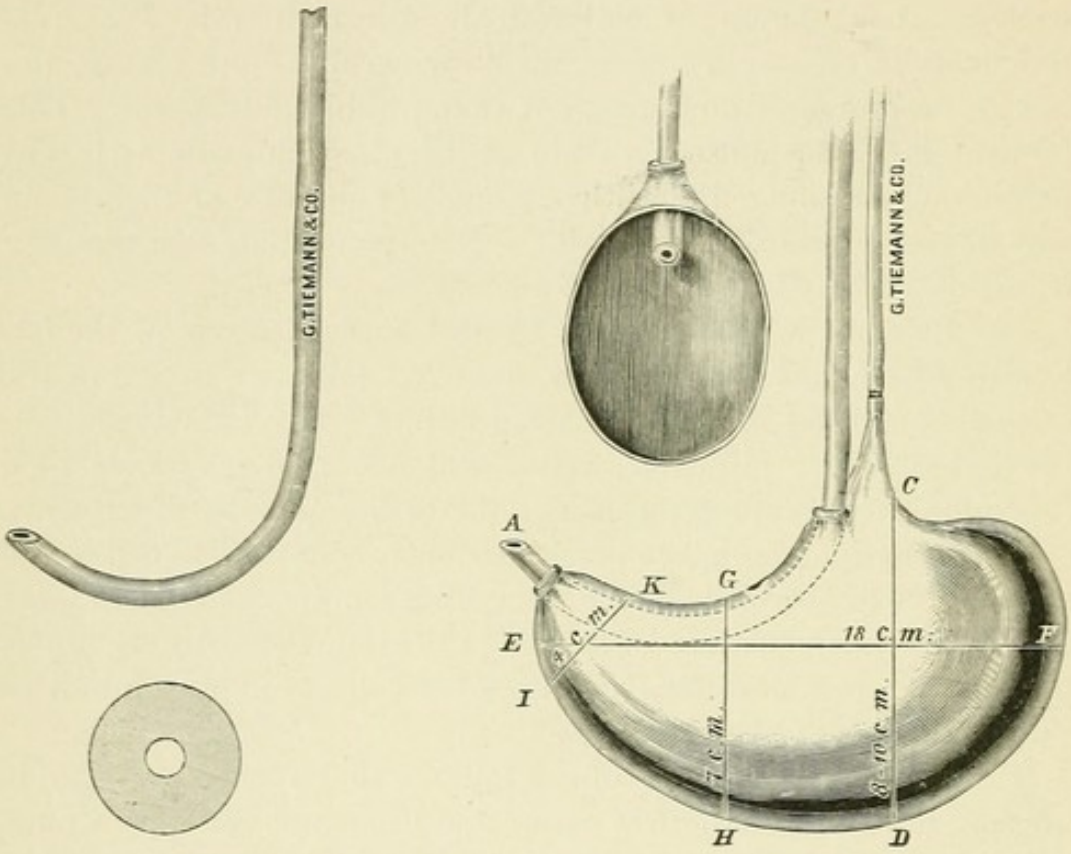


FIG. 5.—APPARATUS FOR OBTAINING INTESTINAL CONTENTS.

intestinal canal of animals. It is a curious historical fact that this intestinal experiment antedated the first investigations of stomach contents which were carried on in 1752 by Reaumur. So up to the present time there was no prospect of getting a better insight into the physiology of intestinal digestion until a method for intubating the duodenum in the living human subject was devised by the author.

This method, which is described in the *Johns Hopkins Hospital Medical Bulletin* for April, 1895, and also in "Boas' Archiv. for

Digestive Diseases," vol. II, page 85, consists, in the first place, of the introduction of a thin, elastic rubber bag into the stomach. This bag, when folded over a tube which runs through it, does not occupy as much space as an ordinary stomach-tube, and has the exact shape of the human stomach when it is distended by blowing it up within that organ, to which it fits itself exactly,—and is closely applied to the gastric walls.

The intragastric bag is distended by the pressure apparatus shown in figure 6. The graduated bottle (*A*) is full of water and elevated above the bottle (*B*), which is empty and also graduated.

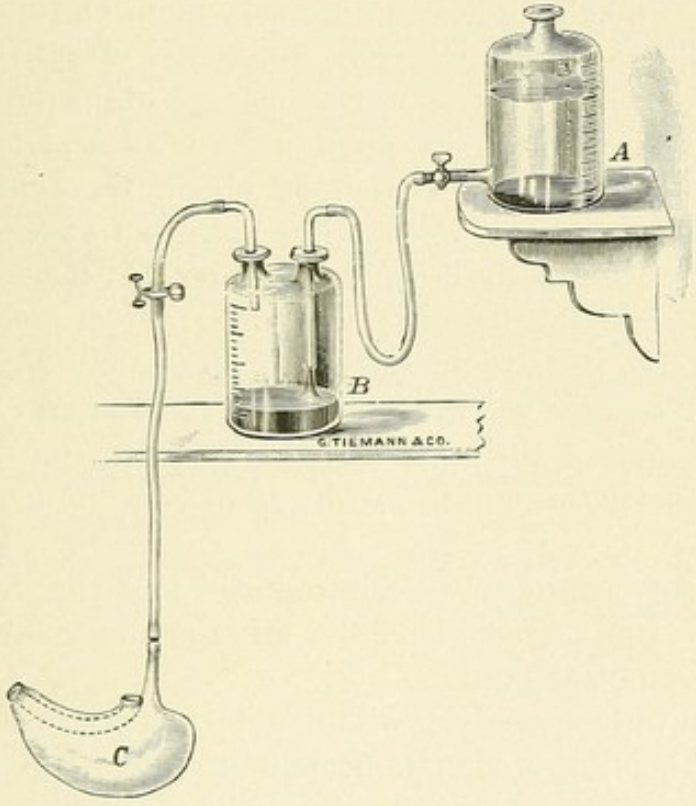


FIG. 6.—PRESSURE BOTTLES FOR DISTENDING THE INTRAGASTRIC BAG DURING DUODENAL INTUBATION.

The stomach-shaped bag (*C*), when it reaches the stomach, is connected with the lower empty bottle (*B*). Then the stop-cock permitting the water to run from *A* to *B* is opened, and the water runs from *A* into *B*, displacing the air in *B*, which distends the bag, *C*, within the stomach, filling it entirely. As is observable on this bag, a guide is contained in it, running along the dotted line parallel to the lesser curvature. In this guide the duodenal tube is inserted, lubricated with oil before the bag is pushed into the

stomach. This tube is provided with very thick walls, by virtue of which it is not easily kinked or bent upon itself.

The relation of the thickness of the walls to the diameter of the lumen is shown in the cross-section of figure 5. When the intragastric bag is blown up, it fills the stomach entirely. The duodenal tube lies in its sheath or guide, and, on being pushed onward from the mouth, it is not possible for it to go anywhere else except through the pylorus into the duodenum. In the figures it can be seen that the bag is not distended by the duodenal tube, but a separate, very small tube runs down the esophagus, ending in the bag, serving the purpose of its distention. Both tubes together do not occupy as much space as an ordinary stomach-tube.

A description of this method is considered essential because it seems to be destined to bring our knowledge of the physiology and pathology of the intestines upon a basis of ascertained facts; because we can at any time thereby obtain the contents of the intestine, and the gut may in any of its parts be reached with safety.

After known test meals, it is possible, after they have passed from the stomach into the duodenum, to draw out samples from this part and subject them to analysis. By alternately distending any part with air or water we will be enabled to locate the part by the percussion sound on the outside of the abdomen, and the distance it is located from the mouth can be seen from the length of tube introduced.

Small electric lamps may be introduced into the duodenum as they are into the stomach, and the location and condition recognized by electrodiaphany.

Hitherto, in all experiments on this subject it has been impossible to obtain either the pancreatic or biliary secretion in a pure condition; this is due to the fact that both the pancreatic and the common gall-duct empty into the descending portion of the duodenum very near each other.

Just at present we have under observation a female patient who has suffered repeated biliary colic. At times she passes small stones without giving her much pain,—at least, they are found in the stools without having given her any colic. She is willing to undergo an operation to be relieved. Through the comparatively thin abdominal walls we are able to feel numerous stones in the gall-bladder. She consented to an attempt at intubation of the duodenum to determine whether there was any bile secreted. The

duodenum was entered without difficulty, and cleansed by running in and aspirating out distilled warm water. Twenty-four hours afterward the duodenum was again intubated according to our method, and washed with 100 c.c. of warm distilled water.

On being aspirated, this water was still clear, but viscid and sticky, similar to a solution of egg-albumen. It contained no bile pigments nor cholesterin, and was free from taurocholates and glycocholates. It was colorless and odorless, and seemed very rich in some form of albumin. That it was pure pancreatic juice was proven by its digesting fibrin and serum albumin.

The juice obtained in this manner will digest from 85 to 95 per cent. of Merck's dried serum albumin in the digestorium at 100° F. in two hours. The amylolytic and fat-decomposing property of the juice was determined in a similar manner. One is therefore justified in concluding that in this case the pancreatic juice was obtained almost pure, as there were no bile elements contained in it, the bile being prevented from entering the duodenum by a calculus or catarrhal occlusion in the common duct. As there are also pancreatic calculi, or occlusions of the duct by neoplasm or catarrhal swelling, it is conceivable that we may yet be able to obtain the bile in a pure condition, and free from pancreatic juice, in the human subject.

The secretion of Brunner's and Lieberkühn's glands will, however, always constitute an admixture of these juices.

The Pancreas; its Secretion and Pancreatic Digestion.—In 1846 Claude Bernard made the first scientific and fundamental investigation concerning the pancreatic secretion. Later on, Kühne, Bidder and Schmidt, Corvisart, Heidenhain, and others enlarged these results.

Its secretion, as Bernard first observed, is dependent upon digestion, and is a clear, colorless, and odorless fluid, very alkaline, and so rich in albumin that it solidifies on boiling. Zawardsky had opportunity of analyzing the normal human pancreatic secretion in a case of pancreatic fistula, which remained behind after removal of a tumor. According to his analysis it contained 86.4 per cent. water, 13.25 organic substances; among the latter are 9.2 proteid bodies and 0.83 extractive substances, soluble in alcohol; lastly, 0.34 per cent. salts.

The chyme which passes into the duodenum from the stomach comes under the influence of formed or organized and unformed or unorganized ferments. The formed or organized ferments are

represented by bacteria, which bring about carbohydrate fermentation, mostly in upper bowel, and proteid putrefaction, mostly in lower bowel.

The unorganized ferments are contained in the pancreatic secretion, the bile, and in the succus entericus. The most important constituents of the pancreas are three ferments or enzymes: (1) an amylolytic, (2) a proteolytic, and (3) a fat-splitting ferment.

According to W. G. Halliburton and T. G. Burton (*Journal of Physiology*, vol. XX, p. 106), pancreatic juice possesses a milk-precipitating substance, causing at 35° to 45° C. a granular precipitate in milk, but there is no solidification until the milk cools, when it sets to a coherent curd. On warming, the curd is broken up, and the milk resumes its granular fluidity. The granular precipitate produced by pancreatic juice seems, according to these observers, to be intermediate between casein and caseinogen.

The amylolytic or pancreas diastase is very similar to ptyalin in its action, and changes boiled starch into maltose exceedingly rapidly at body temperature. In addition, small quantities of dextrin and grape sugar are formed; one gm. of pancreatic juice from a dog will invert 3.6 gm. starch into sugar. Cane sugar and inulin are not affected by it. According to Zweifel, this ferment is absent in the pancreas of new-born children.

The fat-splitting ferment of the pancreas (also called steapsin), which thus far has not been obtained in a pure state, breaks up neutral fats into fatty acids and glycerin. This process occurs very slowly, however. Berthelot found that 15 gm. of pancreatic secretion of the dog required at least twenty-four hours to break up two decigrams of monobutyrin completely into butyric acid and glycerin. The fatty acids formed during this transformation combine with alkalies in the intestine to form soaps, which by emulsifying other fats assist greatly in their absorption. In the laboratory it always requires powerful mechanical action to effect an emulsion of fats; not so in the intestine, where it is evidently accomplished with great facility. That this must greatly assist in fat resorption is evident from the frequent observation that after disease of the pancreas the feces become very rich in fat, which may be present in such a large amount as to congeal on the surface of the stool.

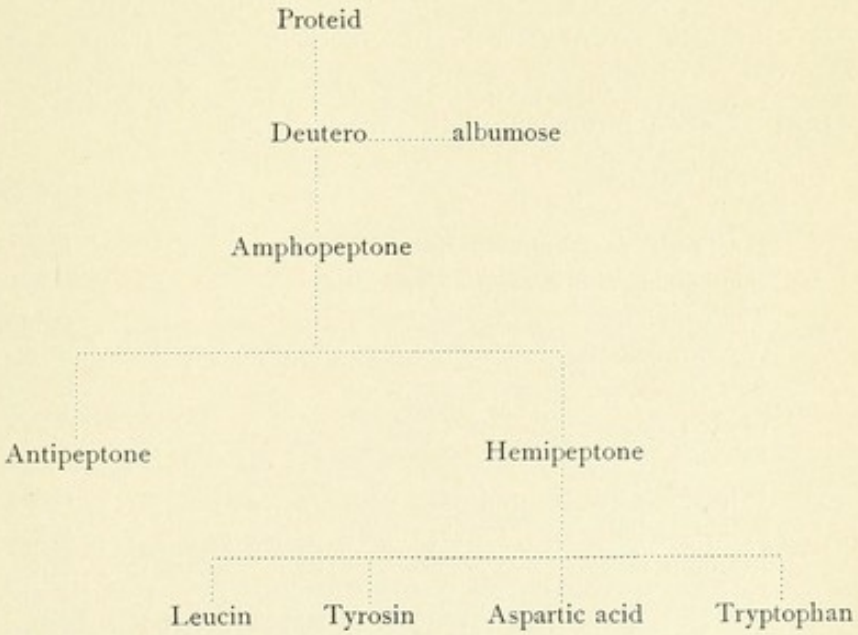
The proteolytic ferment of the pancreas has been called trypsin by Kühne. Using pancreatic juice and boiled blood-fibrin, he found that it did not swell up, but that it became very fragile, and

finally liquefied. As we take in all of our albumin in a boiled or roasted state, which becomes peptone in the stomach and not soluble albumin, the question has arisen—whither do we derive our soluble native albumin? This is obtained from pancreatic trypsin digestion of boiled albuminous bodies, which changes them to albumin soluble in water, or at least in a weak saline solution, from which they can be precipitated by heat. The proteolytic action of trypsin takes place in an alkaline or neutral medium only.

Among the bodies formed from albumins and proteids under the influence of trypsin are a globulin that is insoluble in water, hemipeptone and antipeptone, leucin, tyrosin, and asparaginic acid. Indol, which is found in the jejunum, is a product of bacterial action on albumins. A chromogenic body has been described by Tiedemann and Gmelin which has received the name tryptophan; it is a result of advanced albumin decomposition. Trypsin, then, to sum up, changes proteids to peptones and soluble albumins, casein to casein peptones, gelatin to gelatoses and gelatin peptone, and elastin to elastoses and elastin peptones.

In animals that have been deprived of their pancreas by operation only 44 per cent. of proteid, 57 per cent. to 70 per cent. of carbohydrates, and no fats at all, were absorbed, although four-fifths of the fats were split up into fatty acid and glycerin.

The processes of tryptic digestion are briefly represented in the following schema, according to Neumeister:



Trypsin produces peptone from proteids more readily than does pepsin.

CHAPTER VI.

THE BILE.—THE SUCCUS ENTERICUS.—INTESTINAL FERMENTATION.—PUTREFACTION.—FORMED OR ORGANIZED FERMENTS.

It is known at present that the bile exerts no chemical effects upon the food-materials; nevertheless its presence in the duodenal chyme is significant on account of its alkaline reaction and its effect on the mucous membrane. The most important function of the bile is the excretion of metabolic products that can not be utilized in the organism.

The contents of the gall-bladder represent a concentrated secretion, therefore our knowledge of the physiological action of the bile depends upon the discharge of biliary fistulæ. The bile is a golden yellow, at times olive-brown, secretion; it is never of a green color, but generally very mucoid and stringy. Its alkaline reaction is due mainly to carbonates and phosphates. The quantity poured into the intestine is largest in the first hour after food is taken.

Albumins increase, fats diminish, this quantity, while sugar and carbohydrates appear to exert no influence (Voit). The quantity secreted in twenty-four hours averages from 500 to 600 c.c. (Ranke, Wittich, Hammarsten). The quantitative analyses of Hammarsten have given the following results:

Solid materials,	1.62	to	3.52
Water,	96.47	"	98.37
Mucin and coloring-matter,	0.27	"	0.91
Compounds of bile acids and alkalies,	0.26	"	1.82
Taurocholate,	0.052	"	0.203
Glycocholate,	0.204	"	1.61
Fatty acids,	0.024	"	0.136
Cholesterin,	0.048	"	0.160
Lecithin,	0.048	"	0.065
Fat,	0.061	"	0.095
Soluble salts,	0.676	"	0.887
Insoluble salts,	0.020	"	0.049

At times a diastatic ferment is present in the bile, but it is not a specific constituent (Neumeister), but appears in the bile like the

diastatic ferment which appears in the urine; it seems to be identical with the ptyalin zymogen of the pancreatic juice.

When the bile is prevented from entering the intestine at all, albumins, gelatins, and carbohydrates are absorbed in a normal manner (Voit and J. Munk), but the digestion of fats is very seriously interfered with; a normal animal resorbs 99 per cent. of fats, if not more than 150 to 200 gr. are given, *i. e.*, only one per cent. appears in the feces, but on producing an experimental fistula conducting the bile outward, 60 per cent. of the fats are not utilized (Voit). The subjoined is a synopsis of the uses and functions of the bile:

1. Fats are brought into a fine, permanent emulsion by bile, just as by pancreatic juice.

2. Bile assists the fat-splitting effect of pancreatic juice (Nencki). Without bile, only 61 per cent. of tribenzoicin were decomposed by pancreatic juice; with bile, the total amount.

3. By its alkalinity it necessitates the formation of soaps.

4. Bile dissolves fats in minute quantities.

5. Bile dissolves the saponified alkaline bases which are insoluble in the juices of the intestines.

6. Animal membranes moistened with bile are more permeable to emulsified fats than membranes moistened with water (von Wisting, Heidenhain).

7. Bile is a stimulant to the intestinal epithelial cells, incites their proper functioning and maintains it (Röhmann).

8. It is claimed that albuminous bodies and pepsin, dissolved in the chyme, are precipitated as a resinous, sticky deposit, which adheres better to the duodenal wall, and effects a better utilization of the albuminates thereby.

9. An inhibitory influence over putrefaction is ascribed to bile (Maly and Emmerich).

10. An influence favoring an increase of the peristalsis of the intestine (Röhmann).

The Succus Entericus.—The succus entericus is a secretion of the crypts of Lieberkühn, and was first studied in man by Demant after a herniotomy. This secretion has the color of light Rhine wine, and is very strongly alkaline, owing to the presence of 1.5 per cent. carbonate of sodium. The principal constituents are albumins and mucin. It contains also ptyalin and an inverting ferment, but has no effect on albumins and fats; its purpose seems

to be probably that of a neutralizer of the acids originating from fermentation of carbohydrates; its excess of mucin may be instrumental for the onward movement of the bowel contents.

The Formed, or Organized, Ferments (Bacteria).—Proteids, carbohydrates, and fats are subject to decomposition in the intestines by bacteria. Fats are not decomposed to any considerable extent in the lower intestinal sections (Nencki and Blank), but a small fraction is split up into glycerin and fatty acids.

A greater interest attaches itself to the fermentation of carbohydrates, which occurs principally in the upper small intestine and leads to the formation of acetic, lactic, butyric, and carbonic acids, alcohols, and hydrogen. It is not known how much of the carbohydrates is decomposed in this manner.

The putrefaction of proteids, caused by certain bacteria of the lower bowel, only occurs in an alkaline medium. The first products of this putrefaction are the identical bodies which are formed during pancreatic digestion, viz.: albumoses, peptone, amido-acids, and ammonia. But then the putrefaction goes still further; tyrosin is formed, and from this, through a series of complex oxyacids, the product phenol (carbolic acid) is reached, which may yield phenyl-propionic and phenylacetic acids. A second variety of aromatic substances, not derived from tyrosin, is represented by indol, skatol, and skatol carbonic acid; finally, leucin and ammonia salts of capronic, valerianic, and butyric acids. The gases formed are carbonic acid gas, hydrogen, hydrogen sulphid, and methyl-mercaptan. As bacteria can produce peptone, it might be presumed that such product may be useful to the organism. This peptone is not made for philanthropic purposes,—it is simply one intermediate, probably unavoidable, stage in a long chain of decompositions.

We can not measure the intensity of carbohydrate fermentation, but the aromatic end-products of proteid putrefaction can be approximately estimated by determination of the combined and ethereal sulphates occurring in the urine.

The number of bacteria increases from the duodenum on downward until they become enormously prolific in the colon. They also differ qualitatively. In the small intestine, Gessner found a prevalence of the bacterium lactis ærogenes and streptococcus pyogenes; the colon-bacillus was present, but insignificant in numbers. In the colon, however, the reverse was the case. It was formerly an accepted view, principally defended by Pasteur, that

the intestinal bacteria were absolutely indispensable for digestion, and, therefore, for the nutrition of the individual. From this view we have returned to what seems a more logical belief, based on observations of Escherich, who held that bacteria contributed very little to the digestion of the infant, as they do not affect casein and fats, but only sugar of milk, turning it into lactic and carbonic acids and hydrogen. The work of Nuttal and Thierfelder shows that guinea-pigs can live on absolutely sterile food.

Macfadyen, Nencki, and Sieber arrived at a similar conclusion in their now classical observations on adults. (*Archiv f. exp. Pathologie*, Bd. XXVIII, 1891.) One of their objects of study was a female with a fistula that opened the small intestine on the external abdominal wall, just at the end of the ileum. The entire colon was therefore excluded from the digestive act. As nearly all proteid putrefaction occurs in the colon, this case presented a chance to study the absence of products of albuminous putrefaction and its effects.

It was found that bacteria are not at all essential to digestion, as their patient was very healthy without proteid putrefaction. They declare that the bacterial fermentation of carbohydrates in the small bowel is detrimental, rather than advantageous; inasmuch as the bacteria live at the expense of the ingested carbohydrates, therefore a corresponding amount of food is lost to the organism.

Our knowledge of the bacterial activity in the intestines, though much enriched by valuable researches in the last decad, is, according to our opinion, in its infancy. So, also, our knowledge of the pathogenic significance of intestinal bacteria. There is, undoubtedly, a kind of interaction and correlation between digestive ferments and juices on the one hand, and bacteria on the other, or even between bacteria and bacteria, or between the products of bacterial metabolism. For instance, Metschnikoff has demonstrated that the multiplication of the cholera vibrio is much advanced by the presence of torulæ and sarcinæ in the intestines.

It is conceivable that bacteria wage war upon each other, as well as upon the cells of our tissue, and that we are benefited by this mutual self-destruction of our parasitic inhabitants. It is conceivable, also, that they fall a prey to the poisonous metabolic products of their own or other species of bacteria. Certain very decomposable food, as cheese that was rich in germs, has been found by competent observers to reduce the amount of indican

and of the ethereal sulphates in the urine, which indicates a reduced putrefaction.

The human stomach must not be regarded as an organ that can absolutely sterilize all food. The spores, being more resistant to HCl than the fully developed bacteria themselves, pass through the stomach uninjured. Miller assumes that at the height of digestion only, when the amount of HCl is greatest, the less resistant bacteria are killed. Bunge, some years ago, announced that the principal object of the HCl was one of sterilization. It is undeniable, from recent investigations, that the human stomach is at no time free from germs. Captain and Morau found them at the height of digestion. Abelous found them in his own stomach when it was perfectly empty. Miller (University of Berlin) demonstrated that the mouth contains large quantities of microbes; in one unclean individual he estimated the numbers of mouth bacteria at 1,140,000,000. Of 25 different varieties occurring in the mouth, this observer was able to demonstrate 12 of the same in the feces. Nevertheless, the mouth bacteria, according to Lucksdorff, constitute only three per cent., while those entering through the food constitute 97 per cent., of the bacteria of the intestine. There is no autochthonous vegetation of bacteria in the intestine; they are all introduced from the mouth, or in the food, or reach there by way of the circulation. (By autochthonous bacteria are meant such as are originally developed at the place where they are found.)

From observations made up to the present time it seems probable that catarrhal and other inflammatory diseases of the intestinal mucosa are not produced by specific, constantly recurring microbes, but that many kinds of bacteria are capable of producing these diseases under conditions which are thus far not perfectly understood.

It appears, furthermore, that the same bacterium may at one time be perfectly harmless, or it may cause a light, trivial affection, or, thirdly, at other times a very serious disturbance. This is the case with the bacterium coli communis, which is tolerated without detriment by the majority of mankind; but occasionally it is demonstrated as the producer of colitis, dysentery, and cholera nostras.

The manifold forms of the catarrhal inflammations are explicable by the fact that the intestinal flora is also very manifold. These same bacteria are factors in the etiology of diseases of the perito-

neum, and of all organs that are in connection with the intestines. Even remote organs, not in anatomical connection with the bowel, are not safe from their invasion.

They are known to gain entrance into the blood and lymphatic channels through losses of substance in the intestinal mucosa. The experiments of Posner and Lewin have taught us that even without such portals of entry they seem to be able to pass through the bowel-wall in masses, and threaten the organism. Hans Hensen (*Zeitschr. f. Biol.*, Bd. xxxv, page 110) has shown that bacteria can penetrate natural and artificial membranes, allowing diffusion. There are fine canals, passable for bacteria, that can not be demonstrated by the hemoglobin test. Great harm can be done to the general organism, and to special organs in particular, not only from this invasion, but also from absorption of the soluble products of bacterial metabolism and of food decomposition.

This condition of self-poisoning from toxic substances in the individual's own intestinal canal, is spoken of as intestinal auto-intoxication. Not all auto-intoxications are of intestinal origin; diabetes mellitus, for instance, is an auto-intoxication by grape sugar, which is in this case a product of disturbed metabolism, and does not originate from the digestive canal.

The dangers which threaten the general organism from the intestinal bacteria have given rise to many efforts to sterilize the digestive tract by means of so-called antiseptics. Most of the agents used for this purpose—calomel, salol, naphthalin, beta-naphthol, bismuth, creosote, bismuth salicylate—are themselves toxic, and, in doses sufficiently large to reduce the number of bacteria to any considerable extent, they are harmful to the body. The putrefaction of proteids, as measured by combined and ethereal sulphates in the urine, can only be temporarily diminished by this method.

Intestinal disinfection is therefore an unsolved problem. Efforts in this direction should still be encouraged, because we may be able thereby to attenuate the pathogenic inhabitants of our intestines and render them less virulent. The best disinfectant of the human intestine is its normal action, and the best way to control putrefaction is by selection of adapted, appropriate diet, fresh air, moderate exercise, good sleep, pure water, avoidance of overeating, overdrinking, and of chemicals. (Hemmeter "On Intestinal Putrefation and Albuminuria"—*Maryland Medical Journal*, July 24 and 31, and August 7, 1897.)

CHAPTER VII.

EFFECTS OF CONTEMPORANEOUS ACTION OF SEVERAL DIGESTIVE SECRETIONS.—METHODS FOR TESTING THE MOTOR FUNCTIONS OF THE STOMACH.

When the gastric chyme enters the duodenum the albuminoid and proteid foods appear partly as syntonin, albumoses, and peptones, and partly unchanged. The carbohydrates appear either as erythrodextrin, achroodextrin, or maltoses, and partly unchanged. The fats are unchanged; rarely are they found split up, so that one can detect traces of fatty acids.

Water, according to the interesting investigations of von Mehring, is absorbed only in very small quantities from the stomach. It appears that fully 90 per cent. of all water taken into the stomach is passed into the duodenum; alcohol, and whatever is in solution in it, is absorbed readily. Grape-, milk-, and cane-sugar, also maltose, are absorbed in moderate amounts when they are in aqueous solution; when they are in alcoholic solution, larger amounts are absorbed. Dextrin and peptone are also taken up from the stomach, but in smaller quantities than sugar. The amount of the substances resorbed increases with the concentration of the solution. Simultaneously with this resorption, a more or less active secretion of water occurs into the stomach. This secretion of water into the stomach increases or diminishes as the quantity of substances resorbed or taken up increases or diminishes.

Secretion of water occurs even when no HCl is demonstrable in the normal stomach. The chyme, then, as it enters the duodenum, still contains all of its water, but is minus some of the peptones, dextrins, sugars, and alcohols. It is more or less acid from free HCl. When the bile acts on this acid chyme, a resinous, flocculent precipitate is deposited from it on the walls of the duodenum; at the same time a finely granular cloudiness occurs. The resinous deposit consists of bile-acids and syntonin (Hammarsten), and the granular opacity is due also to bile-acids and small amounts of peptone.

Excess of bile may redissolve these precipitates so that they can not at times be found in animals killed at the height of digestion. The digestion by pepsin is checked by the complete neutralization of HCl by pancreatic juice, bile, and succus entericus. If any precipitation occurs as stated, pepsin is also thrown down and resorbed again or digested by trypsin. The bile does not disturb the proteolytic power of pancreatic juice (Claude Bernard). Boas and the author have shown that the clear duodenal chyme will digest 81 per cent. of serum-albumin in three hours; at 40° C. its alkalinity was 0.8 per cent., Na_2Co_3. It was also shown that this duodenal chyme converted 25 per cent. of starch into maltose, and that it produced 12.1 per cent. fatty acids from neutral olive oil in three hours. Boas obtained his mixture of bile, pancreatic juice, and succus entericus from a patient who had most probably a duodenal stenosis and who vomited this chyme frequently. In the author's experiments the duodenal secretions were obtained by his method of intubation of the duodenum.

It was found in these experiments that the duodenal juices, when filtered, digested 85 per cent. to 95 per cent. of Merck's dried serum-albumin in three hours at 40° C. The author's results with starch conversion show that the filtered duodenal juices will digest 42 per cent. of starches, or rather convert them into maltose, which is considerably in excess of the figures obtained by Boas. The fat-splitting effect observed by us in this juice was again more near the result of Boas, for we found that 15.3 per cent. of fatty acids were obtained from neutral olive oil. In a case of biliary calculi, we have been able to obtain the pancreatic juice free from bile, as the bile-ducts must have been stenosed either by a small calculus or a bit of thickened bile and mucus mixed.

The fat-splitting effect of pancreatic juice is improved by the presence of bile, as is also the amylolytic action of amylopsin.

Demonstration.—The action of pancreatic juice obtained from a dog on neutral olive oil and on a solution of starch should be studied both with and without bile. Pancreatic juice plus bile will split up more fat and convert more starch into maltose than without bile (Martin and Williams).

The effect of trypsin on pepsin is not definitely known, but it is probable that pepsin, being closely allied to proteids, is disintegrated by trypsin, but, in an acid solution, pepsin checks the action of trypsin (Kühne, Langley, Ewald, and Baginsky). According to Baginsky,

rennin is destroyed by a neutral solution at room temperature. The ferment action of bacteria in the small intestine is limited to the carbohydrates. Discharges of food from fistulæ of the small intestine show no fetid decomposition of albuminoids (Ewald and Nencki). The absence of proteid putrefaction in the small intestine is probably due to the rapid downward movement of the food mass in this portion of the bowel and to its acid condition.

Carbohydrate fermentation yields mainly lactic acid, ethyl alcohol, carbon dioxid, and hydrogen. Macfadyen, Nencki, and Sieber found the chyme that passed over into the large intestine (the cecum) from the ileum to be 550 gm., with 4.9 per cent. solid residue in case the chyme was of a very thin consistence; and 232 gm., with 11.23 per cent. solid residue, if the chyme was very condensed. Both of these figures are the amounts passing in twenty-four hours. The shortest time in which food passed into the cecum after it was swallowed was two hours; the longest period, five and and a quarter hours.

The reaction expressed in acetic acid was equal to one per thousand; the acidity is considered to be due to newly formed acetic acid, as the lactic acid and the HCl are neutralized by the succus entericus. This chyme contained 1 per cent. albumin; also peptone, mucin, dextrin, sugar, lactic acid, sarcolactic acid, and traces of fatty acids; it contained no leucin, tyrosin, urobilin, or ammonia. The characteristic products of albuminoid decomposition were absent.

Jaworski's investigations on the contents of the large intestine, which were discharged from a fistula in the ascending colon, showed that the daily fecal discharge of 150 to 200 grs. was decidedly alkaline, and contained the products of proteid and albuminoid decomposition, viz.: urobilin, skatol, phenol, oxyacids, ammonia, leucin, cadaverin, ethyl and butyl alcohol, sulphuretted hydrogen, and methyl mercaptan.

In view of the fact that the putrefaction of albuminoids and proteids occurs mainly in the colon, it is of interest to know how much of this class of food substances is left for the colon, and how much is digested in the small intestine. Nencki found that when the food contained 70.74 gm. albumin, which represent 10.602 gm. nitrogen, the amount of solid material discharged from a colon fistula in twenty-four hours was 26.5 gm., with 1.61 gm. nitrogen, which represented 10.06 gm. albumin. From this it is evident that 14.25

per cent., or, in other terms, only one-seventh of the total albumin, is left for digestion in the colon, and that 85.75 per cent. is resorbed from the stomach and small intestine.

The intensity of putrefaction in the colon depends upon four factors: (1) The amount of decomposable albuminoid materials ingested; (2) the duration of their retention in the colon; (3) the vigor and tonicity of the intestinal peristalsis; and lastly (4) the chemical reaction, for a very strong acid reaction, due to free acids, inhibits putrefaction.

Bile assists in this inhibition. Hirschler has demonstrated that carbohydrates suppress putrefaction considerably; this is due to the lactic, butyric, acetic, and carbonic acids caused by their fermentation. Albumin and peptone are absent from the contents of the rectum (feces), but are present in typhoid fever (von Jaksch). Peptone is found in all diseases that may produce pus in the evacuations,—for instance, dysentery, tuberculous intestinal ulcers, perforation, peritonitis, hepatic cirrhosis, and carcinoma.

A very important inquiry is that into the ultimate fate of the digestive ferments: Do they pass through the entire intestinal tract? Are they absorbed or are they decomposed? Or do they appear in their active form in the feces? This question is a very difficult one to solve, as our only method of detecting pepsin, rennin, trypsin, and ptyalin is by their digestive activity. In all experiments of this kind the feces must be first sterilized by saturated solutions of thymol; before using this it is well to exclude the action of peptonizing bacteria by filtering through a Pasteur filter.

If we found in the glycerin extract of the sterilized feces a substance which would dissolve boiled egg-albumen in a solution of 0.2 per cent. HCl, we should be justified in concluding that it was pepsin. If it did not digest in HCl, but in a one per cent. solution of sodium carbonate, it would probably be trypsin.

For the demonstration of a diastatic ferment a dilute solution of starch is brought into the incubator with about five c.c. of filtered, sterilized feces. After a few hours the HCl and soda solutions of the boiled albumin are tested for peptone by the biuret reaction, and the diastatic test-tube is tested with a dilute solution of iodin in iodid of potassium. If the starch is unchanged the solution will be changed to blue; if not, the color will be brown or yellow.

In this way we have confirmed the fact that pepsin is absent from the colon contents, but trypsin is at times present, since the

perfectly sterile extract of feces will digest fibrin and albumin in alkaline solution.

The digestive action of the succus entericus, which, according to Grützner, has a weak fibrin-dissolving property, does not extend to the albumins, and therefore it will not confuse the result stated above as pertaining to pepsin and trypsin.

The chief digestive action of succus entericus is on the carbohydrates. If peptone occurs in the stools, it is, in the author's opinion, a product either of pepsin or trypsin digestion, not of bacterial origin. Undoubtedly, there are proteolytic bacteria,—for instance, the bacillus subtilis of Ehrenberg, the proteus vulgaris of Hauser, the bacillus putrificus coli of Bienstock, and the bacillus liquefaciens ilei of Macfadyen, Nencki, and Sieber, all of which exist ordinarily in the human intestine; and their first products of action on albumins are the same as occur in normal pancreatic digestion, viz.: albumoses, peptones, amido-acids, and ammonia; but then the action continues uninterruptedly, ending in the formation of decomposition products stated in a previous paragraph. The bacterial product of peptone is probably of no use to the organism in which it occurs,—it is a first stage to proteid putrefaction, and these proteolytic parasites need peptone for their own existence.

The remote possibility that bacteria could produce peptone in the colon (feces) might be excluded by the fact that after sterilization of the feces by a saturated solution of thymol, peptone will, in some cases, still be produced when the above tests are made. It is due most probably to trypsin, which is present in the stools when they have traversed the intestines rapidly.

Starch-inverting ferments are present in the saliva, pancreatic juice, and succus entericus; hence, if such a ferment appears in the feces, it is impossible to decide upon its source.

Amylopsin and steapsin have not been demonstrated as such in normal feces. It is not known whether pepsin and rennin occur in normal feces. We have found a proteid-dissolving ferment in the stool, which acted in a one per cent. solution of carbonate of sodium only, and was studied, in a case of complete atrophy of the gastric mucosa, with total absence of HCl, pepsin, and rennet, and also of the pro-enzymes of these ferments. In the wash water, bits of mucosa were found that proved the absolute destruction of the glandular apparatus of the stomach.

It is probable that this ferment was trypsin. There was a moderate gastrectasia but otherwise no anatomical defect observable.

The stools were not diarrheic. Escherich's assertion that the colon bacteria do not live upon the food introduced,—as, according to his opinion, there is no digestible food left there under normal conditions,—but that they live upon the secretions of the walls of the colon is certainly erroneous—if this statement of his view is correct—(Mannaberg, in Nothnagel's "Erkrankungen des Darms," p. 38).

The conception of some writers on this subject, that food materials are completely used up in the digestive tube, is not proven by actual fact. Even meat, when eaten in a most digestible form, is found undigested to a small extent in the evacuations. It is therefore more than probable that the colon bacteria live at the expense of the ingested proteid food.

Having thus far reviewed the physiology, anatomy, and, in part, the pathology of food digestion in general, let us now return to the special pathology of the functions of the stomach, as a preparation for a better comprehension of its diseased states.

Qualitative and Quantitative Methods for Testing the Motor Functions of the Stomach.—The motor or peristaltic function is the most important one, because a man may be able to live without the secretory and resorptive functions of his stomach, as the intestinal digestion and secretion would suffice for the conversion of carbohydrates and proteolysis, and he depends upon the small intestine altogether for the digestion and absorption of fats; so that even in the total absence of gastric resorption and the falling away of secretions of HCl, pepsin, and rennin ferments, life could be maintained.

But if the motor function is interfered with, the food would remain in the stomach and accumulate. If a normal gastric juice were even possible when the peristalsis is paralyzed, the food could be only partly digested. Carbohydrates and fats would not be digested. When the limit of distention was reached the food would be ejected (pyloric stenosis; gastrectasia).

In all cases of inhibition or loss of motor power, the secretory power is seriously disturbed or may cease absolutely; so also the resorptive power. Many cases of total absence of gastric secretions have been reported in patients whose body weight continued normal and their general health unimpaired. The stomach has

been removed experimentally in dogs, and the animals continued to thrive without it if precautions were taken in providing finely-divided food.

There have been, up to very recently, six different methods proposed for determining the motor functions of the human stomach: the methods of Leube, Ewald and Sievers, Klemperer, Fleischer, Einhorn, and Hemmeter.

Leube's method of estimating the duration of gastric digestion—*i. e.*, to determine after a definite average time of six to seven hours after a meal of 50 gm. bread, 200 gm. beefsteak, and a glass of water, or two hours after an Ewald test-breakfast, whether solid contents were still to be found in the stomach—will serve the practitioner with a simple and ready method, which follows naturally in the line of drawing test-meals from the stomach; it is, however, subject to too many physiological variations to permit of accurate deductions.

Ewald has proposed the use of salol, which, according to Nencki, is not decomposed by acids in the stomach, but is converted, by the alkaline juices of the duodenum, into salicylic acid and phenol. He found, in connection with Sievers, that the appearance of salicyluric acid—the product of the decomposition of the salol in the urine—would indicate that the salol had actually passed out of the stomach.

Normally, salicyluric acid will appear in the urine from forty to seventy-five minutes after taking one gm. of salol. Delay in its appearance will indicate a retardation in the passage of food into the intestines.

Salicyluric acid is recognized in the urine by the violet color produced on the addition of neutral ferric chlorid solution. This method necessitates frequent urination of the patient—every five minutes, at least; otherwise the result will not be accurate.

Brunner, Riegel, and Eichhorst found that the time in which the reaction occurred in the healthy individual varied from forty minutes to two hours. This was to be anticipated, as the period during which a test-meal may remain in the stomach may vary normally between two and three hours.

CHAPTER VIII.

METHODS FOR TESTING THE GASTRIC PERISTALSIS (Continued).

As Ewald's salol test is not applicable in private practice, because of the frequent micturition that is necessary, it is impossible to examine females by this method; and, also, because the excretion of salicyluric acid depends upon the changing energy of the heart's action, intra-arterial pressure, the amount of water in the blood, and the changeable function of the kidneys themselves.

Huber improved this method somewhat by ascertaining that salicyluric acid disappears from the urine, after the administration of salol to healthy persons, in twenty-four hours; but in patients with impaired gastric peristalsis, the reaction continues to be distinct much longer,—sometimes for forty-eight hours. According to Fleischer and Hecker, the duration of excretion of potassium iodid in the urine of healthy individuals varies from twenty-nine to fifty-five hours; of sodium salicylate, from twenty-one to twenty-nine hours; and in cardiac and nephritic patients this may vary from eighty to ninety-six hours. It is evident that methods of such a variable character are not satisfactory for exact research; nor even, on account of the great loss of time, of much value for comparative tests.

Klemperer's method consists in the introduction of 100 gm. of neutral olive oil into the perfectly clean stomach, after lavage, through a stomach-tube. Oil or fatty acids, which are formed in traces, are not absorbed from the stomach. After two hours, all oil yet remaining is washed out by repeated lavage, dissolved in ether, and weighed after removal of the ether by distillation. In the normal subject Klemperer could find but 20 to 30 gm. of oil; the remaining 70 to 80 gm. had passed into the intestine. If larger amounts are found—for instance, 50 to 60 gm., or more—they are, according to Klemperer, an evidence of motor insufficiency. This method requires much time and skilled chemical analysis, and is also open to the same objection as that of Leube.

Fleischer ("Spez. Path. u. Therap. d. Magen- u. Darmkrankh.," p. 791) has proposed a method to determine the gastric peristalsis by giving 0.1 gr. iodoform in a gelatine capsule during meals; this compound does not decompose in acid media, but does break down in the alkaline juices of the duodenum, and one of its resultants is potassium iodid, which can be tested in saliva by starch paper, which, when dipped into the saliva, colors blue on being touched with a drop of fuming nitric acid. Naturally the potassium iodid can also be detected in the urine; but the fact which gives this method the preference over Ewald's salol test is that the KI can be detected in the saliva.

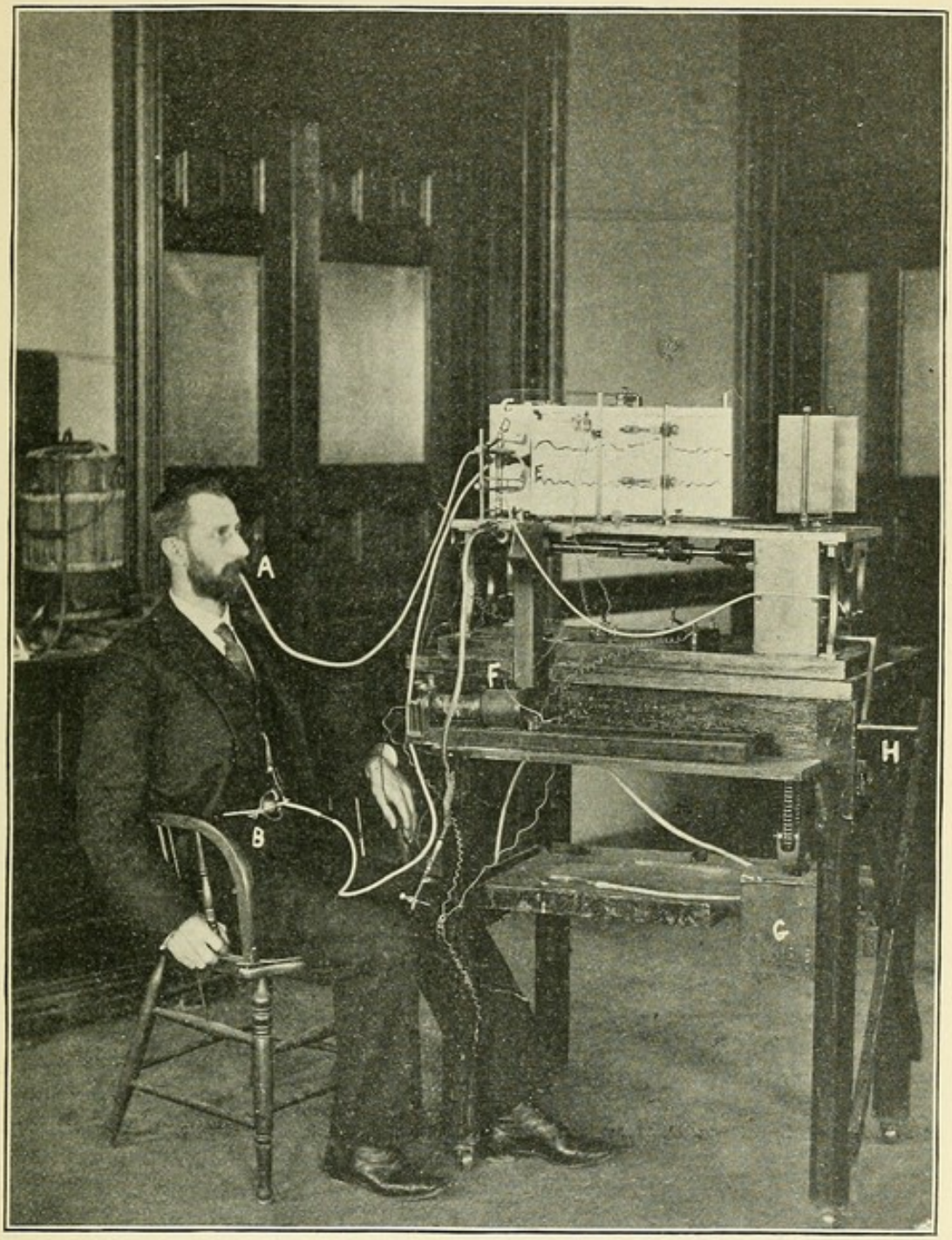
It is quite a variable experiment, as we have discovered. In twenty-three cases in which we have tried it, the reaction coloring the starch paper first occurred just one hour after the meal, in twelve cases; in six cases it occurred first in one hour and twenty to twenty-two minutes; in two cases, in one hour and forty-one minutes; and in one case in two hours. In two cases it took two hours and a half to demonstrate KI in the mouth, after giving 0.1 iodoform.

These were cases in which the gastric secretions were known to be normal. The time of the appearance of the first red and the subsequent blue coloring of the starch paper was carefully noted. Fleischer states that after a test-breakfast the reaction in the saliva should occur in from fifty-five to one hundred and five minutes, which is still a considerable margin for variations—too great for practical purposes.

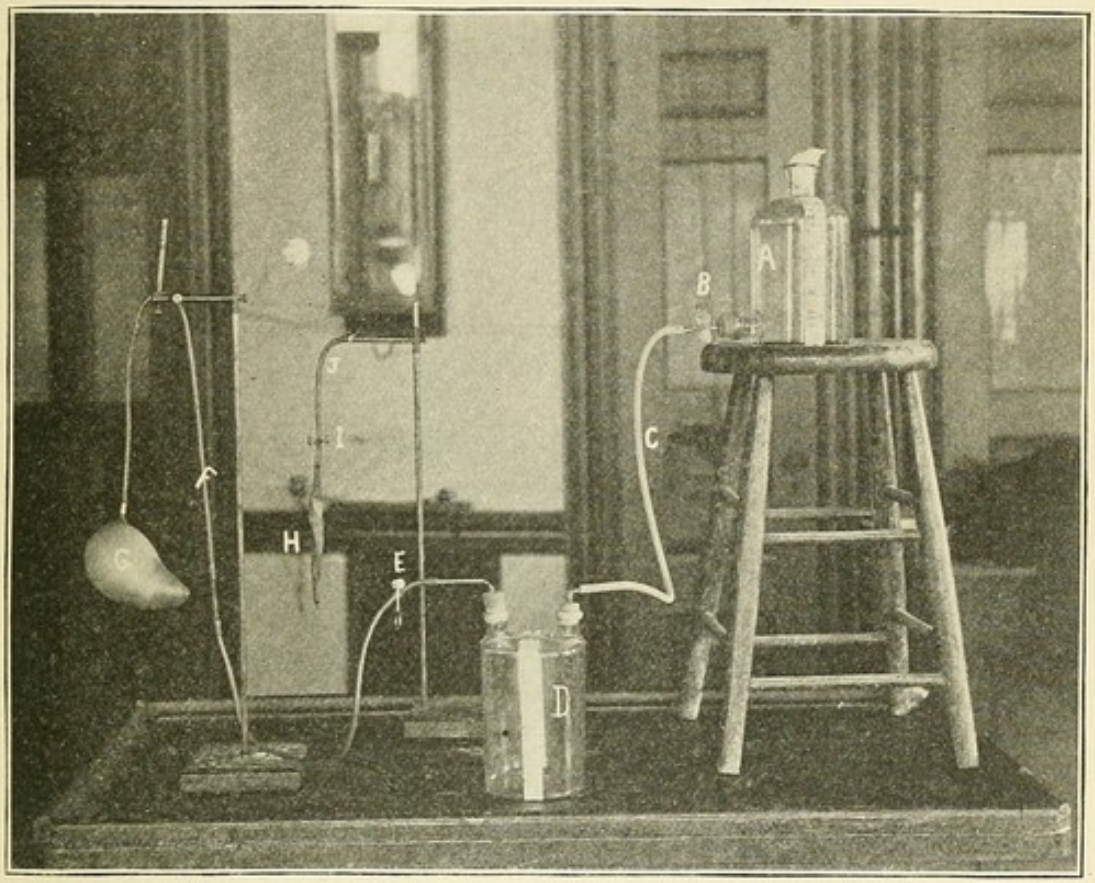
Nevertheless, the method is interesting, and, with exactly known meals, might be available for hospital work.

In Leube's, Ewald's and Sievers', Klemperer's, and Fleischer's methods, it will be observed that the gastric motility was determined by something that was administered (salol, iodoform, and food) or poured into the stomach (oil), and by the absorption of the product of breaking down in the alkaline duodenum, and its subsequent appearance in the secretions and excretions (potassium iodid in the saliva and salicyluric acid in the urine)—an expression in terms of time was arrived at, to denote the intensity of the gastric peristalsis.

In two methods the expression is derived from the quantity of oil or food retained in the stomach after two hours, but here also the result depended upon the passage of something into the duo-



PATIENT WITH INTRAGASTRIC BAG WITHIN STOMACH AND PNEUMOGRAPH IN PLACE, BOTH CONNECTED WITH THE KYMOGRAPH.



The apparatus, not including kymograph. G. Intragastric bag distended. F. The esophageal tube attached to it. H. Intragastric bag collapsed in the shape it is introduced. A. Pressure bottle elevated and filled with water and graduated. B. Stop-cock. D. Lower graduated bottle, empty at first. The bag is distended after it is swallowed by connecting it at E with D; the stop-cock, B, is turned on, and the water then runs from A to D, displacing the air in D and forcing it into the bag. Both bottles being graduated, the amount of air in G is always known and can be utilized as an indication of the gastric capacity.

denum. In all of these methods, therefore, the fundamental idea is the rate of expulsion of gastric contents into the duodenum, as if that were the only object of the motor functions of the stomach.

It is probable that this, which is only a part of the purpose of the gastric peristalsis, was so much dwelt upon because it offered the most expedient means for experimenting, and a greater possibility of solution of the problem. However, a second and most important purpose of the gastric peristalsis, and one concerning which none of the methods referred to thus far can instruct us, is the moving about of the ingesta within the stomach, (1) so that they may be made into a more homogeneous mass, (2) so that they may be brought into thorough contact with the gastric juice, and (3) to stimulate the secretion of this juice by the mechanical irritation of the walls of the organ.

These, in addition to expelling the chyme, are the purposes of the motor function. The secretion of the gastric glands is not only stimulated by the mechanical irritation of the stomach-walls during peristalsis, but by the contraction of a liberal supply of muscular fibers, which arise from the muscularis mucosæ, and are spun around the fundus or bases of the gland tubules (see frontispiece lithograph of normal gastric mucosa); the glands are no doubt themselves contracted and their contents expelled.

In some of the batrachians this contraction of the gastric gland tubules by electric stimulation is visible under the microscope.

Dr. Max Einhorn has described, in the *New York Medical Journal*, September 15, 1894, an instrument which records the gastric movements by dots on a narrow piece of paper.

This apparatus consists of a ball about ⅝ of an inch (14 mm.) in diameter, which is made up of two hollow metallic hemispheres screwed together. Within this is contained a second smaller ball, which is attached to the upper hemisphere by a non-conductor so that it is insulated from it.

The central smaller ball bristles with small metallic spikes which radiate in all directions from the center to the inside of the two hemispheres, but not touching them.

A tiny platinum sphere completes the interior of this apparatus; it lies within the larger round capsule and moves about, knocking at the spikes. When it does so, it completes an electric circuit between the outer hemispheres and the spikes of the central ball,—

for two insulated wires, one connected with the hollow ball, the other with the spiked ball, run up in a very fine, thin, rubber tube and are connected with the two poles of an electric battery. On connecting the ball with another part of the apparatus, the "ticker" (very much like the instrument used at the stock exchanges for reporting the variations in stock by telegraph) each motion of it will be recorded by lines or dots on the paper. The ball is swallowed and brought into the stomach by the aid of a draught of water. It must be borne in mind that the paper records the motions of the ball only; this does not mean that it records every motion of the gastric peristalsis.

In animals upon which we experimented at the biological laboratory of the Johns Hopkins University, a rubber stomach-shaped bag was fitted exactly to the interior of the animal's stomach and connected with a manometer on the Ludwig kymographion. Records were taken with the animal's abdomen intact and compared with those with the abdomen opened, so that the gastric peristalsis could be viewed by the experimenter.

The physiological peristalsis is essentially the same whether the animal's stomach is normally contained within the abdomen or exposed to view.

In our experiments the animals were placed in a large metal case with a glass top; underneath the animal holder about two inches of water was contained in the bottom of the case, which was kept at any desired temperature by a number of Bunsen burners beneath the case. Thermometers were suspended from different parts of the case to keep watch on the temperature, for it is most essential that after an animal's abdomen has been opened it should be kept at a constant temperature by moist steam; this also insures the viscera against becoming dry.

In a similar manner Ludwig and H. Newell Martin studied the physiology of the mammalian heart; Schatz conducted his fundamental investigations on the contractions of the uterus; Engelmann carried on his pioneer work on the contraction of the involuntary muscle-fibers of the ureter. Phlüger and Heidenhain have done similar accurate work on excised organs, and the results have been repeatedly confirmed by other competent investigators.

These epoch-making experimentations are mentioned to emphasize the fact that experiments conducted on organs isolated either

entirely (Martin, Ludwig, Engelmann) or partially (Schatz, Phlüger) are capable of giving perfectly physiological contractions or peristalsis which differ nowise from the perfectly normal ones.

It is frequently urged that these experiments, on account of the operations and the anesthesia necessary, do not present perfectly physiological conditions, and that therefore the deductions made therefrom are not logical, nor represent the true state of normal functioning.

It is undeniable that we never get at the absolutely exact normal functioning of an organ—the stomach, for instance—during an experiment, as ether and chloroform have an inhibiting effect on gastric peristalsis. But we are enabled to produce unconsciousness of the animal after a brief ether narcosis by brain compression, after which the ether is no longer necessary, and then the gastric peristalsis continues perfectly normal. The stomach of the rabbit will show normal peristalsis after complete excision and suspension on a hook or clamp in a warm, moist chamber.

What brought us to the idea of using an intragastric thin rubber bag to record the peristalsis, after many attempts with a small spherical bag that did not exactly fill out the entire lumen of the stomach, was the repeated observation that the small, round bag, such as Professor Moritz, of Munich, used, did not record every peristaltic movement that was visible to the eye when the abdomen was opened.

We frequently noticed peristaltic constrictions of the antrum pyloricum when the rubber bag, of about 12 cm. in diameter, was at the cardia or fundus, and recorded no movement but that due to the pressure caused by the descent of the diaphragm. We concluded, after three months' experimentation, that a small intragastric apparatus could not possibly record every peristaltic movement.

Sometimes one could witness very strong tonic contractions of seemingly every muscle-fiber of the stomach,—it gave that impression,—by which the whole organ contracted from all sides by shortening of every circular, oblique, and longitudinal fiber, and at the same time the bag gave no record of movement, although when it was lying in the fundus it was clearly being lifted up,—it would not record until it was compressed by food or the opposite gastric wall.

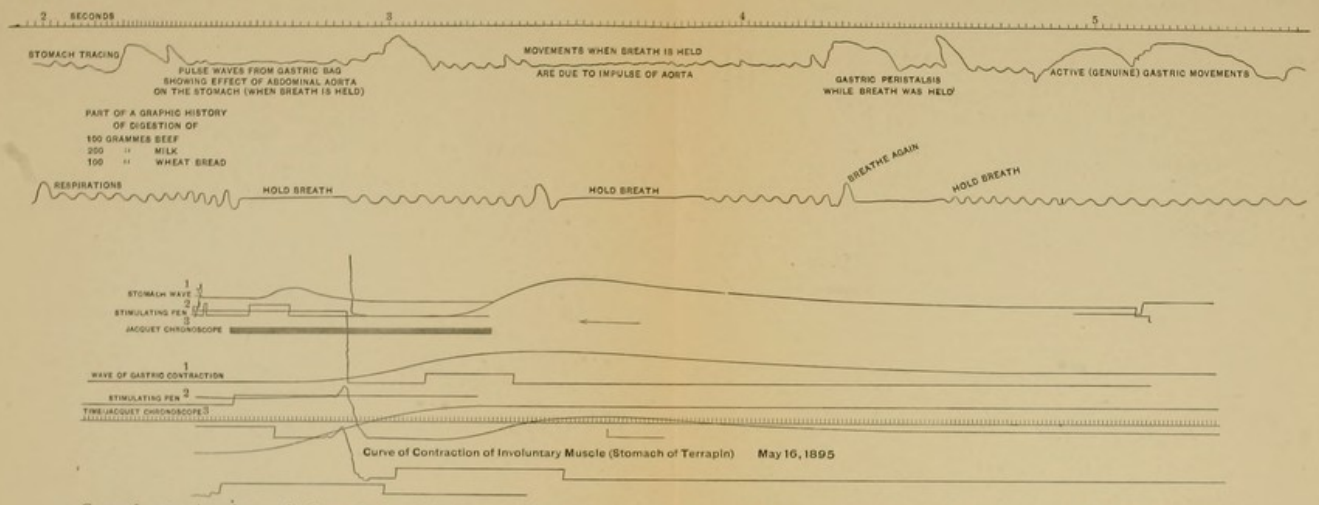
For these reasons a bag was devised which had the exact shape

of the stomach, but could readily be swallowed, and when distended within the organ, exactly adapted itself to its interior, filling every nook and corner in it. If a little food was needed in the organ, we simply did not blow the bag up so far as to fill it out completely.

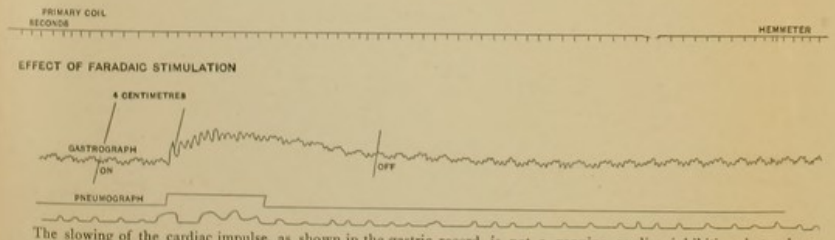
Our apparatus, as has been demonstrated many times on a large variety of cases in the clinical amphitheater of our school, is adjusted with great ease even in patients who are examined for the first time. By a pneumograph the respiration is recorded separately, and thus one is enabled to differentiate the active from the passive movements.

A separate seconds-pen gives on the same paper a record in time, so that the experimenter can tell at a glance the duration, beginning, and end of the peristalsis. While it is the most perfect apparatus yet devised for recording the motor function, it offers a reliable means of ascertaining the size and exact capacity, and finally the intragastric pressure. No apparatus hitherto devised combines these facilities in such a simple bit of mechanism; for, taking away the kymograph, which should be in every medical school, its important parts are simply the intragastric bag and a manometer.

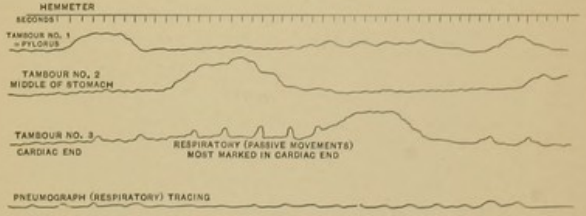
In practice, a manometer connected with the intragastric bag will answer; with watch in hand the experimenter is able to count the peristaltic movements as they are conveyed to the column of water. Einhorn, in his new book, "Diseases of the Stomach," page 96, has gathered the impression that the apparatus is of difficult adjustment, because in our first report (*loc. cit.*) we stated that only such patients are taken as have become accustomed to the stomach-tube, as the nausea and vomiting first attending the initial introduction of the tube make an *exact* record impossible (we lay great stress here on the word *exact*). No intragastric instrument, not even Einhorn's electrode, can be introduced the first time without some nausea; while this may not lead to emesis, it nevertheless has a great influence on the number of gastric movements, as all cases we have tried generally show more contractions in the first experiment than in any other. If the record is to be exact and free from objections that may be urged on account of the influence of nervousness, nausea, suggestion, etc., a certain adaptation and experience of the patient is indispensable, no matter what instrument is used. Probably none of these apparatuses will be regularly used in practice; they are implements for the trained specialists, who know



Curve of contraction of stomach of terrapin, in which slow stimulations—*i. e.*, twenty per second—are more effective than rapid stimulation. The number of stimulations can be so increased per second that the muscle will not contract at all.



The slowing of the cardiac impulse, as shown in the gastric record, is not a genuine cardiac inhibition, but only an apparent one, due to the fact that the stomach draws away from the diaphragm and aorta during violent contractions, and does not receive every impulse; the radial pulse during this period was undisturbed and regular. Distance of secondary coil from primary coil, four cm. on the sliding apparatus.



Hemmeter's triple intragastric bag. Kymographic record of pyloric (No. 1), middle portion (No. 2), and cardiac end of stomach (No. 3) in successive peristalsis.

how to apply them and how to interpret their results. Nevertheless, our intragastric bags are used regularly at the Maryland General Hospital and exact results obtained thereby, even at the first experiment.

Our objections to the Einhorn gastrograph are: (1) That no differentiation between active and passive movements is possible thereby; (2) that there is no coincident record of time in seconds on the paper; (3) that the tonicity or intensity of a contraction can not be adequately determined; (4) that the slow but very extensive general tonic contractions—a narrowing down, as it were, of the entire stomach to one point in the center—will probably be recorded by a single dot, such as would be made by an inspiration also. At the same time, when we reflect that a bag 12 cm. in diameter may miss some of the contractions and fail to record them, it is difficult to imagine that the gastrograph should record them all, being not even an inch in diameter.

Nevertheless, Einhorn's apparatus marks an epoch in the history of the study of stomach motions and their physiology. It is the first attempt, and largely a successful one, to obtain their record by mechanical means.

Passive motions caused by the pulsations of the aorta and the impulse of the heart ventricles against that part of the saccus cœcus cardiæ which touches the arch of the diaphragm, and also the respiratory passive motions due mostly to the muscles of respiration, are, to a small extent, participants in the causes of gastric movements; but they can not of themselves produce evacuations of the contents, as we had occasion to observe in the clinic on a hysterical girl (R. H.), who had no active stomach movements, no genuine peristalsis at all, all of her gastric movements being due to respiration and circulation.

This girl showed a normal state of the secretions after an Ewald test-meal, but at the same time there was stagnation and over-retention of food. It is, therefore, most essential to be able to distinguish between active and passive movements, for a person may have a great many movements of the stomach and yet have no genuine peristalsis at all.

It is necessary to distinguish between methods for physiological study of gastric peristalsis and methods for diagnostic or clinical work. Our method is available mainly for the physiological laboratory.

CHAPTER IX.

HEMMETER'S METHOD FOR TESTING THE GASTRIC PERISTALSIS.

Theories Concerning the Movements of the Ingesta.

One of the intragastric stomach-shaped rubber bags which are used in our clinic consists of three separated compartments: one filling out the pylorus, the second filling out the middle portion of the stomach (the fundus), the third occupying a small part of the fundus and the saccus cœcus cardiæ. (See *N. Y. Med. Jour.*, Sat., June 22, 1895, p. 772, and the accompanying illustration.) Each one of these compartments records on the kymograph by a separate tambour.

In the report referred to we made the assertion, from the results obtained with this bag, that in the human being most, if not all, of the peristaltic waves are executed by and start at the pyloric end. This statement was made before Moritz's investigations were published in the *Zeitschrift für Biologie*, proving that the cardiac end and the fundus of the stomach could not contract, even when stimulated by powerful faradic currents on both the mucous and peritoneal surfaces.

One week before our results were published in the *New York Medical Journal*, Dr. S. J. Meltzer, of New York, published his results with direct and indirect faradization of the digestive canal, which demonstrated quite conclusively that the mucous membrane of the digestive canal offers a considerable resistance to the penetration of the faradic current to the muscular coat, the greatest resistance being found in the mucous membrane of the stomach. Percutaneous and direct faradization of the stomach or intestines can not produce any contraction in these parts.

Meltzer stated explicitly the kind of instruments used,—the sliding inductorium (Schlittenapparat) of DuBois Reymond, a Grove's cell prepared anew for each experiment,—and also the distance, in every case, of the primary from the secondary coil. His device of including the sciatic nerve of an animal (nerve-

muscle preparation of hind leg of frog, most likely) in the circuit is practical, and has for a long time been used in our laboratory.

There is, however, a very important matter which physiologists must insist on knowing, and which Meltzer does not state, perhaps because it was not very readily found out; and that is, the

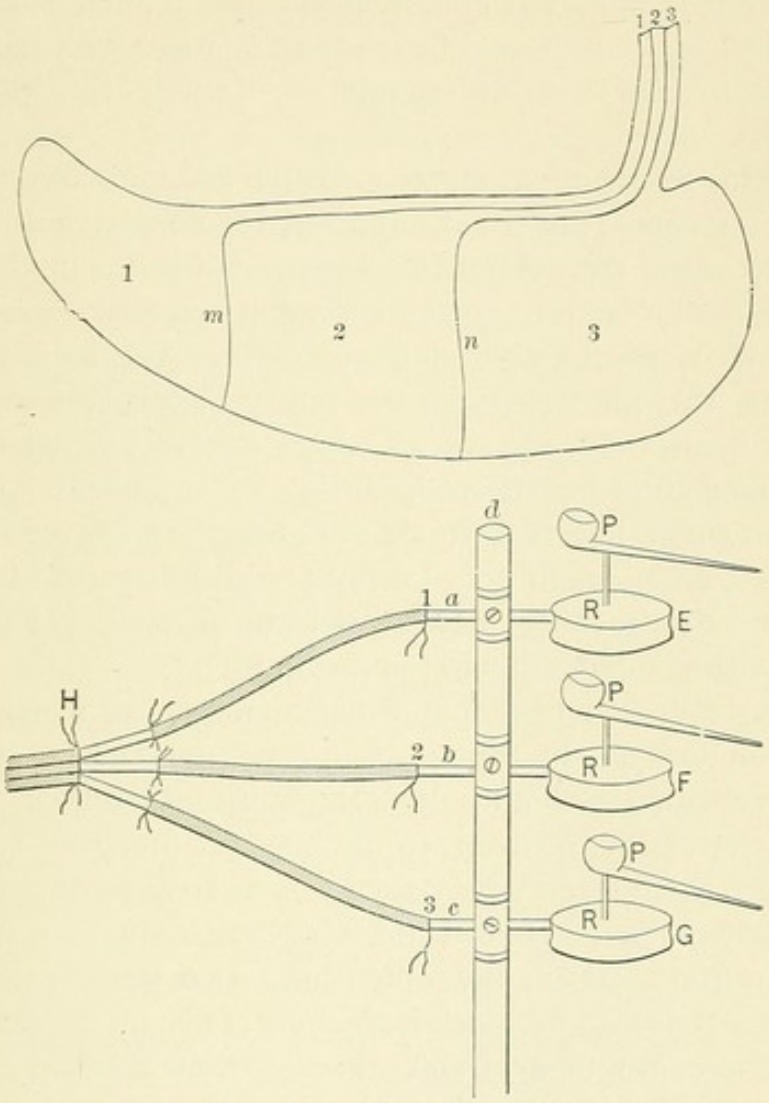


FIG. 7.—INTRAGASTRIC TISSUE RUBBER BAG, with three distinct parts and three separate outlets for recording the origin and direction of gastric peristalsis. Outside of the mouth the triple tube separates into its three component tubes, each being connected with a separate tambour and glass ink pen, writing the gastric contractions and relaxations on the kymograph. Part No. 1 records the contraction of the pylorus; part No. 2, the middle of the stomach; and part No. 3, of the cardiac end.

number of stimulations to the second used by him. Involuntary muscle-fibers are much slower to contract than voluntary muscles, and in electrical stimulation experiments they contract much more readily when the number of stimulations does not exceed 240 per

minute. The best contractions are obtained at a much lower rate of stimulation.

The vibrator on the DuBois Reymond inductorium was found, after months of experimentation, to send too many stimulations into the gastric muscle per second. Later on, when we used the Kronecker interrupter, in connection with a Jacquet chronograph, and no more than 100 stimulations per minute, it was found that the preantral sphincter could be made to contract with the distance of primary from the secondary coil, = 0, and both electrodes on the mucosa.

To get this result, it is best to make the animal starve for twelve hours, as for some reason yet unknown the contractions are more unlikely to occur the sooner the experiment is made after the ingestion of food. Still, it must be emphasized that the mucosa of the stomach is practically a non-conductor. We had occasion to try this in the physiological laboratory recently, with a bit of healthy human stomach-mucosa which one of our students tore off from the wall of his stomach during experimental lavage; the piece was 15 mm. long, five to six mm. broad, and two to four mm. thick. The gentleman in question, after trying to wash his stomach out, and not succeeding to his satisfaction, connected the end of the tube with a suction apparatus (aspirator).

This was followed by copious hematemesis, for which we were hastily summoned. In the stomach-tube, partly projecting from the lower opening, was a bit of fleshy substance, which, on microscopic examination, proved to be gastric mucosa. After the hemorrhage ceased, the young man was treated for one week as if he had gastric ulcer. He did not experience any pain during the accident, nor thereafter; the only thing that frightened him was the blood. He made a good recovery. This bit of mucosa was placed in a continuous circuit generated by a battery of three freshly prepared Grove's cells, with a milliampèremeter, soon after it was found; the meter showed but three milliampères. As it was impossible to get this fresh piece of mucosa into the circuit perfectly dry, it is probable that the indication of three milliampères was brought about through the conducting agency of the moisture on the outside of the tissue.

In the biological laboratory of the Johns Hopkins University we have frequently had persons' stomachs connected with the kymograph, and an intragastric rubber bag blown up to fill out

their stomachs exactly. Through the intragastric bag ran two insulated wires, one ending in a small brass knob near the pylorus, the other coming out against the mucosa near the cardia in a similar knob.

Every active and passive motion was recorded by a manometer pen (*N. Y. Med. Jour.*, June 22, 1895, p. 771). But the strongest faradic currents (distance of primary from secondary coil = 0) could produce no contractions of the stomach.

Dr. George P. Dreyer and myself held one of the poles in the right hand—the plus, for instance—while the negative was in the stomach; with the left hand we touched the back of the person's neck. The current was so strong that it became intolerable to us. Although this current made its circuit through the patient's stomach, it caused no contraction, as was evidenced by the manometer in connection with the intragastric bag.

Frequently we could observe contractions of any skeletal muscle upon which the outer electrode was placed,—for instance, the gastrocnemius,—and still the stomach did not contract. This proves that in some conditions the gastric mucosa may transmit a current, yet the muscular layer give no evidence of contractions. We do not wish to imply that it is absolutely impossible to contract the human stomach by electrical stimulation; but the current required to effect this must be so strong that the experiment becomes hazardous.

Einhorn ("Diseases of the Stomach," pages 78–83) and Paul Cohnheim (*Archiv f. Verdauungskrankheiten*, Bd. 1, S. 274) have described tiny bits of mucosa which are found in the wash-water and vomit of many gastric sufferers. We can confirm this observation, and add that we have found these pieces of gastric mucosa on washing out the stomachs of perfectly healthy persons.

Now, it has occurred to us that in rare instances in which a good contraction of the stomach was obtained, it was due to the fact that the current found its way to the muscular layer, through spots from which the glandular layer had been cast off. It must not be omitted that all stomachs experimented upon by our method in this series were washed out prior to the experiment to insure absence of current-interrupting food-particles in the organ.

Professor Moritz, of the University of Munich, experimented with an apparatus very similar to ours, except that his rubber intragastric bag was round, not stomach-shaped. It did not, therefore, exactly and completely fill out the organ, nor did he use the

graduated pressure bottles, by which it is possible to determine exactly how much air is blown into the bag. Instead of a pneumograph, he used a cork in one nostril of the patient, which was connected with a second manometer, writing on the Ludwig kymograph. The advantage of the pneumograph over this method must be apparent to every one.

The author's first results appeared in print three months before those of Moritz in the *Zeitschrift für Biologie*, Bd. XXXII, which are perhaps the most important contributions to the physiology of the motor function since the investigations of Hofmeister and Schütz (*Archiv f. exper. Pathol. und Pharm.*, 1886, Bd. XX). In order that the mechanism of the gastric peristalsis may be better understood, it is well to bear in mind the arrangement of the muscular layers,—(1) longitudinal, (2) oblique, and (3) circular,—and what was said under the head of anatomy of the gastric layers and the formation of the sphincter of the pylorus. The part of the stomach near the pyloric end is spoken of more specifically as the antrum pylori.

The line of separation between the antrum pylori and the body or fundus of the stomach is made by a special thickening of the circular fibers, forming what is spoken of as the transverse band by older writers,—for instance, Beaumont, in his "Physiology of Digestion," second edition, 1847, page 104. (A pioneer piece of work, very fundamental and thorough in its observations, this book remains a monument to American physiological investigation.) Recent observers describe this transverse band as the sphincter antri pylorici, and locate it at a distance of seven to ten cm. from the pylorus.

In the antrum pylori there is a very strong musculature, and its glands contain only (or rather mostly) chief, central, or ferment cells. The exact character of the gastric movements during digestion were first carefully studied on the human being by Beaumont; his facts and errors have influenced physiologists more or less up to the present time. One can not fail to suspect that the stomach of Alexis St. Martin and its manner of peristalsis were too far from the normal to permit absolutely correct conclusions. The extensive adhesions which Beaumont describes certainly acted at times as irritants, at others as impediments, to normal peristalsis.

Professor W. H. Howell's views on the gastric movements, as expressed in his new "American Text-book of Physiology," page

317, will serve as an expression of a modern specialist in this branch. He says (*loc. cit.*) the movements occur in two phases: "first, the feeble peristaltic movement running over the fundus, chiefly on the side of the great curvature, and resulting in pushing the fundic contents into the antrum; secondly, the sharp contraction of the sphincter antri pylorici, followed by a similar contraction of the entire musculature of the antrum, both circular and longitudinal, the effect of which is to squeeze some of the contents into the duodenum."

"It is possible that either of these phases, especially the first, might occur at times without the other, and in the first phase it is possible that the longitudinal fibers of the stomach also contract, shortening the organ in its long diameter, and aiding the propulsive movement, but actual observation of this factor has not been successfully made. It can well be understood that a series of these movements occurring at short intervals would result in putting the entire semi-liquid contents of the stomach into constant circulation."

"The precise direction of the current set up is not agreed upon, while it is probable that the graphic description given by Beaumont is substantially accurate. A portion of this description may be quoted as follows: The ordinary course and direction of the revolutions of food are, first, after passing the esophageal ring, from right to left, along the small arch; thence, through the large curvature, from left to right. The bolus, as it enters the cardia, turns to the left, passes the aperture, descends into the splenic extremity, and follows the great curvature into the pyloric end; it then returns in the course of the small curvature."

"The average time taken for one of these complete revolutions, according to observations made by Beaumont, seems to vary from one to three minutes."

"It is possible, of course, that this typical circuit taken by food may often be varied, more or less, by different conditions, but the muscular movements observed from the outside would seem to be adapted to keeping up a general revolution of the kind described. The general result upon the food may be easily imagined. It becomes thoroughly mixed with the gastric juice and any liquid which may have been swallowed, and is gradually disintegrated, dissolved and more or less completely digested, so far as the proteid and albuminoid constituents are concerned."

"The mixing actions are aided, moreover, by the movements of

the diaphragm in respiration, since at each descent it presses upon the stomach. The powerful muscular contractions of the antrum serve also to triturate the softened solid particles, and finally the whole mass is reduced to a liquid or semi-liquid condition, in which it is known as chyme, and in this condition the rhythmic contraction of the muscles of the antrum eject it into the duodenum."

"The rhythmic spurting of the contents of the stomach into the duodenum has been noticed by a number of observers, through duodenal fistulæ in dogs, established just beyond the pylorus. It has been shown, also, that when the food is entirely liquid—water, for example—the stomach is emptied in a surprisingly short time, —within twenty or thirty minutes; if, however, the water is taken with solid food, then, naturally, the time it will remain in the stomach may be much lengthened."

Brinton ("Diseases of the Stomach") advanced the view, which differs from Beaumont's, in assuming a central current of the food, moving from the pylorus to the cardia through the central long axis of the stomach. There are, according to this author, two currents, one along each curvature running from the cardia to the pylorus, meeting and turning inward toward the center of the stomach in front of the pylorus, and then running back toward the esophagus as a single central current, there dividing to make again two currents as before, one along each curvature.

According to Poensgen ("Die motor. Verricht. des menschl. Magens," Strassburg, S. 82), Reymond, Donders, and Lesshaft approved of this theory; while Penzoldt and Foster accept the great food-circle of Beaumont.

Although we have made over fifty experiments on dogs, cats, and rabbits to observe a food-circulation within the stomach corresponding to these views, and although we have had an opportunity of seeing into the human stomach, through fistulæ, during digestion, we have not been able to confirm, by actual observation, either Beaumont's or Brinton's views. While we have no new explanations to offer, it has occurred to us that the piston-like backward and forward movements of the food caused by the antral contractions, and especially of the sphincter of the antrum, is a sufficient force to effect the mixture of the chyme with HCl and the ferments such as is found in it when it leaves through the pylorus.

The views of Beaumont and Brinton date from the epoch when it was considered all-important that food must be properly digested

and macerated in the stomach; it was not conceivable then that by far the main bulk of digestion is carried on in the intestines. Hence the complicated theories of Beaumont and Brinton, of circular movements of food, owe their origin to the thought that such a movement was necessary to mix the ingesta with the gastric juice. In dogs this mixture is not proven to occur in every instance. In herbivora (horse, cow), the center of the food mass in the stomach may be alkaline or neutral in animals killed one hour and a quarter after feeding.

The almost vertical position of the stomach was unknown to Beaumont and Brinton. Like many clinicians of the day, they believed the organ was normally in a horizontal position, transversely across the upper part of the abdomen. The amount of force required to lift the food-mass in a vertical line upward is considerable; it is necessary to imagine a still greater force to accomplish the vertical ascent on the side of the lesser curvature, in order to conceive of a simultaneous descent on the side of the great curvature, which descending current must inevitably interfere more or less with the ascending one.

In a number of experiments in which the stomachs of animals on opening the abdomen were found in active motion, we inserted long needles through the gastric walls to determine the direction they would assume under the pressure of the ingesta; according to Beaumont, the ingesta moving from the saccus cœcus along the greater curvature to the pylorus should compel the points of needles to be directed toward the pylorus when run through the greater curvature, and along the lesser curvature they should point toward the cardia.

If Brinton's theory were true, the points of the needles at both curvatures should, at least during a large period of gastric digestion, be directed toward the pyloric end. If needles are inserted to a distance of ½ of an inch along both curvatures during active gastric peristalsis, a great diversity of movements of the outside portions of the needles is observable. They very rarely point the same way along either curvature, and one portion of them may point toward the cardia, while another points to the pylorus. Only when the needles are inserted very deep, so that they dip into the central or axial stream, can one occasionally observe what appears as concerted action.

During active peristalsis, when the pre-antral sphincter at times

contracts so powerfully as almost to obliterate the lumen, those needles inserted into the fundic portion of both the greater and lesser curvatures are strongly turned toward the cardia, but simultaneously those few needles in the antral and pyloric portions are turned toward the duodenum. The same evidence of a central or axial current, which indicates the pumping work of the muscular antrum in pushing back solid particles into the fundus, and squeezing liquid and semi-liquid portions into the duodenum, can be obtained by the intragastric electric lamp when introduced during the height of gastric digestion. These lamps can be seen through the abdominal wall in dogs whose abdomens have been shaven, when introduced in a dark room, though naturally not quite so distinct as when the abdomen is opened.

Once we observed this axial food-current at the clinic in a female patient with very thin abdominal parietes, when the Einhorn intragastric lamp was introduced one hour after a meal. In animals with abdomen opened we have been able to see this lamp carried along the entire greater curvature, from the pylorus toward the cardia, during active digestion, but the occurrence is so rare as to appear accidental.

That the retrogressive current, which is set up by contractions of the antrum forcing the too solid food-particles back toward the fundus, must inevitably set up some new movements among the remaining food-mass in the fundic end is natural, but we doubt whether it ever reaches that systematic circulation described first by Beaumont and Brinton.

It should not be overlooked that if the observations of Beaumont of a complete food-circuit were really true and constituted the only movements in addition to the duodenal extrusion which the food-mass underwent, there must always be a mass of food in the center of the stomach which never touches the gastric wall ; if all the food moves about along the periphery, there must be a central, quiet portion.

Brinton was aware of this defect in Beaumont's statements, and improved upon them by his still more complicated theory of piston movements to explain the axial food-motions.

If the conditions described by these authors exist, they are not well explained by the arrangement of the muscularis of the fundus, which, as far as the work of Meltzer (*loc. cit.*), Moritz (*loc. cit.*), and Goldschmidt (*loc. cit.*) show, is very feeble indeed in its contrac-

tions and hardly sufficient to propel food in any direction; yet, according to the above theory, much work is ascribed to it, but as the pre-antral sphincter is only seven to eight cm. from the pylorus it certainly can not be made accountable for the movements all around the cardia and the saccus cœcus.

The musculature of the fundic end has never been observed in peristaltic motion by us, excepting the peristalsis occasionally arising from the antrum and traveling upward over it. During active peristalsis it is in a condition of tonic contraction with the intragastric bag in the fundus; we have estimated this to be equal to six to eight cm. of water (water manometer).

Moritz, in his work on "The Motor Function of the Stomach," studiously avoids referring to any systematic food-circulation within the organ. It seems rational that sufficient churning and mixing is effected by the powerful contractions of the antrum during the general tonus of the fundus to explain the saturation and softening of the ingesta by gastric juice.

The contrasting relations of the fundus and antrum regarding active peristalsis are evident in the degree of pressure, as observed on a water manometer in connection with our triple intragastric bag. In the fundus the pressure is, on an average, equal to three to six cm. of water. The increase of intragastric pressure due to cardiac action is equal to one to two cm. (In this is included the pressure due to every new heart impulse and aortic impulse.) The inspiratory increase of pressure is equal to 6 to 12 cm. These are very nearly the figures Professor Moritz obtained, and we add them here as merely in support, and confirmatory, of his views.

The physiology of the motor function has been dwelt upon more extensively than seems necessary in a condensed statement of gastric pathology, not only because it is the most important office of the stomach, but because we have become convinced that, in a large majority of disorders of secretion and absorption,—not all,—an abnormality in the motor function lies at the foundation.

The exaggerated or diminished peristalsis can on careful examination be detected sometimes before the secretory and absorptive anomalies are apparent. The secretory disturbances observed after double vagotomy (section of both vagi) are due, according to Contejean, to the motor paralysis caused at the same time (*Archiv. de Physiologie*, vol. IV, p. 640). A similar view is held by H. Borutteau (*Phlüger's Archiv*, Bd. LXV, p. 26).

The relation between motility and secretion and absorption is not at all well understood; the peristaltic movements effecting a churning motion are those mostly concerned in stimulating secretion; when these movements are lost, secretion is generally lost also.

The last vestige of peristalsis left is that by which the stomach is emptied, and it may be present with total absence of secretion. In stomachs with motility much impaired and secretion arrested, the absorptive function is greatly reduced (atrophic gastritis, carcinoma). In temporary arrest of these functions, the secretive and absorptive functions generally return with improved motility.

In our drawing (frontispiece), the manner in which the deep ends of the fundus glands are encircled by fibers from the muscularis mucosæ is very evident. From this it is conceivable that the function of the gland-cells is in a manner dependent upon the contractility of the fibers of the muscularis mucosæ, which can not fail to influence the blood-supply to these cells (see Mall, on "Circulation in the Dog's Stomach," chap. 1).

CHAPTER X.

ABSORPTION FROM THE STOMACH.

Penzoldt's and Faber's, Herschel's, Julius Miller's, and Hemmeter's Tests for Gastric Resorption.

The method most commonly employed to test gastric resorption is that of Penzoldt and Faber, consisting of three to five gr. of iodid of potassium inclosed in a gelatin capsule, which is administered with 100 c.c.—3½ ounces—of water. Iodid of sodium or potassium, when taken internally, will appear, and can be tested for in the saliva and in the urine, where it is excreted in from six and one-half to fifteen minutes.

The test is generally made by wetting starch paper with the saliva of the patient every two minutes after the KI is taken, and touching the wet spot with fuming nitric acid. The first appearance of a blue color indicates that the iodid has reached the point of excretion, and consequently must have been absorbed. If this reaction occurs later than after fifteen minutes, then the rate of absorption is reduced. This, according to Zweifel ("Resorpt. Verhältnisse d. menschl. Magens," *Deutsch. Arch. f. klin. Med.*, Leipzig, Bd. XXXIX, p. 349, 1886), occurs in gastritis, dilatation, and carcinoma; in gastric ulcer the resorption is said to be normal, or nearly so.

Most authorities (J. Wolff, Zweifel, Sticker, Quetsch) differ very much on this question, but agree on the reduced absorption in carcinoma. If the iodid is given during a meal, the reaction occurs much later.

Herschel ("Indigestion," London, 1895, p. 115) estimates the absorptive power by giving two decigrams of powdered rhubarb, which gives a red color in the urine with liquor potassæ normally in fifteen minutes. Our experience with this method is that frequently the urine is so highly colored in digestive diseases that the red color must be very decided to be recognized,—in addition to which it suffers from the same objection as Penzoldt's and Faber's method. In the first place, Brandl's experiments have

shown that sodium iodid is absorbed to a very slight degree or not at all in dilute solutions.

Not until its solutions reach a concentration of three per cent. or more does its absorption become important. Accordingly, all soluble inorganic salts are practically not absorbed in the stomach, since it can not be supposed that they are normally swallowed in solutions so concentrated as three per cent. Brandl also found that condiments, such as mustard and pepper, and also alcohol, very much facilitated the absorption of sodium iodid. Perhaps these substances act by stimulating the epithelial cells, or by causing a marked hyperemia of the mucosa.

The absorption time does not vary much in the same individual, except when the stomach is full; in this case it is not only prolonged, but is very variable in the same individual. This prolongation, according to Sidney Martin ("Diseases of the Stomach," London, 1895), is probably due to a considerable dilution of the iodid by the stomach contents, and also to the fact that the salivary glands are not so active after a meal as in the fasting condition. One must not overlook the fact in these experiments that it is not only the absorptive activity of the stomach that is being investigated, but also the excretory activity of the salivary glands.

In Zweifel's experiments it is probable, from what we know of the absorption of water in the stomach, through the observations of Tappeiner ("Ueber Resorption im Magen," *Zeitschr. f. Biol.*, München, Bd. XVI, p. 497, 1881) and von Mering (*loc. cit.*),—that most of the liquid containing the iodid passes rapidly into the duodenum. Therefore we may be testing not only gastric absorption and excretory activity of salivary glands, but also intestinal absorption.

Zweifel concludes (*loc. cit.*) that in all diseases of the stomach there is a prolongation of absorption time, which is greatest in dilatation and carcinoma and least in chronic gastric catarrh, and very slight in ulcer in the later stages; in the early stages of ulcer, however, he claims, the rate of absorption is also prolonged.

It is very evident that no differentiation between catarrh and ulcer is possible according to this method, and thereby one of the main purposes of such investigations—that of aiding in the establishment of a diagnosis—is thwarted.

In view of these defects, which apply equally well to Herschel's, Penzoldt's and Faber's methods of testing absorption, and are

caused mainly by the fact that water is not absorbed from the stomach, and that the varying secretory activity of the salivary glands is a factor influencing absorption time, we have devised a method which is available for experiments on gastric absorption in the physiological laboratory, and which we have successfully tried on six male and eight female patients and ten healthy students. The methods of testing the urine and saliva were discarded entirely.

Our method consists in washing out the stomach thoroughly; then, by means of our method of duodenal intubation, the entrance into the duodenum was plugged, or closed up, by introducing a small rubber baloon into it and blowing it up just in front of or beyond the pylorus. (This method has been described after the author's publication by Dr. F. Kuhn in the *Münchener Medizin. Wochenschr.*, Nos. 27, 28, and 29, 1896, but his method is founded upon a different principle from ours—the spiral electrode.)

After thus mechanically closing the pylorus, a weighed amount of any harmless inorganic salt,—sodium chlorid or sodium phosphate,—dissolved in 100 c.c. of distilled water so as to make a three per cent. solution, is poured into the organ through a tube; this is indispensable to exclude loss of the salt solution through clinging to the tongue, mouth, and esophagus, or absorption from these tissues.

After a lapse of ten minutes the fluid is again drawn out of the stomach by aspiration, or even, if necessary, by adding known quantities of distilled water, until the last washing gives no indication of containing any trace of the salt by a proper chemical test. (In case NaCl—sodium chlorid, or common cooking salt—has been used, a weak solution of nitrate of silver ($AgNO_3$) can be employed to assure oneself that this last washwater contains no more NaCl. The normal HCl is not secreted so rapidly as to cause confusion in the result.) This entire water is now evaporated to dryness and the residue weighed. The difference between the amount of NaCl poured into the stomach—which in a three per cent. solution is three gm. in case 100 c.c. are used—and the amount regained indicates the degree of gastric absorption.

To simplify matters, the practical suggestion of Julius Miller (Boas' *Archiv. f. Verdauungskrankh.*, Bd. 1, p. 237, "Zur Kennt. d. Sek. u. Resorpt. im menschl. Magen") has been utilized and can be recommended. It consists in noting the specific gravity of salt solutions before pouring them through the tube, and, after

any desired time, the solutions are washed out or aspirated, and the specific gravity again determined.

The difference between these specific gravities taken before the salt solution enters the stomach, and after it is regained, affords a satisfactory index of the rate of absorption from the stomach if escape of the solution into the duodenum is prevented. It is not necessary to evaporate the whole solution to dryness in case sodium chlorid or any other harmless neutral salt is used. But after measuring the total quantity of liquid regained—say, for instance, it amounts to one liter (1000 c.c.)—the amount of NaCl in ten c.c. can be determined by evaporation in platinum, and the weight of the total remaining NaCl calculated by multiplying the result by 100, or whatever the figure may happen to be.

This method of determining the rate of gastric absorption gives approximately accurate results, even without duodenal intubation and mechanical closing of the pylorus, provided that by several preliminary experiments the motility of the patient's stomach has been relatively determined.

By observing how much of 500 c.c. of water he will pass into the duodenum in, say, ten to twenty minutes, this also requiring the drawing out again of what is left of the 500 c.c. of water that were taken, von Mering (*loc. cit.*) found that of 500 c.c. of water given through the mouth, to a large dog, the entire amount, or at least 495 c.c., had been passed out of the stomach through a duodenal fistula within twenty-five minutes.

In the human being the passage of water out of the stomach is not nearly so rapid. Julius Miller (*loc. cit.*) found that the human stomach was not even rid of 200 c.c. NaCl solution of the specific gravity 1028 in thirty minutes; after this time he regained in one case 75 c.c.; sometimes he regained more liquid than he poured in.

In 30 tabulated measurements which he gives with sodium chlorid solution (p. 240, *loc. cit.*), he regained more than he poured in, five times, the same amount once, and a less quantity 25 times. But his figures go to prove that even with an open passage into the duodenum comparatively small amounts of salt solutions are passed out in fifteen minutes.

Hence, if in any individual the average amount passing into the duodenum in fifteen minutes is known by previous experiments, the closing of the pylorus is not necessary to reach an approximate result concerning the rate of absorption. Miller confirms von

Mering's conclusions, that, contemporaneous with absorption, a secretion of water occurs into the stomach.

This secretion increases with the concentration of the solutions. In the five instances mentioned where more was regained than was poured in, the specific gravities, which are a good indication of concentration, were 1066, 1061, 1052, 1088, and 1035. (Regarding the taste of three per cent. solution of NaCl, it might be explained that this is the percentage of salt in the water of the Atlantic Ocean, which has been recommended for internal use—A. Levertin, "Hygieina," XLVII, XLVIII. Svenska läkaresällsk Förh. S. 138, 1885.)

In the studies with occlusion of the pylorus we experimented also with known solutions of sodium sulphate, peptone, maltose, cane sugar, milk sugar, and alcohol. As water is poured out on the surface of the mucosa, in return for salts absorbed, the specific gravity will not always instruct us as to the contents of NaCl, which had best be arrived at by weighing.

From experiments on animals it is known that a concentrated solution may cause the stomach to secrete water, thereby diluting it, but that at the same time it is possible that there may be no resorption, so that weighing the residue from evaporating the liquid regained may be unavoidable for a correct result.

Maltose was found a very practical substance for absorption experiments, though dextrose will also answer this purpose, as their quantity can be readily determined in solution by titration with Fehling's solution, and also by the fermentation test, for which the Einhorn saccharimeter is most serviceable. Maltose will not reduce as much Fehling's solution as dextrose, the exact relation between the two being, according to Brown and Heron, for maltose, 60.8; for dextrose, 100.

According to Soxhlet one c.c. Fehling's solution corresponds to 7.78 milligrams = maltose in one per cent. solution (provided the Fehling's test was not diluted). Though maltose is converted into dextrose in the stomach, the amount converted in ten to fifteen minutes is, according to our observations, small enough to be disregarded. If desired, a test by Barfoed's reagent may be made to detect if any dextrose is present in the liquid regained.

The amount of sodium chlorid in the solution regained can also be determined by titration (Salkowsky u. Leube, "Die Lehre vom Harn"; also, Neubauer u. Vogel, "Analysen d. Urins"). The

method is given in the laboratory manual of my assistant, Dr. Edward L. Whitney ("An Introduction into the Laboratory Methods of Clinical Pathology," p. 18, Baltimore, 1896). Our method for absorption testing is, in brief, the following:

To determine the amount of 500 c.c. of a three per cent. NaCl solution passed into the duodenum in ten minutes:

1. Allow 500 c.c. three per cent. NaCl solution to run into a clean stomach through a tube and remain ten minutes.

2. Draw out as much as possible, washing out the last with known quantities of distilled water.

3. Determine the amount of NaCl as stated above, and add the average deficit of escape into the duodenum.

The difference between the original amount NaCl and the amount regained is a fairly accurate index of gastric absorptive power; or, by our method of duodenal intubation, occlude the pylorus by blowing up a balloon in front of or beyond it; pour into the stomach through a tube a known quantity, say, 100 c.c., of a one per cent. solution of maltose; in ten to twenty minutes aspirate or wash out the amount of maltose left as above. The deficit will indicate the amount absorbed.

CHAPTER XI.

METHODS FOR DETERMINING THE LOCATION, SIZE, AND CAPACITY OF THE STOMACH.

Percussion and Palpation.—Gastrodiaphany of Einhorn.

Distention very much facilitates percussion and palpation of the stomach.

Percussion of the stomach gives varying results, according to its contents and to the degree of its distention. The fundus is closely applied to the concavity of the diaphragm, and five-sixths of its volume is to the left of its median line; only one-sixth to the right (observe the accompanying illustrations from Eichhorst's "Klin. Untersuchungs-Methoden"). The highest point is the fundus, which reaches the level of the ninth thoracic vertebra. The lesser curvature runs along the left of the spinal column, and crosses to the right at the level of the first lumbar vetebra. The lesser curvature is entirely covered by the liver, and can only be percussed or palpated when it is located lower than normal. The pylorus is covered by the right lobe of the liver, about three to four cm. from the median line; it is seven cm. lower than the cardia. The pars pylorica (antrum pylori) extends further to the right than the pylorus itself. The greater curvature in its upper part is largely covered by the lung; its lower and anterior part is in apposition with the left hypochondrium and epigastrium. When the stomach is full the greater curvature is two to four cm. above the umbilicus. To the right of the median line it ascends along the median edge of the gall-bladder, and is continued into the pyloric part. (See illustration.)

The conviction has been forced upon us that the degree to which the stomach can be distended is a very limited one. This statement is made after many distentions with the intragastric stomach-shaped bag. Most stomachs that are in a normal state will refuse to be distended more than 100 c.c. beyond their natural capacity. Only in pathological thinning of the gastric walls and in atrophy

of the muscularis is an over-distention conceivable; even then some of the gases will escape by the cardia before painful distention will ensue.

For these reasons distention with air or carbon dioxid is an expedient and safe way of determining the form and location of the stomach, and its relation to any tumors that may be present.

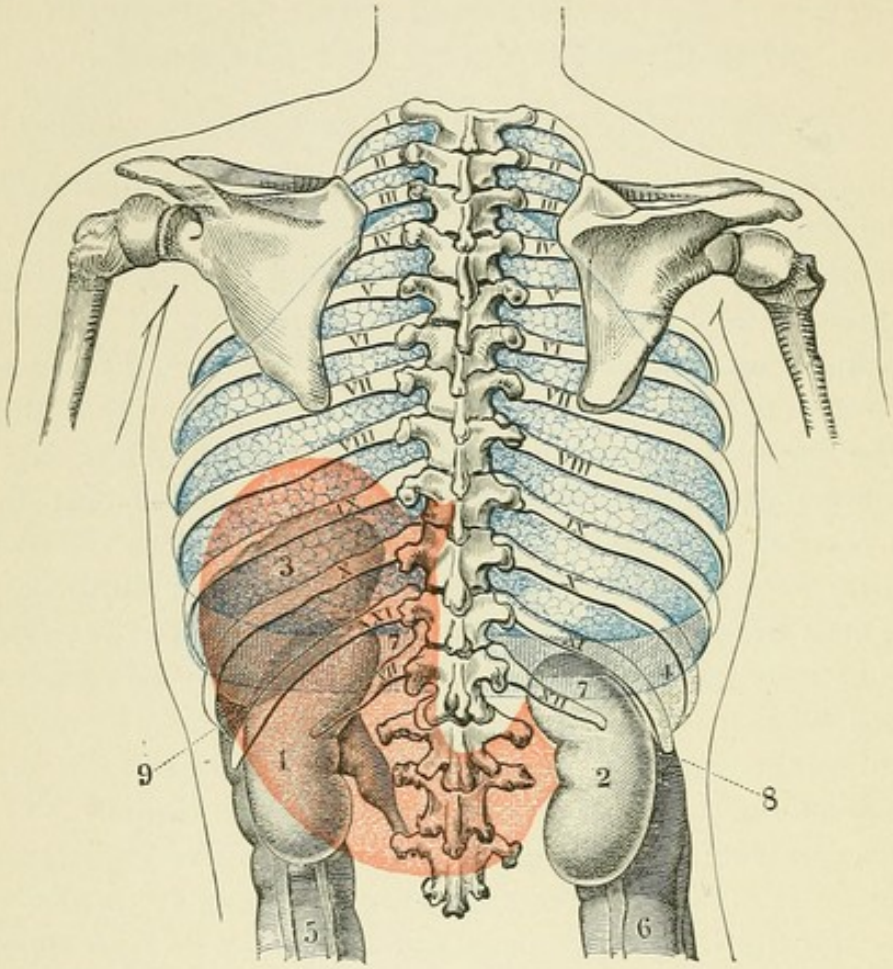


FIG. 8.—LOCATION OF THE STOMACH—DORSAL VIEW.

1. Left kidney. 2. Right kidney. 3. Spleen. 4. Lungs. 5. Descending colon. 6. Ascending colon. 7. Complementary space occupied by expanding lungs in inspiration. The stomach occupies the space colored in red.

Riegel thinks that there is no better way of differentiating gastric dilatation from gastroptosis (falling) than by this process of distention.

This method is carried out by introducing a stomach-tube, to the upper end of which is attached a double-bulb pump arrangement such as is used in some spray apparati (Runeberg, *Deutsch. Archiv f. Klin. Med.*, Bd. XXXIV). Bouveret ("Traite des

Maladies de l'Estomac," Paris, 1893) recommends that the air be forced into the stomach by blowing with the mouth through the tube. Riegel and Boas are very fond of gastric distention by carbon dioxid gas. A teaspoonful of bicarbonate of sodium, and about the same amount, or perhaps a little less, of tartaric acid, are dissolved, each in a separate glass containing 250 c.c. of water.

First the solution of tartaric acid is administered, and immediately afterward the sodium bicarbonate; within the stomach a

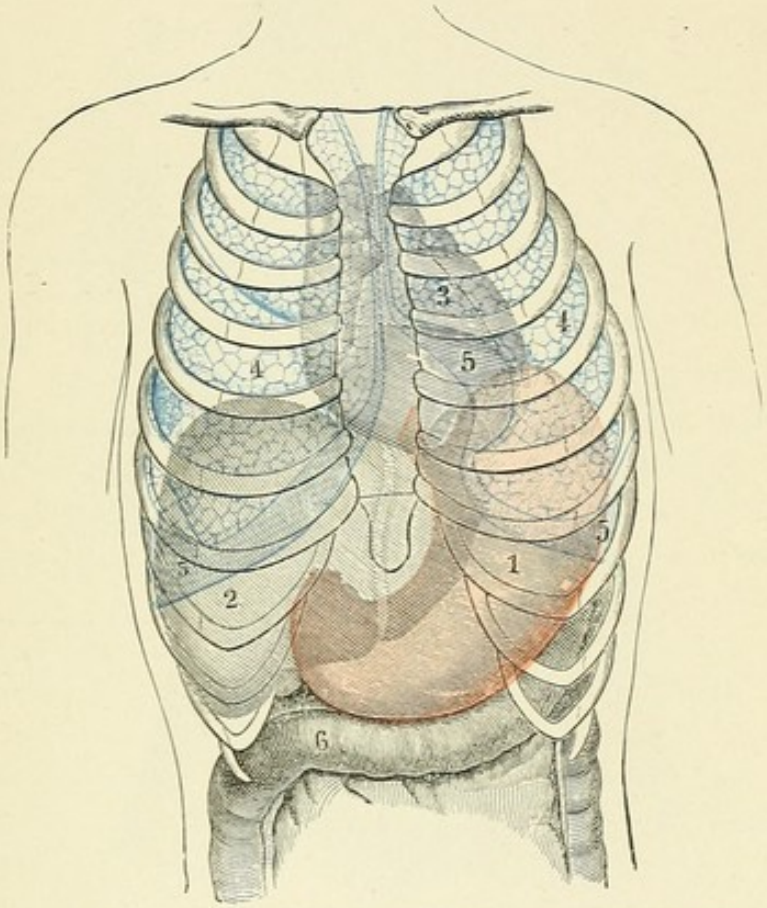


FIG. 9.—LOCATION OF THE STOMACH—ANTERIOR VIEW.
1. The stomach. 2. Liver. 3. Heart. 4. Lungs. 5. Complemental pleural spaces. 6. Transverse colon.

brisk evolution of CO_2 occurs, at once distending the organ so that it stands out prominently, and is evident as a sharply defined, arched elevation; the greater curvature becomes very apparent, not so the lesser one.

The stomach under distention can be readily palpated or percussed. If tumors were made out before the distention, it is important to determine their seat after the distention. It is possible in many cases to demonstrate the connection or non-connection of

the tumor with the stomach after distention. The movability of the tumor gives us some information as regards its seat, particularly if it be carried out before and after distention, for under this method the organ is not only stretched out more, but undergoes a certain amount of turning or twisting around its long axis.

Accordingly, tumors which, when the stomach was empty, were palpated in the line of the umbilicus and to the right, and for that rea-

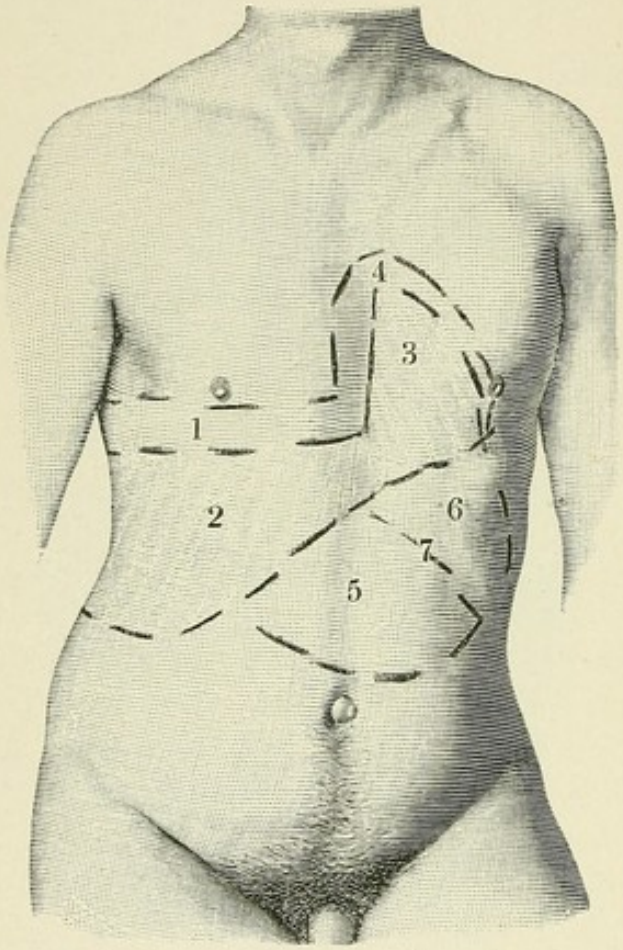


FIG. 10.—NORMAL PERCUSSION LIMITS OF THE ADULT STOMACH.—(*Eichhorst.*)
1. Pronounced liver dullness. 2. Lesser liver dullness. 3. Smaller heart dullness. 4. Larger heart dullness. 5. Limits of stomach percussion. 6. Traube's semilunar space. 7. Left edge of short ribs.

son it might be doubted whether they belonged to the stomach, after distention may move upward to the right, and toward the anterior arch of the short ribs. One may see and feel the direct transition of the tumor mass into the substance of the stomach, or trace its extent over the small curvature toward the pylorus, or ascertain that it is entirely independent of the stomach.

Even the disappearance or the becoming less distinct of a tumor is very important, if it occurs after distention. This is observed in

tumors of the posterior wall. If it is easily movable, very close and tight adhesions may be excluded; if it is absolutely immovable, it is abnormally attached or fixed. It is evident that distention of the stomach with air or gas not only enables one to get a better percussion area, but it serves another purpose: that of facilitating the palpation of tumors. In his new book on "Diseases of the Stomach," Riegel gives, in addition to the above, ten other methods for determining

Location, Size, and Capacity.—Most of which being more or less fallacious, we must refer those specially interested in this matter to Riegel's book, pages 41–56. In our opinion, all of these methods will, before many years, have only a historical value. There is one method for accomplishing the above objects, however, which we can recommend from a very large experience, and which is used exclusively at our clinic, and with the accuracy of which we have had many opportunities to be convinced. It is described and pictured on page 32 of this work.

With our stomach-shaped intragastric rubber bag (see plate IV) and the pressure bottles *A* and *B*, the location and capacity can be determined with great ease. The rubber bag used for this purpose has no sheath or guide for the duodenal tube. The stomach is distended by blowing up the bag within it; the amount of air necessary thereto is measured afterward by allowing it to escape into a spirometer; a less accurate, though a quite practical method, is to catch the escaping air in a glass cylinder filled with water and inverted over a basin.

It might be claimed that our method is a combination of von Kelling's, Schreiber's, and Jaworski's methods, and it does indeed partake of part of the devices of all these (see Riegel, pp. 51, 52, and 54). Schreiber used a small, round—not a stomach-shaped—distensible balloon, but no pressure bottles nor spirometer. Jaworski used two pressure bottles, but no balloon or intragastric bag, and no spirometer, while von Kelling used simply the spirometer to measure the air, which he forced into the stomach with a double bulb, as is used on sprays.

Our method of arriving at the capacity of the stomach is really, then, not entirely original, as it combines the best of three older methods, but it is most convenient and reliable; the bag, as has been shown, can at the same time be used for determining the nature of the motor function. It can be asserted, from obser-

vations on a large number of patients, that there is no other single method which is so useful, and can combine instruction concerning size, location, and capacity of the stomach with that concerning its motor function.

The method is as easy in its application as any which Riegel describes. The capacity can, for practical purposes, be read off on bottle *B*, from the amount of air that has been displaced into the intragastric bag. The expense of the bag is one dollar, and a good idea of the motor function can be gotten from a water manometer in connection with it after distention; with one hand on the epigastric region, the respiratory movements can be felt, and thus distinguished from the active movements as expressed by the rise and fall of the water column in the manometer. It may thus be used without the kymograph.*

Gastrodiaphany of Einhorn.—In 1889 Dr. Max Einhorn succeeded in transilluminating the human stomach in the dark by means of a small Edison lamp attached to a soft rubber tube; from the lamp, through this tube, insulated conducting wires run to a storage battery. (See illustration p. 103.) At some distance from the rubber tube is a current-interrupter. By this apparatus the inventor claims to be able to ascertain the exact position and size of the stomach, and to recognize tumors and thickenings of the front wall by their lack of translucency.

In 1867 Milliot had succeeded in transilluminating the stomachs of animals by platinum wires contained in glass tubes and connected with a Middeldorph's apparatus.

Fleischer, in his text-book ("Path. u. Therap. der Magen- u. Darmkrankh.," p. 789), claims to have succeeded in transilluminating the human stomach, together with Hüfler, before Einhorn. If this is really so, Fleischer did not publish his investigations, as far as we know, and certainly is not entitled to call the method after himself—the gastrodiaphany of Fleischer and Hüfler.

To Einhorn is due the credit of developing the method as an

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* In the shops of Baltimore small toy balloons are sold; they are made of very thin but quite tough rubber, which my assistants have frequently used for intragastric distention. These balloons accompany a game called "pillow dex," and are sold six for 25 cents. For studying the motor function they answer as well as the more expensive stomach-shaped bags, as I have assured myself that on distention they fill every inch of space in a dog's stomach. For determining the capacity, however, the stomach-shaped bag is more accurate.

aid to diagnosis. The patient, in a fasting condition, drinks a liter of water; the apparatus is passed into the stomach just as the lavage tube is passed, and connected with the storage battery. The stomach transmits the electric light through the abdominal walls, becoming visible as a red zone at the place which corresponds to its location.

In case the anterior gastric wall is occupied by a tumor the light will not be transmitted at that spot, but all around it the rays will penetrate, thus evincing a dark, shaded area in a luminous zone.

We are in the habit of marking the ribs, particularly the umbilicus, xiphoid cartilage, and symphysis pubes, with phosphorus, so that they can be seen in the dark and serve as landmarks to the

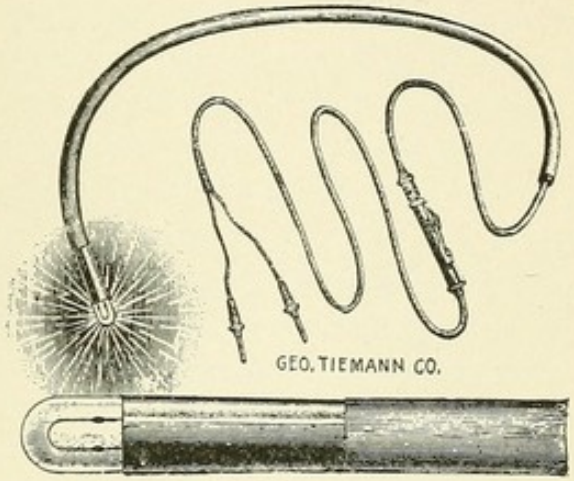

FIG. 11.—THE ELECTRODIAPHANE.

exact abdominal area in which the light permeates. Dr. Howard A. Kelly prompted us to attempt transillumination of the colon by this method, and we succeeded admirably.

We have been able to illuminate, in successive portions, the entire colon in this manner, and demonstrated prolapse of the colon thereby; even into the duodenum and ileum the diaphane has been introduced by us, and we claim these advances of the method as original to our clinic.

Notwithstanding the conservatism of Riegel and Fleiner ("Lehrbuch d. Krankh. d. Verdauungs-Organe," p. 223), and the objections of Boas, and Debove, and Remond, we consider the method valuable; it certainly is convenient for the rapid diagnosis and the differentiation between gastrectasia and gastroptosis.

It should be added, however, that we use a much stronger light (namely, eight to ten volts) than Einhorn, and have one-half of the lamp coated by a reflecting mirror of mercury, which can, of course, be controlled by turning the tube outside of the mouth. At a demonstration which we were requested to give before the Clinical Society of Maryland, the apex impulse of the heart was visible in the dark after transillumination.

CHAPTER XII.

THE STOMACH-TUBE AND TECHNICS OF ITS INTRODUCTION.

Examination of Stomach Contents.—Test-meals: Their Effect upon the Amount of Acid Secreted.

We never use any other kind but a soft elastic stomach-tube, and before introducing it for the first time in any patient, we always instruct him or her carefully about the object and utility of the procedure and its harmlessness. Whenever we can do so we give very timid patients an opportunity of observing with what ease more experienced patients introduce the tube on themselves. This has a most comforting effect. Weak and old persons should always be treated on the bed, several thick towels being placed on the patient's chest and beneath the chin; if the case is to be examined in an erect position, linen gowns are drawn over the breast and lap, or an additional rubber sheet to protect the clothing. Dr. Fenton B. Turck, of Chicago, has devised a useful rubber pocket, which is suspended under the chin during lavage, and protects the garments of the patients from the mouth discharges. If the throat and fauces are very tender (often found in excessive smokers), it is advisable to precede the introduction of the tube by spraying the throat with a three per cent. solution of cocain hydrochlorate or the following anodyne spray:

℞. Three per cent. solution of Cocain hydrochlorate in Benzoinol, . f℥j
One per cent. solution of Menthol in liquid Vaselin oil, ... f℥ss.
Use in atomizer for spraying the throat. Mix.

It is not necessary to lubricate the stomach-tube with any oil or vaselin,—there is generally mucus enough in the esophagus to facilitate the passage.

In the *New York Medical Journal* for December 28, 1895, volume LXII, No. 26, page 822, a new double-current stomach-tube has been described by the author, through which the inflow and outflow goes on uninterruptedly at the same time. This tube is recommended only as a time-saver for the specialist in practice: the simple tube will fulfil every requirement, even though lavage of progressed gastrectasia may require much more time.

From 20 measurements of living female patients, the author has found that the average distance from the incisor teeth to the deepest portion of the stomach is 55 cm., and in 36 measurements of healthy males the same distance was found to be 60 cm. In cadavers this is in both sexes, according to the author's experience, shortened by postmortem rigor, it having been found to be 52.5 cm. on the average for females in 12 different subjects. In 12 male cadavers, the average distance from the incisor teeth to the deepest part of the stomach was 54 cm.

In ten cases of dilatation of the stomach, the average distance from the incisor teeth to the deepest portion of the stomach, as measured by as rigid a sound as could safely be introduced, was 69 cm. In ordering the new double-current tube, however, from Tiemann & Co., the manufacturers were directed to make the proportion which is introduced into the body 70 to 72 cm. long, which length, on the basis of the foregoing measurements, was considered

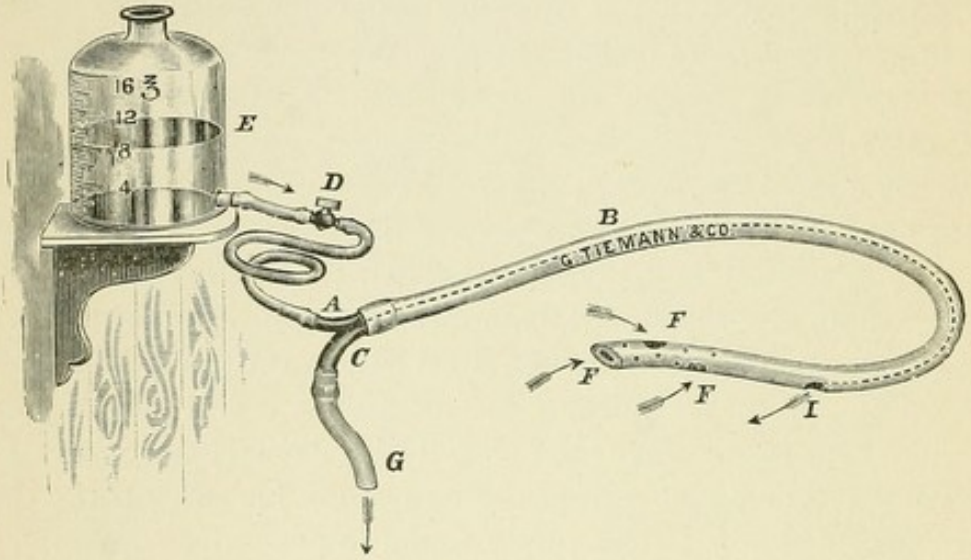

Fig. 12.—Hemmeter's Double-current Stomach Lavage Tube.

sufficient for all requirements. The amount flowing down through the inflow tube will vary with the height of the pressure bottle, but should never exceed more than one liter in two minutes; in the same tube the outflow channel should be able at the same time to discharge by simple siphonage two liters.

The inflow and outflow tube should be tested as regards their caliber, the former by pressure, the latter by siphonage, before they are used on patients.

When the outflow tube is tested as regards the amount of water it will discharge in a given time, the tube should be arranged as in figure 13, so that the water must rise 70 cm., the distance from the deepest portion of the stomach to the incisor teeth, before it can descend into the measuring graduate.

The procedure of lavage must be attentively watched in cases where much solid *débris* is expected, and if the outflow becomes choked with solid matter the inflow must be cut off at once until the outward passage is made clear.

A piece of glass tubing two inches long, firmly tied to the outflow tube, is useful, in that it permits the operator to observe the material which is running

out. The single stomach-tube has been swallowed entirely and disappeared into the stomach. At least two such accidents are on record, one by Leube (33), the other by Jackson (35); they are both quoted by William H. Welch, in his article on "Dilatation of the Stomach" in Pepper's "American System of Medicine," volume II.

With a double, or current, tube this accident is impossible, as the instrument is tied to the supply reservoir. Aspiration of the mucosa, and tearing of pieces of healthy membrane by suction, which has occurred in Leube's experience (2), is also a matter of impossibility with the recurrent tube. Physicians who have used gastric lavage with a simple funnel, know how difficult it is to keep

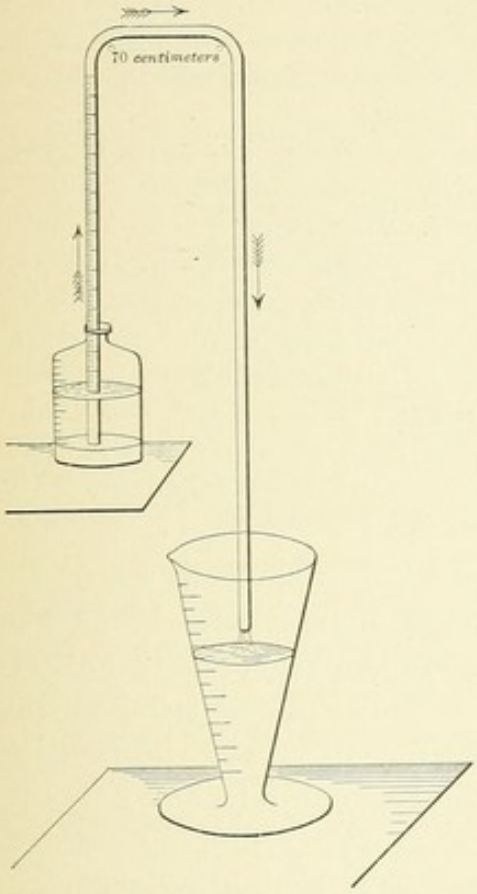

FIG. 13.—ILLUSTRATING THE PRINCIPLE OF SIPHONAGE.

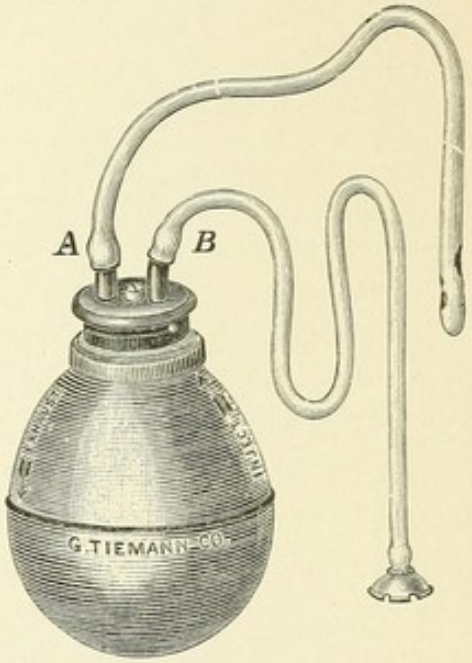

FIG. 14.—BULB USED FOR THE ASPIRATION OF TEST-MEALS WITH PATIENTS HAVING VERY RELAXED ABDOMINAL WALLS.

air from being sucked into the stomach; a deep depression of air forms in the center and is occasionally aspirated into the stomach; all of this is avoided in the recurrent tube. The author, on visiting Professor F. Penzoldt, in Erlangen, in July, 1895, was surprised to find this pioneer of digestive pathology still advocating the use of a guide in the shape of a flexible stick or whalebone, which, during introduction, is inserted into the gastric tube to facilitate its entering the esophagus after it curves over the base of the tongue.

In his most recent contribution to the subject, Penzoldt (*loc. cit.*, 27) gives minute details as regards the method of application of the *Leitungsstab* or *Man-*

drin within the tube, and says that it should be oiled to facilitate its removal when the tube has reached the middle of the esophagus. He also suggests catching the tip of the lavage tube between the index and middle finger of the left hand, which are inserted into the patient's mouth, and bending the tip down over the base of the tongue until it enters the esophagus. This is the method advocated by his teacher, Professor Leube (*loc. cit.*, 2), and also by Rosenheim (36).

In the writer's experience the intratubal whalebone guide and the insertion of the fingers into the patient's month are superfluous. The tube can always be introduced without a guide, and without touching the patient. The main point is, that the point of the tube, when it has reached the wall of the pharynx, shall be deflected downward. This will occur without exception, and in a very natural, easy, manner, if the patient is directed to swallow at this moment. In the moment of this act of deglutition the point of the tube is bent downward into the esophagus.

Beginners in using the tube need have no fear that it will enter the trachea. To make it enter the trachea is, in the writer's experience, a difficult undertaking, and requires special training and dexterity. He was present on an occasion when a class of ten students were taking a private course in diseases of the throat, during which lesson they were trying to mop the larynx. What they really did was to mop out the superior portion of the esophagus. Direct the patient to keep taking deep inspirations, and as soon as the tip or point of the tube is felt touching the pharyngeal wall tell him to swallow, and almost immediately the tube follows into the esophagus and can be pushed into the stomach without further resistance. The double or recurrent tube is still in its experimental stage and can not be recommended as practical.

It is not necessary for the patient to open his teeth any wider than just to admit the sound; at the same time, caution him not to bite on it, but to breathe naturally. No patient should be subjected to gastric lavage without a previous examination of the thorax. Penzoldt tells of a case in which the stomach should have been washed out in the morning, but on account of lack of time this was postponed until the same evening. On the same afternoon the patient died of rupture of an aortic aneurism into the esophagus.

Lavage and introduction of the tube is contra-indicated—

I. In all constitutional and local diseases which could be aggravated or life endangered by the irritation and exertion of lavage. Among these could be mentioned:

1. Pregnancy (though this is not a disease).

2. Heart disease in a state of defective compensation—heart neuroses, angina pectoris, myocarditis, and fatty heart in an advanced stage.

3. Aneurism of the large arteries.

4. Recent hemorrhages of all kinds, including apoplexies, pul-

monary, renal, vesical, gastric, rectal hemorrhages, and hemorrhagic infarctions.

5. Advanced pulmonary tuberculosis.

6. Advanced pulmonary emphysema, with bronchitis.

7. Apoplexy and cerebral hyperemia.

8. Advanced cachexia.

9. Presence of continued or remittent fever.

II. The stomach and intestinal diseases which are contra-indications of the use of tube are:

1. Ulcer, with recent hematemesis and dark stools.

2. Palpable carcinoma of the pylorus, with vomiting of coffee-ground material and the classical symptoms of cancer.

3. Many gastric neuroses in which the character of the malady is clear without lavage.

4. Stomach or intestinal troubles, with acute fever.

5. Gastric mucosa easily started to bleeding.

6. Secondary gastric affections whose dependence upon a distinct and more important primary disease is evident.

These are not invariable rules, however; cases may occur under some of these exceptions, that at times peremptorily require lavage on account of depressing self-intoxication from the stomach or advanced gastric fermentation. Thus, according to Boas, it has been employed with success in pregnancy, and the author has once washed out the stomach in a case of typhoid fever with favorable result, and also performed lavage in a case of aortic regurgitation, with Bright's disease and gastrectasia, where much relief was experienced from the procedure. Professor Moritz, of Munich, has frequently passed the stomach-tube in pregnant women to ascertain the intragastric pressure (25).

In a normal position of the abdominal viscera the location of the cardia corresponds to the spinous process of the ninth thoracic vertebra. By counting off this process on the back of the patient and placing the upper eye of the sound against it, one can measure the length of tube necessary to reach the stomach by applying it from this point along the back, passing alongside of the ear to the front incisor teeth. At this point, which reaches the incisors, it is of assistance to make a mark on the rubber with a nitrate of silver point; this will avoid pushing the tube out or in to discover whether it has reached the stomach after being introduced.

In dilations and falling of the organ, the length of tube required

can only be learned after a previous lavage. When the sound is used to draw out a test-meal, direct the patient to press in his abdominal muscles as if in the act of having a stool. Frequently the accompanying nausea will bring this about involuntarily. If no contents arise, push the tube gently further in or pull it slowly out, and try both ways. If the abdominal walls are flabby, external manual compression will sometimes produce the desired result. If all these manipulations are of no avail, the stomach is either empty or the tube is plugged up with food-particles too large to pass.

To find out which is the case, allow 300 c.c. of pure water to run in and then lower the funnel and siphon out; if nothing but comparatively clear water returns, the test-meal has passed into the duodenum. One should be very cautious in moving the tube out and in when no stomach contents appear in the funnel, as it is possible that the eyes of the tube may have sucked in the gastric mucosa itself, and by moving too suddenly a piece may be torn away. If there is the least resistance, avoid moving; rather pour in a small amount of water, which will push away the adherent mucosa or the food-particle, and the next attempt will bring up the test-meal.

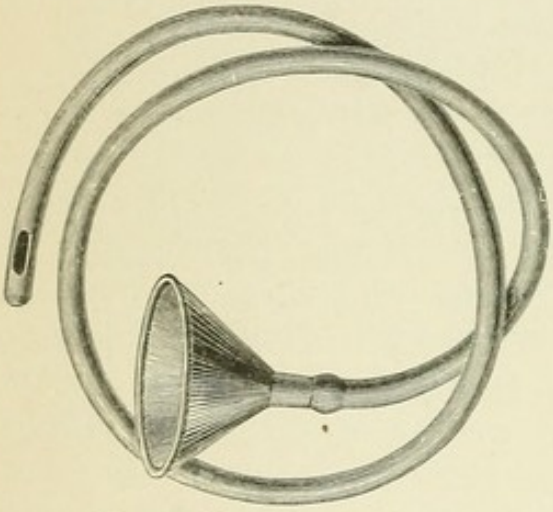

FIG. 15.—EWALD STOMACH-TUBE.

If the stomach is already empty, the test-meal must be given again at another time. We never recommend any apparatus for aspiration, not even the rubber bulb; with patience the simple expression method will suffice. For small samples of test-meals the Einhorn stomach bucket (Einhorn, "Diseases of the Stomach," p. 63) is an available instrument. We do not recommend the recurrent stomach-tube, except as a time-saver, and are aware that its usefulness is very limited. Before using the tube, all artificial teeth should be removed.

In very rare cases of intense food and mucus putrefaction, and in extensive gastrectasis, a recurrent tube may be used with success. To give an idea of the time it takes to cleanse some stomachs, we quote Dr. Herman Strauss, assistant to Prof. Riegel, who claims

to have washed out rice particles after 40 liters of water had been allowed to flow in and out. After washing a dilated stomach for one hour personally, we found bread and stringy mucus in the last washing. Before introducing a stomach-tube, assure yourself that its lumen is not obliterated, and warm it in a pitcher of warm water.

Test-meals.—The test-meal most frequently employed is that of Ewald and Boas, consisting of a roll or a piece of wheat bread and 500 c.c. of water or tea, without milk or sugar. The time for examination is one hour after the meal.

Leube and Riegel advocate a test-dinner of 400 c.c. soup, a portion of beefsteak or roast beef, potatoes, and a roll. The time for examination is three to four hours after this meal.

Jaworski and Gluczinski employ the white of a hard-boiled egg and 100 c.c. water.

Klemperer recommended ½ of a liter of milk and 70 gm. of wheat bread, and examined two hours later.

Germain Sée used 60 to 80 gm. scraped meat and 150 gm. white bread. Examination two hours later.

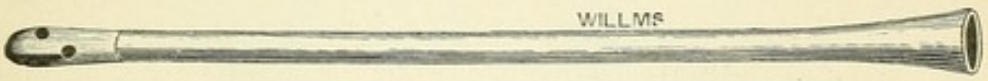

FIG. 16.—THE ESOPHAGEAL TUBAL PROBE.

The Ewald and Boas test-breakfast seems the most convenient, and in cases of enfeebled digestion, where much food is retained from previous meals, the least confusing.

Fleiner's test-meal consists of soup, roast beef, and potato purée, he examines three to four hours after the meal.

At the Maryland General Hospital we generally use a double test-meal consisting of:

8 A. M.—1 small piece of beef, scraped and broiled = 80 gm.; 1 soft-boiled egg; 30 gm. boiled rice; 1 glass of milk=250 c.c., and a piece of bread.

Four hours later an Ewald test-meal is given, and one hour after this the stomach contents are drawn. In giving a test-meal, always insist on good chewing, and urge that all food-substances be very finely cut up so that they can not plug up the tube, even if not digested.

The double test-meal, about which the late Dr. Henry Salzer, of Baltimore, was quite enthusiastic, really offers some advantages over others. In the first place, it permits of as easy a study of the

various stages of the digestion and of the motility and degree of retention as Riegel's test-dinner; but the main advantage of the double test-meal—a full meal at 8 or 9 A. M. and an Ewald test-meal at 12 M. or 1 P. M., examination at 1 or 2 P. M.—is, that after drawing it, we may, in a large number of instances, recognize conditions of gastric motility and secretion before we analyze the contents. For instance, disappearance of the entire breakfast-meal points to a normal digestion.

Absence of all proteids,—beef and egg,—and presence of considerable carbohydrates,—rice and bread,—points to hyperchlorhydria; and, again, absence of all carbohydrates and presence of some of the beef and egg points to hypochlorhydria, subacidity, or anacidity. Presence of the entire meal, with perhaps milk uncurdled, means impaired motility, with atrophy of gastric mucosa, absence of acid, enzymes, and pro-enzymes. If the entire breakfast has disappeared, the status of the gastric secretions may be ascertained from the Ewald test-meal which is still present.

The objection which has been made, that the double meal is uncleanly to handle during analysis, has also been urged against Riegel's. Whether the morsels of an Ewald test-meal are more appetizing and esthetic to handle than remnants of our double test-meal, is a matter concerning which it does not pay to quarrel. It is a very important matter to state what test-meal is used in giving out the various acidities obtained, because some test-meals are greater stimulants to the gastric mucosa than others.

The Ewald test-breakfast really makes very slight demands upon the working capacity of the stomach.

The total acidity one hour after an Ewald test-breakfast is normally about 60; the lowest total acidity observed by us one hour after a test-breakfast of this kind in a healthy individual was 22. Fleiner, who uses a test-meal of soup, roast beef, and potato purée, asserts that three to three and a half hours after this test-meal the total acidity is normally 70 to 100 (Prof. Wilhelm Fleiner, "Lehrbuch d. Krankheiten d. Verdauungs-Organe," p. 186). Dr. Julius Friedenwald has found that the gastric secretion of HCl appears sooner, and reaches a higher degree after our double test-meal than after an Ewald meal.

An amount of HCl equal to 0.1 to 0.2 per cent. may be regarded as normal; everything below that means subacidity, above this, hyperacidity. The total acidity can not correctly be regarded as an

unfailing indication of the amount of HCl present; the latter should always be determined separately in addition to the total acidity.

Apparently there are climatic, barometical, and geographical factors which influence the total acidity. In 170 cases at Riegel's clinic, Strauss found the total acidity after a test-breakfast equal to

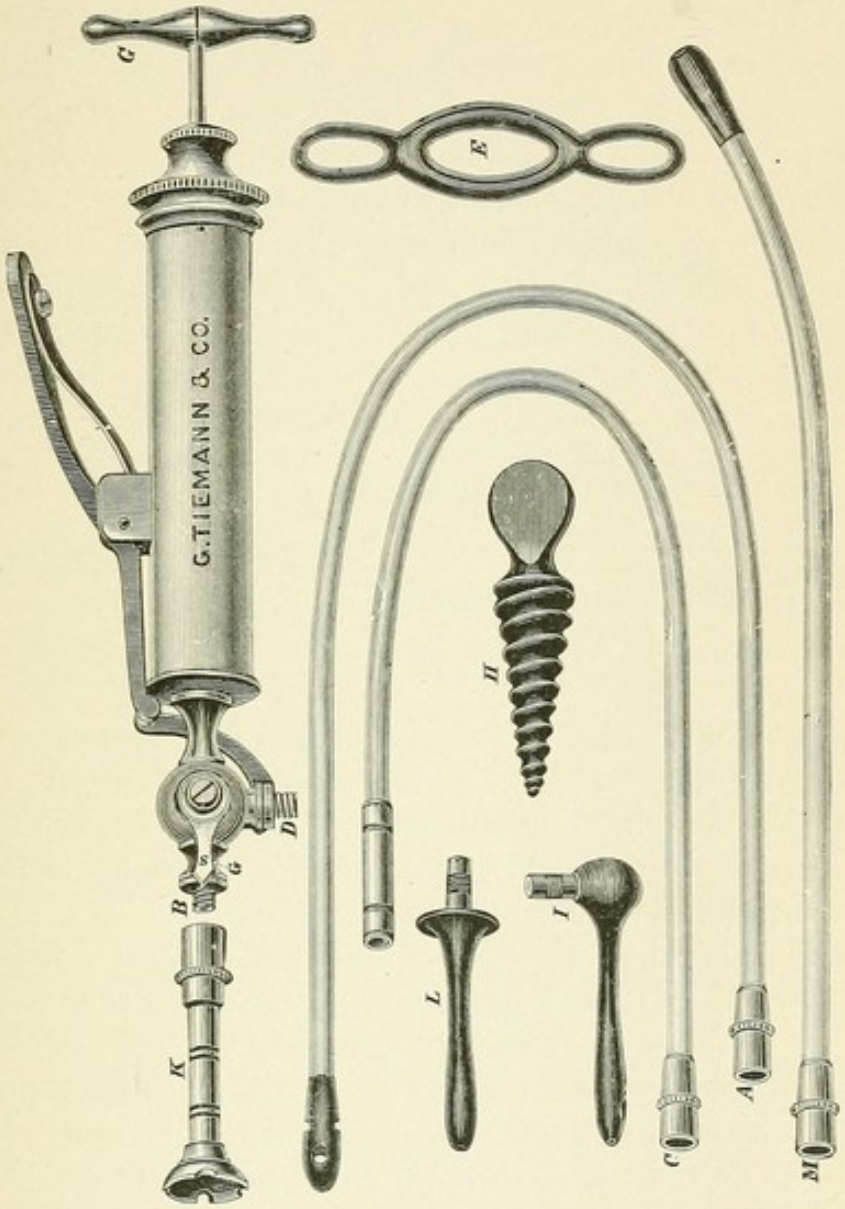

FIG. 17.—STOMACH-PUMP USED ONLY FOR RAPID EVACUATION OF POISONS.

68; in 92 cases at Berlin after a test-breakfast the average total acidity was estimated at 47; the average amount of free hydrochloric acid at Riegel's clinic was found to be 37. Normal values, one hour after a test-breakfast of a roll and water, are, for average total acidity, 40 to 60; for free HCl, 20 to 30, for Baltimore.

After the complex meal of Salzer, *i. e.*, five hours after, 50 to 60 grs. beef, 500 c.c. milk, 70 grs. rice, and one egg, the total acidity on the average was found to be 95° and the free HCl 46°, for Baltimore. It should be emphasized that these figures represent only relative values. One often finds every symptom of hyperacidity with relief following the use of alkalies, when the total acidity was found to be only 56° (one hour after an Ewald breakfast), the free HCl only 24°. On the other hand, cases have presented themselves showing, under the same conditions, a total acidity of 80° and free HCl = 50°, still no symptoms of hyperacidity. All this goes to show that some stomachs may do their work normally very well on relatively low amounts of free HCl, and, of course, suffer from hyperacidity from comparatively slight increase of free HCl, which would not affect a stomach used to higher amounts of acid.

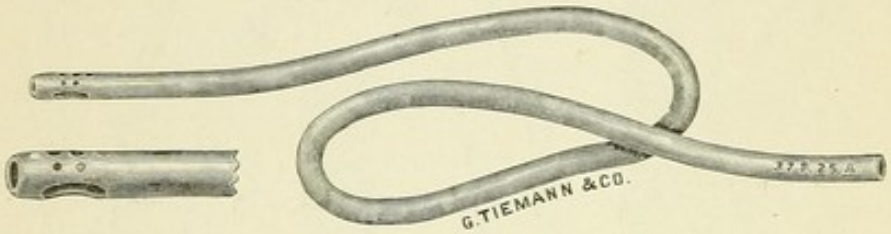

Fig. 18.—Modified Ewald Tube, with numerous smaller and larger Lower Openings.

Most modern observers that can speak with authority on the subject, agree that the total acidity should not be employed to express hyperacidity, but only the amount of free HCl, as this is the only acid which, when increased, gives rise to the complex of symptoms technically recognized as hyperacidity.

Before closing this chapter it might be added, that where it is impossible to use the tube on account of prejudice of the patient, to obtain a test-meal, emesis may be resorted to. The stomach contents obtained after a test-meal, as a rule, filter slowly, and if much mucus is present, not at all. The filtration can be accelerated by rubbing the material first through a small coarsely-grained sieve (strainer), then through a finely-grained strainer, and then filtered through Swedish filter paper.

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CHAPTER XIII.

METHODS FOR QUALITATIVE AND QUANTITATIVE ANALYSIS OF STOMACH CONTENTS.

Presence of Bits of Gastric Mucosa.—Examination of Stomach Contents for Mucus, Saliva, Bile, Duodenal Secretions, Blood, and Pus.—Tests for Blood in Stomach Contents.—Demonstration of the Presence of Iron in Stomach Contents or Vomited Matter.—Spectroscopic Examination of Stomach Contents for Blood.—Examination of Portions of Mucosa or Tissue found in the Wash-water or Vomited Matter.—Literature.

The stomach contents should be examined for—

1. The character and amount of the undigested food.
2. The presence and kind of bacteria.
3. The bile, mucus, pus, and blood.
4. The total acidity.
5. The amount of free hydrochloric acid.
6. The presence of inorganic, lactic, butyric, or acetic acids.
7. The combined hydrochloric acid and acid salts.
8. The presence of products of digestion, viz., syntonin, propeptone, albumoses, peptone.
9. The presence of pepsin and rennin; if these are absent, their pro-enzymes.
10. The products of starch digestion, dextrin, erythrodextrin, achroodextrin, and maltose.
11. Fragments of mucosa.
12. Fragments of neoplasms.

Character and Amount of Undigested Food.—The examination for undigested food-particles may demonstrate the presence of substances eaten twenty-four hours before the expression of contents, and thus, at once, establish a dilatation or stenosis. As already pointed out, excess of rice and bread, and absence of beef

and egg, indicates a higher acidity, while absence of bread and rice, and presence of egg and beef, indicates sub- or anacidity. This, of course, can be most conveniently studied when the contents are drawn out about five hours after the double meal, as employed at the Maryland General Hospital.

Bacteria.—For bacteriological examination, a few slides are stained with methylene blue, and, also, cultures made, the latter especially when there is any disease of the air-passages, the microbes of which may get into the stomach with swallowed mucus or run down unconsciously during sleep. This is particularly important in pulmonary or laryngeal tuberculosis. Instead of methylene blue, Lugol's solution of iodin should be used on other slides for examining bits of tissue, mucosa, and cellular detritus.

The normal stomach contains many micro-organisms; only very large numbers of bacteria have a pathological significance if by culture experiments they can be shown to be still capable of multiplication.

Microbes only propagate luxuriantly when stagnation of gastric contents occurs. The secretory disturbances are then a secondary effect, a consequence of the stagnation. But primary reduction of HCl secretion has been known to cause a luxuriant gastric flora, since it is the HCl which, to a great extent, inhibits their development and also destroys a large number of them. If there be deficient peristaltic power, the diminution of HCl causes further disturbances in the stomach and intestines by accumulation of bacteria. But no degree of gastric acidity, no matter how great, can destroy all bacteria introduced.

Hyperacidity is as detrimental in its consequences as anacidity, because it inhibits normal intestinal digestion, which is the best means of combating fermentation and putrefaction. Hydrochloric acid undoubtedly inhibits or checks gastric fermentation to a certain extent, but all ferment-producing microbes are not destroyed by it in the stomach. Therefore, one frequently finds gastric fermentation with hyperacidity of HCl, and, reversely, fermentation may be absent where hydrochloric acid is entirely absent, provided the motility is good.

This will again impress the importance of an intact gastric peristalsis, a certain time of action being indispensable for organized ferments to set up their characteristic decomposition even at the body temperature; with a good motility, however, the gastric

chyme may reach the intestine, meeting a vigorous digestion before the bacteria get a chance to forge ahead of the normal unorganized ferments.

The most frequent of fungi in gastric contents is ordinary yeast, and there should be no difficulty in recognizing it; unless occurring in very large numbers, it has no pathological significance. Two more germs found in the contents are of interest—the sarcinæ and the Oppler-Boas bacillus, the latter occurring in the gastric contents of carcinoma. Sarcinæ may be seen under the microscope without staining; they are, indeed, preferably to be examined that way, as they stain so deeply with anilin dyes as to look like black patches.

Sidney Martin recommends drying and fixing a drop of stomach contents on the slide or cover-glass, and placing in a very dilute solution of gentian violet for three minutes, washing out in water and mounting in Canada balsam. The gentian violet must be so diluted as to be nearly transparent. Yeast can similarly be stained by magenta or methylene-blue solution (two per cent.). If the latter is used, the preparation requires washing out in water.

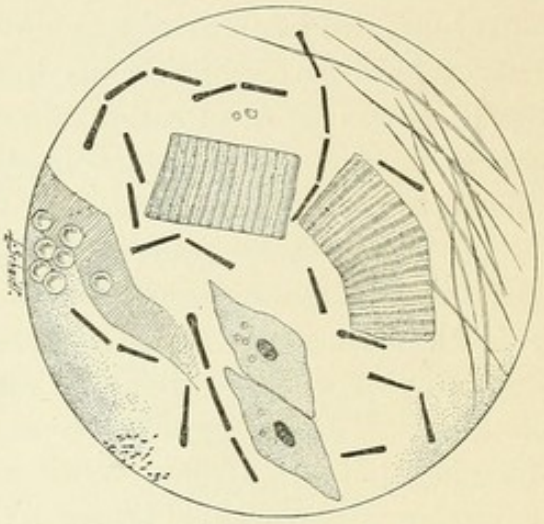

FIG. 19.—OPPLER-BOAS BACILLUS FROM CONTENTS OF A CARCINOMATOUS STOMACH.

Sarcinæ can hardly be said to have any pathological significance, according to Oppler (*München. Med. Wochenschr.*, 1894, No. 29). They are found in ectasias, occurring on a non-malignant basis, and in very atonic conditions; also in acute and chronic gastritis, in ulcer, in the gastric neuroses, and the gastroptoses.

Riegel agrees with Oppler in the assertion that sarcinæ are very rarely found in gastric carcinoma. They are generally observed in biscuit or bale-shaped groups of four, eight and 16 individual sarcinæ bunched together; their occurrence as single individuals is seen rarely.

The Oppler-Boas bacillus (Oppler, "Zur Kentniss d. Mageninhalts bei Carcinoma Ventriculi," *Deutsche Med. Wochenschr.*, 1895, No. 5) is an unusually long and non-motile bacterium, which was

observed in many cases of gastric carcinoma (see Fig. 19). In 20 cases of carcinoma, Kaufmann found these bacilli 19 times, and, according to his investigations, they have the power of abundantly forming lactic acid from various kinds of sugar. In the only case of the 20 just mentioned in which the Oppler-Boas bacilli was absent, the lactic acid was absent also.

According to Schlesinger and Kaufmann (*Wiener Klinische Rundschau*, 1895, No. 15), the presence of a large number of these bacilli in the stomach contents is an indication of carcinoma, and their absence is of similar significance to the absence of lactic acid. If a stenosis of the pylorus is present, then the absence of these bacilli is an argument against carcinoma. Riegel (*loc. cit.*) confirms the occurrence of these bacilli in enormous numbers in carcinoma, and adds that, although there are numerous fungi that have the property of forming lactic acid in stomach contents, this can not alter the significance of the Kaufmann and Schlesinger observation. He does not consider these organisms as pathognomonic of gastric cancer, but as very important for the diagnosis.

Our knowledge concerning the bacteria occurring in normal and pathological stomach contents is very incomplete as yet. It appears, however, that in all pathological processes we are not confronted with qualitatively new bacteria, but with excessive multiplication of those normally present. The disturbances produced by abnormal augmentation of bacteria in the stomach are explained by Minkowski ("Ueber d. Gährung im Magen," *Mittheilung a. d. Medic. Klin. Königsberg*, edited by B. Naunyn, Leipzig, 1888, S. 156) in the following manner:

1. Substances may be formed which irritate the mucosa and provoke catarrhal inflammation.

2. Gas may be formed in considerable quantities, causing distress by distention, and increase the mechanical insufficiency already present.

3. The fermentation may give rise to toxins.

4. Putrefaction of albuminous bodies may produce alkaline bodies that will neutralize the hydrochloric acid or what little of it may yet be secreted.

5. Gastric fermentations may have a detrimental influence on the intestinal functions.

Examination of Stomach Contents for Mucus, Saliva, Bile, Duodenal Secretions, Blood, and Pus.—The presence of *mucus* is

evident to the naked eye by its stringy and tenacious character. Its chemical demonstration is carried out by dissolving the mucus in liquor potassæ, in which it is slightly soluble, and from which it can be reprecipitated by acetic acid. When pharyngitis, laryngitis, and bronchitis can be excluded, large quantities of mucus in stomach contents are indicative of gastritis.

If the gastric contents consist largely of *saliva*, this can be demonstrated by the potassium sulphocyanate, otherwise known as rhodankalium, KCNS, which is a normal constituent of healthy saliva. Potassium sulphocyanate gives a dark, purplish-red color upon the addition of a solution of chlorid of iron.

Bile, if present to any considerable extent, is noticeable at once to the naked eye by the compound greenish-yellow tinge it imparts to stomach contents. Very slight amounts of bile and duodenal secretions are occasionally observed under normal conditions, particularly if the stomach be washed out early in the morning before breakfast, for there is no absolute closure of the pylorus when the stomach is empty.

Boas has, however, pointed out that constant presence of very evident admixture of bile and duodenal secretions points to stenosis of the descending portion of the duodenum (Boas, *Deutsche med. Wochenschr.*, 1791, No. 28., "Ueber die Stenose des Duodenum"). As a rule, it will be necessary to assure oneself of the presence of bile by the Gmelin test, or the demonstration of bile acids or cholesterin.

Gmelin's test is carried out by adding 20 drops of fuming nitric acid to 10 c.c. of officinal nitric acid in a test-tube. Ten c.c. of stomach filtrate are drawn into a pipette, and, holding the test-tube with the HNO_3 in the left hand in a slanting, horizontal position, the filtrate is allowed to flow slowly from the pipette held in the right hand over the nitric acid. If the stomach contents contain bile, there will be formed several characteristic rings of color, which, going from above downward, are (1) green, (2) blue, (3) violet, and (4) red, but only the green color is an evidence of the presence of bile.

Better results are obtained by using a conical glass on a broad foot instead of a test-tube. In the clinical laboratory they are of 60–79 c.c., or about two ounces in capacity. It is of some advantage to be able to place them alternately on and in front of a white and black background during the reaction. First, 20 c.c. of gastric juice,

if necessary previously filtered, are placed in the glass, then ten c.c. of nitric acid added by a pipette, which is carefully carried to the bottom of the vessel; here the nitric acid is very gradually permitted to escape by diminishing the pressure of the finger on the end of the pipette. In this manner it is easier to get the nitric acid under the gastric juice. The display of the colors, yellow, green, blue, violet, and red, occurs from above downward; the green color is the only one that is characteristic of bile elements.

The demonstration of the *bile acids* is effected by first precipitating all albuminous bodies by boiling or by alcohol; a few drops of a solution of cane sugar are added, and then, drop by drop, pure concentrated sulphuric acid. If the solution is now heated a beautiful purple-red color is obtained, between 60° and 70° C. (Pettenkofer).

The presence of duodenal secretions is demonstrated by testing the stomach contents for the specific ferment activity of trypsin, amylopsin, and steapsin. (See p. 58.)

Tests for Blood in Stomach Contents.—Although blood may be present in the material drawn by a stomach-tube, or in vomit, it is not always easy to decide whether it was derived from the lungs or from the stomach. Vomiting may produce a cough, and, vice versâ, coughing may lead to an attack of vomiting, and in cases where either organ is liable to hemorrhage, as in tuberculous patients with a congestive state of the mucosa, it is, except in rare instances, impossible to decide the origin of the blood.

In cases with copious arterial, gastric hemorrhage, the blood is bright red and clotted. A slower but still quite profuse hemorrhage generally shows as a black clot or mass of black clots. In very slow but continuous hemorrhage the blood collects, and may be partially digested or decomposed in the stomach before it is vomited as a black, coffee-ground material. The diagnosis of blood in the vomit is not always easily made. There are four methods of determining the presence of blood, and by one or more of them it may generally be accomplished.

The first is by the microscopical demonstration of the red blood-corpuscles. In cases of suspected ulcer, all vomited matter should be microscopically examined, even when blood is not evident to the naked eye.

The second is known as the guaiacum test. Two or three drops of freshly prepared tincture of guaiacum are added to five c.c. of stomach contents in a test-tube, and ozonized ether poured on the

surface; if blood is present, a blue color develops where the two liquids meet. Equal parts of tincture of guaiacum and turpentine that have been exposed to the air may be used instead of ether. This test for blood is fallacious, as almost any carbohydrate, bile, or saliva will produce the same color in the total absence of blood.

The guaiacum test, which was originally proposed by Almén and van Deen, becomes more rĕliable when executed by an improved method suggested by H. Weber. A considerable quantity of the filtrate is extracted or mixed with water; glacial acetic acid, to the amount of one-third of the entire quantity of water and filtrate mixture, must be added.

Of this acid extract, about ten c.c. are poured off after settling; then ten drops of tincture of guaiacum and 20 to 30 drops of turpentine are added. If blood is present, the mixture becomes violet-blue; in case blood is absent, the color will be red-brown. The blue coloring matter that indicates blood can be extracted by shaking the mixture with chloroform. Coffee-ground vomit will not permit of the correct finding of blood with either of the two preceding tests.

This kind of vomit may have to be differentiated from genuine tea or coffee vomit, or from bile, by Gmelin's test. In this form of vomit, the corpuscles are disintegrated and the hemoglobin transformed into insoluble hematin. Still, there are two ways left to diagnose the blood present, if any: first, the formation of crystals of hemin, and, secondly, the demonstration of the presence of iron.

1. Preparation of hemin crystals: Three to four drops of the thick sediment is mixed on a glass slide with a little common salt, then one to two drops of glacial acetic acid are added, and the mixture carefully heated over a small flame of a spirit-lamp or a Bunsen burner until bubbles begin to form. If blood is present, on examining the preparation with the microscope, reddish-brown, oblong crystals of hemin hydrochlorate will be recognized: their color, form, and occurrence is characteristic. This test may fail in cases where blood is present.

2. Demonstration of the presence of iron: Naturally, the patient whose stomach contents are to be examined must not have been taking iron in any form, nor any raw meats.

Demonstration of the Presence of Iron in the Stomach Contents or in Vomited Matter.—In case one is dealing with coffee-ground material this test may become necessary. Some of the black

sediment is placed in a porcelain dish, and a few crystals of potassium chlorate and two to three drops of strong hydrochloric acid are added. On heating over a flame and adding a few drops of a five per cent. solution of potassium ferrocyanid, $4KCN, Fe(CN)_2 + H_2O$, Prussian blue will be formed. Boas and Sidney Martin consider this a very delicate test. The Prussian blue, upon the occurrence of which this test depends, is a complex cyanid of iron, $4Fe(CN)_3 . 3Fe(CN)_2$.

Spectroscopic Examination of Stomach Contents for Blood.—A spectroscopic examination is possible when the red blood-corpuscles have become dissolved, and the filtrate of gastric contents contains oxyhemoglobin. The compound of oxygen with hemoglobin is distinguished by two absorption bands in the spectrum, which occur between the Fraunhofer lines *D* and *E* in the yellow and green. If after the recognition of these lines a reducing agent is added to the solution of oxyhemoglobin, for instance, if it is shaken with ammonium sulphid, the two bands observed before fuse into a single broad band, occupying the space between the two distinct and separate bands, or move beyond *D* toward the red of the spectrum. (Compare Eichhorst, *loc. cit.* p. 523; also Richard C. Cabot, "Clinical Examination of the Blood," Wm. Wood & Co., Publishers, New York, 1897, and von Jaksch, *loc. cit.*)

Examination of Portions of Mucosa or Tissue Found in the Washwater and in Vomited Matter.—In the washwater from almost every stomach, also in the samples of test-meals gained by the Ewald expression method, and in vomited matter, small portions of the superficial mucosa of the stomach can frequently be found on careful searching. Stimulated by reading the accompanying literature, particularly the work of Hayem, Boas, Einhorn, and Cohnheim, we have during the last three years made a study of such tiny bits of mucosa.

To detect them more easily, the stomach is best washed in the morning, before breakfast, with 500 c.c. of warm water, which is poured into a shallow papier-mâché or hard rubber dish, the bottom of which is colored white and black; on this background the tiny bits of tissue from the mucosa, or from any neoplasm that may be in the stomach, can be more easily recognized. These particles are usually of a reddish color; they may seem at times colorless, so that in a glass or pitcher they may be overlooked, while on the dark flat dish they are quite apparent. These fragments come from

very superficial erosions, which are possibly caused by very slight local congestions or by traumatism (Ewald, *loc. cit.*).

It is conceivable that the contractions of the muscularis of the stomach may, if sufficiently powerful, effect an arrest of the flow of circulation in the folds and cause intense congestion of the veins and capillaries, which may give rise to small hemorrhages into the mucosa. These hemorrhagic areas are very poorly nourished by the blood-current, and may eventually succumb to the autodigestive action of the gastric juice; other gastric contractions then loosen, and cast off these tiny spots of necrosis (Hartung, *loc. cit.*).

According to Virchow (*loc. cit.*), circulatory derangements of the larger vessels of the stomach—the acute and chronic gastritis especially—if accompanied with vomiting and colicky contractions, are the cause of ulcers and erosions. Small erosions represent only the superficial stratum of the mucosa, generally only the vestibule or alveolus and the first third of the gland-ducts; the entire lower half of the mucous membrane is not cast off (Gerhardt, *loc. cit.*). The gland-duct remaining shows nothing pathological. At the sides and edges the glands become longer, and the first ones that are intact usually curve themselves over the defect, partly covering it. Recovery takes place by the simple after-growth of the remaining portions of the glands.

In three stomachs which were taken immediately after death (not later than two hours after), we observed what was undoubtedly a superficial epithelial sequestrum resting loosely upon the mucous membrane in many places of what we had every reason to believe was a perfectly normal stomach. The autodigestion in this case had been prevented by pouring 80 per cent. alcohol into the organ about fifteen minutes after death. In places, portions of mucosa half as large as a lentil seed could be dislodged by a gentle stream of water from a wash-bottle. The erosions included the inner third of the gland-duct proper (inneres Schaltstück of Stöhr), and it seems that even before they were dislodged the process of repair had already begun; for underneath small areas of necrosed superficial epithelium that were lifted from the true glandular stratum by a thin layer of lymph containing few red blood-corpuscles, cell proliferation was going on in the parietal or oxyntic cells, and in the cylindrical cells of the adjoining intact epithelium, formation of mitosis and karyokinetic figures were evident in picrocarmin and eosin stains of these sequestrations of mucosa.

It seems possible that a process of exfoliation is constantly going on in the lining membrane of the gastro-intestinal tract just as in the epidermis. It is not conceivable that the constant and continuous impact and friction of the ingesta should go on daily without causing necrosis of epithelium in places. If we should hold the normal acid chyme in the palm of our hands for three or four hours three times or more every day, we would very soon notice exfoliations of the epidermis.

In the digestive tract (for it occurs all along the small intestine) this exfoliation goes deeper than in our hands because of immediate autodigestion of the exfoliated spot. Although we have examined nearly 50 human stomachs with especial regard for this phenomenon, we have failed to detect it in but four cases, and in these the examination was limited to a very small portion of the stomach.

Even in stomachs obtained within one hour after death, and preserved by pouring alcohol or solutions of formalin into the organ, these erosions can be seen in places. We generally request a strip which begins in the esophagus, runs through the cardia saccus cœcus, entire greater curvature, and pylorus, and has a piece of duodenum attached to it. This is hardened, and in many places pieces are excised half an inch apart and imbedded in celloidin, and, cut into serial sections with the revolving microtome, stained in eosin and hematoxylin and mounted in balsam. In some cases we sectioned strips running along the lesser curvature.

In this way it was found that most of these erosions and exfoliations occur in the vicinity of the sphincter antri pylorici, about seven to ten cm. from the pylorus. At this point the muscularis has its most powerful development, and the peristalsis, and consequently the impact of the food with the mucosa, is most vigorous; hence the epithelium here has most to suffer from friction. Slight erosions can be detected in the lower part of the esophagus, where no peristalsis normally occurs but that accompanying deglutition. So the conclusion seems justifiable that very tiny exfoliations and erosions occur in all stomachs, and, in adult life, perhaps at all times. This precludes the presumption that the pieces of mucosa are lesions produced by the stomach-tube.

Boas (*loc. cit.*) thinks that coughing or defecation may cause the dislodgment of such loosened epithelium. When this process reaches such an exaggerated type as described by Einhorn (*loc. cit.*,

"Erosions of the Stomach"), it is very probable that the mucosa is made less resistant by some well-developed gastric disease (one of the forms of gastritis, carcinoma, etc.), for his patients suffered from pains, emaciation, and weakness.

Among the 46 stomachs examined by myself were 19 in which no symptoms referable to the stomach were given during life. The pieces varied from five mm. in length, and nearly as wide. Einhorn recommends intragastric spraying of a solution of 1 : 1000 of argentic nitrate for the excessive exfoliation, combined with intragastric galvanization, diet, and tonics, with a hygienic outdoor life.

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CHAPTER XIV.

THE DIAGNOSTIC SIGNIFICANCE OF FRAGMENTS OF GASTRIC MUCOSA.

One of the first to utilize these fragments for diagnostic purposes was Boas, who attributed great importance to this way of finding out the real state of the mucosa. He held that in certain conditions of suppressed secretion the differential diagnosis between a possible neurosis and a genuine gastritis with glandular atrophy was only possible by examination of such pieces of mucosa. Rosenheim (*loc. cit.*), Boas (*loc. cit.*), and Julius Friedenwald (*Medical News*, June 22, 1895), emphasize the value of qualitative and quantitative testing of rennin zymogen to differentiate between chronic gastritis with glandular atrophy and carcinoma on the one hand, and nervous dyspepsia and secondary gastritis on the other. However, Ewald (*loc. cit.*), and also Einhorn (*loc. cit.*), have asserted that absolute deficiency of rennin zymogen is not pathognomonic of atrophy, therefore it would indeed seem as if a certain diagnosis could only be made by a small piece of mucosa.

Is there any clue which can be derived from these pieces regarding the state of the mucosa in the secretory disorders? This we will try to answer in the following. Hayem, to whom we are indebted for the best histological investigations of the gastric mucosa, emphasizes that the individual elements of the mucosa, gland-ducts, superficial epithelium, and interstitial tissue can become diseased in a variety of ways; the various portions of the stomach fundus, pylorus, and cardia may exhibit different affections; and, finally, the mucosa may at different parts show different phases of disease. He distinguishes a parenchymatous and an interstitial gastritis. First, the parenchymatous:

1. Gastrite parenchymateuse hyperpeptique chloro-organique. Under this he has two sub-classes: (*a*) D'emblée—dyspeptic distress coming on at once—in the first stage of digestion. (*b*) Tardive—dyspeptic distress coming on in later stages—in one and one-half to two hours. Under this hyperpeptic parenchymatous

gastritis, Hayem means, clinically, a hyperpepsia with hyperacidity and, anatomically, degeneration of the principal central or chief cells, with proliferation of the parietal border or oxyntic cells.

2. Gastrite parenchymateuse muqueuse (gastritis mucipara), by which he means a mucous degeneration, a process taking place principally in the vestibules to the gland-ducts (which are lined

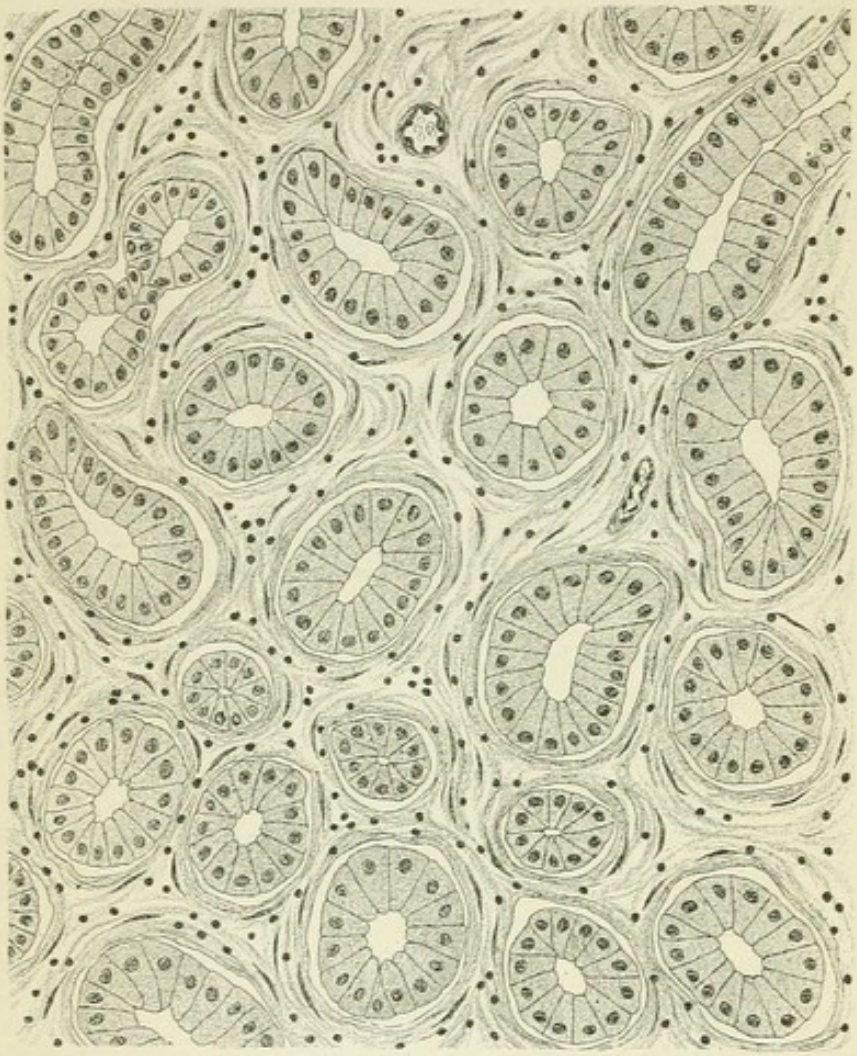

FIG. 20.—FRAGMENT OF MUCOSA SHOWING A NORMAL CONDITION OF GLANDS: very slight round-celled infiltration. The piece became detached above the level at which the oxyntic cells are found. × 600.

with columnar epithelium) and corresponds to the *Schleimkatarrh* of most German writers. This is associated with hypopepsia and subacidity.

3. Gastrite parenchymateuse atrophique, which signifies, anatomically, the total atrophy of the glands without interstitial processes, and, clinically, anacidity or achylia. The interstitial forms he separates into two classes:

(*a*) Those in which the round-cell infiltration;

(*b*) Those in which the sclerosis, *i. e.*, connective-tissue proliferation, predominates.

These processes are described as occurring purely as such, or mixed with forms of parenchymatous gastritis, and as leading to sub- or anacidity. In order to bring our results in critical consideration with those of Einhorn (*loc. cit.*), we have adopted his

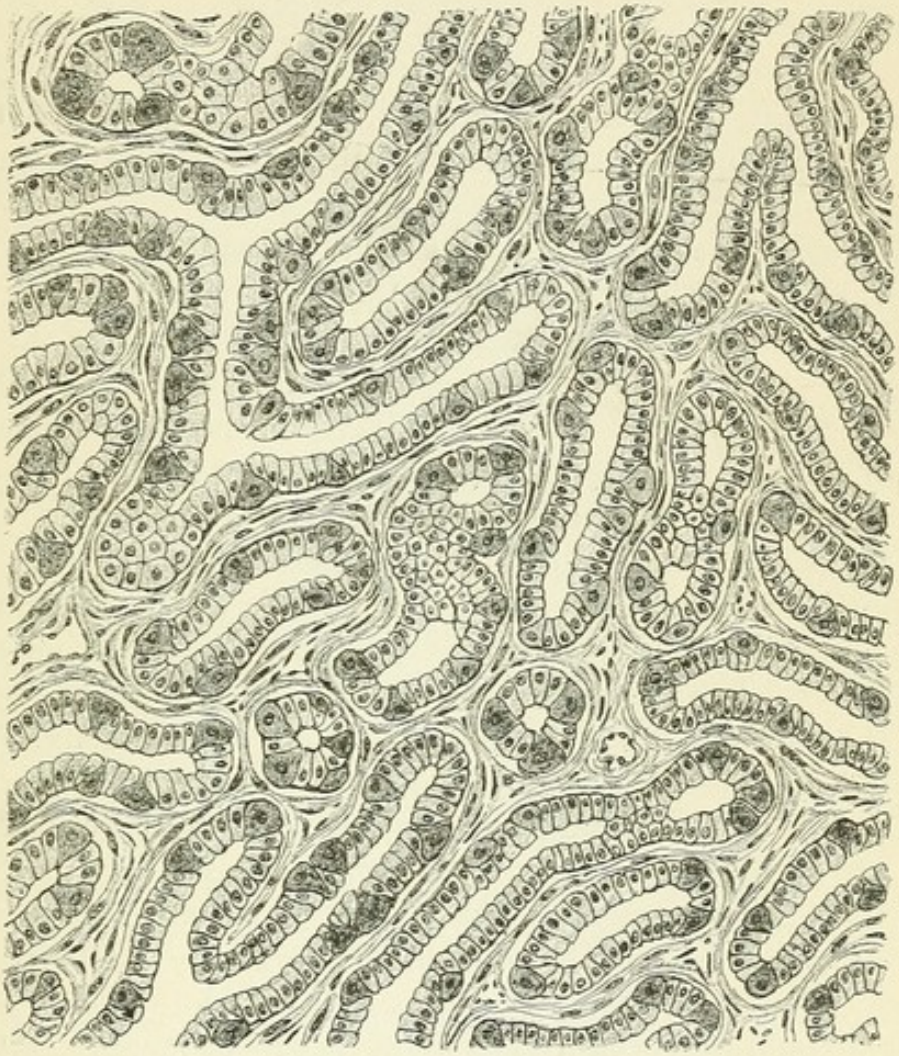

FIG. 21.—HYPERTROPHY AND PROLIFERATION OF GLANDULAR ELEMENTS.—(*From a case of persistent hyperacidity found in the eye of the tube.*) × 500.

classification of the anatomical conditions found in these fragments. There is, however, one objection that can be urged against it, and that is the apparent fact that he has based his system upon conditions of the gland-tubes and interglandular tissue exclusively, and mentions the state of the cells only once in six types described. We will therefore supplement his categories by

adding the state and condition of the vestibular or alveolar columnar cells (Vorraumzellen), and the condition and numerical relations of the chief central or ferment cells (Hauptzellen), and the parietal, border, or oxyntic cells (Belegzellen).

1. *Normal.*—The gland-ducts and interglandular tissue exist in normal proportions. The columnar epithelium of the surface and of vestibule is normal, with scattered cells showing at their free ends slight mucoid metamorphosis. Average number of parietal or oxyntic cells in six ducts which were sectioned very nearly down the center = 22–40. (Fig. 20.)

2. *Connective-tissue Excess.*—Proliferation of connective tissue around the glands—glands and epithelial cells as in normal condition.

3. *Proliferation of Glands.*—Under this class we have in 19 cases been impressed with the probability that there must be three types of this condition.

Type *a*—In this subtype there is a proliferation of gland-tubules. Under the same field of microscope there will be more than under normal conditions, since they are much closer to each other, but the number of central and oxyntic cells are from 18–42, or the same as under the normal condition.

Type *b*—Increase of oxyntic or parietal cells with normal number of gland ducts. Here there seems to be no proliferation of the gland-ducts. The connective tissue and the ducts bear the same relation as in class 1, but the anilin-staining oxyntic cells may be so increased that they lie in juxtaposition, giving the whole duct the appearance of a peptic duct of the dog; the number may reach 70 in one duct. The oxyntic cells are increased in size.

Type *c*—Increase of the number of ducts in which the number of oxyntic cells appear normal in size and number, and, in the same fragment or section, portions of mucosa in which the ducts are not augmented but the oxyntic cells are increased in number and size; this third type is a combination of types *a* and *b*. When there are many oxyntic cells above the normal the entire gland duct assumes a tortuous or elongated shape. It seldom extends down into the mucosa in the same plane, therefore it is very rare that a section will strike down the middle of a duct. Generally the counts in six ducts struck fairly along the central canaliculus are taken as an average.

4. *Incipient Atrophy.*—To the same field, under the micrometer,

there are fewer glands present than normally; they appear shrunken and smaller, at the same time the spaces between the glands are larger than normal owing to an increased connective-tissue formation; the latter is thickly invaded, as a rule, with small round-cell infiltration. The next type is—

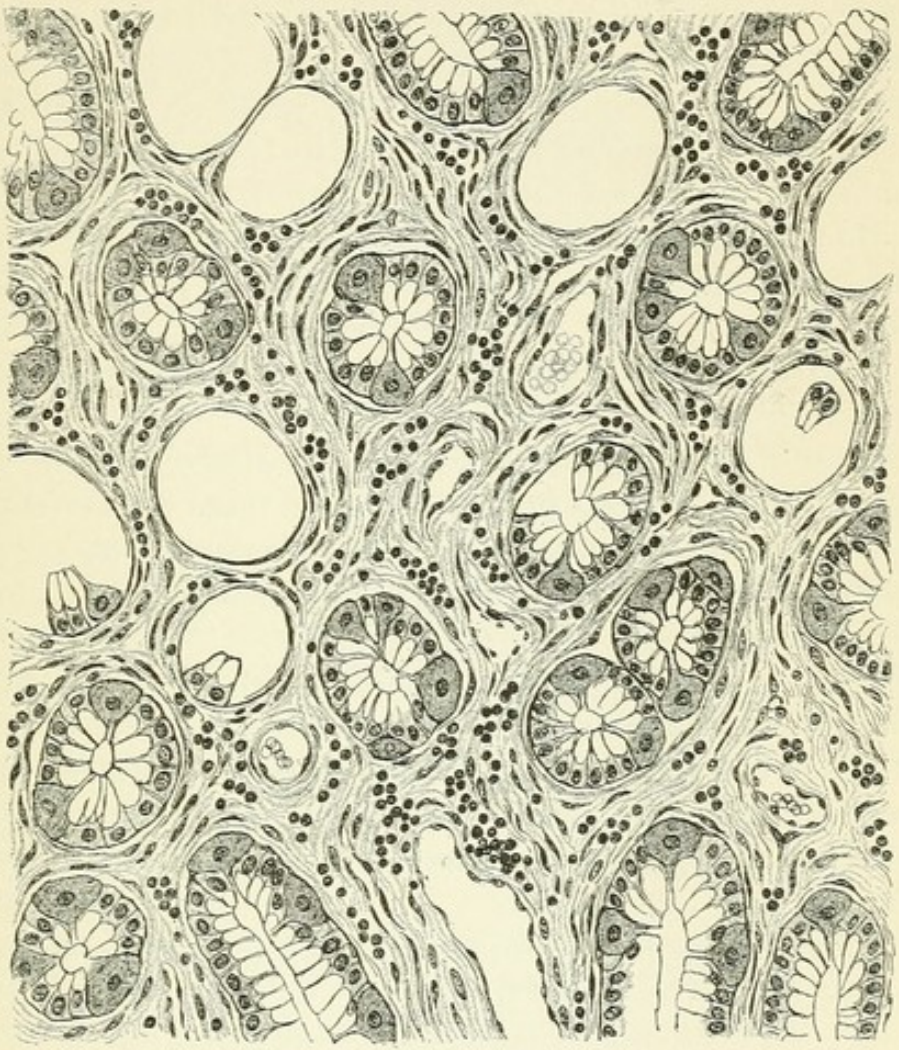

FIG. 22.—ATROPHY AND VACUOLIZATION OF GLANDULAR ELEMENTS—MUCOID DEGENERATION OF PEPTIC CELLS—INCREASE OF INTERSTITIAL CONNECTIVE TISSUE—SMALL ROUND-CELLED INFILTRATION.—(*From a case of chronic alcoholic gastritis. Found in the washwater.*) × 500.

5. *Atrophy.*—In complete atrophy there are only remnants of glands left, a few degenerated cells lying in empty circular spaces where glands had previously existed; there is also a diffuse round-celled infiltration (see Fig. 22).

6. *Vacuolization.*—Round or ovoid vacuoles exist within the glands in large numbers, being the result of mucoid degeneration of some of the glandular cells; this is generally associated with

connective-tissue proliferation (see Fig. 22). Vacuoles are present in the gland-cells normally, as can be seen in the drawings of Kupher and Stöhr. We have also seen them in both longitudinal and cross-section of the gland-tubules, but rarely more than two or three to the entire duct. It is conceivable that they may be produced by the process of hardening and imbedding. Some of the fragments obtained from stomachs may show characteristics of two types.

Deductions from 36 Cases.—In eight *healthy* persons, the mucosa fragments were normal in six; proliferation and autodigestion marked in one, which showed also beginning small round-cell infiltration between the ducts; connective-tissue increase in one. In the first of these cases the examination showed proliferation in one fragment, and a normal condition in a second found in the same washwater.

In 18 cases of *hyperacidity* the fragments of gastric mucosa found were apparently normal in four.

Atrophy of gland-tubules and connective-tissue increase, so that there were fewer glands,—but in these few there were contained a larger number of oxyntic cells than normal,—in two cases.

Proliferation of gland-ducts, with apparently normal oxyntic cells, in six cases.

Proliferation of oxyntic cells, generally without marked increase in the gland-tubules, in six cases.

In twelve cases of *anacidity* or *subacidity*, the fragment was apparently normal in two cases.

Proliferation of glands, with marked small round-cell infiltration, was found once.

Atrophy in some form was found in the fragments from the nine remaining cases.

In establishing the classification of euchlorhydria and hyperchlorhydria, we could not be guided exclusively by the amount of free HCl found after the double test-meal.

Thus, a young, vigorous farmer, aged 25, who never had any disease, showed on repeated examination an amount of free HCl equal to 60°, with a total acidity of 80°. Ordinarily, judging simply from the analysis, such a case would be diagnosed as hyperacidity, according to the principles defined in the chapter on the normal amount of HCl; however, these cases can be diagnosed justly and accurately when considered together with concomitant signs and symptoms only. Although this case had the large amount of free HCl, there was no starch indigestion, no erythrodextrin, no pyrosis; there were no symptoms referable to the stomach at all; the man was in perfect health.

Another case, a neurasthenic female, had intense suffering from hyperacidity and occasional gastroxynsis, and the amount of free HCl was never over 40°. This case showed hypermotility; the stomach, as a rule, was empty twenty-five minutes after an Ewald test-meal; with our intragastric rubber bag, in connection with the kymograph, she showed very frequent and sudden gastric peristalsis of unusual tonicity.

Summary of results from examination of fragments of mucosa:

Eight healthy persons, .	Perfectly normal in six. (*a*) Glandular proliferations, (*b*) Normal in one (same person). Connective-tissue increase in one.
Hyperacidity in 18 cases,	Normal in four. Atrophy in two. Proliferation of glands in six. Proliferation or hypertrophy of oxyntic cells in six.
Anacidity or Subacidity, .	Normal in two. Proliferation of glands in one. Atrophy in nine.

Proliferation, therefore, according to this table, is present in two-thirds of these cases of hyperacidity, and atrophy in three-quarters of these cases of anacidity or subacidity. Einhorn (*loc. cit.*) does not give any results from examination of perfectly healthy individuals, as his cases of euchlorhydria seem to be in patients.

Of the 12 hyperacid cases, three were normal, or very nearly so, six showed proliferation, and three showed connective-tissue proliferation. In his cases of anacidity, or, rather, what he calls achylia gastrica, of which there were seven cases, there was atrophy three times, marked vacuolization once, proliferation once, and normal condition twice.

On the whole, judging from Einhorn's results, Cohnheim's, Hayem's, and our own, the conclusions seem justifiable that proliferation of glandular elements is present in from one-half to two-thirds of the cases of hyperacidity; and atrophy is present in from one-half to two-thirds of the cases of anacidity.

Adolph Schmidt (*Virchow's Archiv*, Bd. CXLIII, S. 478) asserts that the epithelium of the surface of the stomach is preserved better than the gland-cells in inflammatory conditions of the mucosa. This, he says, is particularly so in chronic gastritis, which forms island-like foci in stomachs otherwise not much changed. Our experience, and that of W. D. Booker, is not in accordance with this observation (see pathology of simple, acute, and chronic gastritis in the clinical portion of this work). Although

we preserved the stomachs by injecting them immediately after death (within twenty minutes) with alcohol, also with formalin and sublimate, so that autodigestion was at once checked, our sections showed generally a more serious destruction of the surface epithelium than of the gland-cells. At times both are so much altered that it is impossible to say which is most or least affected. It seems in chronic gastritis that new epithelium will be re-formed quite rapidly where the old has been lost or destroyed.

In cases of suspected malignant neoplasm, fragments of the growth are occasionally found and are of importance in the diagnosis. In carcinoma of the cardia or the esophagus, they are most frequently found in the lower or side opening of the tube, as it must pass through or over the growth on its way into the stomach. But even in malignant growths of other parts of the stomach, patient searching in the sediment of the washwater will sometimes reward the clinician by the discovery of tumor fragments. Of the first washwater in the morning, about 500 c.c. should be permitted to settle twelve hours in a conical glass such as is used for the settling of solid urinary constituents, or the gastric contents should be brought to settle out minute particles by use of the centrifuge. The sediment should be examined under a low power (about 50 diam.). The centrifuge is preferable, as long standing of the fluid causes putrefaction.

Once we made the diagnosis of carcinoma when no tumor was evident, from repeatedly finding involuntary muscle-fibers when no meat had been eaten for six days after thorough lavage. It proved to be a broad, flat carcinoma of the posterior wall. The method of recognition of neoplastic fragments will be fully considered in the chapter on Carcinoma.

The drawing of a longitudinal section of the secreting gland-tubules, showing beautifully the well-preserved cylindrical epithelium of the gastric surface and well-differentiated oxyntic and chief cells, was made from several sections of a piece of mucosa that was torn loose by the stomach-tube, inserted by a medical student, who tried to aspirate by means of the pump a meal that had disagreed with him. The tearing off must have occurred in an instant, as there were no signs of inflammation in the sections. The sections were stained in a variety of ways, principally in the eosin, hematoxylin, Golgi, and Bismarck-brown stains. The minute communications of the oxyntic or parietal cells with the

central duct are best brought out by the Golgi method (see frontispiece).

The drawings of fragments found in the washwater, illustrating glandular proliferation, with glands closely packed and connective tissue diminished and of glandular atrophy, mucoid degeneration, vacuolization, and small-cell infiltration, are all explained by the text accompanying the illustrations. We have seen that histological changes approaching or actually representing pathological states may be going on in perfectly healthy stomachs. Furthermore, the stomachs of diseased patients may, on serial sections, show a different pathological state at different places of the mucosa. Therefore it must be borne in mind, that although the findings in hyperacidity and anacidity appear to be in some relation to the disease, this kind of investigation must not be relied upon as representing in a given fragment the condition of the entire mucosa. It represents the state of the location whence it sequestrated; that location not being accurately known, generalizations must be made with caution.

It should be emphasized that the most important conditions in these fragments are not the number of gland-ducts and the state of the connective tissue, but the relative number of oxyntic or border and chief or central cells. A fragment may show a normal or subnormal number of gland-ducts, and at the same time these may contain an abnormally large number of cells.

CHAPTER XV.

OCCURRENCE OF SECRETIONS IN THE EMPTY STOMACH.

*Stimulations to Secretions of Gastric Juice.—Significance of Foam.—Preparation of Gastric Contents.—Quantitative Analysis.—Methods.—Standard or Normal Solutions.—Indicators.—Titration.—Apparatus.**

Most authors are of the opinion that no secretion is contained in the empty stomach. Schreiber (*Arch. f. exper. Pathol. u. Phar.*, Bd. XXIV, S. 365; also, *Deutsche med. Wochenschr.*, 1894, Nos. 18 to 21), however, concludes that a secretion is found also in the empty stomach; that is, he denies a continuous secretion or gastrosuccorrhea as a disease *sui generis*, and claims to be able to obtain 60 c.c. of a secretion possessing good digestive power from a jejune, or fasting, stomach.

Pick (*Prager med. Wochenschr.*, 1889, No. 18), who obtained similar results, believed that the secretion was set up by the stimulation of the sound used. . Rosin (*Deutsche med. Wochenschr.*, 1888, No. 47), A. Hoffmann (*Berlin. klin. Wochenschr.*, 1889, No. 12), and Martius (*Deutsche med. Wochenschr.*, 1894, p. 638), have also obtained a digestive secretion from the fasting stomach.

Although there may be found 50 to 60 c.c. of a secretion possessing digestive powers in the empty, normal stomachs of perfectly healthy individuals, this does not prove that a continued secretion exists normally (Riegel, *Deutsche med. Wochenschr.*, 1893, p. 735). Leo ("Krankheiten d. Bauchorgane," p. 54) considers this digestive secretion a residuum of the last previous meal, and seems to have shown conclusively that such a residuum is constantly present in

* The section on quantitative chemical analysis of gastric contents and the chapters on the condition of the blood and urine in gastric diseases and on the gases of the stomach have been written by my associate, Dr. Edward L. Whitney, whose experience as demonstrator of clinical pathology has admirably fitted him for the concise and clear account of this department. It gives me pleasure to express my thanks to him for his assistance. —(J. C. H.)

the stomachs of infants after a night's sleep (see Leo, *Berlin. klin. Wochenschr.*, 1888, No. 49). For the practical objects of diagnosis he concludes that a secretion of 50 to 60 c.c. of digestive fluid found in a fasting organ must not be considered pathological. Only when the amount gained reaches 100 to 300 c.c. does it indicate hypersecretion, which is often associated with hyperacidity (Reichmann, *Berlin. klin. Wochenschr.*, 1887, S. 12; Bouveret [*loc. cit.*]; Débove, and Rémond, "Les Maladies de l'Estomac"). Riegel and Reichmann do not distinguish sufficiently between so-called continuous secretion of gastric juice with a stomach of normal capacity and normal exit to the duodenum and continuous secretion which appears as a concomitant symptom of gastrectasia with probable pyloric stenosis. Einhorn asserts that, with more accurate differentiation between these states, it will probably be found that the normal stomach in a fasting condition contains very little if any secretion. We have seen a number of cases whose stomachs were of natural size and where there was no disturbance, but which contained this secretion early in the morning before breakfast.

J. Schreiber (*Deutsche med. Wochenschr.*, 1894, No. 53) has experimented upon two healthy persons, before any food had been taken, and found gastric juice with hydrochloric acid in both. The amount of secretion thus obtained varied from 10 to 22 c.c. Martius (*Deutsche med. Wochenschr.*, 1894, No. 32) and Huber (*Korrespondenzblatt f. Schweizer Aerzte*, 1894, No. 49) confirm Schreiber's results. According to Ewald, who sums up the literature (in Lubarsch and Ostertag's "Ergebnisse d. spez. Pathologie," Bd. III, S. 27), and gives his own observations in a large number of cases, this problem is represented in the following manner: In many individuals small quantities of a digestive secretion containing free hydrochloric acid can be obtained from the fasting, or jejune, stomach. Sometimes it is mixed with bile, coloring matter, and duodenal contents. But he claims that the stimulation to this secretion has been furnished by swallowed saliva (Martius), remnants of food, pharyngeal secretion, etc., and that the state of things lies between a normal and an abnormal one, and that there is no diseased condition of the gastric mucosa.

In the case of typical gastrosuccorrhea, however, there is a much increased irritability of the mucosa, giving rise eventually to a profuse secretion, which, when found in empty stomachs, is quantitatively more considerable than that found in normal jejune

stomachs. Huber compares it to a slow, gradual dying away of the tonus of secretory irritability ("Abklingen des Sekretionsreizes") that has been set up by the ingesta and seems to linger after they have passed into the duodenum.

In order to obtain gastric secretion a variety of methods have been suggested:

By chemical stimulation, according to Leube's method, which consists in allowing 50 c.c. of a three per cent. solution of sodium bicarbonate to flow into the stomach. After twelve minutes this is washed out again, and should be found neutral. By thermic stimulation, according to Jaworski's method, consisting of the introduction of 100 c.c. of ice-water and washing it out again after ten minutes, when it should contain acid and pepsin. These methods, if successful at all, bring out the gastric juice in a most diluted state, and therefore give no adequate means of determining the secretion by chemical analysis. It has been claimed by Einhorn (*New York Medical Record*, November 9, 1889) and Allen A. Jones (*ibid.*, 1891) in this country, and Hoffmann (*Berlin. klin. Wochenschr.*, 1889, No. 13), Ewald (*loc. cit.*), and Ziemssen in Germany, that the gastric secretion, as evinced by the amount of hydrochloric acid, could be increased by faradic or galvanic stimulation. While we have our doubts about this matter, we do not wish to imply that electricity is not a very valuable therapeutic agent in the treatment of secretory diseases; we could not, in fact, dispense with it as an auxiliary to treatment. In our opinion, the influence of electricity on secretion is still doubtful.

As a means of obtaining gastric secretion, this method is certainly not available. The normal secretions are best obtained by the natural stimulation of one of the test-meals as stated in a previous chapter.

Mathieu and Rémond (*Société de Biolog.*, 1890) have published a method of determining the total quantity of stomach contents by finding out the acidity of the undiluted contents as much as can be drawn; then that of the contents as much as can be gained by washing out the stomach with a known quantity of water, and from this the acidity of the total amount of contents that were originally in the stomach are calculated. Strauss (*Therapeutische Monatshefte*, März, 1895) has simplified this procedure, but for the practitioner it is sufficient to know the amount gained by the simple methods of drawing the contents by expression or aspiration. Concerning

the recognition of proteid and carbohydrate indigestion from the food-remnants, it should be added this is much facilitated by the double test-meal used at our hospital (Maryland General Hospital).

In gastrectasias, presence of foam indicates gas-fermentation. Gas may be found even in presence of normal or supernormal amount of hydrochloric acid, since F. Kuhn (*Zeitschr. f. klin. Med.*, Bd. XXI, and *Deutsche med. Wochenschr.*, '92, No. 49) has demonstrated that the hydrochloric acid of gastric juice has no detrimental effect on large amounts of yeast. Whenever there is stagnation of gastric contents this gas-formation can occur.

After the contents of the stomach are withdrawn, they must be prepared for and submitted to chemical examination. The contents may be beaten up thoroughly to make a homogeneous mixture, and the chemical examinations conducted on this mixture, or this mixture may be filtered and the clear filtrate subjected to analysis. The former method gives the more accurate results, with slightly higher acidity, than the latter method, which has the advantage, however, of allowing better observation of color changes in the solution during titration.

Before entering upon a discussion of the chemical methods as applied to the gastric juice, a short description of the methods, solutions, and apparatus required in quantitative analysis will be given.

The solutions required can be made up, and, if preserved from the influence of light and air, can be kept indefinitely.

The methods used in quantitative chemical analysis may be divided into two general classes: *Gravimetric* and *Volumetric*. The gravimetric methods consist of the isolation of the substance or one of its compounds, which is weighed. The isolation of substances in a pure state often requires long training in chemical methods, and if a small amount of the substance in question is present it may be very difficult to separate a weighable amount unless large quantities of the mixture are available. Many substances can not be separated from mixtures without losing at the same time their relation to other substances in the same solution. The great objection to the gravimetric method, however, is the large amount of costly apparatus necessary, and the length of time needed for the manipulations.

The volumetric methods are more easily performed; in this, the quantity of the substance under examination is ascertained by a

calculation based upon a measured quantity of a solution of a known strength required to perform a certain reaction with it. These solutions, called standard solutions, are of two kinds, normal solutions and empirical solutions.

A normal solution is one which contains in a liter a quantity of the active reagent expressed in grammes and chemically equivalent to one atom of hydrogen. In other words, a normal solution contains in 1000 c.c. an amount of the active constituent just sufficient to combine with or replace one gramme of hydrogen.

Decinormal solutions, N_{10}, are one-tenth the strength of normal solutions.

Centinormal solutions, N_{100}, are one-hundredth the strength of normal solutions.

Empirical solutions are those which do not contain an exact atomic proportion of the reagent, but are made up of such strength that one c.c. is equivalent to some definite weight of the substance sought.

Residual titration, or back titration, consists in treating the substance under examination with standard solution in excess of that known to be required; the excess is then ascertained by residual titration with another standard solution.

In general, titration results in the formation of a compound that can be distinguished by its properties from those substances present in either solution.

1. It may form a precipitate.

2. It may cause the complete solution of some precipitate.

3. A slight excess of either reagent may produce some visible change in some constituent of the solution, or a change in some substance added for the purpose (indicators).

4. The indicator in some cases can not be added to the solution, but from time to time a few drops of the solution are added to the indicator on a watch-glass at the side.

Of the above the normal solution is the most used, the empirical solution being only of limited application.

It would seem a simple matter to make up a standard solution which would be perfectly accurate, but in practice the problem is not so simple. Absolutely pure chemicals are not easily obtained, and such as are easily obtained, unmixed with other mineral substances, contain a variable amount of water, and are, moreover, exposed to more or less danger of contamination from the impuri-

ties of the air. The following methods of obtaining a tenth-normal solution are recommended as a basis for the preparation of other solutions.

1. Pure, dry oxalic acid is obtained, and the crystals that show no sign of efflorescence selected. From the formula, $C_2H_2O_4 + 2H_2O$, it is seen that the molecular weight is 126, and as it is a dibasic acid the normal solution would contain one-half of this (63 gm.) dissolved in distilled water and made up to one liter at a temperature of 15° C. As a tenth-normal, N_{10}, solution is required, one-tenth of this, or 6.3 gm., are made up to a liter as before, and used to correct the solutions employed in analysis. It must be noticed that oxalic acid in dilute solution soon decomposes, so that it must be freshly prepared as required.

To prepare an equivalent solution of caustic soda (decinormal NaOH) about five gm. of caustic soda are dissolved in about 900 c.c. of distilled water and well mixed. To this there is added lime-water or baryta water $Ca(OH)_2$ or $Ba(OH)_2$ as long as a precipitate forms, to get rid of carbonates or sulphates. The solution is allowed to stand until the impurities have settled. Twenty-five c.c. of the solution are then measured with a pipette into a clean flask or beaker and titrated with the above solution of oxalic acid, using a few drops of phenolphthalein as an indicator, until the red color of the solution just disappears. The solution is then diluted to the strength of a decinormal solution.

As an illustration of the method of ascertaining the amount of dilution necessary to make the two solutions exactly equivalent, we will suppose that the 25 c.c. of caustic soda solution required 28.3 c.c. of the oxalic acid solution to discharge the red color. If 25 c.c. of the caustic soda solution neutralize 28.3 c.c. of the acid solution, then the amount of caustic soda solution necessary to neutralize 1000 c.c. of the acid solution will be found by the following proportion:

$$28.3 : 25 : : 1000 : (X) \qquad X = 883.4$$

X = amount of caustic soda solution necessary for 1000 N_{10} NaOH. Dilute 883.4 c.c. of the caustic soda to 1000 c.c. with distilled water.

After diluting the solution, it should be again titrated to ensure its accuracy, and if properly standardized it should be changed from red to colorless and vice versâ by the addition of a drop or two of

the acid or alkaline solutions, respectively. The titration should be conducted as rapidly as possible to avoid the error produced by absorption of CO_2 from the air, and all solutions kept in well-stoppered bottles for the same reason.

2. About eight gm. of pure, dry sodium carbonate are heated in a platinum crucible for ten minutes at a *dull-red* heat, stirring occasionally with a platinum wire. After heating, it is powdered in a warm mortar and allowed to cool in a desiccator. When cool, 5.3 gm. of the powder are weighed rapidly, washed into a flask with hot distilled water, and made up to a liter. This constitutes a decinormal solution of sodium carbonate.

A decinormal solution of sulphuric acid is prepared in the following manner: About three c.c. of the pure, strong acid, of a specific gravity of 1.840, is made up to about 900 c.c.

The approximate solution is standardized against the sodium carbonate solution prepared as above, using a drop or two of a 0.1 per cent. solution of methyl-orange as an indicator. Twenty-five c.c. of the acid solution is titrated with the decinormal sodium carbonate until the red color shown by this indicator in acid solution turns to a light yellow. The correction of the approximate solution is made from a proportion upon exactly the same principle as in the former case (No. 1).

To correct this decinormal solution of sulphuric acid for very accurate work, the following method is recommended: One hundred c.c. of the decinormal solution of sulphuric acid is alkalinized with a strong solution of pure ammonia (ammonium hydrate). The solution is evaporated on the waterbath, heated to 105° C. in hot-air bath, cooled, and weighed. The amount of sulphuric acid is calculated from the amount of ammonium sulphate formed.

Indicators.—An indicator is a substance used in volumetric analysis, which indicates, by change of color or some other visible effect, the exact point at which a given reaction is complete.

Generally the indicator is added to the substance under examination, but in a few cases it is used outside, a drop of the solution being brought in contact with a drop of the indicator.

The particular uses of the indicators will be more fully explained in their proper places, under the quantitative examination of the gastric juice, but the chief ones in use in such examinations may be briefly mentioned.

Tincture of litmus, which turns red in acid solution, blue in an

alkaline solution. It is used in solution, and also in the form of test-papers. (It is not used when carbonates are present.)

Phenolphthalein solution, a one per cent. solution of phenolphthalein in alcohol, colorless in acid solutions, red in alkaline solutions. Is not reliable for alkaline phosphates, bicarbonates, or ammonia.

Methyl-orange solution, a 0.1 per cent. solution of methyl-orange in water, turns red with acids, yellow with alkalies. It is not affected by carbonic acid, and is valuable for titration of alkaline carbonates.

The other indicators and their uses in analysis of the gastric juice will be mentioned later.

Apparatus.—The apparatus needed for volumetric work is comparatively simple,—burettes, measuring-flasks, measuring-cylinders, and pipettes. An accurate balance is required in all chemical work, delicate to a milligramme and weighing up to say 50 gm. Burettes are glass tubes graduated to tenths of a c.c. and holding from 25 to 100 c.c. They are provided at the lower end with a rubber tube and pinch-cock, by means of which the amount of the solution can be accurately regulated. The tube is graduated upon its outer surface, and the amount of the solution used can be read off from this graduation. The simplest form of burette is the one already described, known as Mohr's, of which various modifications are in use.

Measuring-flasks are vessels made of thin glass having a narrow neck, and so constructed that a certain amount of fluid reaches a graduation placed about the middle of the neck. These flasks are of various sizes,—100, 200, 250, 500, and 1000 c.c.

Measuring-cylinders are of various sizes, from 25 to 1000 c.c.; graduated from 0.5 to five c.c.

Pipettes are of two kinds,—those graduated for one quantity only, and those graduated on the stem to deliver various quantities. A convenient set for stomach-work is the following: two, five, ten, and 25 c.c.

In addition to the above apparatus, one should be supplied with funnels, crucibles, beakers, flasks, test-tubes, Bunsen burners, etc.

All apparatus should be kept scrupulously clean, rinsed before and after using with distilled water. It is well to wash the inside of any measuring-apparatus two or three times with small portions of the solution for which it is to be used.

Greasiness interferes very much with accurate reading. It may be removed by dilute alkaline solution.

The burette should be placed perfectly perpendicular, and firmly fastened. Fill by a funnel, the stem resting against the inner surface of the burette to avoid the formation of bubbles. Always fill above the zero mark; gently tap the burette until the bubbles disappear should they be formed. Then run out a small portion (or down to the zero mark), remembering to run out enough to remove all air-bubbles from the bottom of the burette.

In reading the results, always read from the bottom of the meniscus formed by the rising of the outer borders of the liquid along the sides of the burette.

CHAPTER XVI.

CHEMICAL EXAMINATION OF GASTRIC JUICE.

Tests for Presence of Free Acids.—Tests for Free Hydrochloric Acid.—The Dimethyl-Amido-Azo-Benzol Test.—The Resorcin Test.—Combined Hydrochloric Acid.—Lactic Acid: Formation, Significance, Detection.

Reaction.—The reaction of the gastric juice, obtained by means of the stomach-tube or otherwise, after the administration of a test-meal, is always acid in the normal individual. The reaction is best determined by dipping into the juice a piece of very delicate blue litmus paper. In juice of acid reaction the paper immediately turns red. Very rarely is the reaction alkaline, this being found only in a few cases of atrophy of the gastric mucosa, occasionally in acute gastritis, and when, for some reason, a portion of the intestinal contents and the alkaline bile has been forced through the pylorus in sufficient quantity to neutralize the acid of the stomach.

In severe cases of gastric atrophy the reaction is usually acid, even in absence of fermentative changes. This is due to the presence of acid salts, such as acid sodium phosphate (NaH_2PO_4), and of traces of organic acids, which occur in nearly every test-meal in quantities sufficient to produce an acidity of from six to ten degrees.

Tests for Presence of Free Acids.—A delicate test for the presence of free acids is found in Congo-red. This substance occurs as a fine reddish-brown powder, dissolving readily in water to form a clear deep-red solution, which changes in the presence of free acids to a dark blue. It may be used in two ways as an indicator.

1. A solution is prepared by dissolving one gm. of the powder in 100 c.c. of water, and adding a drop to a few c.c. of the gastric juice. If the juice contains even a slight trace of free hydrochloric

acid, or the organic acids in slightly larger quantities, the solution immediately turns a bright blue.

2. A test-paper may be prepared by soaking bibulous paper in the above solution of the dye for several hours and then carefully drying. This paper is simply dipped into the filtrate, or into the contents before filtration, and exhibits the same color reaction as the solution mentioned above, and has the additional advantages of being more convenient and exhibiting as readily, slight changes in color. It has been found, also, that when the acidity is due to organic acids and not to free hydrochloric acid, the color can be made to disappear by warming gently over the open flame. If the acidity is due to hydrochloric acid, on the contrary, the dark-blue stain on the paper changes to a lighter tint, but does not disappear except when strongly heated.

It must be emphasized that this color-change from red to blue does not occur in solutions of acid salts or in the presence of combined hydrochloric acid, and therefore indicates the presence of some free acid,—inorganic or organic.

Tests for Free Hydrochloric Acid.—Many tests have been proposed for free hydrochloric acid, the following, given in the order of their accuracy and delicacy, being probably the most reliable:

1. Dimethyl-amido-azo-benzol, ... 0.02 pro 1000
2. Phloroglucin-vanillin, ... 0.05 "
3. Resorcin, ... 0.05 "

The Dimethyl-Amido-Azo-Benzol Test.—This test, recently introduced by Töpfer, is probably destined to replace all others in the clinical laboratory, both on account of its simplicity and also on account of its ready application to the direct quantitative estimation of the amount of free hydrochloric acid in the gastric juice. This indicator occurs in the form of a brown powder, readily soluble in alcohol, only slightly soluble in water. A few drops of the alcoholic solution, added to a solution of hydrochloric acid, turns a bright cherry-red, increasing in intensity as the strength of the acid solution is increased. In the absence of free hydrochloric acid or other mineral acid the solution turns a bright lemon-yellow.

In actual practice a 0.5 per cent. solution of the substance in alcohol is employed. A few drops of this solution are added to the stomach contents, which need not be filtered for this purpose, or to the residue left in the receptacle in which the stomach con-

tents were received. If free hydrochloric acid is present the cherry-red color develops and spreads in beautiful rings from each drop of the indicator, usually leaving in the center a clear, yellow area. In case the indication is doubtful the following modification may be employed: A small porcelain evaporating dish (or white butter plate) is thoroughly rinsed with distilled water and dried. Upon one side of the dish a few drops of the filtrate are placed, and upon the opposite side a single drop of the indicator. By inclining the dish gently the two solutions may be made to mix, and at the line of junction the cherry-red color may be seen, the white background rendering the detection of the tint less difficult.

It has been stated by Einhorn and others that this test is liable to mislead in cases in which there is a large amount of organic acidity. It is true that in the presence of lactic acid amounting to 0.2 per cent. or more in gastric juice this test yields a red color, resembling that due to inorganic acids, but the objection is more theoretical than real, as the presence of such an amount of organic acids seldom occurs in the stomach, and in the presence of proteids, peptones, mucin, etc., still stronger solutions of the organic acids are required to produce the characteristic reaction.

Furthermore, the quantitative estimation of organic acidity to be described presently will show the necessity of employing further tests for the presence of free hydrochloric acid, on account of a specially great acidity of organic acids, which does not, as a rule, occur in a stomach secreting a normal amount of hydrochloric acid.

The Phloroglucin-vanillin Test.—The modification of this test proposed by Boas gives the most satisfactory results. Two gm. of phloroglucin and one gm. of vanillin are dissolved in 100 gm. of 80 per cent alcohol. The solution must be kept in a dark-colored, well-stoppered bottle, as the solution soon decomposes when exposed to the light. The original Günzberg formula was composed of the same amount of the ingredients dissolved in 30 c.c. of absolute alcohol. This solution still more readily undergoes decomposition and has no advantages over the above modification. The solution is employed in the following manner: Four or five drops of the reagent are mixed on a small porcelain dish or small butter plate with an equal amount of the filtered gastric juice or the unfiltered gastric contents. This is placed on a waterbath, kept just below the boiling point, and evaporated slowly. If free hydrochloric acid be present in the proportion of 0.5 pro thousand

or more, a fine rose tint will develop at the edge of the drop where the mixture is dried.

The mixture may be evaporated over a naked flame with the same results, provided the temperature is not raised above the boiling point. If too much heat is applied, a brown or brownish-red color may develop, which resembles the color produced where free hydrochloric acid is absent. The rose color produced by this reagent comes only from free mineral acids; organic acids, acid salts, combined hydrochloric acid, peptone, and albumose produce only a brown or yellowish discoloration.

The Resorcin Test.—The solution consists of five gm. of resorcin (resublimed), and three gm. of cane sugar dissolved in 100 c.c. of 94 per cent. alcohol. Six drops of the filtered gastric juice and three drops of the solution are mixed on a porcelain plate and slowly evaporated as in the phloroglucin-vanillin (Günzberg) test. Care must be employed that too much heat is not applied, as heating too strongly simply yields a brown or black deposit. If the operations be properly conducted and free hydrochloric acid be present, a fine vermilion-red line forms at the edge of the drops, following down the edge of the solution as evaporation proceeds, while the color at the periphery gradually fades, disappearing entirely after a short time, leaving a reddish-brown stain. This test has the same degree of delicacy as the phloroglucin-vanillin test and the advantage of much greater stability, retaining its delicacy for months, while the latter lasts only a few weeks.

Many other tests might be mentioned, some of them much less delicate, among them Tropæolin 00, Mohr's reagent, methyl-violet, and emerald green, but the three described will be found the most reliable and easily applied.

Combined Hydrochloric Acid.—If albuminous bodies are treated with a weak solution of hydrochloric acid, it is found that a certain amount of the hydrochloric acid combines with the albuminous bodies to form compounds which do not give the reactions of free hydrochloric acid. In other words, certain affinities of the albuminous substance must be saturated before hydrochloric acid appears in the free state. In the stomach the same reaction must take place, except probably to a greater extent, due to the more complicated chemical processes through which these substances pass. This is shown by the fact that even after a simple test-meal a certain amount of time elapses before the presence of free hydro-

chloric acid can be demonstrated. In the Ewald meal, from twenty to forty minutes elapse before free hydrochloric acid can be demonstrated in the normal individual, while in the more complex meals considerably more time is required. This form of hydrochloric acid is important, inasmuch as it constitutes a part of the physiological hydrochloric acid, and stomach digestion will proceed in a fairly normal manner, if enough hydrochloric acid is secreted to saturate these affinities, while not enough is secreted to form the excess or reserve supply called free hydrochloric acid. It is evident, therefore, that if free hydrochloric acid be present, all these affinities must be saturated, while in its absence some hydrochloric acid, enough to more or less saturate these affinities, may have been secreted. The entire absence of hydrochloric acid, both free and combined, if more than temporary, is a serious condition, indicating an atrophy of the gastric mucosa, a severe gastric catarrh, or achylia gastrica. From these considerations it will be seen how important the determination of the combined hydrochloric acid is, in all conditions of anacidity. The estimation and quantitative determination of the combined hydrochloric acid will be deferred to the paragraphs devoted to the quantitative determination of hydrochloric acid.

The amount of pure hydrochloric acid necessary to combine with 100 gm. (or 100 c.c.) of the various food-stuffs will be given in the chapter on the Therapy of HCl.

Lactic Acid; Formation, Significance, Detection.—It was formerly supposed that lactic acid was secreted by the stomach, but by the more accurate investigations of later years it has been shown beyond doubt that lactic acid in the gastric contents is either introduced as such in the food or is the product of abnormal fermentative changes in the food after ingestion.

Lactic acid may be introduced in food either as sarcolactic acid from meat or fermentation lactic acid found in bread and other starchy foods. Lactic acid may be formed after the food is ingested, in cases of carcinoma of the stomach, and probably also in small amounts in other conditions, of subacidity or anacidity associated with deficient motility.

In the great majority of cases of carcinoma of the stomach lactic acid is present in considerable amounts, except in those cases in which the motility is not impaired. In such cases only a small

amount of lactic acid can usually be demonstrated, and in some cases it is absent. There are cases of carcinoma of the fundus or body of the stomach in which the motility is so good that at the end of one hour no remains of the test-meal can be regained.

Small traces of lactic acid can usually be detected for some time after the administration of the Ewald breakfast or similar meals, though at the height of digestion the usual tests are negative, due either to the absorption of the lactic acid, or the interference of free hydrochloric acid, or the products of digestion with the delicacy of the tests. In cases in which it is desirable to prove the formation of lactic acid within the stomach, it is necessary to employ some meal which is entirely free from lactic acid.

Such a meal has been proposed by Boas, consisting of oatmeal gruel to which only a little salt has been added. The stomach is washed out on the evening preceding the administration of the meal until no food-particles can be found, the gruel given in the morning and the contents removed one hour after.

Only rarely, under such conditions, is any notable amount of lactic acid to be demonstrated except in cases of carcinoma of the stomach. The easiest clinical test for the presence of lactic acid is that of Uffelmann. Ten c.c. of a 4 per cent. solution of carbolic acid are mixed with 20 c.c. of water, and a drop of a strong solution of ferric chlorid added. A beautiful amethyst-blue color is produced which turns a canary-yellow when treated with a solution of lactic acid or gastric juice containing lactic acid. The delicacy of this test is interfered with by the presence of free hydrochloric acid and peptones. Glucose, acid phosphates, and alcohol give a reaction resembling that of lactic acid, butyric acid giving a much lighter tint. In case of doubt, a modification that has given good results is the following: Five or ten c.c. of the filtered gastric juice are treated with ten times its volume of ether, free from alcohol, and then shaken in a stoppered separating funnel for fifteen or twenty minutes and allowed to stand till the layers have separated. The ethereal solution is allowed to evaporate, the residue dissolved in five or ten c.c. of water, and the solution tested for lactic acid as above. While this test is not a very delicate one, lactic acid when present in considerable amounts gives a more decided reaction than any of the substances mentioned as having a similar reaction, and it is a good test for clinical purposes.

Boas' method is to be employed in doubtful cases. This method is based upon the fact that when lactic acid is treated with strong oxidizing agents, formic acid and acetic aldehyd are formed.

$$C_3H_6O_3 = CH_3COH + HCOOH.$$

Acetic aldehyd may be easily recognized by its action on Nessler's reagent, or upon an alkaline solution of iodin in iodid of potassium. Nessler's reagent is prepared in the following manner:

One hundred c.c. of a four per cent. solution of iodid of potassium is warmed, and while warm treated with iodid of mercury until a small amount remains undissolved. After cooling, 40 c.c. of water are added. Two parts of this solution are then treated with three parts of a strong solution of caustic potash; any precipitate which may form is filtered off and the reagent kept in a well-stoppered bottle.

The solution of iodin is prepared by mixing a solution of iodin in iodid of potassium with caustic potash or potassium carbonate.

Method.—The filtered gastric juice is tested for the presence of free acids as above, and, if present, 10 or 20 c.c. are treated with an excess of barium carbonate. If no free acids are present, this is not necessary. The solution is now evaporated to a syrup on the waterbath to drive off the fatty acids. The syrup is treated with a few drops of phosphoric acid and brought to a boiling point to expel carbon dioxid. After cooling, it is extracted with 100 c.c. of ether by shaking for half an hour. After standing for a short time to allow separation to take place, the ethereal layer is drawn off and evaporated (avoiding a flame), the residue taken up in 45 c.c. of water, shaken and filtered. The filtrate is treated in an Erlemeyer flask with five c.c. of strong sulphuric acid and as much black oxid of manganese as will lie on the point of a knife-blade. The flask is closed with a perforated stopper, in which is placed a bent glass tube, the long arm passing into a cylinder filled with 10 or 15 c.c. of Nessler's reagent or alkaline iodin solution prepared as described. Carefully heat the flask, and if lactic acid is present, aldehyd will distil over forming aldehyd mercury, yellowish-red in color, if Nessler's reagent is used, and yellowish crystals of iodoform, which may be recognized by their odor, if the alkaline solution of iodin is employed.

Butyric acid can usually be determined by its odor alone, which is that of rancid butter. In case of doubt, ten c.c. of the

gastric juice are extracted with 50 c.c. of ether, the ethereal solution evaporated, and the residue taken up with water. The odor is more evident in this concentrated aqueous solution. A small amount of calcium chlorid causes the separation of an oily layer of butyric acid; strong mineral acids also separate the oily layer or drops of the acid.

Acetic acid may also be detected by its odor.

Ten c.c. of the gastric juice are extracted with ether, the ether evaporated, the residue taken up with a small amount of water, accurately neutralized with caustic soda solution, and mixed with a few drops of a very dilute solution of ferric chlorid. In the presence of acetic acid this gives a dark-red color.

The ethereal residue after evaporation is taken up with a small amount of strong sulphuric acid and alcohol. If acetic acid is present, the fragrant odor of ethyl acetate is easily detected.

Fatty acids do not occur normally in the stomach contents. Butyric acid may be formed when a large amount of milk or carbohydrates have been ingested, usually associated with an excess of lactic acid. It has been shown also that butyric acid can be formed from lactic acid.

Acetic acid, on the contrary, is a product of alcohol, and may be formed from alcohol ingested or from alcohol produced by the action of yeast upon the sugar contained in the stomach contents. Hence it follows that it is necessary to exclude alcoholism before significance is attached to the presence of acetic acid in the stomach contents. If, in the case of acetic acid, alcoholism be excluded, and, in the case of butyric acid, the ingestion of butter or fats in general be excluded, the presence of these acids has the same significance as the occurrence of lactic acid, viz., stenosis of the pylorus with dilatation and fermentation.

CHAPTER XVII.

QUANTITATIVE ANALYSIS OF THE STOMACH ACIDS.

Numerous methods have been devised for the estimation of the amount of free hydrochloric acid present in the gastric juice. The most convenient method of estimation for clinical purposes is that of Töpfer, which at the same time estimates the acidity due to organic acids and acid salts, and that due to the combined hydrochloric acid.

Töpfer's Method.—Three indicators are used in this method:

1 A 0.5 per cent. alcoholic solution of dimethyl-amido-azo-benzol.

2. A one per cent. aqueous solution of alizarin (alizarin monosulphonate of sodium).

3. A one per cent. alcoholic solution of phenolphthalein.

1. As has been mentioned under the head of tests for free hydrochloric acid, dimethyl-amido-azo-benzol reacts to very faint traces of mineral acids, particularly hydrochloric, but to organic acids only when present in very large amounts, and not at all to combined hydrochloric acid or acid salts. It will be seen that by this indicator we can easily find the amount of free hydrochloric acid. Töpfer's method gives results as reliable as those of the improved Sjöqvist's or Braun's method, according to Paul Hàri (*Arch. f. Verdauungskrankheiten*, Bd. 11, S. 332).

Ten c.c. of the filtered gastric juice are measured into a small clean flask, and a few drops of dimethyl-amido-azo-benzol added. The solution turns a bright red in the presence of free hydrochloric acid. The solution is now titrated with a decinormal solution of caustic soda (prepared as above), until the red color of the solution changes to a clear yellow.

2. Into a second beaker or flask ten c.c. of the gastric juice are measured, a few drops of the alizarin solution added, and the solution titrated with the decinormal solution of caustic soda until the solution turns to a clear violet color.

As this tint is difficult for the unpracticed eye to recognize, Töpfer recommends the following preliminary tests:

(*a*) To five c.c. of distilled water add two or three drops of the alizarin solution. A clear yellow color results.

(*b*) To five c.c. of a one per cent. solution of disodium phosphate add the alizarin solution as above. A reddish color with a slight tinge of violet results.

(*c*) Five c.c. of a one per cent. solution of sodium carbonate when treated with alizarin, as above, give a clear violet tint, which is the tint to be reached in the titration. Until the eye becomes accustomed to the reaction, it is well to prepare this solution as a guide in the titration.

3. To a third portion (ten c.c.) of the filtered gastric juice two or three drops of phenolphthalein solution are added and the solution titrated with the decinormal solution of caustic soda. After a certain amount of the solution has been added, a light-rose color develops, which is not, however, the end of the reaction. It will be noticed that as the drop of caustic soda solution falls into the solution, a dark-red color is produced at the point of contact, fading into rose color on agitation. The titration must be carried on until the entire solution has reached this color and no line of separation can be made out on adding a drop of the caustic soda solution.

There are two ways of stating the result of the titrations. The simplest method is to state the number of c.c. of the caustic soda solution which would be necessary to neutralize 100 c.c. of the gastric juice, as that number of degrees of acidity. For example, the number of c.c. of the caustic soda solution necessary to neutralize ten c.c. of the gastric juice, using dimethyl-amido-azo-benzol as an indicator, is 2.3 c.c. One hundred c.c. would then require ten times that, the amount of acidity being stated as 23 degrees = 23 c.c.

The second method of stating the results is to give the amount of acid per thousand in terms of hydrochloric acid. As each c.c. of the solution of caustic soda will neutralize 0.00365 gm. of pure hydrochloric acid, the above example would show 0.8395 gm. of hydrochloric acid per thousand, or 0.08395 per cent.

As an example of the calculations employed in Töpfer's method, let us suppose that in the titration (1) with dimethyl-amido-azo-benzol as an indicator, 3.5 c.c. of caustic soda solution were employed, (2) with alizarin 4.9 c.c. of the caustic soda solution were required, and (3) with phenolphthalein 7.5 c.c. of caustic soda solu-

tion were required to produce the proper tint, using in each case ten c.c. of the stomach contents.

1. As dimethyl-amido-azo-benzol reacts only with free hydrochloric acid, the acidity referable to this is 35 degrees, or .12775 per cent.

2. Alizarin shows the tint of an alkaline reaction when the free hydrochloric acid, organic acids, and acid salts have been neutralized, combined hydrochloric acid having no effect upon it. Hence it follows that by subtracting the amount of free hydrochloric acid from the acidity found by alizarin, the amount of acidity due to organic acids and acid salts will be found, in this case $49 - 35 = 14$ degrees, or .0511 per cent.

3. Phenolphthalein only turns to a dark-red color when all the acidities of the solution have been saturated, including the combined hydrochloric acid. The amount of combined hydrochloric acid may be found by subtracting the acidity found by alizarin from that found by phenolphthalein, in this case $75 - 49 = 26$ degrees, or .0949 per cent.

Method of Martius and Lüttke.—By this method the amount of physiological hydrochloric acid, the free and combined hydrochloric acid, are found, as well as the total chlorin of the gastric juice, by determination of the amount of chlorin. The method is based upon the fact that by moderate incineration the free hydrochloric acid can be driven off, while the chlorin in combination with the inorganic bases is not affected.

For this method the following solutions are required:

1. A decinormal solution of hydrochloric acid, which can be prepared by standardizing against the decinormal caustic soda solution as described in a former chapter.

2. A decinormal solution of nitrate of silver, containing 25 per cent. of pure nitric acid. This solution is approximately made up by dissolving 17 gm. of pure crystallized nitrate of silver in 900 c.c. of a 25 per cent. solution of nitric acid, and adding 50 c.c. of the liquor ferri sulphur oxydati of the German Pharmacopeia (the liquor ferri oxysulphatis ["National Formulary,"] will serve the same purpose). The solution is then standardized against the solution of hydrochloric acid and diluted to the proper volume. Each c.c. of the solution is equivalent to 0.00365 gm. of pure hydrochloric acid.

3. A decinormal solution of ammonium sulphocyanate. Eight

gm. of the pure salt are dissolved in 900 c.c. of distilled water and titrated against the decinormal solution of silver nitrate. After ascertaining the strength of this solution it is diluted so that it is exactly equivalent to the decinormal solution of nitrate of silver.

Method.—1. To determine the total amount of chlorin present in the gastric juice, ten c.c. of the stomach contents, after thorough mixing, are measured into a small cylinder graduated to 100 c.c., and treated with 20 c.c. of the solution of nitrate of silver. The mixture is thoroughly shaken and allowed to stand for ten minutes. The mixture is then diluted to 100 c.c., once more agitated, and filtered through a dry filter into a dry flask. Fifty c.c. of the filtrate are then titrated with the decinormal solution of ammonium sulphocyanate until a permanent red color appears. Multiply the number of c.c. of ammonium sulphocyanate by two, as only half the filtrate was taken, and subtract from the number of c.c. of nitrate of silver added (20); the result will be the number of c.c. of the nitrate of silver solution precipitated by the total chlorin of the gastric juice and correspond to the same number of c.c. of decinormal solution of hydrochloric acid, the whole amount of chlorin being expressed in terms of hydrochloric acid.

2. To determine the amount of chlorin in combination with inorganic bases.

Ten c.c. of the filtered gastric juice, or of the well-mixed stomach contents, are evaporated to dryness in a platinum or porcelain crucible, over a water-bath or on a plate of asbestos, to avoid loss from sputtering. The incineration is carried only to the point when the residue ceases to burn with a luminous flame. After cooling, the residue is treated with distilled water up to about 100 c.c., or until the filtrate comes away free from chlorids, which may be shown by treating with a drop of silver nitrate. If the filtrate remains perfectly clear after the addition of a drop of nitrate of silver, the residue is free from chlorids. To the clear filtrate is now added ten c.c. of the decinormal solution of nitrate of silver, and the excess titrated by means of the decinormal solution of ammonium sulphocyanate as before. The amount of ammonium sulphocyanate solution subtracted from the amount of the silver solution (ten c.c.) gives the amount of silver precipitated by the chlorids remaining after incineration in combination with the inorganic bases. By subtracting the result of the second process from that of the first, the amount of free and combined hydrochloric acid is determined.

Modifications.—1. By titrating with decinormal caustic soda solution, using dimethyl-amido-azo-benzol as an indicator, we obtain the amount of free hydrochloric acid; this subtracted from the sum of the free and combined hydrochloric acid together, as arrived at by the method No. 2 on previous page, will give the amount of combined hydrochloric acid.

2. By determining the total acidity with phenolphthalein and subtracting from it the amount of free and combined hydrochloric acid, we can estimate the acidity due to organic acids and acid salts.

3. The amount of organic acid present may be estimated in terms of hydrochloric acid by the method of Hehner-Seeman (to be described later). This result deducted from the result of the preceding modification gives the amount of acidity due to acid salts.

Leo's Method.—Leo bases his method upon the fact that when calcium carbonate is added in a fine powder to the gastric juice, the free and combined hydrochloric acid combine with the calcium carbonate to form calcium chlorid, a neutral salt, while the acid salts are not affected. During the reaction, however, the calcium chlorid reacts with the phosphates to form acid calcium phosphate (monocalcium phosphate, $CaHPO_4$). As this requires double the amount of caustic soda solution to neutralize that would be required for the acid sodium phosphate, it is necessary to add each time an excess of calcium chlorid solution before titration.

Method.—Ten c.c. of the gastric juice are shaken up with 50 c.c. of ether to remove organic acids. The residue after drawing off the ethereal layer is treated with five c.c. of a concentrated solution of calcium chlorid and titrated with the decinormal solution of caustic soda, using phenolphthalein as an indicator. This determines the acidity due to free and combined hydrochloric acid and to acid salts. A second portion of 15 c.c. is treated with a small amount of pure, dry calcium carbonate, the mixture stirred and immediately filtered through a dry filter. The carbon dioxid is expelled from the filtrate by passing a current of air through it. Ten c.c. of the filtrate are then treated with five c.c. of the saturated solution of calcium chlorid and titrated as above. The acidity found is due to the acid phosphates. By subtracting the result found in the second titration from that of the first, the amount of free and combined hydrochloric acid is determined.

Boas' Method.—This method is an easily applied test for free hydrochloric acid, which gives fairly accurate results in the absence

of organic acids or when they are present only in traces. Ten c.c. of the filtered gastric juice are titrated with decinormal caustic soda solution until a small amount (a drop) removed by a platinum loop fails to change the tint of Congo paper. Instead of using the paper as an indicator outside, a small bit of the Congo paper may be dropped into the solution and the titration conducted slowly, with constant shaking, until the paper regains its original red color. This test, however, can not be employed in the presence of any considerable amount of free organic acids.

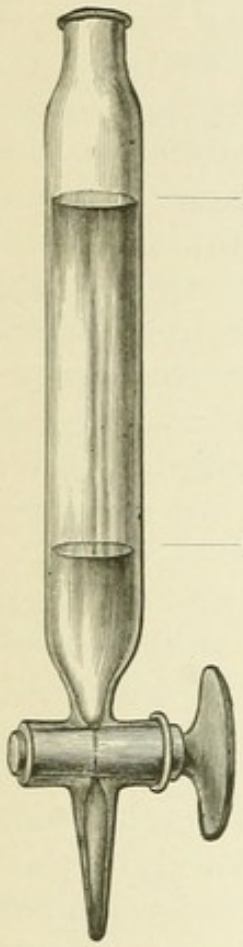

FIG. 23.—STRAUSS' MIXING FUNNEL FOR LACTIC ACID DETERMINATIONS.

Lactic Acid—Quantitative Estimation.—A simple clinical test for lactic acid has been devised by Strauss (*Berlin. klin. Woch.*, 1895, No. 37). A separating funnel is graduated to five c.c. below and 25 c.c. above. The funnel is filled to the five c.c. mark with gastric juice and ether added to the 25 c.c. mark. The funnel is corked and well shaken, and after standing for a short time to allow the fluids to separate, the liquids are run out to the five c.c. mark. Distilled water is added to the 25 c.c. mark and the mixture treated with two drops of a solution of the officinal tincture of the chlorid of iron, diluted 1 : 10. On shaking the mixture, an intense green color is produced if lactic acid is present in the proportion of 1 per 1000 or more. If present in the proportion of from 0.5 to 1 per 1000 only a pale-green color is produced.

Boas' Method.—This method of estimating the amount of lactic acid depends upon its oxidation into aldehyd and the estimation of the latter by means of a standard solution of iodin.

Solutions required: 1. A decinormal solution of iodin is prepared by dissolving 25 gm. of potassium iodid in about 200 c.c. of water, and dissolving in this 12.6 gm. of resublimed iodin. The solution is diluted with distilled water to 1000 c.c., and requires no correction.

2. A decinormal solution of sodium arsenite: Dissolve 16.5 gm. of sodium arsenite in about 900 c.c. of distilled water. It is

then titrated against the decinormal solution of iodin and diluted so that the two solutions are equivalent.

3. Hydrochloric acid (sp. gr. 1018).

4. Normal solution of potassium hydrate (56 gm. in 1000 c.c.).

Method: Ten or 20 c.c. of the filtered gastric juice are tested for the presence of free acid; if present, a small amount of barium carbonate is added (if free acid be absent, this addition is unnecessary), and evaporated to a syrup. A few drops of phosphoric acid are added and the solution boiled slightly to expel carbon dioxid.

Allow the syrup to cool; extract with 100 c.c. of ether free from alcohol; after the two fluids have separated draw off the ethereal solution; evaporate; take up the residue in 45 c.c. of water, and filter. The filtrate is treated in an Erlemeyer flask with five c.c. of sulphuric acid and a small amount of manganese dioxid. The flask is closed by a two-holed rubber stopper, one aperture being closed by a glass tube and rubber tubing clamped off, the other opening receiving a bent glass tube leading to the distilling apparatus. The distillate is received in a large flask well stoppered. The mixture is distilled at a gentle heat until about four-fifths of the fluid has passed over. The distillate is then treated with 20 c.c. of the decinormal solution of iodin and the same amount (20 c.c.) of the normal potassium hydrate solution, thoroughly shaken and allowed to stand for a few minutes in the flask. Twenty c.c. of hydrochloric acid and an excess of sodium bicarbonate in powder are then added, and the excess of iodin determined by titration with the solution of sodium arsenite. The sodium arsenite is added until the solution is decolorized; fresh starch solution and the iodin solution is then added until the blue color becomes permanent. Each c.c. of the iodin solution in excess of the sodium arsenite solution is equivalent to 0.003388 gm. of lactic acid.

Fatty Acids.—The method of Cahn and Mehring modified by McNaught is simple and fairly accurate. The total acidity is determined in ten c.c. of the filtered gastric juice by titrating with a decinormal solution of caustic soda, using phenolphthalein as an indicator. Ten c.c. are evaporated to a syrup on the water-bath, made up to the original volume with distilled water, and the acidity determined as before. The difference in acidity will be the amount due to fatty acids.

Total Organic Acids.—The total organic acids are best esti-

mated by the method of Hehner-Seeman, called, by Leube, Braun's method.

Ten c.c. of the gastric juice are accurately neutralized with a decinormal solution of caustic soda, using phenolphthalein as an indicator. This solution is then evaporated to dryness, carefully avoiding sputtering, and incinerated as long as the residue burns with a luminous flame. After cooling, the residue is extracted with boiling distilled water, filtered, and the amount of sodium carbonate formed determined by titration with a decinormal solution of hydrochloric acid. As the presence of free carbon dioxid interferes somewhat with the delicacy of the reaction when phenolphthalein is used as an indicator, the following modification has given better results: After the incinerated mass has been extracted with boiling water and filtered, a known excess of the decinormal solution of hydrochloric acid is added, the solution boiled to expel any carbon dioxid in solution, and the excess of acid determined by back titration with a decinormal solution of caustic soda.

This method is based upon the fact that when salts of the organic acids with the alkalies are incinerated at a low heat, the carbonates of the alkalies are formed with the liberation of water and carbon dioxid. This method is simple. Martius and Lüttke speak favorably of it, and Hemmeter has obtained accurate results with it.

CHAPTER XVIII.

DIGESTIVE FERMENTS.—PRODUCTS OF DIGESTION.—TESTS FOR SAME.

Saliva.—The saliva as found in the mouth is the mixed secretions of all the salivary glands. It may be readily obtained for testing by requesting the individual under examination to chew a piece of soft rubber or other insoluble substance, to stimulate the secretion, and as it forms it is placed in a clean receptacle. It is a clear, slightly opalescent fluid, of a mucoid consistency, having a specific gravity of 1002 to 1006. Under normal conditions it has a slight alkaline reaction, its alkalinity averaging in man 0.08 per cent., expressed as sodium carbonate (Chittenden).

Its active constituent, ptyalin, acts most readily upon boiled starch, raw starch being protected from its action by the coating of cellulose surrounding each granule. Its action is entirely amylolytic, as it has no action upon other food-products.

Its action upon starch may be demonstrated in the following simple manner: A few c.c. of boiled starch paste are treated in a test-tube with a small amount of saliva. A few drops removed and treated on a testing plate with a drop of iodin solution give the characteristic blue color of starch. After a moment or two a few drops removed will show a violet color, and by treating a portion at intervals the color changes gradually to a deep reddish-brown, and finally disappears. Different products of the action of the ferment are found at different stages of digestion. The violet color first found is a color which results from a mixture of erythrodextrin and starch when treated with iodin. Later the color becomes reddish-brown, due to the change of the starch entirely into dextrins and sugar. When digestion has gone on until the solution gives no color whatever with iodin, the solution still contains some form of dextrin (achroödextrin), as may be shown by the addition of alcohol, which throws down a profuse white precipitate. It may be shown, also, that the solution contains sugar by treating a small amount of the mixture with Fehling's solution. This sugar, ac-

cording to the investigations of Nasse, von Mehring, and Musculus, is not dextrose, as formerly taught, but maltose.

The action of ptyalin is most energetic at the temperature of the body. It acts best in a neutral medium, though a small trace of alkali has little or no effect upon it. Its activity is stimulated by the addition of enough acid to combine with its proteid constituents. A minute trace of acid still allows the action to continue, but for practical purposes we may say that the addition of free acids, in such quantity as are found in the gastric juice, not only stop its action, but possibly destroy the ferment, so that after neutralization it is no longer able to digest starch.

In the stomach, the action of the ptyalin probably continues until the presence of free acid destroys the ferment. As no free acid can be demonstrated in the stomach until the lapse of fifteen or twenty minutes normally, the greater portion of the starch is transformed into sugar and achroödextrin. Under normal conditions, then, we should find in the gastric juice removed for examination, sugar, achroödextrin, and a faint trace of erythrodextrin. The presence of a marked reaction of erythrodextrin, then, is valuable presumptive evidence of hyperacidity, its absence indicating either normal acidity or subacidity.

Only in rare instances has absence of ptyalin from the saliva been seen.

There are some unexplained cases in which, with a normal or diminished acidity, the digestion of starches is very poor, as is shown by the marked reaction of erythrodextrin and the small percentage of sugar found by quantitative test. The activity of the salivary excretion ought always to be examined in such cases.

Pepsin.—The proteolytic ferment of the gastric juice is active only in an acid medium, and is destroyed by very dilute solutions of the alkaline carbonates. Pepsin is probably not secreted as such, its precursor being pepsinogen or propepsin, which is transformed by weak acids into the active ferment, pepsin. While hydrochloric acid acts best in thus transforming pepsinogen into pepsin, other acids to a lesser degree can perform the office. Pepsin, like the other ferments, has the property of changing an almost unlimited amount of proteids, providing the products of its action are removed when formed, and the temperature kept at a favorable point, as it appears to act by its presence, not being itself destroyed or changed by the reaction.

While no quantitative methods have been devised, and it has never been isolated in a pure state, we know that a product can be obtained by complex chemical methods which, while intensely proteolytic, exhibits none of the reactions of proteids, so that the ferment, whatever its nature, probably is not a proteid.

The only tests which can be used for its detection, therefore, are of a qualitative nature: its effect in acid solution on proteid substances. Comparative tests may also be instituted, using for comparison gastric juice from a healthy stomach.

The amount of acid necessary for the most vigorous action of pepsin varies with the form of proteids employed. For example, pepsin acts best on fibrin when the acidity is about 1 : 1000, while coagulated egg-albumen is digested most rapidly when the acidity amounts to two or three per thousand of hydrochloric acid.

Test.—Three test-tubes or small wine glasses are taken, and a small thin slice of boiled egg-albumen placed in each. To the first is added three c.c. of the gastric juice; to the second, three c.c. of the gastric juice to which hydrochloric acid has been added in sufficient quantity to bring the acidity to two or three per thousand; the third is acidulated as in number two, and a few grains of pepsin added. The three tubes or glasses are now placed in the incubator, at a temperature of 40° C., and allowed to remain for three hours.

If at the end of this time all three tubes show digestion by the rounding-off and solution of the egg-albumen, the specimen contained pepsin; if number two and three only show digestion, the contents contained pepsinogen but no pepsin; while if only the third tube or glass shows traces of digestion, the specimen contained neither pepsin nor pepsinogen.

Pepsinogen.—This substance is supposed to be secreted by the cells of the gastric mucosa, and to be changed into pepsin by the action of the hydrochloric acid of the gastric juice. This action has been differently explained by various experimenters, the most plausible theory being that a combination of the two takes place with the formation of pepsin—hydrochloric acid.

In the absence of hydrochloric acid, this body, pepsinogen, may be present in normal amount, and require only the addition of a sufficient quantity of hydrochloric acid to bring the gastric juice to a normal acidity to render the stomach contents active.

In the absence of free hydrochloric acid, we may test for the presence of this substance by acidulating with hydrochloric acid, as

in number two of the pepsin test, adding a small bit of boiled egg-albumen and placing in the thermostat at a temperature of 40° C. for three hours, at the end of this time noting the presence or absence of signs of digestion.

Boas following out the observations of Brücke ("Vorlesungen über Physiologie," p. 311, 1884. Quantitative determination of pepsin, etc.) employs a comparative test which in doubtful cases may yield valuable information. Properly labeled tubes are prepared, and in them are placed measured quantities of gastric juice diluted with a solution of hydrochloric acid of the normal strength of the gastric juice (two or three per 1000), so that the tubes contain the gastric juice in dilutions of 1 : 10 and 1 : 20. To each tube a small flake of egg-white or fibrin is added and put in a thermostat at the temperature of the body. From the amount of dilution at which digestion ceases, an idea may be gained of the amount of pepsin or pepsinogen which any gastric juice contains. For comparison, similar tubes may be prepared of normal gastric juice, and the digestive power of the two compared.

Rennin and Rennin Zymogen.—In addition to pepsin, the gastric juice also contains a ferment, or its zymogen, whose special property appears to be the precipitation of casein from milk. As in the transformation of pepsinogen into pepsin hydrochloric acid is required, so rennin zymogen in the gastric juice is not transformed into rennin except in the presence of hydrochloric acid. Certain neutral salts of lime, such as calcium chlorid, however, have the power of transforming rennin zymogen into rennin, even in neutral or slightly alkaline solutions.

The following tests for the presence of rennin and its zymogen have been devised by Boas:

Rennin.—Five c.c. of the gastric juice are exactly neutralized with a decinormal solution of caustic soda, five c.c. of neutral milk added, and the mixture, after being well shaken, is placed in an incubator at the body temperature.

If rennin is present, the casein will form a firm coagulum in from ten to fifteen minutes.

A relative quantitative estimation of the rennin ferment may be performed by the following method:

The gastric juice is accurately neutralized and portions of this diluted with distilled water, in known proportions, 1 : 10, 1 : 20, etc. To five c.c. of each of these dilutions, five c.c. of neutral milk are

added, and the tubes placed in the thermostat at the body temperature for fifteen minutes. At the end of this time the tubes are removed and the dilution at which no coagulation takes place is noted. In stating the dilution, note must be taken of the fluid added in neutralizing.

Rennin Zymogen.—Five c.c. of the gastric juice are rendered faintly alkaline by the addition of a decinormal solution of caustic soda; one c.c. of a one per cent. solution of calcium chlorid and five c c. of neutral milk are added. The tube is placed in the thermostat, and after fifteen minutes should show a firm cake of casein if rennin zymogen is present.

Quantitative.—The gastric juice is rendered faintly alkaline by adding a decinormal solution of caustic soda and dilutions prepared, 1 : 10, 1 : 20, etc., estimating in the dilution the amount of fluid added in alkalinizing. Five c.c. of each of these dilutions are placed in test-tubes with five c.c. of neutral milk and one c.c. of a one per cent. solution of calcium chlorid. These are placed in a thermostat at the body temperature, and at the end of fifteen minutes the dilution at which the enzyme fails to act is noted. From the observations of Boas and others, it appears that the secretion of the ferments and the pro-enzymes is less affected by the minor disturbances which may cause a temporary arrest of the acid secretion of the stomach. Decrease in the activity of the ferments, on the other hand, is usually the result of some organic change in the gastric mucosa.

By experiment upon normal individuals it has been found that rennin is active in dilutions of from 1 : 30 to 1 : 40, and rennin zymogen in dilutions varying from 1 : 100 to 1 : 150. It has been found that, even in the absence of free hydrochloric acid, the ferments may be active up to the limit observed in normal individuals, and that in such cases the condition of anacidity was a temporary matter, due to some mental or circulatory disturbance, the acid reappearing when the cause of the disturbance was removed.

On the other hand, in cases of anacidity in which the rennin zymogen was active only in the stronger dilutions, 1 : 5, 1 : 10, etc., the anacidity is due to some organic change in the gastric mucosa from which recovery is usually rare.

It will be seen from these considerations of what importance a quantitative investigation of the gastric ferments is from the prognostic standpoint.

Action of Pepsin on Proteids.—The action of pepsin upon proteids, as has been shown, only takes place in an acid medium. The action is a very complex one and is not as yet fully understood. The first observable result of the action of a hydrochloric acid solution of pepsin upon a coagulated albumen, such as egg-white, is apparently a partially mechanical change. The egg-white swells up, its edges become rounder, and it becomes clearer and more glassy in appearance. The egg-white then begins to dissolve, as is shown by the presence in the solution of a substance precipitated by neutralization, which may be called syntonin, or acid albumen. This action takes place also in acid solutions to which pepsin has not been added. The next step is one in which the pepsin plays an important part. The syntonin or acid albumen is changed first into the primary albumoses, proto- and hetero-albumose. These undergo further change and become deutero-albumoses, and, finally, peptones. These substances may be distinguished from each other by the following reactions :

(*a*) Native albumens may be removed from the solution, if present, by rendering the stomach contents faintly acid, if not already so, and boiling. The precipitate will consist of the native proteids, viz., albumen and globulin.

(*b*) The solution is carefully neutralized by the addition of a weak caustic soda solution. The precipitate will consist of syntonin or acid albumen. The neutralization must be exact, as the precipitate is dissolved by an excess of acid or alkali to form acid-albumen or alkali-albumen, respectively.

(*c*) The filtrate from which the albumen and acid albumen has been removed is now saturated with magnesium sulphate and filtered. The precipitate, which consists of the primary albumoses, proto- and hetero-albumoses, is dissolved in water, placed in a dialyzer, and the salts removed by dialysis. As hetero-albumose is insoluble in pure water, it is precipitated by the removal of the salts as in a dialyzer. The proto-albumose remains in solution, as it is soluble in water, and may be tested for by acidulating with nitric acid in the cold, the precipitate redissolving on heating.

(*d*) Deutero-albumose, or secondary albumose, is detected in the following manner: A sufficient quantity of the gastric juice is freed from albumen and acid albumen, according to (*a*) and (*b*). The filtrate is saturated with powdered ammonium sulphate and the precipitate which forms, consisting both of primary and secondary

albumoses, is filtered off, and washed thoroughly with a saturated solution of ammonium sulphate.

The precipitate is redissolved in the least amount of water possible, faintly acidulated with acetic acid and saturated with common salt, which precipitates the primary albumoses, leaving the deutero-albumose, or secondary albumose, in solution. After filtration the secondary albumose may be detected by saturating again with ammonium sulphate any precipitate which may form consisting of deutero-albumose. It may be detected also by adding a considerable amount of common salt to its solution and acidulating with nitric acid. A precipitate will form in the presence of deutero-albumose, redissolved on heating.

(*e*) Peptone may be detected by precipitating all the other proteids by saturating with ammonium sulphate and filtering. The filtrate contains the peptone, which may be tested for by the biuret reaction. The filtrate is treated with an excess of caustic alkali and a few drops of a very dilute solution of copper sulphate. If peptones are present in the solution a pink or rose-red color appears.

CHAPTER XIX.

GASTROSCOPY.

The first one to use a gastroscope was Mikulicz ("Ueber Gastroskopie u. Oesophagoskopie," *Wien. med. Presse*, 1881, No. 43; also *Wien. med. Wochenschr.*, 1883, Nos. 23 and 24). The instrument used was made by Leiter, of Wien, and was curved at an obtuse angle. The following account of the technic and value of the method is quoted from Rosenheim (*loc. cit.*):

"Gastroscopy is founded on the fact discovered by this author, that in the majority of cases (80 per cent.) it is possible to introduce, without special difficulty, a straight, rigid tube, 12 mm. in diameter, the patient having first been placed in the dorsal position. It is possible to introduce such a tube far into the stomach, often as far as the navel, and eventually below the same. The establishment of this fact first furnished him with a foundation on which gastroscopy could be developed, after he had come to the conclusion that an optical apparatus, to be suitable for the stomach, must be straight as in the cystoscope (Rosenheim, 'Gastroskopie,' *Berlin. klin. Wochenschr.*, 1896, No. 13).

"Apart from complications that are due to tumors, exudations, enlargement of the liver, etc. (that is, to the pathological-anatomical conditions), apart also from congenital anomalies (abnormally contorted course of the esophagus or abnormal contraction), two facts are to be considered in the light of an impediment to a successful probing by means of introducing a rigid tube into the stomach: in the first place, the bend to the left, or spiral twist, which the esophagus shows so frequently in its subphrenic part; and, secondly, the occurrence of spasm at the lower physiological contraction of the organ. With continued practice it becomes apparent that the anatomical obstruction, caused by the change in the direction of the esophagus, may usually be overcome if the instrument is introduced from the right angle of the mouth, preferably while the head is turned slightly to the right, laterally.

"The obstruction before the cardia, caused by spasm of the

muscles of the esophagus, can not be eliminated mechanically; here the manner of introducing the tube makes no difference, and soothing the patient, persistence, and adaptation can alone lead to the desired result. Local anesthesia is useless. How much the occurrence of the spasm is due to the psychic condition of the patient, was shown by numerous observations with invalids, particularly neurasthenic persons, who were timid and restive when the probe was first introduced, and with whom it was impossible to penetrate to the stomach; while later, after they had become familiar with the proceeding, this was easily accomplished. It is necessary to keep in mind, also, that the spasm appears more frequently with persons who are suffering with an ulcer or carcinoma near the cardia."

Description of the Instrument.—(See Fig. 24.) The gastroscope is a straight metal instrument, 68 cm. in length, 12 mm. in diameter, consisting of three concentric systems of tubes, and terminating in a larger head-piece for the different conduits. The inner tube (1) forms an optical apparatus, the ocular of which is situated at *O*, and a rectangular prism, *P*, is located in front of its objective lenses. The visual angle of the telescope (otherwise constructed according to the principle which has been approved in the cystoscope, viz., as a terrestrial telescope) amounts to 60°, so that it is possible to inspect an area five cm. in diameter at a distance of five cm. from the object. The center of the portion in view lies vertically over the small side (cathetus)

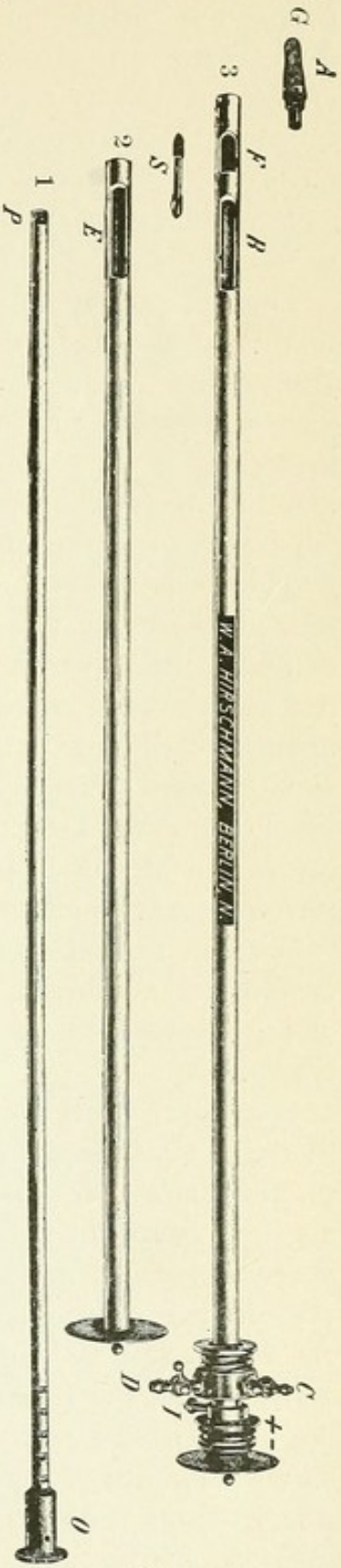

FIG. 24.—GASTROSCOPE.

of the rectangular prism which receives the image. In order to inspect a surface the center of which does not lie at right angles over the cathetus, the rectangular prism may be replaced by an acute angular prism ; by this means those surfaces also can be examined that are situated above, which can be only partially viewed by means of a rectangular prism. The absolute necessity of inspecting parts of the stomach which appear at varying heights—for instance, the region of the pylorus—explains this arrangement.

The optical apparatus is inclosed by a tube (3) that is closed at the lower end by a head-piece, *A*, carrying a tip of rubber, *G*. Just above the tip there is an aperture, *F*, which is closed by a glass window, behind which there is situated an incandescent lamp, as shown in *S*. At the upper and lower ends of the lamp the metal contacts that conduct the current are fastened. Above the window there is a second aperture, *B*, in which the prism is adjusted, and inside of which it may even be moved up or down. In the tube (3) there are four canals separated from one another. Two of these canals, which end at *C* and *D*, serve to conduct water through the instrument and around the lamp, to prevent excessive heating of the tubes caused by the incandescent lamp. The third canal is used to receive the wires that conduct the current to the lamp; while the fourth canal, which begins at *I*, and opens at the lower end of the instrument behind the window, *F*, is used to introduce air, which must be pumped into the stomach, by means of a blast, to distend its walls. Toward the top, the thin tube terminates in a larger head-piece that establishes the connection of the canals with the different conduits for water, air, and electricity.

Fig. 24 (1) shows the sliding tube, a tube with a centimeter scale, that can be shoved over the instrument (3) and easily revolved on the same. It has an aperture at *E* corresponding to the aperture *B*, and, by being turned 180 degrees, it serves to cover this aperture, as well as the prism lying behind the same, so as to prevent the optical apparatus from being soiled by mucus while the instrument is being introduced. If the external tube is so adjusted that the aperture *B* is closed, and if, to further protect the optical apparatus, the latter is turned 180 degrees so that the exposed surface of the prism faces the side of the tube, then the prism enjoys a double protection, and in consequence the instrument can not be soiled while being introduced. Small metal knobs

are attached to the top of all three tubes, to enable us to control from the outside their position in the stomach; when these stand in a straight line, the observer knows that the prism is not covered by the revolving tube, but faces the cavity of the stomach through the aperture. The electric current + and — is introduced at the points of contact by means of a movable cable that is equipped with an interrupter. The intensity of the electric current is 16 volts. In conducting water through the apparatus, a stand carrying an irrigator is used. The two rubber tubes conduct the water through the instrument. By means of a cock the flow of the water can be interrupted. Another tube carries the water that has passed through the instrument into the water-bucket. To cool the instrument it is advisable to use, not cold water, but water of about 40° C., in order that the lenses of the optical apparatus and the surfaces of the prism may not be covered with a film of moisture caused by sudden condensation. The stand carries the accumulator (storage battery) used to furnish the electricity; this is supplied with a rheostat for regulating the current, and also with an interrupter.

. . . . . .

It is absolutely necessary in every case to convince ourselves, before carrying out the gastroscopic investigation, that the way from the teeth, as far as the great curvature, is really unobstructed, and no special difficulties are offered to the passage of a straight rigid tube while the patient occupies the dorsal position. This test should never be neglected; at the same time it should be carried out with the greatest caution.

Rosenheim employs for this purpose a hollow steel probe 70 cm. long, and having the diameter of the gastroscope (12 mm.), or a smaller one, ending likewise below in a rubber appendage, in the side of which there is a small aperture provided with a blast; the parts can be screwed off to facilitate cleansing; a centimeter scale is engraved on the sides. This probe is introduced in the dorsal position, preferably from the right corner of the mouth; after measurements along the back have been made to determine the distance of the cardia from the teeth, and after having applied a four per cent. solution of cocaine to the pharynx, the patient is directed to breathe quietly and deeply, and to lift his right hand on feeling a pain in the region of the stomach or above the same. If the patient shows pain, the procedure must cease at once. If

resistance is felt, a moment of rest intervenes, or eventually the instrument is retracted a little, only to try again whether the resistance yields under gentle pressure, the reaction on the part of the patient meanwhile determining the degree of energy that is to be employed in this manipulation. The absolute law in probing is to avoid all strong pressure, otherwise lesions of the membrane, even perforation of the esophagus or stomach, may be the consequence. After the diaphragm has been passed, air is pumped into the stomach and we determine how far the instrument is able to penetrate into the inflated organ.

The correct guiding of the instrument from the right corner of the mouth plays an important part in the success of introducing the instrument in the majority of cases.

If we wish to get our bearings and inform ourselves by means of the telescope about the vast cavity of the stomach, it is preferable to start from the normal position just described: The point of the instrument far down at the great curvature, the window turned to the front. In this position the front wall of the stomach approaches the eye closely, within from two to three cm., so that we see it magnified. A hasty glance suffices to recognize the condition of the mucous membrane here, and we then immediately change the position of the instrument by revolving it slowly to the right so that the prism faces the pylorus. This part of the stomach and the adjoining portion of the small curvature varies in its distance from the prism in various cases. The distance is from 6 to 12 cm., and the image which we receive of this section is therefore usually somewhat reduced in size (to about ½). We now are examining a part of the organ that, from a practical point of view, is perhaps the most important, since ulcers and cancers are so frequently located there. We exert ourselves now, starting from the opening of the pylorus, to investigate systematically the whole hollow cone, situated to the right. This part does not escape us as a rule, if we move the tube gradually from the great curvature upward while revolving the apparatus generally in both directions. After we have found the orifice of the stomach, as a fixed point, it is not difficult to espy from the same the neighboring section of the small curvature, at least, and something of the rear wall. The higher the portio pylorica lies behind the liver, the more it (as is normal) bends away to the rear on the right, the more difficult it is to inspect, while a low position greatly facilitates our investigation.

In the former case (for which we may be somewhat prepared by the preceding inflation of the stomach) the optical apparatus provided with an acute-angled prism is recommended. It is possible to recognize how different the distance is between the prism and the pylorus during the normal position of this segment of the organ and during dislocation of the same. In the former case

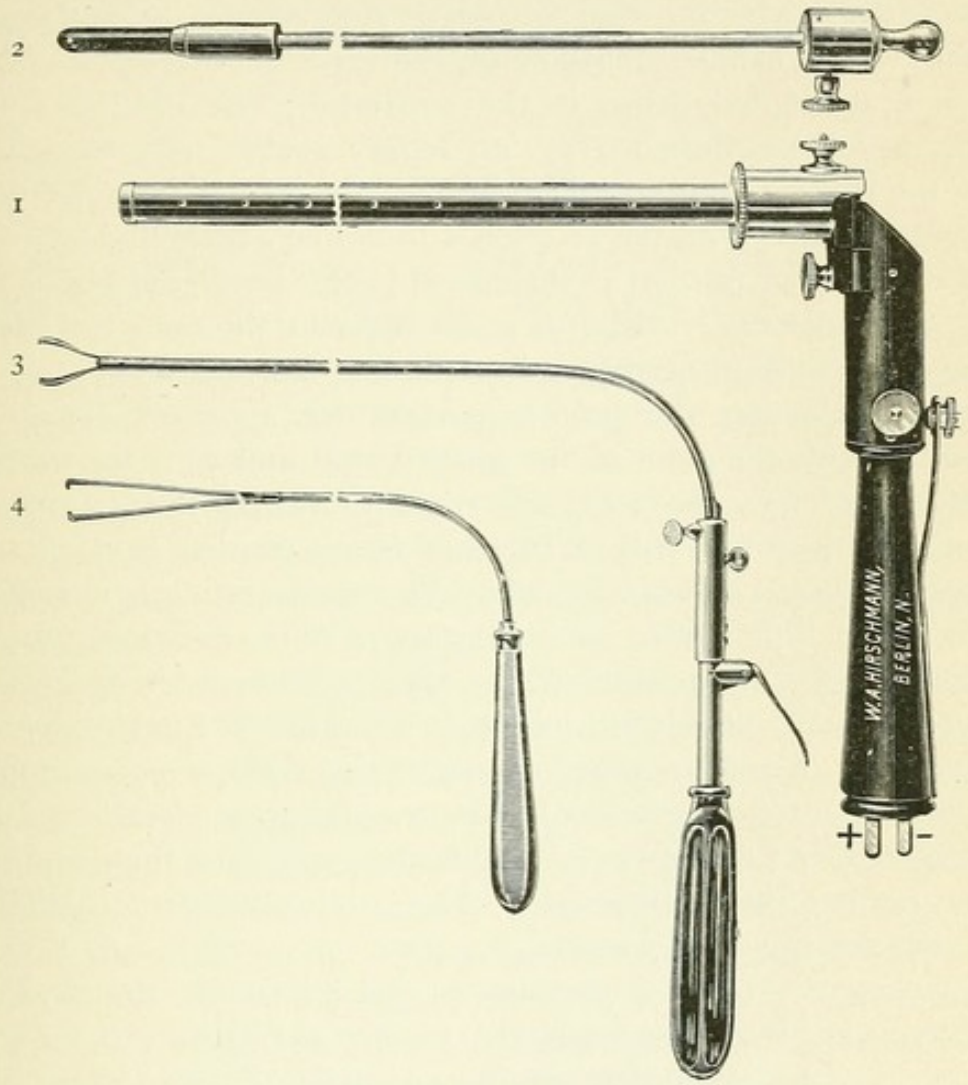

FIG. 25.

1. Esophagoscope. 2. Obturator. 3. Esophageal forceps. 4. Esophageal applicator.—(*Rosenheim.**)

the distance is more considerable; we must withdraw the instrument further, to bring at least a part of the portio pylorica within the angle of the prism; and if the point of the instrument

* Our thanks are due to Prof. Theod. Rosenheim (Berlin) for presentation of these illustrations.

diverges a little further to the left from the vertebral column, this approach to the cardia avails nothing; under all circumstances we receive only an image of the part beneath the orifice of the stomach. During these manipulations we are in danger of being surprised by an obscuring of the field of vision, since we are compelled to approach closely the descending part of the small curvature adjoining the cardia. These disturbances are avoided if we take a view of the pyloric portion from a deeper point, a thing which can be conveniently effected by the employment of an acute-angled prism in the apparatus; the center of the circle, which we then survey, no longer stands perpendicularly over the prism. We no longer receive the image from a region at the same level with the prism, but from one a little higher.

If the pyloric portion is dislocated to the lower margin of the liver, or deeper, the rectangular prism opposite the same can easily be adjusted without needing a correction.

After inspecting the pyloric portion we approach the great curvature with the point of the gastroscope, and turn the instrument to the left by 180°; while slowly withdrawing the instrument, we next inspect the part of the fundus and cardiac portion that belong to the left half of the body. The investigation is now completed; the illumination is discontinued, the revolving tube is pushed in front of the window, the blast is removed in order that the gases may quickly escape; only after this the instrument is withdrawn. Rosenheim has devised a gastroscope more recently in which the stream of water for the cooling of the electric lamp is dispensed with; the lamp is only flashed now and then, and not kept incandescent continuously. The latter instrument is thinner and only ten mm. in diameter.

Conclusion.—(1) Not all parts of the interior of the stomach can be inspected. Portions of the greater curvature—of the posterior wall, the immediate neighborhood of the cardia, are not visible. It can not be practised on all individuals.

(2) All suspected cases of ulcer must be excluded if recent pain and hemorrhages have occurred. Ulcers at pylorus are less liable to be injured than those near the cardia.

(3) Rosenheim suggests that gastroscopy may be employed for the early diagnosis of carcinoma and its differentiation from ulcer. He admits that it is an inconvenient procedure.

PART SECOND.

THERAPY AND MATERIA MEDICA OF STOMACH DISEASES.

CHAPTER I.

THE PRINCIPLES OF DIETETIC TREATMENT OF GASTRIC DISEASES.

In the chapter on the physiology of digestion we have briefly considered the various food-substances, their nutritious and innutritious constituents, the amounts of each requisite to maintain a healthy organism, and their caloric values, etc. It is one of the far-reaching deserts of the great Father of medicine to have first methodically developed dietetics for the sick as a special discipline and an integral part of therapy.

In his classical dissertation on the conduct of febrile diseases (Hippocrates, "De victus ratione in morbis acutis"), in his aphorisms, and in many other treatises, he emphasizes the great importance of careful regulation of nutrition for patients. His principles, based upon analytical experience, are stated with unsurpassable precision. His dietetics are free from speculation, and regard the nature and stage of the disease, the constitution, age, and habits of the patient; above all, they show what is in our days termed an individualizing principle. It would seem probable that a therapeutic aid that had been logically considered at the very dawn of medical knowledge, and by such an able mind, would at the present time be one of the most highly developed in medicine, particularly when one reflects upon the declaration of Donders ("Die Nahrungsstoffe des Menschen," Crefeld, 1853): "Whoever works at the development of our knowledge on food-substances is working on a broad basis for the development of mankind." Fortunately for us, many bright intellects have already applied themselves to this work, and our knowledge has been enriched by treasures of valuable informa-

tion. But the well-advised special student can not fail to recognize that we have only entered a vast territory, and that the greater part of it remains to be explored. Even the small portion which by hard toiling is clearly our own, is, we regret to say, far from being the common property of the profession, at least it does not seem to realize that a logical and individualizing diet is a more potent therapeutic factor than medicine.

The results so far obtained show great domains of research and inquiry yet to be explored for truth bearing on dietetics. And many of our present results demand reconsideration for correct interpretation. Various eminently fitted observers disagree on vital dietetic questions, because the special point of view from which each one's research ("Fragestellung") was undertaken was not identical, sometimes not defined with precision. Sometimes the intricacy of the question to be solved did not permit of direct methods of investigation, and indirect methods had to be employed.

The scales of digestibility of various foods, as devised by Leube and Penzoldt, for instance, were arrived at by determination of the time which the stomach required to discharge these foods into the duodenum. Evidently the term digestibility means the rate of solution of the various food-substances by the constituents of the gastric juice, or of the intestinal juices, as the case may be. Digestibility, therefore, has reference mostly to secretion, but the rate of the gastric expulsion of chyme is a problem of motility.

To be of easy digestibility food-substances must—

(1) Offer only a slight resistance to the digestive juices; *i. e.*, they must be of easy solubility.

(2) They must not impede nor accelerate peristalsis.

(3) They must not excessively irritate the digestive organs, either mechanically or chemically.

(4) They must not increase the processes of fermentation or putrefaction.

(5) The greater portions of the substance must be absorbable either in the stomach or intestines.

To say that veal in amounts of 100 gm. leaves the stomach in one to two hours does not imply that it is digestible, for the same may be said of sawdust (from actual experiment of a colleague, made upon himself). By our method of duodenal intubation, we succeeded in regaining from the duodenum 56.4 per cent. of a weighed

amount of ingested veal two hours and fifteen minutes after it had been eaten. The veal was weighed and was easily recognizable; besides, nothing else had been eaten at the time. The celerity with which a food disappears from the stomach is not so much an indication of its digestibility as it is of the gastric motor power.

A more correct way to determine the digestibility of various foods—one which we have systematically experimented with on a number of volunteers from our classes who had a normal digestion—is to find out how much by weight of a known amount of ingested food is converted into peptone or dextrose, as the case may be, in a given time,—for instance, one hour or thirty minutes.

In a large number of these experiments we aspirated some of the weighed test-meals from the duodenum (method of the author, Boas' *Archives for Digestive Diseases*, vol. II). For approximately accurate results it is sufficient to weigh the insoluble residue of the particular food that is drawn out of the stomach. It is necessary to have those experiments which are to serve as crucial tests of digestibility made with comparatively pure proteids, such as meat and egg, and pure carbohydrates, such as rice. The amount of water used in the cooking and the amount ingested must be known, and can be found out by evaporating control samples to dryness.

It may thus be learned how much proteid is rendered soluble by the pepsin hydrochloric acid, how much casein is digested, or how much of starch is converted into dextrose in the fifteen to forty-five minutes, or any desired period during which the particular ferments are permitted to act. The results can naturally not be absolutely correct, but only relatively so; at least, they are as correct as the conditions of the experiment will allow the results of Leube and Penzoldt to be.

The absolute amount of food need not be regained; all that is required for a comparative study is to learn in a given sample, say 30 c.c., the proportion of soluble and insoluble chyme. Dextrose present can be determined by titration, as we have shown elsewhere; and from the reactions of the various transition products from proteid to peptone the amount of the latter can also be approximately known, particularly if the amount of solid residue of proteid that can be regained is learned first.

It is an interesting fact that the results of these tests of digestibility performed directly on the normal stomach can be confirmed

by control analysis, made with animals (making allowance for the increased secretion of HCl in dogs), and by analysis made with artificial digestive mixtures in the incubator. In the chapter on digestion by pepsin it has been explained why the exact gastric digestion can not be imitated in a test-tube, mainly because the formation of peptone remains at a certain percentage by the absorption of peptones over that amount. As soon as the amount of peptone exceeds a certain percentage, it retards, inhibits, and may even suspend proteolysis; besides, the stomach attempts to maintain a fairly constant degree of concentration of contents, by removal of chyme into the duodenum.

Notwithstanding all these differences, test-tube or artificial digestion experiments are very valuable for comparative studies in digestibility, particularly when deductions are made in combination with test-meals on the normal and diseased human stomach. In the stomach, we must bear in mind, there is a carbohydrate and a proteid digestion; we can rarely give food exclusively from the standpoint of gastric digestion, as Leube and Penzoldt's tables show that the greater portion of the digestive work is executed in the intestine.

Our results, so far as we can judge at present, agree with the main ones of these observers. Leube studied the duration of retention of various foods in the stomach of diseased patients, and Penzoldt in the stomach of healthy individuals before they were expelled into the duodenum. We have confirmed their principles by experiments, ascertaining the amounts of proteid and carbohydrates converted into a soluble form in a given time in normal and pathological stomachs. These experiments were supported by tests made with artificial digestive mixtures, and on dogs.

The explanation of the agreement of these various methods of testing digestibility is probably the fact that food-substances which are most rapidly and thoroughly converted into a soluble form, are also most easily expelled into the intestines. Easily soluble proteids, though solid, are readily converted into a liquid, or at least semi-solid, form in which they are readily propelled onward. Proteid soluble with difficulty is retained longer, because the pre-antral sphincter has, to a degree, a selective action, and will not readily permit the passage of consistent food. The matters of solution and rate of propulsion of foods are, then, the factors which are intimately correlated and largely go to make up

the quality of digestibility. The definition of a digestible food, then, is one that makes relatively small demands upon the secretory and motor functions of the stomach, which is readily absorbed and produces no subjective complaints or feeling of discomfort.

From a pathological point of view, however, the conception of digestibility is a variable one. Foods that may be easily digestible for a gastric-ulcer patient may be very indigestible for a cancer case. Leube and Penzoldt's method of estimating gastric digestibility by the rate at which various foods are expelled into the duodenum, gives a relatively correct indication for the sound normal organ, because secretory and motor functions are equally taxed as they go hand in hand.

The results can not be unconditionally applied to abnormal states where one or the other function, or both, are disturbed, sometimes in opposite directions, secretion increased, motility diminished, or vice versâ. There are conditions in which gastric digestion is completely destroyed and must be replaced by the intestinal function. There are states of absolute and permanent loss of gastric secretion, in which the propulsion of food from the stomach is not delayed. Now, one can not speak of gastric digestibility in this case, because there is very little, if any; but such cases may have a perfect intestinal digestion, so that the expressions "gastric" and "intestinal digestibility" are plausible.

The diet of stomach patients must be varied and adapted to the conditions of the secretion, motility, and absorption; but the diet must also—and this is generally overlooked—be adapted to the sensibility of the stomach. The neuroses of sensation, considered in the clinical portion of this work, offer a fertile field of work to the thoughtful dietarian. An abnormally increased feeling of hunger, in which this intensely heightened sensation can hardly be appeased by food,—*bulimia* and *akoria*,—as well as absence of hunger, in which the appetite is very readily appeased, can in many cases be successfully treated by diet.

By treating *bulimia* dietetically, we do not mean to suggest unlimited ingestion of food, but rather a painstaking investigation of the cause, which may be an unduly large stomach or convalescence from infectious disease (typhoid, diabetes). We do not class the increased desire for food observed in men performing exceptional physical work, in women during pregnancy and lactation, as well as in rapidly growing children, as bulimia. This augmentation of

hunger is due to a greater requirement of food, because the organism has greater expenses in supplying material for growth or energy. Many forms, perhaps 60 per cent., of bulimia cases are due either to hyperesthesia, hyperacidity, or hypermotility. If these are causes, the treatment given under these diseases should be administered (clinical part). The meals should be allowed every two or three hours, and consist largely of such proteids as have a great combining affinity for HCl. These are given under Fleischer's list of the HCl binding power of foods. But if an irritative state of the glandular layer can be ascertained, the diet should be largely amylaceous. If the motility be exaggerated with the hyperacidity, it is well to direct the patient to drink frequently of cold alkaline waters, such as the Saratoga Vichy, particularly when the stomach is empty.

Anorexia, in its severe forms, is most often due to organic changes in the gastric walls. In the nervous forms it is often benefited by a course of forced feeding with the stomach-tube. Persistent anorexia in highly neuropathic individuals had, in fact, best be treated this way as soon as the patients positively refuse food, because a complete cure can frequently be accomplished by gavage alone. The feeding through the tube has a moral and educational effect not to be underestimated. In our experience as physician in charge of Bay View Asylum, many cases were observed to resume taking their meals with good appetite as soon as they became convinced that forced feeding would be insisted upon. But aside from this moral effect there is also a physiological one: this consists in the supplying of a stimulant to the stomach in the form of food. Nourishment is the proper stimulant to secretion, and if it is wanting for a long time the functions of the stomach soon become arrested, and with them the appetite. The nutritive stimulant to the gastric mucosa is food; it causes a filling of the blood- and lymph-vessels, thus indirectly bringing about a better nutrition of the histological elements of the mucosa and a resumption of HCl formation with ferments, which in anorexia is, as a rule, suppressed. In fact, as appetite causes eating in the healthy, so eating will cause appetite in these cases of anorexia. In mild cases of anorexia a sensation of hunger is frequently started up by salty and "piquante" articles, such as caviar, sardelles, herrings, etc., and at the same time small doses of alcohol with bitter tonics, such as the Angostura Bitters, are advisable. Lavage with solutions of chlorid

of sodium or a .04 per thousand HCl is most effective. The most essential condition to a proper dietetic treatment is, of course, that the patient should have appetite. A great point is gained if he can be made to take food with pleasure. For the management of those cases with anorexia we must refer to the article on this subject.

Sir William H. Broadbent (*Brit. Med. Jour.*, vol. II, 1893, p. 1268) says: "In all cases in which the cause has been overfeeding or improper food, or food taken at a wrong time, an extremely strict and meager diet for a few days will be the best treatment. No advantage is gained from a low diet in neurotic cases.

"The object we set before ourselves must be, not to *level down* the diet to the digestive capabilities of the stomach, but to *level up* the digestion till it can deal efficiently with the amount of food for the due support of the nervous system. No hard and fast rule can be laid down." Speaking generally, such a (neurotic) patient will digest food which he relishes better, even if it have the reputation of being indigestible, than the most digestible and scientifically prepared food which he eats by order, and dislikes. A very common experience is that he is tempted by a good dinner, eats largely and indiscriminately, and then, instead of a bad night and great discomfort, which he thinks he has deserved, he sleeps well and feels all the better for his indiscretion.

A very important point will be to disabuse the patient's mind of the idea that pain after meals necessarily indicates that the food has been unsuitable. One day, and under one set of circumstances, anything will agree; on another day, under different circumstances, nothing is digested. Directions must be given not to eat when exhausted or excited or anxious, not to jump from meals and rush off to work of any kind, and to eat very slowly.

The state of the *gastric absorption* has to be considered in the selection of a diet. In most text-books this factor of dietetics is entirely neglected. In the light of the most modern knowledge on absorption, that furnished by the work of von Mehring, according to which it is almost limited to cane-, grape-, and milk-sugar, maltose, dextrin, alcohol, and peptone, whilst water is not at all absorbed (see Absorption, p. 90), it is not at once evident why the state of absorption should be considered in selecting a diet. Von Mehring's results, however, seem to point the way in selecting peptones, maltose, dextrin, alcohol, etc., where we must depend on rapid diffusion of nutritious material, and also in avoiding

water, or foods containing water, where the gastric walls are weak, because it is not absorbed and overdistends by its weight. Not only this, but simultaneously with resorption a more or less active excretion of water occurs into the stomach. The amount of this excretion of water increases or diminishes with the quantity of substances resorbed or taken up. Certain gastric diseases connected with much fermentation are supposed to be in etiological relation with tonic muscular spasms—forms of tetany of gastric origin. Bouveret and Devic ("Rech. clin. et experim. sur la tetanie d'origine gastrique," *Revue de Méd.*, 1892, XII, p. 48) assert that alcohol is instrumental in favoring the formation of an intragastric diffusable toxin in dilations and hypersecretive states, and that these poisons bring about the spasms. Fleiner (*loc. cit.*) and Kussmaul recommend that no alcohol in any form be given in dilations with pyloric stenosis, where naturally the absorption of the alcohol must be very much retarded. The latter authors do not accept the toxic origin of the spasms, but suggest that the alcohol causes a tremendous excretion of water into the stomach, thus robbing the organism of a requisite amount,—a tetany, therefore, due to drying of muscles and nerves; both views are merely hypotheses. The practical deduction is that where the absorption has been found defective by tests, alcohol had best be avoided.

Boas recommends that explicit written directions be given to each patient after the diagnosis has been made, concerning—

(1) Exact time of meals.

(2) An exhaustive account of articles of diet and luxury that are allowed.

(3) An exact statement of the weights and measurements of the foods and beverages.

(4) Brief instructions on the preparation of the food, temperature of drinks, seasoning, etc.

(5) Special account of foods that are forbidden.

The time for the ingestion of food is an essential factor in dietetics, particularly with our American business men, with whom it is a common practice to sacrifice meal hours to business. The hours for meals should be religiously observed by gastric sufferers, and the hours for stomach-rest or fasting also. Hyperacidity and forms of nervous dyspepsias require small meals frequently repeated; the same is true of some types of atonic and stenotic gastrectasias.

Other stomach diseases require long pauses of rest between the meals, and it is not always possible to state *a priori* how much digestive work and how much rest any particular diseased stomach may require. It is only after a prolonged study of the various gastric functions that the physician can give correct instruction in chronic cases.

There is much need for enlarging the dietetic menu of dyspeptics; nothing should be forbidden, except there are actual facts founded on experiment, the nature of the disease, or the idiosyncrasies of the case proving it to be harmful. Our experience is that when the menu is too limited a certain disgust for the diet eventually becomes manifest, resulting either in temporary anorexia or a disregard of the directions and indulgence in forbidden foods.

Directions as to **Preparations of the Food** are sometimes necessary. Here the physician must be able to indicate, for instance in an- or subacidity or atrophic gastritis, that the meats should be finely scraped or cut, then cooked in a steam broiler with a liberal seasoning of pepper and salt. In hyperacidity, gastroxynsis, gastritis acida, and the convalescence from gastric ulcer, all seasoning except a little salt must be avoided; wherever there is excess of HCl the meats should also be finely divided before cooking. The amounts of paprika, red and black pepper, mustard, horse-radish, lemon, vinegar, and ginger, that can be allowed in cases of absolute suppression of secretion (as these materials have some effect in stimulating secretion), must be stated.

The preparation of soups and gravies, the amounts and kinds of fats and sugars to be used in the cooking, are points of importance. For a more detailed account of these indispensable methods and directions for preparations of food, reference must be had to works on Dietetics,—*vide* Gilman Thompson, Munk and Uffelmann, Wiel (Tisch für Magenkranke), Boas ("Diät u. Wegweiser f. Magenkranke"), Woltering ("Diätetisches Handbuch"), Yeo, Penzoldt (vol. IV. of the "Handbuch f. speziel. Therapie"), Honigmann (*Zeitschr. f. Krankenpflege*, 1894, No. 8), Wegele ("Diätetische Behandl. d. Magen- u. Darmkrankh.).

The Diet as Influenced by the State of the Secretion.—The anomalies of secretion are: (1) Hyperacidity, (2) Sub- and Anacidity, which form one group of gastric neuroses. In another group we may classify hypersecretion (of normal gastric juice in

which the HCl is not increased or diminished.) This is the "Magensaftfluss" of Reichmann, the gastrosuccorrhea chronica or periodica, of which Schreiber holds that it is not a disease *sui generis*. We class the so-called "gastroxynsis" of Rossbach with the hypersecretions, because it impresses us to be a gastric neurosis with excessive secretion and hemicrania, and is hardly entitled to be classed as a distinct and separate disease.

As far as diet is concerned, the hypersecretions do not exactly coincide with the hyperacidity in the treatment. For the augmented gastric juice in the super- or hypersecretions may be a passive act on part of the glands,—their activity may be kept up by retained food. But in hyperacidity the excessively high percentage of HCl is an active process, an irritative state of the mucosa in which it responds with excessive formation of acid to all food stimuli. In the hypersecretions the diet should be selected with regard to favoring rapid gastric evacuation. In hyperacidity there is no better diet than rest. These are states in which there is an accelerated digestion of albuminous and proteid foods, and a retardation of carbohydrate digestion, which is caused by an inhibition of the inverting action of the diastase of the saliva, the ptyalin by the excessive amount of HCl. The same is true of the pancreas diastase. Boas has shown (*loc. cit.*) that a neutralization of the chyme will restore the diastatic action, but we have assured ourselves that if the gastric acidity has once reached 0.3 per cent. the action of the ptyalin can not again be so perfectly restored by neutralization with sodium carbonate as it was before. In other words, excessive hyperacidity permanently damages the ptyalin. It may resume some inverting action after neutralizing, but it is not equal to that it evinced during the first forty-five minutes of normal gastric digestion. An intensely acid gastric juice will produce a deleterious effect on the bile by precipitating from it a substance up to the present time not isolated, by which it effects partial digestion of the fats. In a similar way the secretion of the pancreas is prevented from performing its work, because it can do so only in an alkaline medium. There are three organic diseases which dietetically come under this group of excessive acidity or secretion; these are ulcer, gastritis acida, and ulcus carcinomatosum. Concerning the dietetic treatment of hyperacidities, uniformity of opinion does not exist. As a general rule, it can be stated that in the simple forms a bland, unirritating diet, which at

the same time binds as much hydrochloric acid as possible, should be prescribed. We are in favor of a diet that does not irritate the mucosa any more than is absolutely necessary. There are two indications: (1) An etiological one, directed to the condition of the mucosa and demanding rest for the irritative state present. (2) A symptomatic one, directed to neutralization of the excess of HCl by diet having the greatest HCl-binding affinity. These two indications are to some extent opposed to one another. The etiological indication necessitates avoidance of albuminous food, for in our experience proteid and albuminous foods produce an increased secretion of HCl. The second or symptomatic indication calls for a large ingestion of albumen to combine with the HCl. In case of ulcer the food must be the least irritating, the mildest that our menu contains. Not the total quantity of acid secreted constitutes hyperacidity, but the amount secreted in *excess* of what is *required* for combining with the proteids. For instance, a case may show hyperacidity after a simple Ewald test-breakfast of a roll and a glass of water, because the acid secreted has nothing to combine with and remains free, whilst the same case may show very little excess or normal acidity after the first of our double test-meals, as employed at the Maryland General Hospital, consisting of beefsteak, eggs, rice, milk, and bread, because the acid, in this instance, at once enters into combination. The more abundant secretion of HCl is more completely used up when the meals consist of a preponderance of proteid food than when they consist of carbohydrates. Therefore, the dietetics of these cases, as usually recommended, include the red meats, venison, game, turkey, eggs, chocolate, etc., liberally, a certain limitation of carbohydrates, and the alkaline carbonated waters. In hyperacidity and supersecretion spices are to be forbidden, and only so much salt as is indispensable to make the food palatable. All acids, such as vinegar or lemon-juice, in the food simply aggravate the trouble.

There are undoubtedly different kinds of hyperacidities. We feel justified in distinguishing two classes: (1) Those in which there is a preponderance of nervous symptoms and fragments of the mucosa show no increase in the number of gland-tubules or in the oxyntic or acid cells; these cases are, then, of a purely neurotic type.

(2) Secondly, those in which there is an increase in the number

of gland-tubules or in the oxyntic cells. There is no hard and fast line to separate these classes, but they demand somewhat different treatment. A number of competent observers have recommended an exclusion of proteid and an increase of the carbohydrate foods in hyperacidity.

For, although proteid foods combine with more HCl than any other, they are also the greatest stimulants to the secretion of acid. See Dujardin-Béaumetz ("Traitement des maladies de l'estomac") and von Sohlern (*Berlin. klin. Wochenschr.*, XCI, Nos. 20 and 21); Fleiner (*Volkmann's klin. Vortr.*, No. 103); Rummo (*Terapia clin.*, 1892, Nos. 10, 11, 12); v. Jaksch (*Zeitschr. f. klin. Med.*, Bd. XVII, 1896.) These writers argue that carbohydrate food is not so irritating and calls forth much less secretion of HCl. W. Roux ("Entwicklungsmechanik der Organismen," 1895) states that increased activity heightens the specific force of the organs, whilst diminished activity lowers it. The existence of the cells of the organism depends upon their work; those that work most are nourished best and grow strongest. In other words, the elements in any tissue that are incited to greatest activity and function will gain supremacy over others and increase in strength and numbers. The deductions are not purely theoretical, for not only do we find proliferation of acid cells in hyperchlorhydria to be present in from 50 to 75 per cent. of the cases, but in animals with a high acidity of HCl (dog, fox, wolf, etc., carnivora) there is a tremendous multiplication of acid cells. It seems logical, therefore, that there are cases in which the hyperacidity may in the long run be kept up by a proteid diet, although for the time being this diet may render the acidity less by combining with the free HCl. Experience teaches that the most annoying symptoms, the gastralgia and pyrosis, are promptly relieved by the proteid diet, and we shall endorse the latter as most eminently proper in selected cases. When, however, the symptoms are relieved only very briefly, particularly when the ratio of the ethereal to the preformed sulphates in the urine is found to become very high under a rich albuminous diet, and the indican increases, we advise a diet rich in carbohydrates and fats. See Chas. E. Simon, on "The Relation of Indican to Gastric Diseases" (*Am. Jour. Med. Sciences*, August, 1895). This can be filled by all breads and articles made from flour, rice, peas, beans, potatoes, the cereals, oatmeal, and rich milk and butter. It is true that in some forms of hyperacidity these substances can be

found sometimes six hours after they are ingested, unchanged in the stomach; here the motility is seriously at fault. As alkalies must be given even with a proteid diet, they should, in case the food consists largely of carbohydrates and fats, be given immediately after meals and combined with ptyalin or diastase to hasten amylolysis. It is frequently observed, that the amount of free HCl becomes less and less, and the alkalies and artificial ferments may be dispensed with if the amylaceous diet is persisted in. This diet we suggest particularly after the albuminous diet has failed, for there are cases of hyperacidity which are undoubtedly maintained by an exclusive proteid diet. It must not be overlooked that such a thing as a *pure* carbohydrate diet does not exist, because all articles of this class contain protein, and some very considerable quantities of it; peas, beans, and lentils, for example, contain more protein than beef, ham, or fish. It is not a total exclusion, but simply a reduction of proteid that is practically recommended.

All cases of hyperacidity require a certain amount of carbohydrates. It is a matter of experience that proteid diet alone will not permanently satisfy their cravings. Flour and the many articles prepared from it are not readily converted into dextrin in an excessively acid medium. It is expedient, therefore, to recommend dextrinized flours, such as Avenacia, Maggi, and Kuffeke's flour. The American product, "Horlick's Food," is a flour in which the wheat starch has been almost entirely converted into dextrin by malt diastase. It has a high caloric value, and its price is sufficiently moderate for humbler practice when artificial flours seem indicated.

Regarding the preparation of carbohydrates, we refer to the special lists given in the text under the various diseases, and to Wegele's "Diätetische Küche." In the hyperacidity of ulcer, the diet must be of the least irritating quality, and the coarse-fibered meats—beef, mutton, lamb, veal, venison—are not to be allowed, even during the periods of convalescence, and when they are finally conceded, they should all be reduced to a pulpy (scraped) form.

In sub- or anacidity, when the motor function is good, the problem of diet is not so complicated, because the deficient HCl can be supplied if it is found necessary, and the intactness of the peristalsis insures a good intestinal digestion. As the motility is the only safeguard against malnutrition, great care should be taken to avoid injuring it by overloading the organ. Small meals frequently repeated are indicated, consisting of very tender meat (in fine sub-

division), soft, tender vegetables, such as finely chopped spinach, cauliflower, ends of asparagus, purée of potatoes, peas, beans, lentils. The fats, which are best given in form of rich cream and good butter, have a high caloric value. They must be forbidden as soon as it is discovered that they cause gastric irritation by formation of fatty acids. The diet must vary according to the cause of the sub- or anacidity. If it can be ascertained that there is no injury of the glandular apparatus, but simply an inhibition of secretion, the salty and spicy articles, even pepper and ginger, may be advised.

Such sub- or anacidities are improved by taking caviar, sardelles, small pickeled herrings, or anchovies before meals, because salt is an approved stimulant to secretion. In these cases HCl is not only supplied because of its deficiency, but also because it is actually curative in hastening the resumption of secretion. If, however, the absence of HCl and ferments is due to results of inflammation still going on, all spices and unnecessary salt and foods containing them must be forbidden, since they may act as irritants. Although HCl is absent, it will be found best not to administer it when it causes symptoms of gastric distress. In these cases, where the mucosa is extremely sensitive and an atrophic gastritis exists, gastric digestion had best be converted into an alkaline proteolysis and amylolysis by supplying pancreatin and sodium carbonate. In these extreme cases of sub- or anacidity it is sometimes found that hydrochloric acid gives pain and even causes emesis. Meats that are given in anacidity must not be too fresh, but properly seasoned and very tender; they must be thoroughly cooked in a steam broiler until they almost fall apart into the primitive muscle-bundles. A practical way is to rub, cut, or scrape the meat prior to cooking it. Finally, if in addition to the anacidity one has reason to believe that duodenal digestion is also disturbed (from chronic duodenitis, occlusion of the pancreatic or bile duct, or from carcinoma of the duodenum, pancreas, gall-bladder, or liver), then the administration of meat powders and beef peptones is in order. These substances, which are really albumoses, though capable of satisfying the requirements of metabolism, are not palatable and are relatively expensive. The peptones most frequently used in Germany are those of Kemmerich, Denayer, and Maggi. Ewald and Gumlich (*Berlin. klin. Wochenschr.*, 1890, No. 44) have investigated the qualities of a "peptone beer" and found it quite nutritious. Boas speaks favorably of the American product "Mosquera"

Julia Beef Meal. Professor R. H. Chittenden (in a report to the Philadelphia County Medical Society, May, 1891) has given the results of his analysis of American beef products, which are found in the following table:

PERCENTAGE COMPOSITION OF BEEF PRODUCTS, ANALYZED 1891.

Constituents.	Liebig's Extract of Beef.	Armour's Extract of Beef.	Valentine's Meat Juice.	Wyeth's Beef Juice.	Bovinine.	Murdock's Liquid Food.	Johnston's Fluid Beef.	Arlington Chem. Co.'s Beef Peptonoids.	Mosquera Beef Meal.
Water (at 110° C.),	20.06	14.03	60.31	57.88	81.09	83.99	39.58	6.80	6.68
Solid matter (at 110° C.),	79.94	85.97	39.69	42.12	18.91	16.01	60.42	93.20	93.32
Soluble in water,	. .	. . .	. .	. .	.	. . .	50.40	48.14	31.26
Insoluble in water,	0	0	0	0	0	0	10.02	45.06	62.06
Inorganic constituents,	24.04	28.29	11.30	17.52	1.02	0.66	13.52	5.08	4.23
Phosphoric acid (P_2O_5),	9.13	7.28	4.00	3.94	0.03	0.09	3.91	1.40	1.71
Fat, ether extractives,	0.91	1.27	0.78	0.85	1.49	0.27	1.29	2.95	13.60
Soluble in 80 per cent. alcohol,	55.72	67.92	29.15	35.08	.	. .	34.10		. . .
Total nitrogen,	9.52	8.80	2.68	3.25	2.43	2.29	7.38	4.42	12.36
Nitrogen of insoluble matter,	. . .	. . .	. . .	. . .	. .	.	1.46	3.25	7.65
Insoluble proteid matter,	. . .	. .	. . .	. .	. . .	. . .	9.12	20.30	47.81
Soluble albumin coagulable by heat,	0.06	0.68	0.55	0.47	13.98	14.29	0	0	0
Soluble albumoses,	0	0	0	0	0	0	0	5.44	11.09
Peptone,	0	0	0	0	0	0	0	1.87	18.34
Total proteid matter available as nutriment,	0.06	0.68	0.55	0.47	13.98	14.29	9.12	27.61	77.24
Nutritive value as compared with fresh lean beef (lean beef = 100),	0.30	3.50	2.80	2.40	72.40	74.00	47.20	143.0	400.00

It should be stated in justice to other manufactured products which Prof. R. H. Chittenden has included in his list for comparison, that extract of beef is, by a number of firms, offered not as a nutritive or food for the sick, but as a culinary article, the same manufacturers making also other highly nutritious foods.

Armour & Company, of Chicago, manufacture a valuable product which is called "Vigoral," containing 68 per cent. albuminoids. It is a saturated solution, or rather suspension, of pure powdered beef

in beef extract. This is not a patented or proprietary article, and can be conscientiously recommended. So the table of Chittenden's comparisons is valuable only to show the superiority of foods containing the beef in powder or insoluble form, to the extracts, which represent only the soluble salts of the beef and very little of the nitrogenous constituents—rarely more than eight per cent.

The Mosquera beef meal is a product that undoubtedly has an exceptionally high nutritive value, the total proteid matter available as nutriment being 77.24 per cent. It also contains 13.60 per cent. fat, 11.09 per cent. soluble albumoses, and 18.34 per cent. of peptone. With Chittenden's authority for this analysis, and our own experience as to its easy digestibility and perfect absorption, this product commands an important place in our dietary for sub- or anacidity and gastric atrophy, particularly when associated with intestinal disease. The juice of the pineapple contains a proteolytic ferment, thus adapting this fruit for the treatment of cases where no gastric juice is secreted. For its digestive effect the juice of the fresh fruit only should be swallowed and the fiber removed from the mouth. By boiling the pineapple the proteolytic ferment is destroyed.

When *atony* or pronounced *dilatation* accompany any gastric disease, particularly those already referred to, the dietetic management is most important. Weakening and loss of motility are among the most serious affections of the organ and in the gravity of their consequences outweigh any disturbance of secretion. Motor insufficiency may supervene upon any gastric disease. As a rule, the chronic affections rarely become manifest until the motility is disturbed; that is, until the muscular tonus relaxes. Many times a co-existing secretory or organic disease is the cause of the dilatation; for instance, it is generally admitted that hyperacidity can produce spasm of the pyloric sphincter which the gastric peristalsis will be unable to overcome.

The section on motor insufficiency in the second part of this work will consider fully the etiology of this affection. When ingesta remain in the stomach longer than normally, fermentation, gas formation, and distention eventually supervene, causing stasis in the muscular layer and dilatation (Naunyn, *Deutsch. Arch. f. klin. Med.*, 1882, Bd. XXXI). In dilatation proper—the deciding sign of which is presence of ingesta in the stomach in the morning after a test-meal taken twelve hours previously—we must, from a dietetic as well as from a therapeutic standpoint, distinguish between the

myasthenic, atonic form due to simple relaxation of the muscularis, and the obstructive form due to pyloric or duodenal stenosis.

In the first variety dietetic and other treatment may effect a cure. In the stenotic types little beyond transient improvement must be expected, except when the obstacle (cicatrix, carcinoma, hypertrophied pylorus, gall-stones, peritoneal adhesions, etc.) can be removed permanently or temporarily, or where a new route can be devised for the passage of the chyme. Here abdominal surgery asserts itself, and gastro-enterostomy has thus far produced the best results. (See chapter on "Operative Treatment of Gastric Diseases.")

The dietetic management of dilatation will have regard not only to the injured muscular tonus, but also to the secretory or organic disease with which it may be associated. For example, if it is associated with chronic gastritis, the diet must be that for this disease and dilatation; and if it is combined with hyperacidity, the diet is the one devised for this trouble and dilatation. As the fundamental thing in dilatation is to prevent burdening of the muscularis, the diet must be as light in weight as possible, and especially, as far as practicable, exclude liquids, for these are not only all heavy, but are, according to von Mehring, not at all absorbed from the stomach (excepting alcohol). The requisite amount of water is best given by high sigmoid enemata: ½ liter, slightly warmed, two or three times a day. In this manner a water impoverishment can be prevented (see Wegele, "Die atonische Magenerweiterung u. ihre Behandlung"). An absolutely dry diet was suggested by van Swieten, Chomel ("Des Dyspesies," Paris, 1857), and Fossagrives, but its strict execution, as was developed by Schroth, is impracticable, since fully developed dilatations require months, even years, of strict dieting, which, if followed out on these lines, would inevitably produce dangerous drying out of the organs (Kussmaul, "Z. Behandl. d. Magenerweit.," *Deutsch. Arch. f. klin. Med.*, Bd. VI).

Although it is not strictly a dietetic therapy, yet lavage must be mentioned here. If large masses of decomposed food are vomited the stomach-tube is indispensable. Milk diet, used exclusively, aggravates the symptoms without exception. Should the vomit or test-meals reveal that carbohydrates habitually disagree and ferment, an exclusive beef or meat diet for a few weeks is rational, and is followed by less distention (Minkowski).

In very severe and extreme cases of dilatation one may be compelled to feed exclusively by rectal enemata, for the preparation of

which we refer to the paragraph on that subject. Soups and drinks during meals must be avoided. Great thirst can be quenched by taking small pieces of ice into the mouth. Patients that are being treated with lavage may be permitted to quench their thirst before the evacuation. Moritz has shown that solid food is retained longer than semi-solid; the latter form is therefore preferable. Meats are given best in scraped or finely chopped state, and must be of the red varieties and free from fat. Meat dumplings or balls, hash of fresh beef or lamb, Mosquera, Valentine's, Wyeth's, or Wiel's beef jelly, are adapted to dilatations in which secretion is preserved. When gastric digestion is much lowered the cereal and leguminous products are useful. We recommend gruels made from arrowroot, tapioca, rice, sago, cerealin, strained oatmeal, to which we are accustomed to add some form of diastase, either the taka diastase or malt extract. Aleuronat flour, containing much digestible albumin (prepared by Dr. Hundhausen, Hamm, Westphalia, Germany), and the soup meals of C. H. Knorr (Heilbronn, Germany) are of use when prepared according to our dietetic directions. To be digestible, even for healthy stomachs, all leguminous foods must be cooked a long time. For gastric sufferers they must be used only in a condition of very fine subdivision, and partial dextrinization of their starch, rendering it more soluble. Besides the products of this character just mentioned, we have the Liebig malto-leguminose (prepared by William Roth, Jr., in Stuttgart), and the biscuit-leguminose (Theodor Fimpe, in Magdeburg). The firm of Hartenstein & Company, Chemnitz, Germany, prepare several good leguminous flours. (Further reference in Penzoldt and Stintzing's "Handbuch d. Therapie," vol. IV, pp. 256-258.)

The collection of dietaries will contain menus for gastric atony and dilatation (1) with loss of secretions or anacidity, (2) with normal or augmented secretions, (3) with serious stenotic symptoms. In severe cases of the latter type even the most sparing diet by the mouth will be impossible, and as a last resource we must fall back on rectal enemata. Sometimes after a week to ten days of rectal alimentation the diseased condition, if it be within the stomach,—as, for instance, an ulcer or carcinoma at the pylorus,—becomes so improved that partial mouth-feeding may be resumed.

The diet in the various types of **carcinoma** coincides with that of motor insufficiency and dilatation whenever the neoplasm is causing the stenosis. In carcinoma of those portions which do

not form an obstacle to the exit of the chyme, and where the motility is good, the patient's appetite must be stimulated as much as possible by strychnin, HCl, bitter tonics, condurango, etc., and nothing forbidden, as the physician must be satisfied if the patients eat anything. As chronic gastritis is always present even in these cases, the diet list as given for this disease is advisable.

In cancer arising on the basis of old gastric ulcers, the *ulcus carcinomatosum*, there is often a pronounced hyperacidity which naturally is best met by the diet recommended for augmented gastric secretion, provided the stenosis permits it.

Where the stenosis is at the *cardia* the matter of proper alimentation becomes difficult.

As long as the stenosis, no matter from what cause, can be kept open by bougies and sounds, a highly nourishing liquid diet of milk, eggs, beef jelly, beef meal, peptone, nourishing soups containing somatose, and wines are indicated. In rare cases we have seen life prolonged by allowing the esophageal tube to remain *in situ* and feeding through it every two to three hours. Leyden (Leyden-Renvers, *Deutsche med. Wochenschr.*, 1887, No. 50) and Gersunny (*Wien. med. Wochenschr.*, 1887, No. 43) have strongly indorsed this procedure for esophageal strictures of carcinomatous origin. After the stenosis is no longer passable and gastrotomy has become necessary, nutrition must be carried on through the gastric fistula. According to Julius Friedenwald's studies on salivary digestion it would be logical to advise these unfortunates to chew their food first, and removing it from the mouth insert it through the fistula. But where this is objected to, it is expedient to add 15 grs. of ptyalin to the food, which must always be liquid or in the form of paste, soup, or gruel. Finally, when gastrotomy can not be done, or permission thereto is refused, the only way to nourish the patient is by rectal enemata. So it is evident that the symptoms and dietetic management of carcinoma vary greatly according to its location. At present we have under observation a patient who has a gastric tumor; as far as can be palpated it is about four inches long and two inches wide. There has been no evidence of HCl secretion for over a year, but, as the motility is very good, there is no lactic acid formation. The Oppler-Boas bacillus has been repeatedly found in the gastric contents. Still, this patient, with an undoubted carcinoma, has gained 12 pounds in six weeks, and

has no subjective complaints while in our sanitarium for digestive diseases.

With anacidity and a fair peristalsis a carbohydrate diet is applicable to carcinoma, but when HCl is well tolerated the various meats must not be forbidden. Of these, Boas prefers the meat from various fishes. Where HCl is not well tolerated, pancreatin is in place. Lavage can not be avoided when much fermentation and signs of dilatation are marked; in case these signs are very annoying, a few days of exclusive meat diet or of rectal feeding may be necessary to restore somewhat of the lost gastric tonicity. Before pyloric stenosis is complete, the case should be transferred to the surgeon, as the resorption from the stomach itself, even if it were normal, is insufficient to maintain life. Those materials that are readily absorbed from a healthy stomach, such as peptone, glucose, alcohol, etc., are given in these cases. There is little evidence, however, of their being absorbed. The good effects of gastro-enterostomy consist not only in the entrance made for the food into the intestine, whereby better digestion becomes possible, but also in an improvement in the inflammatory process around the neoplasm, which is no longer kept in constant irritation by stagnating, fermenting masses of food in continual contact with it. The main reason why operations do not bring as much relief and improvement as is expected is to be sought in delay in performing them.

The Dietetics of Gastric Ulcer and Erosions. — There are three types of gastric ulcer which demand a varying or separate dietetic treatment. These are: (1) Light attacks with pain, hyperacidity, and pyrosis, but no vomiting of blood. (2) Serious cases that have had hematemesis, and still have signs of it at the time of presentation. (3) Old, chronic, frequently relapsing gastric ulcers. There are forms that run a latent course, void of symptoms until a sudden severe hemorrhage surprises the patient and physician, and possibly terminates the case. Erosions which have no great extension laterally nor toward the depth, and can be recognized by fragments of mucosa found in the washwater, yield very readily to an exclusive diet of milk combined with rest. During the gastric hemorrhage we advise that nothing at all be given by the mouth, not even water; nor ice pills,—positively nothing; but absolute rest, a hypodermic injection of 30♏ of ergotol, and, if pain and restlessness are marked, ¼ of a gr. of morphin sul-

phate hypodermically, and a small ice bag placed over the epigastrium. Wiel claims to have arrested gastric hemorrhage by lavage with cold (10° C.) or hot (42° C.) water. Such treatment in profuse bleeding impresses one as hazardous. Our course has, in a very large number of cases, been, as a rule, very satisfactory.

If there is much weakness we use the Boas or Ewald nutritive enemata on the day of and following the hematemesis; but if the pulse is good we dispense with them. On the day following the hemorrhage, milk in teaspoonful doses is given every half hour; egg-albumen, if it is taken willingly, is more fitting, as it combines with a larger amount of HCl, and when diluted it does not stimulate secretion in small doses.

Brandy and wine are to some extent irritating to ulcers and excite more secretion; they are accordingly not given except there be great prostration. On the third day it will be safe to proceed to carrying out a Leube rest-cure, with the consecutive order of diet suggested by him or the diet lists proposed by Penzoldt, both of which present four different groups of food-materials. Beginning with the simplest and most digestible, they gradually lead up to a more consistent ordinary household menu.

Each of these four diet orders must be persisted in from one week to ten days. Penzoldt's and Leube's diet orders, together with our explicit diet lists for various stages of gastric ulcer, will be appended. The principle underlying all treatment by food in these cases is to secure the greatest amount of rest and such substances as will combine with the largest amount of HCl and relieve the hyperacidity. In a number of cases of ulcer we have found that an amylaceous diet was retained better and caused less pain than scraped beef or soft eggs. It is advised on the same principles as stated on page 189.

In chronic and frequently recurring cases of ulcer, McCall, Anderson (*Brit. Med. Jour.*, May 10, 1890), and H. B. Donkin (*The Lancet*, September 27, 1890), have had excellent results from total exclusion of the stomach from digestion, by feeding with rectal enemata altogether; some of their cases were nourished in this way for twenty-three days. Riegel (*Zeitschr. f. prakt. Aerzte*, 1890, No. 2) speaks enthusiastically of this treatment in stubborn cases of ulcer, and Boas reports ten cases, all of which but one were cured by this method, by giving three to four of his nutritive enemata daily for fourteen days. We have a personal experience of 14

persistent cases of ulcer treated in this manner, together with nitrate of silver, bismuth subnitrate internally, and rest-cure, and are disposed to look upon the treatment with great favor. An ulcer must not be considered cured until there is no more epigastric pain on pressure and the patient gains weight (Gerhardt).

The treatment of the *sensory* gastric neuroses, to be effective, must combine a number of remedial agents with diet.

Hyperesthesia, *gastrodynia* or *gastralgia*, and *neurasthenia gastrica* are morbid states, the treatment of which must be largely directed to the central nervous organs. The same must be said of the motor neuroses: *Cardiospasm* and *pylorospasm*, *nervous vomiting*, *rumination*, *Kussmaul's peristaltic unrest*, *incontinence of the cardia* and of the *pylorus*. These diseases demand electric, hydropathic, climatic, and medicinal measures, and massage.

A very careful investigation into possible causative constitutional morbid states (anemia, gout, rheumatism, tuberculosis, chlorosis, uric acid diathesis) will often reveal a removable underlying etiological foundation. Fliess has reported cases of gastralgia and vomiting, emanating reflexly from the nasal mucosa, and has cured them by local treatment ("Neue Beitr. z. Klinik u. Therapie d. Nasal-Reflexneurose"). In a similar way attention to gynecological disorders in the female and genito-urinary diseases in the male have led to the cure of distressing nervous dyspepsias. In the gastric neuroses of motor or sensory type the diet must be based, as in all previous diseases, as far as is expedient, upon the state of the secretion and motility. When the general nutrition is disturbed, the plan of treatment most generally adopted is a fattening rest-cure according to the principles laid down by Weir Mitchell and Playfair. This treatment, though not universally applicable, is the one most to be employed in nervous vomiting of hysterical, anemic, and chlorotic origin, and in stubborn cases of anorexia and gastralgia. It is somatic and psychical at the same time. We have had ample opportunity to test the dietetic part of this treatment as it has been developed by Burkart in Germany (*Volkmann's klin. Vorträge*, No. 245). And cases of the types described have, in our local sanitarium and in the Maryland General Hospital, been completely cured. Of course, organic and structural changes in the digestive organs render the proper nutrition impossible.

Contra-indications to fattening cures are: Cerebral excitation or depression; hysteria, with uncontrollable vomiting; and visceral

neuralgias, which are expressions of sympathetic nerve diseases.

The best results with Weir Mitchell's rest and fattening cures are obtained in neurasthenic or hysterical anorexia with much emaciation but where there is no digestive disease. Wherever the nervous dyspepsia is accompanied by gastritis, atony, or dilatation, fattening cures may easily cause pain, pressure, vomiting, and diarrhea; so that if such cures are attempted because everything else has been tried, one must be cautious not to persist in systematic introduction of large amounts of food, even in divided portions, as the frequently repeated small portions may accumulate in atony, etc., and aggravate the symptoms.

Burkart begins with 100 gm. of milk with Zwieback every two hours, and increases it so that two to three liters of milk per day are taken after fourteen days of treatment. The milk may be flavored with sugar, cocoa, tea, lime-water, or salt, according to taste; and after a few days other articles of diet are cautiously sandwiched in. We give Burkart's complete menu among our diet tables.

Neurasthenia gastrica (Ewald), or the *nervous* dyspepsia of Leube, when it occurs in males, is, in our experience, not benefited by the methods of Weir Mitchell or Burkart. Here more than ever the physician must endeavor to remove the cause if possible (excess in tobacco, overwork, uric acid diathesis, sexual overindulgence). A definite diet can not be given because there are rarely two cases alike; the dietetic treatment of nervous dyspepsia is difficult, the strictest individualization is requisite.

The prognosis as regards perfect recovery is doubtful. An attempt with the Leube or Penzoldt order of dieting is not only rational but sometimes productive of lasting improvement.

The Indications for Predigested Foods: Peptones, Albumoses, Dextrose, etc.—The idea of supplying foods that would replace the lost digestive function of the stomach or by presenting them in an absorbable form that would spare the work which it had become incapable of performing, was suggested by the recognition of diseases that tended to destroy the glandular apparatus, or caused emesis of the ingested food.

In such instances in which the amount of albuminous foods that can be taken is very small, the question has been raised whether peptones are able to equalize the deficit of albumin re-

quirement. As a certain amount of albuminous food is indispensable to life, whenever the quantity ingested sinks below the so-called threshold value of albumin ("Schwellenwerth"), health begins rapidly to decline. Deiters has shown (von Noorden, "Beitr. z. Lehre v. Stoffverlust d. gesund. u. krank. Menschen," Heft 1, 1892) that even when the amount of albumin ingested sinks below the threshold value, peptones and albumoses (Denayer's mixture) are capable of maintaining the body in nitrogenous equilibrium. Kuhn confirmed this observation in Riegel's laboratory with regard to an albumose mixture selling under the name of somatose; so that we may conclude that these products can replace food-albumin for a time at least; still, we doubt very much whether it is expedient or necessary to give peptones and albumoses in larger quantities. Their expensiveness would be no serious objection if the advantages of their use were very obvious.

In secretory insufficiency of the stomach it is well known that the proteolytic power of the intestine will utilize most of the unaltered proteid.

Even where the stomach is excluded from digestion entirely, albumin is used up to a sufficient degree (Ogata, *Arch. f. Anat. u. Physiol.*, 1883, S. 89). Loss of peptic function, therefore, does not prevent sufficient utilization of albumin. There is an apparent advantage in the bland and unirritating quality of peptones, but certainly this is possessed also by certain undigested foods (milk, egg-white). Peptones and albumoses are not absorbed more readily when ingested ready-formed, than when they are first developed from albumin in the stomach (Cahn, "Die Verwendung d. Pepton als Nahrungsmittel," *Berlin. klin. Wochenschr.*, 1893, No. 24).

They are said to produce diarrhea; but to establish this fact the quantity of artificial peptone and the amount of nitrogen in the remaining ingesta must be considered together.

As an indication for the use of the predigested foods, we may state the conditions where the albumin-dissolving power of the stomach is permanently reduced or lost, where the amount of meat- and egg-ingestion becomes insufficient because of efforts necessary to avoid mechanical irritation; if duodenal digestion is also deranged, the indication becomes more urgent. When the secretory function is lost but the motility preserved, the vicarious intestinal proteolysis will digest sufficient proteid. When peristalsis is, however, also lost, then the administration of peptones is of no benefit,

for we agree with Cahn (*loc. cit.*) that in gastrectasia dependent upon pyloric stenosis, peptones are not absorbed, but remain in the stomach just as water does. Riegel (*loc. cit.*, p. 241), considers this deduction of Cahn's as erroneous, because von Mering has shown that the stomach never absorbs water, but does absorb peptone. To this we would reply, that von Mering did not experiment upon dilatations dependent upon stenosis, but upon normal stomachs.

In his experiments, Cahn found that peptone causes an increased flow of the gastric juice, and it was suggested that therefore it should not be given in hyperacidity and hypersecretion, in which evidently it is quite unnecessary, as in these secretory abnormalities meat and egg are digested excellently. In ulcer, peptones may find temporary usefulness because of their unirritating qualities, which, of course, would be counterbalanced by their effect in increasing the HCl, should this be confirmed.

In conclusion we may state our personal custom in the use of these substances. Whenever the ingestion of albumin in the food becomes insufficient, or even where it is ingested in sufficient amounts for a healthy individual, but owing to some consuming disease, such as carcinoma (not hindering peristalsis), tuberculosis, etc., it can not cover the nitrogen equilibrium, in the amounts possible to eat in ordinary diet, there we employ peptones and albumoses liberally, and generally so mixed with the food (soups, scraped broiled meats, in purée) that the patient can not detect them; for this purpose the taste- and odorless products,—somatose, Mosquera beef meal, etc.,—are preferable.

Rectal Alimentation.—In diseases in which the approach to the stomach is stenosed (carcinoma, or stenosis of the esophagus or cardia), or in which the organ requires a temporary but absolute exemption from work (ulcer and some forms of gastritis), or in cases in which the mildest and most digestible diet is not tolerated, it becomes necessary to support the strength by nutritive rectal enemata.

The history of the evolution of the nutritive enema is, from a physiological standpoint, very interesting; and as we have at various times done considerable experimental work in this line, a brief review of the same is believed essential to a proper understanding of the subject.

The first to discover that the human colon and rectum absorbed an emulsion of eggs and water only when sodium chlorid was

added, were Voit and Bauer (*Zeitschr. f. Biol.*, 1869, Bd. v). They found that these foods were not absorbed in the absence of salt, and in 1871 Eichhorst (*Phlüger's Archiv, Jahrgang* iv, 71) confirmed their results. Injections of bouillon, milk, and eggs had been used long before this time, but no one ever attempted to ascertain to what degree the mucosa of the large intestine would absorb it. In 1872 Leube proposed a meat-pancreas injection (*Deutsch. Archiv f. klin. Med.*, Bd. x, Reihe iii), the plan emanating from the idea to transpose something of the character of pancreatic digestion into the large intestine. The preparation of this useful enema is as follows: Take 150 to 300 gm. of very finely scraped beef and 50 to 100 gm. of finely chopped pancreas of the calf or pig, and mix with the addition of 150 c.c. luke-warm water in a bowl; if desired, 25 to 50 gm. of fat may be added in the form of oil or butter; the injection must be made at the body temperature.

We have convinced ourselves that this mixture digests thoroughly in the large intestine; its preparation is complicated, however, requiring very intimate mixing. Leube reported a case which he kept alive six months by this enema exclusively, and in a similar way Riegel nourished a case of esophageal stricture for ten months. Ewald demonstrated that egg emulsion is absorbed without being peptonized or salted (*Zeitschr. f. klin. Med.*, Bd. ii), and Huber, while confirming Ewald's observation, added that the addition of salt, or previous peptonizing, really doubled the amount of emulsified eggs that was absorbed in the colon. Eggs, it must not be overlooked, contain a considerable amount of normal salt, and this may explain Huber's results (*Zeitschr. f. klin. Med.*, Bd. xlvii) as to their absorption in part, even without the addition of salt. But it is an established fact that the addition of sodium chlorid very much increases the amount of eggs that is absorbed.

In a very interesting series of experiments Grützner offered a physiological explanation of this phenomenon (*Deutsche med. Wochenschr.*, 1894, No. 48), having demonstrated that under certain conditions particles of charcoal, finely cut horse-hair, or sawdust, impregnated with normal (0.6 per cent.) salt solution and injected into the rectum of rabbits, guinea-pigs, and rats, are found six hours later all along the small intestine, even in the stomach, while the rectum is empty. During a period of twenty-four hours before these injections, the animals were starved. When the suspensions of these particles were made in distilled water, HCl

solution, or potassium chlorid solution, instead of physiological salt solution, the particles did not ascend in the digestive tract. Grützner injected starch-suspensions in normal NaCl solution into the rectum of human beings, and after a number of hours demonstrated starch grains in the gastric contents, microscopically. Nothnagel first showed that sodium chlorid placed on the serous surface of the intestine is capable of starting antiperistaltic movements ("Beitrag z. Physiol. u. Pathol. d. Darms," 1884), and Grützner interprets this observation as an explanation of the digestion of egg-enemata containing salt. He assumes that the injected mass is moved upward through the entire small intestine, and so becomes digested and absorbed. Even Riegel (*loc. cit.* p. 245) is satisfied with this interpretation, and adds that it explains the negative results of Voit and Bauer without salt, and the positive ones with salt, and also those of Huber.

In our opinion, the evidences that food-substances move antiperistaltically upward in the intestine are not satisfactorily given in Grützner's work. It is undeniable that minute particles of starch, charcoal, etc., are moved from the rectum toward the stomach in man; and we have been able to confirm this part of his results, as well as the fact that salt favors the ascent and HCl and KCl impede it. But this antiperistaltic motion we conceive to be only a very feeble marginal ascending movement, effected by surface contact of the particles with the epithelium, which in turn is moved by the muscularis mucosæ. This very slight marginal antiperistalsis is never visible to the eye, and can only be demonstrated by the progress of particles; it is not capable of propelling food masses; on the contrary, we have proven that at the same time that the marginal peristalsis drags visible particles of charcoal toward the stomach with infinitesimal slowness, there is an uninterrupted current of central food masses toward the anus. The marginal antiperistalsis may be a physiological thing, present at all times, and its object may be the raising or drawing up of portions of mucosa from one place to another in order to bring new surfaces in contact with the ingesta, or to replace a portion of surface to its normal topography after it has been dragged away from it by the downward current. The cohesion of these small particles with the mucosa can be seen when a piece of fresh animal intestine is sprinkled with lycopodium or finely cut horse-hair particles and held under a gentle stream of water; the gut may be moved

upward on the surface of the hand while the water moves downward, and still many of the particles will adhere.

The antiperistalsis that Nothnagel produced by placing crystals of salt upon the serosa is quite a different thing, for it is plainly visible to the eye, and never occurs under physiological conditions (Nothnagel, "Erkrank. d. Darms," portion on "Die Darmbewegung," p. 6, 1896). Among the abnormal conditions that may cause this visible antiperistalsis, Nothnagel states stronger solutions of sodium chlorid and the introduction of food at an unphysiological entry, as which in man we must consider the rectum. He adds (*loc. cit.*) that from a physiological entry, *i. e.*, from the stomach, the strongest chemical irritants produce only peristaltic movements toward the anus. There are, then, two kinds of antiperistaltic movements: (1) Those of Grützner, being marginal, invisible, possibly physiological, and not capable of moving food masses; (2) those of Nothnagel, being visible, strong, and occurring only under abnormal conditions. Christomanos, a pupil of Nothnagel, has urged an objection against Grützner's results that seems to invalidate the conclusions of the latter; namely, he found (Christomanos, "Z. Frage d. Antiperistaltik," *Wien. klin. Rundschau*, 1895, Nos. 12 and 13) that when his animals were prevented from licking up the expelled rectal contents his results as to finding the particles in the stomach were negative. Dauber came to the same conclusion as Christomanos, namely, that the occurrence of particles of rectal injections in the stomachs of animals did not take place when they were prevented from eating their excrement. These objections are, however, set aside by Grützner's and our own observations on the human subject. The experiments of Swiezynski (*Deutsche med. Wochenschr.*, 1895, No. 32) also confirmed Grützner's statements, in that lycopodium injected into the rectum was found in the stomach.

We must, however, again emphasize that this antiperistalsis is not capable of moving ingesta, and that it can not logically be taken as an explanation of the digestion of enemata.

If liquid enemata can be worked well up into the small intestines, why is it that the liquid and semiliquid stools are not pushed up into the stomach also under normal conditions? The fact that normal salt solution favors the invisible marginal ascent of particles, and that other chemicals impede it, is perfectly natural. For if this antiperistalsis be physiological, and be assumed to be going on at all times, normal salt solutions can not disturb it, for they are

the physiological environment in which all intestinal movements occur. But HCl and KCl are chemical irritants, to which the muscularis mucosæ reacts by efforts at expulsion.

The Occurrence of Proteolytic Ferments in the Colon and Rectal Contents.—The author's explanation of the digestion of egg and milk emulsions in the rectum and colon is quite different from Grützner's and from that accepted by Riegel and others, and is based on a very carefully conducted series of experiments on animals and human beings. We desire to speak only of actual digestion, for there is good reason for believing that albumen, fats, etc., may be absorbed from the rectum as such—without digestion.

Without going into the details and technic of these experiments we will briefly state the conclusions. After the rectal contents of dogs or cats are sterilized with saturated solutions of thymol (so strong that the crystals float on top) and passed through a Pasteur filter, control cultures are made to ascertain that the watery extract of the excrement is sterile. For this purpose the meat pepton-gelatin and agar plates recommended by Nothnagel (*loc. cit.*, p. 22) are most convenient. The reaction of human excrement is generally weakly alkaline or neutral, very rarely weakly acid, under normal conditions. This watery extract of normal rectal contents contains a substance which in a digestorium (thermostat at 40° C.), and *in an alkaline medium* (equal to from 0.8 to 1 per cent. Na_2CO_3), *dissolves from 36.5 to 50 per cent. of Mercks' dried serum-albumin* in three hours. It will also digest fibrin, and has, in addition a faint amylolytic power, converting from 10 to 14.5 per cent. of starch into maltose in an alkaline medium of 0.3 per cent. Na_2CO_3. We have not been able to find any fat-splitting effect.

Now, as there are many bacteria that produce peptone in the breaking down of proteid, and others that ferment carbohydrates, the previous sterilization is necessary in order to exclude their action. Bacteria do not make peptone for any philanthropic purposes; the peptone they give rise to is an intermediate stage in a long series of decomposition products. It does not remain peptone, but is rapidly decomposed into amido-acids, ammonia, tyrosin, etc. We state this because even among medical men the opinion has been encountered that the bacterial peptone might be of utility to the organism in which it is formed. The feces for our purposes can not be sterilized by heat, because that would destroy any possible enzymes present.

It is certain, therefore, that rectal contents contain a proteolytic ferment; also one having a slight amylolytic power, acting only in a faintly alkaline medium, the action being destroyed in an acid medium. Whether these two digestive actions are carried out by one and the same ferment or by two different ferments we are unable to say. That it can not be pepsin is proven by the fact that it does not act in an acid, but only in an alkaline, medium.

It would be interesting to learn whether the walls of the large intestine secrete any proteolytic ferment. The colon of a dog that is kept clear of fecal masses by making an abdominal fistula and sewing it to the abdominal wall at the ileocecal valve, secretes an alkaline fluid, which, however, has no proteolytic powers whatever, but there is an evident amylolytic ferment contained in it. The human colon can be plugged up in the transverse portion by introducing a balloon and blowing it up; thereafter the part between the rubber balloon and the anus is washed out with sterile normal salt solution. A secretion is formed in two to three hours and can be collected on absorbent cotton, placed in the rectum, and later squeezed out into a small beaker. The secretion is alkaline, but has, after filtration through a Pasteur filter, no proteolytic power. Therefore, it is reasonable to assume that the ferment we have demonstrated is derived from the pancreas. In two patients with total atrophy of the gastric mucosa (atrophic gastritis), as evidenced by fragments of the mucosa found in the washwater, the same proteolytic ferment was demonstrable in the colon contents. It was hitherto assumed that the ferments of the pancreas were destroyed in the intestine (see Rosenheim, "Die Erkrank. d. Darms," p. 46). A large number of similar experiments as above described justifies the belief, however, that trypsin and perhaps amylopsin may survive the passage through the bowel. Busch has shown that digestion may go on in the human intestine without gastric or pancreatic juice, without bile and secretion of Brunner's glands (Brücke's "Vorlesungen über Physiologie," Wien, 1885, p. 352). The patient on whom Busch experimented had received an abdominal injury by an accident in such a manner that the gastric juice, together with the chyme, pancreatic juice, duodenal secretions, and bile, ran outward through a fistula. Thereafter, Busch fed him through the fistulous opening communicating with the lower bowel, and succeeded in maintaining the nitrogenous equilibrium. He lowered coagulated albumin

inclosed in small cotton bags into the bowel, and drew them out by a string five hours later, finding that from five to 35 per cent. of the albumin was dissolved. In Busch's experiments the action of the bacteria can not be excluded; the action of the succus entericus may explain the carbohydrate digestion, but as no proteolytic ferment could enter the small intestine, the digestion of albumin was probably due to bacteria.

In conclusion we may say that rectal enemata are digested probably by pancreatic ferments passing through the bowel, by bacteria, and by the succus entericus, which, even in the colon, has an amylolytic action; that certain foods—egg-albumen, fats, milk—can be absorbed as such without being digested. Grützner's marginal ascending motion of particles can not move ingesta upward. F. Mall ("Johns Hopkins Hospital Reports," vol. 1, p. 70) holds that the propelling force of the intestines normally acts in one direction only; the antiperistalsis is only found as a pathological phenomenon, and all of his efforts to force the intestine to work in the wrong direction by reversal (*loc. cit.*, p. 93) were negative. Under conditions of great irritation rectal contents may be vomited.

Preparation of Rectal Enemata:

Indications and Methods of Administration.—The preparation of Ewald's, Leube's, and Boas' nutritive enemata is given under the dietetic tables. Jaccoud recommends 250 gm. of bouillon, 120 gm. of wine, yolks of two eggs, 5 to 20 gm. of peptone. Rosenheim uses peptone (one to two drams), two eggs, 15 gm. of glucose, and sometimes, if desired, emulsions of cod-liver oil. Singer's enema (*Centralblatt d. ges. Therap.*, März, 1895) is very much like that of Boas, with the addition of peptone. These examples will amply suffice for all purposes.

Method:

(1) Every nutritive injection must be preceded by a cleansing injection one hour previously.

(2) The amount of injected nutriment must not exceed ¼ of a liter (℥viij) at a time.

(3) After the injection the patient must remain in the recumbent position for one hour, and a hot towel should be held firmly against the anus for fifteen or twenty minutes.

(4) The patient should lie on his left side with his hips raised upon a pillow, and the injection must be given very gradually.

(5) If the rectum is very irritable, Riegel recommends the addition of a few drops (10 to 20) of tr. opii.

(6) The injection should be made with a funnel or an irrigating bottle, never with a syringe. The best tube to use is that named after Langdon, as it is sufficiently soft and flexible and can not kink upon itself.

(7) The tube should, in adults, be passed high up into the colon; if possible 14 to 18 inches should be introduced, but 12 inches will, as a rule, suffice. The higher up the enema is placed the less will be the liability of its rejection. An anatomical and physiological reason for placing injections high is found in the nature of the anastomoses of the vascular supply of the rectum, sigmoid, and colon. The superior rectal and sigmoid veins communicate with the inferior mesenteric vein, therefore these veins conduct whatever they have absorbed directly to the liver through branches of the vena porta. In the liver the very important secondary digestion takes place. The veins from the lower third of the rectum communicate with the inferior vena cava, and their contents are not conducted to the liver.

(8) The temperature of the injection should be that of the body —98.6° F.

Indications Necessitating Rectal Feeding.—There are two classes of conditions in which nutritive enemata are indicated.

I. The first class comprises patients that are still able to swallow food and willing to do so, but on account of the existence of some gastric, esophageal, or duodenal disease it is expedient to rest the stomach and exempt it from work. These are:

(1) Gastric Ulcer.—For the purpose of keeping the ulcer free from irritation and permitting it to heal or to prevent the starting up of hematemesis.

(2) Dilatations.—Either in the atonic or benign forms to attempt a cure by relieving the stomach of the weight of ingesta and the constant fermentation; or in the malignant, pyloric, and stenotic forms, because food positively can not pass the pylorus and gastro-enterostomy is refused or impossible.

(3) Severe gastric irritations, as in toxic gastritis.

(4) Exhausting diseases, especially the infectious types, where secretion and absorption are inhibited and food not retained, though swallowed.

(5) Ulcer of the esophagus or duodenum, stricture, ileus, invag-

ination, volvulus, stenosis of any part of the alimentary tract between stomach and rectum.

II. The conditions in which the patients are unable to swallow food are:

(1) Temporary obstruction to the entrance of food into the alimentary canal; presence of new growths; foreign bodies; acute inflammations about mouth, pharynx, esophagus.

(2) Extreme sensitiveness of the mouth and esophagus excited by corrosive poisons.

(3) Carcinoma, cicatricial contraction, diverticulum, neoplasms, of esophagus, carcinoma of cardia.

(4) Reflex vomiting, as in pregnancy and sea-sickness.

There are *other states* in which the patients are either unable or *unwilling* to swallow food, but in these *feeding by the tube* is preferable to rectal feeding. These are: (*a*) Inability to swallow from coma, delirium, or paralysis of the muscles of deglutition;, post-diphtheritic paralysis. (*b*) Insanity, refusal of food. (*c*) Total anorexia (hysterical, etc.).

Intravascular and Hypodermic Feeding.—In 1850 Hodder first practised intravenous injection of milk in cases of collapse from cholera Asiatica. T. G. Thomas about this time published a case in which ½ of a pint of milk warmed to body temperature was injected into one of the brachial veins with the result of saving life. According to Gilman Thompson (*loc. cit.*, p. 383), Fowler has practised intravenous injection of peptone and has also given six ounces of digested beef solution in this manner. There seems to us no physiological reason why intravenous or even intra-arterial feeding should not be practised in emergencies. As a safeguard, however, we would suggest that every precaution be taken to have the injection absolutely sterile, and composed of such substances as are normal to the blood, such as serum-albumin, sterile plasma, defibrinated fresh sterile blood. Much careful experimenting is required, however, before we can be justified in using such methods on the sick human being. Intravenous and intra-arterial injections of warm, sterile, normal salt solutions have been extensively used in Asiatic cholera and in exhausting hemorrhages. We have had occasion to use them in hematemesis after gastric ulcer, with the conviction that life was saved thereby.

Subcutaneous Feeding.—In 1869 Menzel and Perco injected fats,

albumin, and sugar into dogs and human beings, and showed that liquid oils were resorbed without causing local or general reaction. They injected nine gm. of oil into one patient, at Billroth's clinic, who had spinal caries; a swelling as large as a silver dollar ensued, but disappeared entirely in thirty hours (*Wiener med. Wochenschr.*, 1869, No. 31).

Attempts have been made in the human being with injection of defibrinated calf's-blood by Landenberger (*Würtemberg. med. Correspondenzbl.*, Bd. XLIV, No. 20), with olive oil by Krueg (Referat in *Wien. med. Wochenschr.*, 1875, No. 34), with olive oil and milk by Whittaker (*Schmidt's Jahrb.*, Bd. CLXXVII, Heft 1), who in eight sittings injected 124 gm. in one day—in all he made 68 injections. Karst recommended defibrinated blood (*Berlin. klin. Wochenschr.*, 1873, No. 49). Eichhorn was so enthusiastic with his injections of milk-peptone and cod-liver oil that he believed the normal nutrition of an animal could be supplanted by this method (*Wien. med. Wochenschr.*, 1881, Nos. 32, 33, and 34). Leube proved that oils injected subcutaneously were actually used up in the metabolism of the body (Leube, "Verhandlung. d. XIV. Congresses f. innere Medicin," 1895). In spite of these experiments it is very doubtful whether subcutaneous injections of nutritive materials can ever be utilized to even supplement normal feeding. The caloric value of the amounts that are available for injection is comparatively insignificant, the method quite irritating, and in progressed sufferers hardly justifiable.

TABLES OF DIETETICS.

APPROXIMATE ANALYSES OF A MAN.—(*Moss.*)

(Height, 5 feet 8 inches; weight, 148 pounds.)

	Pounds.		Pounds.
Oxygen,	92.4	Sodium,	0.12
Hydrogen,	14.6	Iron,	0.02
Carbon,	31.6	Potassium,	0.34
Nitrogen,	4.6	Magnesium,	0.04
Phosphorus,	1.4	Silica,	?
Calcium,	2.8	Fluorin,	0.02
Sulphur,	0.24		
Chlorin,	0.12	Total,	148.00

Landois and Stirling give the following table, which differs somewhat from the other tables in the relative proportion of fats and starches. An adult doing a moderate amount of work takes in as food per diem:

	C.	H.	N.	O.
120 gm. of albumin, containing,	64.18	8.60	18.88	28.34
90 gm. of fats, containing,	70.20	10.26	. . .	9.54
330 gm. of starches, containing,	146.82	20.33	. . .	162.85
	281.20	39.19	18.88	200.73

Add 744.11 gm. of O. from the air by respiration.
" 2,818.00 " of H_2O.
" 32.00 " of inorganic compounds (salts).

The whole is equal to three kilogm. and a half (seven pounds), *i. e.*, about one-twentieth of the body weight, so that about six per cent. of the water, about six per cent. of the fat, about one per cent. of the albumin, and about 0.4 per cent. of the salts of the body are daily transformed within the organism.

An adult doing a moderate amount of work gives off in gm.:

	Water.	C.	H.	N.	O.
By respiration,	330	248.8	. . .	?	651.15
By perspiration,	660	2.6	. . .	. . .	7.2
By urine,	1,700	9.8	3.3	15.8	11.1
By feces,	128	20.0	3.0	3.0	12.0
	2,818	281.2	6.3	18.8	681.45

STANDARDS FOR DAILY DIETARIES.—(*Compiled by Atwater.*)

Weights of nutrients and calories of energy (heat units) in nutrients required in food per day:

	Nutrients.				Potential Energy.
	Protein.	Fats.	Carbo-hydrates.	Total.	
	Gm.	*Gm.*	*Gm.*	*Gm.*	*Calories.*
Children to a year and a half, .	28 (20–36)	37 (30–45)	75 (60–90)	140	767
Children of two to six years, .	55 (36–70)	40 (35–48)	40 (100–250)	295	1,418
Children of six to fifteen years,	75 (70–80)	43 (37–50)	325 (250–400)	443	2,041
Aged women, ...	80	50	260	390	1,859
Aged men, ...	100	68	350	518	2,477
Woman at moderate work, *Voit*,	92	44	400	536	2,426
Man at moderate work, *Voit*, .	118	56	500	674	3,055
Man at hard work, *Voit*, . . .	145	100	450	695	3,370
Man at moderate exercise, *Playfair*, ...	119	51	531	701	3,139
Active labor, *Playfair*, . . .	156	71	568	795	3,629
Hard labor, *Playfair*, ...	185	71	568	824	3,748
Woman with light exercise, *Atwater*, ...	80	80	300	460	2,300
Man with light exercise, *Atwater*, ...	100	100	360	460	2,820
Man at moderate work, *Atwater*,	125	125	450	700	3,520
Man at hard work, *Atwater*, .	150	150	500	800	4,060
Man at moderate work, *Moleschott*, ...	130	40	550	720	3,160
Man at moderate work, *Wolff*,	120	35	540	695	3,032

Table of analyses made by Dujardin-Béaumetz, showing the proportion of nitrogen present and also the combustible carbon and hydrogen:

	Nitrogen.	C + H Combustibles Calculated as Carbon.
Beef, uncooked, ...	3.00	11.00
Roast beef, ...	3.53	17.76
Calf's liver, ...	3.09	15.68
Foie gras, ...	2.12	65.58
Sheep's kidneys, ...	2.66	12.13
Skate, ...	3.83	12.25
Cod, salted, ...	5.02	16.00
Herring, salted, ...	3.11	23.00
Herring, fresh, ...	1.83	21.00
Whiting, ...	2.41	9.00
Mackerel, ...	3.74	19.26

Table of analyses showing the proportion of nitrogen and combustible carbon and hydrogen (*Continued*).

	Nitrogen.	C + H Combustibles Calculated as Carbon.
Sole,	1.91	12.25
Salmon,	2.09	16.00
Carp,	3.49	12.10
Oysters,	2.13	7.18
Lobster, uncooked,	2.93	10.96
Eggs,	1.90	13.50
Milk, cow's,	0.66	8.00
Cheese (Brie),	2.93	35.00
Cheese (Gruyère),	5.00	38.00
Cheese (Roquefort),	4.21	44.44
Chocolate,	1.52	58.00
Wheat (hard southern, variable average),	3.00	41.00
Wheat (soft southern, variable average),	1.81	39.00
Flour, white (Paris),	1.64	38.50
Rye flour,	1.75	41.00
Winter barley,	1.90	40.00
Maize,	1.70	44.00
Buckwheat,	2.20	42.50
Rice,	1.80	41.00
Oatmeal,	1.95	44.00
Bread, white (Paris, 30 per cent. water),	1.08	29.50
Bread, brown (soldiers' rations formerly),	1.07	28.00
Bread, brown (soldiers' rations at present),	1.20	30.00
Bread from flour of hard wheat,	2.20	31.00
Potatoes,	0.33	11.00
Beans,	4.50	42.00
Haricots, dry,	3.92	43.00
Lentils, dry,	3.87	43.00
Peas, dry,	3.66	44.00
Carrots,	0.31	5.50
Mushrooms,	0.60	4.52
Figs, fresh,	0.41	15.50
Figs, dry,	0.92	34.00
Plums,	0.75	28.00
Coffee (infusion of 100 gm.),	1.10	9.00
Tea (infusion of 100 gm.),	1.00	10.50
Bacon,	1.29	71.14
Butter, fresh,	0.64	83.00
Olive oil,	. . .	98.00
Beer, strong,	0.05	4.50
Wine,	0.15	4.00

THE RELATIVE VALUE OF FOODS.—(*Scammell.*)

(The figures represent percentages.)

Articles.	As Material for the Muscles.	As Heat Givers.	As Food for the Brain and Nervous System.	Water.	Waste.
Wheat,	14.6	66.4	1.6	14.0	3.4
Barley,	12.8	52.1	4.2	14.0	16.9
Oats,	17.0	50.8	3.0	13.6	16.9
Northern corn,	12.3	67.5	1.1	14.0	5.1
Southern corn,	34.6	39.2	4.1	14.0	8.1
Buckwheat,	8.6	53.0	1.8	14.2	22.4
Rye,	6.5	75.2	0.5	13.5	4.3
Beans,	24.0	40.0	3.5	14.8	17.7
Peas,	23.4	41.0	2.5	14.1	19.0
Lentils,	26.0	39.0	1.5	14.0	19.5
Rice,	5.1	82.0	0.5	9.0	3.4
Potatoes,	1.4	15.8	0.9	74.8	7.1
Sweet Potatoes,	1.5	21.8	2.9	67.5	6.3
Parsnips,	2.1	14.5	1.0	79.4	3.0
Turnips,	1.2	4.0	0.5	90.4	3.9
Carrots,	1.1	12.2	1.0	82.5	3.2
Cabbage,	1.2	6.2	0.8	91.3	0.5
Cauliflower,	3.6	4.6	1.0	90.0	0.8
Cucumbers,	0.1	1.7	0.5	97.1	0.6
Milk of cow,	5.0	8.0	1.0	86.0	. . .
Milk, human,	3.0	7.0	0.5	89.5	. . .
Veal,	17.7	14.3	2.3	65.7	. . .
Beef,	19.0	14.0	2.0	65.0	. . .
Lamb,	19.6	14.3	2.2	63.9	. . .
Mutton,	21.0	14.0	2.0	63.0	. . .
Pork,	17.5	16.0	2.2	64.3	. .
Chicken,	21.6	1.9	2.8	73.7	. . .
Codfish,	16.5	1.0	2.5	80.0	. . .
Trout,	16.9	0.8	4.3	78.0	. . .
Smelt,	17.0	very little	5 or 6	75.0	. . .
Salmon,	20.0	some fat	6 or 7	74.0	. . .
Eels,	17.0	"	3 or 4	75.0	. . .
Herring,	18.0	"	4 or 5	75.0	. . .
Halibut,	18.0	"	3 or 4	74.0	. . .
Oysters,	12.6	. . .	0.2	87.2	. . .
Clam,	12.0	very little	2 or 3	. . .	. . .
Lobster,	14.0	"	5 or 6	79.9	. .
Eggs, white of,	13.0	. . .	2.8	84.2	. . .
Eggs, yolk of,	. . .	29.8	2.0	51.3	. . .
Butter,	. . .	100.0	. . .	. . .	. . .
Artichoke,	1.9	19.0	1.8	76.6	0.7
Asparagus,	0.6	5.4	0.4	93.6	. . .
Bacon,	8.4	62.5	0.5	28.6	. . .
Carp,	18.0	0.8	2.9	78.3	. . .
Cheese,	30.8	28.0	4.7	36.5	. . .
Cherries,	0.6	21.0	1.0	76.3	1.1
Chocolate,	8.8	88.0	1.8	. . .	1.4
Cream,	3.5	4.5	. . .	92.0	. . .
Currants,	0.9	6.8	0.3	81.3	10.7
Dates, fresh,	. . .	73.7	. . .	24.0	2.3
Figs,	5.0	57.9	3.4	18.7	15.0
Ham,	35.0	32.0	4.4	28.6	. . .

THE RELATIVE VALUE OF FOODS (*Continued*).

Articles.	As Material for the Muscles.	As Heat Givers.	As Food for the Brain and Nervous System.	Water.	Waste.
Horse-radish,	0.1	4.8	1.0	78.2	16.0
Kidney,	21.2	0.9	1.4	76.5	. . .
Lard,	. . .	100.0	. . .	. . .	. . .
Liver,	26.3	3.9	1.2	68.6	. . .
Onions,	0.5	5.2	0.5	93.8	. . .
Pearl barley,	4.7	78.0	0.2	9.5	7.6
Pears,	0.1	9.6	. . .	86.4	3.9
Pigeon,	23.0	1.9	2.7	72.4	. . .
Prunes,	3.9	78.6	4.5	13.0	. . .
Radishes,	1.2	7.4	1.0	89.1	1.3
Suet,	. . .	100.0	. . .	. . .	. .
Venison,	20.4	8.0	2.8	68.8	. . .
Vermicelli,	47.5	38.0	1.7	12.8	. . .
Whey,	. . .	4.6	0.7	94.7	. . .

ATKINSON'S TABLE OF DIGESTIBILITY OF NUTRIENTS OF FOOD MATERIALS.

In the Food Materials Below.	Of the Total Amounts of Protein, Fats, and Carbohydrates, the Following Percentages were Digested:		
	Protein.	Fats.	Carbohydrates.
Meat and fish,	Practically all	79 to 92	. . .
Eggs,	"	96	. . .
Milk,	88 to 100	93 to 98	?
Butter,	. . . . .	98	. . .
Oleomargarine,	. . . . .	96	. . .
Wheat bread,	81 to 100	?	99
Corn (maize) meal,	89	?	97
Rice,	84	?	99
Peas,	86	?	96
Potatoes,	74	?	92
Beets,	72	?	82

PERCENTAGES OF NUTRITION IN VARIOUS ARTICLES OF FOOD.—(*Moss.*)

Raw cucumbers,	2	Broiled venison,	22
Raw melons,	3	Potatoes,	22½
Boiled turnips,	4½	Fried veal,	24
Milk,	7	Roast poultry,	26
Cabbage,	7½	Raw beef,	26
Currants,	10	Raw grapes,	27
Whipped eggs,	13	Raw prunes,	29
Beets,	14	Boiled mutton,	30
Apples,	16	Oatmeal porridge,	75
Peaches,	20	Rye bread,	79
Boiled codfish,	21	Boiled beans,	87

PERCENTAGES OF NUTRITION (*Continued*).

Boiled rice,	88	Boiled barley,	92
Barley bread,	88	Butter,	93
Wheat bread,	90	Boiled peas,	93
Baked corn bread,	91	Raw oil,	96

The average percentage of the different food classes needed to sustain a man in perfect health is given in Kensington Museum "Handbook on Food":

	Per cent.
Water,	81.5
Albuminoids or flesh-formers,	3.9
Starches and sugars,	10.6
Fat,	3.0
Salt (NaCl),	0.7
Phosphates, potash salts, etc.,	0.3

AN IDEAL RATION WITH SOLID FOOD.—(*Mrs. E. H. Richards.*)

Material.	Amount.		Proteid.		Fat.		Carbohydrates		Calories.
	Gm.	Ozs.	Gm.	Ozs.	Gm.	Ozs.	Gm.	Ozs.	
Bread,	453.6	16	31.75	1.12	2.26	0.08	257.28	9.04	1,206.82
Meat,	226.8	8	34.02	1.20	11.34	0.04	. . .	. . .	243.72
Oysters,	226.8	8	12.52	0.44	2.04	0.07	. . .	. .	70.01
Breakfast cocoa,	28.3	1	6.60	0.23	7.50	0.26	9.60	0.34	135.42
Milk,	113.4	4	3.63	0.13	4.42	0.16	4.88	0.17	75.55
Broth,	453.6	16	18.14	0.64	18.14	0.64	90 72	3.20	613.21
Sugar,	28.3	1	. . .	. . .	. .	. .	27.36	0.96	112.17
Butter,	14.17	½	0.14	. . .	12.27	. . .	. . .	. . .	118.62
Total,	. .	. . .	106.80	. . .	57.97	. . .	389.84	. . .	2,575.52

The following table is a fair average work ration in round numbers, based on such data as those in the other tables:

ESTIMATED WORK RATION, MAXIMUM AND MINIMUM.—(*Mrs. E. H. Richards*).

	For One Day.
Proteid, gm.,	125 110
Fat, gm.,	125 90
Carbohydrates, gm.,	450 450
Calories,	3,500 3,000

About 30 gm. of salts should be added to this (Landois). The bare subsistence ration is much less, as follows:

ESTIMATED LIFE RATION.—(*Mrs. E. H. Richards.*)

	For One Day.		For One Day.
Proteid, gm.,	75	Carbohydrates, gm.,	325
Fat, gm.,	40	Calories,	2,000

It will be observed that the totals are somewhat less in this diet than those of the preceding table, which is designed for a working man who is developing more calories.

TABLE OF ENERGY.

Estimated in Foot Tons instead of Calories.—(*Yeo.*)

Energy developed by one ounce of the following foods when oxidized in the body:

Food Stuff.	With Usual Percentages of Water.	One Ounce Water-free.
	Foot Tons.	*Foot Tons.*
Beef (best quality), uncooked,	48.5	199
Meat (served to soldiers), uncooked,	57.8	243
Beef (fattened), uncooked,	96.0	280
Meat, cooked,	102.6	240
Corned beef (Chicago),	124.0	217
Salt beef,	52.0	138
Salt pork,	71.6	166
Fat pork,	202.0	336
Dried bacon,	292.3	346
Smoked ham,	179.6	267
Whitefish,	44.3	209
Poultry,	50.7	204
Bread,	87.5	147
Wheat flour,	123.6	146
Biscuit,	173.3	189
Rice,	126.5	141
Oatmeal,	130.0	154
Maize,	132.0	160
Macaroni,	122.7	146
Millet,	125.9	149
Arrowroot,	116.4	138
Peas (dried),	118.9	151
Potatoes,	33.0	141
Carrots,	14.3	137
Cabbage,	13.0	158
Butter,	344.5	367
Eggs,	67.3	265
Cheese,	149.9	245
Milk (cow's), new,	26.9	225
Cream,	109.2	365
Skimmed milk,	20.4	181
Sugar,	126.4	128
Pemmican,	270.1	293
Ale (Bass's bottled),	30.0	260
Stout (Guinness),	41.5	360

Prof. Egleston's standard of nutrition is high. He places the daily allowance of nutritive material at 700 gm., divided as follows: Carbohydrates, 400 gm.; fats, 150 gm.; proteid, 150 gm.,—yielding in all 3650 calories.

PERCENTAGE COMPOSITION OF EDIBLE PORTIONS OF GARRISON RATION.—(*Captain C. E. Woodruff, M.D., Asst. Surgeon, U. S. A.*)

	Water.	Protein.	Fats.	Carbo-hydrates.	Salts.	Energy Calories, per lb.
Bacon, fat,	20.0	8.00	69.5	. . .	2.5	3,080
Beans,	12.6	23.10	2.0	59.2	3.1	1,615
Pork, salt and fat,	12.1	0.90	82.8	. . .	4.2	3,510
Sugar, ground,	2.0	. . .	. . .	97.8	0.2	1,820
Sugar, brown issue,	3.0	. . .	. . .	96.5	0.5	1,795
Flour,	12.5	11.00	1.0	74.9	0 5	1,644
Beef,	55.0	17.10	27.0	. . .	0.9	1,460
Potatoes,	78.9	2.10	0.1	17.9	1.0	375
Onions,	87.9	1.4	0.3	10.1	0.6	225
Oatmeal,	7.6	15.10	7.1	68.2	2.0	1,850
Cornmeal,	15.0	9.20	3.8	70.6	1.4	1,645
Canned apples,	83.2	0.20	0.4	15.9	0.3	315
Dried apples,	25.0	0.90	1.8	71.5	1.4	1,418
Tapioca or corn-starch,	2.0	. . .	. . .	97.8	0.2	1,820
Butter,	10.5	1.00	85.0	0.5	3.0	3,615
Syrup,	43.7	. . .	. . .	55.0	2.3	1,023
Lard,	12.0	0.60	83.4	. . .	4.0	3,570
Rice,	12.4	7.4	0 4	79.4	0.4	1,630
Canned corn,	81.3	2.80	1.1	13.2	0.6	345
Canned tomatoes,	96.0	0.80	0.4	2.5	0.3	80
Macaroni and vermicelli,	13.1	9.00	0 3	76.8	0.8	1,406
Milk, fresh,	14.1	0.843	0.802	1.069	0.164	418
Milk, condensed,	25.0	17.00	11.0	44.00	3.0	1,595
Peas,	12.3	26.70	1.7	56.40	2 9	1,565
Raisins,	40.0	0.40	. . .	24.00	0.6	440
Cheese,	35.0	33.00	22 0	5.00	5.0	1,600
Prunes,	30.0	2 50	. . .	12.0	0.6	140
Cabbage,	92.0	2.10	0.6	5.5	1.1	155
Ham,	41.5	16.7	39.1	. . .	2.7	1,960
Apricots, canned,	50.0	2.00	. . .	30.0	0.6	460
Barley,	. . .	13.00	2.7	76.0	3 0	1,800
Chocolate,	12.0	20.00	50 0	10.0	4.0	2,650
Sausage,	41.2	13.80	42.8	. . .	2.2	2,065
Oysters,	87.1	6.00	1.2	3.7	2.0	230
Salmon, canned,	63.6	21.60	13.4	. . .	1.4	965
Crabs,	. . .	15.0	1.0	. . .	. . .	526
Crackers,	. . .	10.3	9.4	70.5	. . .	1,900

Church furnishes the following table showing the number of tons which it is calculated could be raised through the height of one foot by the complete combustion of a single pound of each kind of food. In the body only about a fifth of this energy would develop work, the rest going into heat production.

1 pound beef fat	raises	5,649	tons	1 foot	high.
1 pound oatmeal	"	2,439	"	"	"
1 pound gelatin	"	2,270	"	"	"
1 pound lean beef	"	885	"	"	"
1 pound potatoes	"	618	"	"	"
1 pound milk	"	390	"	"	"
1 pound ground rice,	"	2,330	"	"	"

CHAPTER II.

DIETETIC KITCHEN. DIET LISTS.

In the following we give the diet orders of Penzoldt, which agree essentially with those mentioned by Leube, but have this advantage over the latter, that they contain at the same time the permissible quantities of each article of food, and are also expanded in other directions.

The following diet list, consisting of four different kinds of diet, is, like that of Leube, especially intended to be a basis for a mild dietetic treatment in cases of diseases of the stomach in general (the so-called ulcer cure of Leube). By means of a gradual transition from a very light to a stronger and richer diet, it endeavors not to tax the diseased organ in the beginning, and gradually to accustom it to increased service.

It is self-evident that this diet list may not with impunity be extended in the same manner to all diseases of the stomach. According to the state of the secretion, the peristalsis, and of sensation, other problems concerning the diet may arise. We will revert to the special details of the several forms of disease when we come to them. It is necessary only to give the principal rules for the chief types of diseases of the stomach in this chapter.

PENZOLDT'S DIET ORDERS FOR GRADUAL TRAINING OF THE DIGESTIVE CAPACITY.

FIRST DIET (ABOUT TEN DAYS).

Foods or Drinks.	Largest Quantity at One Time.	Preparation.	Character.	How to be Taken.
Bouillon.	250 gm., ¼ liter.	To be made from beef.	Lean, very little salt, or none at all.	Slowly.
Cow's milk.	250 gm., ¼ liter.	Well boiled, or sterilized (Soxhlet's apparatus).	Pure milk, or eventually ⅓ lime-water and ⅔ milk.	Eventually with a little tea.

PENZOLDT'S DIET ORDERS FOR GRADUAL TRAINING OF THE DIGESTIVE CAPACITY.—FIRST DIET (ABOUT TEN DAYS) (*Continued*).

FOODS OR DRINKS.	LARGEST QUANTITY AT ONE TIME.	PREPARATION.	CHARACTER.	HOW TO BE TAKEN.
Eggs.	One or two.	Very soft, merely warmed or raw.	Fresh.	If raw, stir into the warm, not boiling, bouillon.
Meat solution (Leube-Rosenthal's).	30–40 gm.	See Dietetic Kitchen.	It may have only a faint odor of bouillon.	By teaspoonfuls or stirred up into bouillon.
Cakes (Albert biscuits).	Six.		Without sugar.	Not soaked or softened, but to be well masticated and insalivated.
Water.	⅛ liter.		Ordinary or natural carbonated, containing a little carbonic acid (Selters), Saratoga Vichy, Londonderry Lithia, Poland.	Not too cold.

SECOND DIET (ABOUT TEN DAYS).

FOODS OR DRINKS.	LARGEST QUANTITY AT ONE TIME.	PREPARATION.	CHARACTER.	HOW TO BE TAKEN.
Calf's brain.	100 gm.	Boiled.	To be freed from all membranes and fiber.	Preferably in the bouillon.
Sweetbread (thymus gland).	100 gm.	Boiled.	Similar to above, especially to be peeled carefully.	Similarly to above.
Pigeons.	One.	Boiled.	Only young ones, without skin, tendons and the like.	Similarly to above.
Chickens.	One, the size of a pigeon.	Boiled.	Like above (no fattened chickens).	Similarly to above.
Raw beef.	100 gm.	Finely chopped or scraped, with a little salt.	To be taken from the fillet (tenderloin).	To be eaten with crackers.

PENZOLDT'S DIET ORDERS FOR GRADUAL TRAINING OF THE DIGESTIVE CAPACITY.—SECOND DIET (ABOUT TEN DAYS) (*Continued*).

FOODS OR DRINKS.	LARGEST QUANTITY AT ONE TIME.	PREPARATION.	CHARACTER.	HOW TO BE TAKEN.
Raw beef sausage.	100 gm.	Without additions.	A little smoked.	Similarly to preceding.
Tapioca.	30 gm.	Cooked to a homogeneous gruel with milk.		

THIRD DIET (ABOUT EIGHT DAYS).

FOODS OR DRINKS.	LARGEST QUANTITY AT ONE TIME.	PREPARATION.	CHARACTER.	HOW TO BE TAKEN.
Pigeon.	One.	To be fried with fresh butter, not too much.	Only young ones, without skin, etc.	Without sauce.
Chicken.	One.	Like above.	Like above.	Like above.
Beefsteak.	100 gm.	With fresh butter, half raw (English).	The meat from the fillet, or tenderloin, well pounded.	Like above.
Ham.	100 gm.	Raw, scraped fine.	Smoked, not strong, without bones, the so-called "Lachsschinken."	With wheat bread.
French roll, toast, or Freiberg pretzel.	50 gm.	Baked crisp.	Stale (rolls and the like.)	To be chewed very carefully and to be well salivated.
Potatoes.	50 gm.	*a*. As purée, being forced through a strainer. *b*. As salt potatoes, mashed.	The potatoes must be mealy, crumbling when mashed.	
Cauliflower.	50 gm.	To be cooked in salt water as vegetables.	Only the "flowers" to be used.	

PENZOLDT'S DIET ORDERS FOR GRADUAL TRAINING OF THE DIGESTIVE CAPACITY (*Continued*).

FOURTH DIET (ABOUT EIGHT TO FOURTEEN DAYS).

FOODS OR DRINKS.	LARGEST QUANTITY AT ONE TIME.	PREPARATION.	CHARACTER.	HOW TO BE TAKEN.
Venison.	100 gm.	Roast.	Saddle, hung, not gamy, without high flavor.	
Partridge.	One.	Roast, without lard.	Young birds, with skin, tendons, feet, etc., removed, after having hung in pure cold air for twenty-four hours.	
Roast beef.	100 gm.	Fried until red.	From well-fed cattle, pounded.	Warm or cold.
Fillet.	100 gm.	In same manner as the above.	In same manner as the above.	In same manner as the above.
Veal.	100 gm.	Roast.	Saddle or leg.	Finely cut.
Pike. Perch-pike. Carp. Trout.	100 gm.	Boiled in salt water without any additions.	Carefully remove the bones.	In fish sauce.
Caviar.	50 gm.	Raw.	Russian Caviar with but a little salt in it.	
Asparagus.	50 gm.	Boiled.	Soft, without the hard portions.	With a little melted butter.
Rice.	50 gm.	As gruel, forced through a strainer.	Soft, boiling rice.	Likewise.
Poached eggs.	Two eggs.	With a little fresh butter.		With salt.
Egg soufflé (Auflauf).	Two eggs.	With about 20 gm. sugar.	Must rise well.	To be eaten at once.
Stewed fruits.	50 gm.	Fresh boiled, forced through a strainer.	Freed of all skins and seeds.	
Red wine.	100 gm.	Light, pure Bordeaux, or reliable California.	Or any similar kind of pure red wine.	Slightly warmed.

All of these foods should be prepared according to directions given in the "Dietetic Cooking."

DIET LIST OF EWALD FOR CHRONIC GASTRITIS.

8 A. M.—150 to 200 gm. of tea, with 100 gm. of stale wheat bread, toast, or Zwieback.

10 A. M.—50 gm. of wheat bread, 10 gm. of butter, 50 gm. of cold meat or ham, and either one glass of light wine or ⅓ of a liter of milk.

2 P. M.—150 to 200 gm. of water, milk, or bouillon of white meats; 100 to 125 gm. of meat or fish, 30 to 100 gm. of vegetables, 80 gm. *compote*.

4.30 P. M.—½ of a liter of warm milk, chocolate, or one-half milk and one-half coffee.

7 to 8 P. M.—300 gm. of soup, 50 gm. of wheat bread, 10 gm. of butter.

10 P. M.—Occasionally 50 gm. of wheat bread, biscuit, or Zwieback; one cup of coffee.

Boas gives two lists; the following contains the better and richer diet:

	Calories.
8 A. M.—200 gm. of milk, with 40 gm. of cocoa and 30 gm. of sugar,	462
50 gm. of cakes, or 50 gm. of Zwieback, either one,	187
10 A. M.—50 gm. of wheat bread with 30 gm. of butter,	343
100 gm. of calf's brain (or 100 gm. of sweetbread, 90 calories),	140
Or 100 gm. of broiled pike, 71.75 calories.	
12 M.—Soup of 30 gm. of tapioca, 10 gm. of butter, 1 egg,	282
100 gm. of noodles,	352
Or 100 gm. of spinach, 165.0 calories; 100 gm. of bean purée, 193 calories; 100 gm. of carrots, 40.0 calories; 50 gm. of potato purée, 63.7 calories.	
100 gm. of breast meat of young chicken,	106.4
100 gm. of veal cutlets (250 calories), or in its place 100 gm. of broiled veal, pigeon, venison, or fish.	
100 gm. of farina or omelette, or egg-pancake,	288
3 P. M.—100 gm. of milk, with 20 gm. of sugar, flavored with tea,	147
25 gm. of cakes,	93.5
7 P. M.—50 gm. of wheat bread, 130 gm. of butter,	343
50 gm. of scraped raw beef,	459.5
	3203.4

HEMMETER'S DIET LIST FOR CHRONIC GASTRITIS WITH UNIMPAIRED MOTILITY AND INTESTINAL DIGESTION.

ALSO AVAILABLE FOR LOWERED NUTRITION WHERE DIGESTIVE FUNCTIONS ARE NORMAL.

7.30 A. M.—If the bowels are regular, ½ of a pint of hot normal saline solution. If the bowels are constipated, a pint of cold Saratoga Vichy, Bedford Magnesia Spring, or plain cold water.

	Calories.
BREAKFAST, 8 A. M.—3½ ounces or 100 gm. of farina, boiled with milk,	127
Or 100 gm. of cerealin, boiled with milk.	
Or 100 gm. of breakfast wheat (strained), boiled with milk.	

	Calories.
One soft boiled egg,	80
Two ounces of wheat bread, toasted,	156
One ounce of best fresh butter,	212
One cup of wheat coffee (made of 100 gm. of roasted choice wheat, 250 c.c. of boiling water, and 150 gm. of milk). Instead of this the same portions of tea and milk or cocoa can be used,	100
Sugar, 10 gm. (2½ drams),	40

The farina or cerealin will taste better if eaten with a roasted apple.

As the digestive power improves, the egg is presented in form of omelette, or poached, on toast.

	Calories.
10.30 A. M.—100 gm. of scraped ham (3⅓ ounces),	120
30 gm. of crackers or toast (one ounce),	107
226 gm. or eight ounces of broth. Instead of broth, milk, kefir, and matzoon may be permitted in the same quantity,	306
DINNER, 1 P. M.—Soup made of 250 gm. or eight ounces of bouillon, 30 gm. or one ounce of rice or tapioca, ten gm. or 2½ ounces of butter, and one egg,	282

In case of much weakness and emaciation, ½ of a tablespoonful of *somatose* should be added.

The patient must not be aware of the addition of artificial foods.

	Calories.
120 gm. of breast meat of broiled fowl,	228
Or scraped tenderloin formed into patties and broiled;	
Or steamed or broiled bluefish, trout, white or yellow perch;	
Or broiled rockfish, or sweetbreads.	
50 gm. or two ounces of potato purée,	637
100 gm. or 3⅓ ounces of carrots, steamed,	40
Or 100 gm. of purée of beans or peas;	
Or 100 gm. of strained tomato purée.	
100 gm. of finely divided spinach.	
One cup custard made of two eggs,	160
Or instead of this, 100 grs. of sherry gelatin, or stewed apples, or plums, or rice in form of very light pudding made with slices of apple, no raisins.	
One glass (100 gm. or 3⅓ ounces) of Hungarian Tokay (J. Palugyay & Sons, Pressburg),	50

Instead of the meats given, the patient may, for a change, be allowed broiled pigeon or venison, which must not be gamy; also meat dumplings of scraped beef, scraped pork made into balls with bread crumbs, Zwieback crumbs, egg, and butter, cooked in bouillon, and a separate sauce is made and flavored with scraped sardelles.

Calories.

3 P. M.—One cup of chocolate made with 30 gm. or 1 ounce of breakfast cocoa, or v. Mering's Kraft-chocolate, and ½ of a pint of milk, 135.5

30 gm. of crackers, coffee-cake without grated nuts, cinnamon shortcake with but the faintest trace of cinnamon, ... 107

If the sweet chocolate is not agreeable, plain milk, or a glass of light Rhine wine with crackers, is allowable. Coffee in small quantities may be added to the milk at this hour.

Calories.

SUPPER, 6.30 P. M.—Broiled, panned, or raw oysters, 240 gm. or eight ounces, ... 70

If there is sub- or anacidity, the addition of a little grated horse-radish, lemon juice, or catsup to the raw oysters should not be forbidden.

Crackers, two ounces or 60 gm., ... 107

Butter, one ounce or 30 gm., ... 212

½ of a pint of reliable Rhine wine, ... 50

Or ½ of a pint of imported beer, ½ of a pint of tea and milk.

Instead of the oysters, little-neck clams, fresh scraped beef, finely cut roast lamb or beef, cold, smoked chipped beef, or smoked tongue will answer.

NOTE.—If the gastritis is evidently due to abuse of alcohol, the wines must be excluded.

BILL OF FARE FOR CHRONIC CATARRH OF THE STOMACH, WITH THE DIGESTION OF THE STOMACH ONLY REDUCED.—(*Wegele.*)

	ALBUMIN.	FAT.	CARBO-HYDRATE.	ALCOHOL.
MORNING:				
150 gm. of pepton cocoa, ...	8.00	6.0	7.50	. . .
25 gm. of butter (on toasted roll), .	0.18	20.8	0.15	. . .
FORENOON:				
1 soft egg, ...	6.00	5.0	. . .	. . .
NOON:				
200 gm. of oatmeal soup, ...	12.50	0.3	18.00	. . .
150 gm. of fowl, ...	28.00	13.5	1.80	. . .
200 gm. of carrot, ...	2.14	0.4	16.30	. . .
AFTERNOON:				
150 gm. of pepton cocoa, ...	8.00	6.0	7.50	. . .
25 gm. of butter and Albert or banquet crackers, ...	0.18	20.8	0.15	. . .
EVENING:				
1 egg, ...	6.00	5.0	. . .	. . .
100 gm. of scraped ham, ...	25.00	8.0	. . .	. . .
100 gm. of macaroni, with toasted bread crumbs, ...	9.00	0.3	76.70	. . .
DURING THE DAY:				
200 gm. of wine, ...	. . .	. . .	6.00	16.0
75 gm. of toast, ...	9.00	1.5	63.90	. . .
Total, ...	117.20	94.6	236.01	16.0
Calories about, ...	480	890	970	100

Entire combustion value about 2440 calories.

BILL OF FARE FOR ATROPHIC CATARRH.—(*Wegele-Penzoldt.*)

	Albumin.	Fat.	Carbo-Hydrate.	Alcohol.
Morning:				
150 gm. of maltoleguminose cocoa,	6.00	4.00	13.50	. . .
Forenoon:				
150 gm. of wine, ...	. .	. . .	4.00	12.0
20 gm. of butter (on toasted bread),	0.15	16.60	0.12	. . .
Noon:				
100 gm. of maltoleguminose soup, .	2.60	0.10	6.20	. . .
100 gm. of scraped beefsteak, . . .	20.00	6.00	. . .	. . .
100 gm. of mashed potatoes, . .	3.10	0.50	21.30	. . .
10 gm. of malt extract, ...	0.50	. . .	5.50	. . .
Afternoon:				
1 cup of tea (with toast), ...	. . .	. . .	. . .	. . .
20 gm. of butter, ...	0.15	16.60	0.12	. . .
30 gm. of honey, ...	0.40	. . .	22.00	. . .
Evening:				
250 gm. of rice mush, ...	22.00	8.25	71.00	. . .
During the Day:				
75 gm. of toast (or toasted bread), .	9.00	1.50	63.90	. . .
10 o'clock at Night:				
250 gm. of milk, ...	8.70	9.30	12.00	. . .
10 gm. of cognac brandy, ...	. . .	. . .	. . .	7.0
Total, ...	72.70	62.85	219.64	19.0
Calories about, ...	300	580	920	130

Entire combustion value about 1930 calories.

BILL OF FARE FOR ATONY OF THE STOMACH, WITH GASTRIC DIGESTION REDUCED.—(*Wegele.*)

	Albumin.	Fat.	Carbo-Hydrate.	Alcohol.
Morning:				
150 gm. of leguminose cocoa, . .	6.0	4.0	13.5	. . .
50 gm. of cream, ...	1.8	13.3	1.8	. . .
Forenoon:				
1 soft egg, ...	6.0	5.0	. . .	. . .
20 gm. of toast, ...	2.5	0.4	15.0	. . .
Noon:				
100 gm. of scraped beefsteak, . . .	17.1	6.0	. . .	. . .
200 gm. of mashed potatoes, . . .	4.2	2.7	42.6	. . .
20 gm. of malt extract, ...	1.0	. . .	11.0	. . .
Afternoon:				
150 gm. of leguminose cocoa, . . .	6.0	4.0	13.5	. . .
50 gm. of cream, ...	1.8	13.3	1.8	. . .
Evening:				
250 gm. of tapioca pulp, ...	12.0	8.0	11.0	. . .
15 gm. of diastase malt extract, . .	0.8	. . .	9.0	. . .

BILL OF FARE FOR ATONY OF THE STOMACH, WITH GASTRIC DIGESTION REDUCED.—(*Wegele*) (*Continued*).

	ALBUMIN.	FAT.	CARBO-HYDRATE.	ALCOHOL.
DURING THE DAY:				
50 gm. of toast, ...	6.0	1.0	35.0	. . .
10 O'CLOCK AT NIGHT:				
200 gm. of milk, ...	6.4	7.2	9.6	. . .
10 gm. of cognac, ...	. . .	. . .	. . .	6.9
Total, ...	71.6	64.9	163.8	6.9
Calories about, ...	290	600	670	50

Total combustion value about 1610 calories.

At noon, of course, other kinds of meat could be chosen, such as fowl or game; likewise at night rice or thick gruel.

With fermentation of the stomach, however, the following bill of fare had best be used after a few days:

MORNING:

100 gm. of scraped ham (can) or smoked meat, and 20 gm. of bread crust.

FORENOON:

One soft egg and 20 gm. of bread crust or toast.

NOON:

100 gm. of scraped beefsteak and scrambled eggs (two).

AFTERNOON:

Same as forenoon.

EVENING:

Same as noon.

Two clysters of ½ to 1½ per cent. common salt solution.

HEMMETER'S DIETARY FOR ANACID DILATATION.

Calories.

7.30 A. M.—Lavage with NaCl solution or a decinormal solution of HCl.

8 A. M.—Cerealin with cream, 150 gm., ... 395

Mosquera beef chocolate, 200 gm., ... 140

Malt extract, 10 gm., ... 24.5

10 A. M.—Toast or aleuronat bread (see dietetic directions), 60 gm., . . . 135

Butter, 20 gm., ... 163

12 M.—Boiled round of beef, 150 gm., ... 440

Mashed potatoes, 50 gm., ... 63

Spinach or carrots, 100 gm., ... 165.5

In place of these, purées of peas, beans, lentils, or turnips are allowed.

Omelette soufflé, 100 gm., ... 244

3 P. M.—100 gm. of tea, 50 gm. of Albert biscuits, 10 gm. of milk, . . . 254

Calories.

7 P. M.—100 gm. of scraped ham in omelette, ... 244
Or 60 gm. of scraped ham (262 calories).
200 gm. of farina with milk, ... 432
60. gm. of toast, 20 gm. of butter, ... 298
9.30 P. M.—Milk, 300 c.c., ... 202
Two ounces of banquet crackers or Albert biscuits, ... 200
Or in place of the milk a glass (two ounces) of approved Tokay or Malaga.

BILL OF FARE FOR ATONY OF THE STOMACH, WITH THE PRODUCTION OF HYDROCHLORIC ACID SUSTAINED OR INCREASED.—(*Wegele.*)

	Albumin.	Fat.	Carbo-Hydrate.	Alcohol.
Morning:				
150 gm. of pepton cocoa, ...	8.0	6.0	7.5	. . .
50 gm. of cream, ...	1.8	13.3	1.8	. . .
Forenoon:				
30 gm. of French roll, ...	3.0	0.2	20.0	. . .
50 gm. of ham, ...	12.5	4.0	. . .	. . .
1 egg, ...	6.0	5.0	. . .	. . .
Noon:				
120 gm. of roast meat, ...	21.0	8.0	. . .	. . .
200 gm. of mashed potatoes, . . .	4.2	2.7	42.6	. . .
Afternoon:				
150 gm. of pepton cocoa, ...	8.0	6.0	7.5	. .
50 gm. of cream, ...	1.8	13.3	1.8	. . .
Evening:				
120 gm. of cold roast meat, ...	21.0	8.0	. . .	. . .
200 gm. of rice, ...	9.0	6.6	28.6	. . .
10 o'clock:				
100 gm. of wine, ...	. . .	. . .	3.3	7.8
During the Day:				
50 gm. of toast, ...	6.5	1.6	41.0	. . .
Total, ...	102.8	74.7	159.1	7.8
Calories about, ...	420	700	640	55

Total combustion value about 1815 calories.

Instead of ham, caviar and butter with slices of toasted roll, or scrambled eggs with smoked meat, may be given in the forenoon. At noon, beefsteak, fillet, game, or fowl are allowed, and for side dishes some mashed carrots or spinach. At night, calf's-foot jelly and omelette soufflé.

In convalescence, 10 to 15 gm. of condensed milk or malt extract three times daily after meals can be prescribed, through which the nutritive value of this diet is considerably increased.

BILL OF FARE FOR ENLARGEMENT OF THE STOMACH WITH STENOTIC APPEARANCES.—(*Wegele.*)

	Albumin.	Fat.	Carbo-Hydrate.	Alcohol.
Morning:				
100 gm. of scraped ham, ...	25.0	8.0	. . .	. . .
Tea with 50 gm. of cream, ...	1.8	13.3	1 8	. . .
Forenoon:				
2 eggs, ...	12.0	10.0	. . .	. . .
20 gm. of sugar, ...	. . .	. . .	16.0	. . .
10 gm. of cognac, ...	. . .	. . .	. . .	13.8
Noon:				
100 gm. of scraped beefsteak, . . .	20.7	1.5	. . .	. . .
100 gm. of mashed potatoes, . . .	3.1	0.5	21.3	. . .
Afternoon:				
Tea with 50 gm. of cream, ...	1.8	13.3	1.8	. . .
Evening:				
100 gm. of roast chicken (hashed), .	20.7	1.5	. . .	. . .
100 gm. of flour puff paste, ...	4.2	4.3	22.0	. . .
During the Day:				
80 gm. of toast, ...	8.5	1.2	55 0	. . .
Night:				
200 gm. of milk, ...	6.4	7.2	9.6	. . .
Total, ...	104.2	60.8	127.5	13.8
Calories about, ...	427	565	722	100

Total combustion value about 1814 calories.

With this bill of fare it is most difficult to have a variety. Beefsteak, scraped fine from lean meat, chicken, pigeon, lean ham, smoked meat, cold roast beef, and fillet are recommended.

In the evening one may often serve also calf's-foot jelly, tapioca, or milk jelly. With occasional improvement condensed milk, cream, malt extract, and milk jellies may be tried by spoonfuls between meals. Besides these a nutritive clyster (following a cleansing enema) is to be given twice a day in these severe cases.

One can waive the somewhat tedious meat-pancreas clysters, since a considerable quantity of meat is taken in *per os*, and one can adopt either Ewald's or Boas' method, since according to the investigations of Eichhorst (*Pflüger's Archiv*, Bd. IV, 1871), Ewald (*Zeitschrift f. klin. Med.*, Bd. XII, 1887), and Huber (*Deutsch. Archiv f. klin. Med.*, Bd. XLVII), the digestion of the albumen of eggs and milk proceeds very well without previous peptonization in the rectum, while it is considerably increased by the addition of com-

mon salt (one gm. to one egg) (see chapter on rectal alimentation). Boas ("Diagnostik und Therapie der Magenkrankheiten," zweite Aufl., 1891, S. 244), has followed out rectal nutrition for ten to fourteen days, in cases of severe gastrectasia with symptoms of fermentation, and attained not only the disappearance of the symptoms of fermentation, and a considerably better general state of health, but also temporary increase in weight,—a success which lasted from three to four months.

If to the preceding bill of fare two more nutritive clysters are added, the patient receives:

	Albumin.	Fat.	Carbohydrate.	Alcohol.
4 eggs,	20	24		
100 gm. of red wine,			3.3	7.8
Total,	20.0	24.0	3.3	7.8
Calories,	82	224	31	54

Total combustion value about 391 calories.

If we assume that of this only ten gm. of albumen, two gm. of carbohydrate, ten gm. of fat, and four gm. of alcohol should attain resorption, we would obtain a total combustion value of about 1750 calories.

With two nutritive clysters, according to Boas, the following increase would be attained:

	Albumin.	Fat.	Carbohydrate.	Alcohol.
500 gm. of milk,	17.0	18.2	24.2	. . .
4 eggs,	24.0	20.0	. . .	. . .
30 gm. of red wine,	. . .	. .	. .	2.0
40 gm. of leguminose flour,	8.8	3.6	25.0	. . .
Total,	49.8	41.8	49.2	2.0

If half be assumed as resorbed, then there would be an addition of about 25 gm. of albumin, about 20 gm. of fat, about 25 gm. of carbohydrate, about one gm. of alcohol. This would give a total combustion value of about 1850 calories (Wegele).

BILL OF FARE FOR GASTRIC CARCINOMA WITHOUT PERCEPTIBLE STENOTIC APPEARANCES.—(*Wegele.*)

	Albumin.	Fat.	Carbo-hydrate.	Alcohol.
Morning:				
150 gm. of maltoleguminose cocoa, .	6.0	4.0	13.5	. . .
Forenoon:				
200 gm. of kefir, ...	6.6	4.5	3.8	. . .
Noon:				
150 gm. of maltoleguminose soup, .	4.0	0.15	9.3	. . .
100 gm. of scraped beefsteak, . . .	20.0	6.0	. .	. . .
Afternoon:				
150 gm. of maltoleguminose cocoa, .	6.0	4.0	13.5	. . .
Evening:				
100 gm. of scraped ham, ...	25.0	8.0	. . .	. . .
150 gm. of tapioca, ...	7.0	5.0	8.0	. . .
10 o'clock:				
200 gm. of kefir, ...	6.6	4.5	3.8	. . .
With the cocoa, 30 gm. of honey, .	0.4	. . .	22.0	. . .
With the kefir, 20 gm. of cognac, .	. . .	. . .	. . .	14.0
During the Day:				
50 gm. of toast, ...	6.6	1.0	35.0	. .
Total, ...	87.6	37.1	108.9	15.0
Calories about, ...	360	350	450	100

Total combustion value about 1260 calories.

For a change, tea can be often given instead of cocoa; where kefir does not agree with the patient, or is refused by him, one can try condensed milk with cognac instead; further, one can let him eat butter upon toast, or toasted bread with the tea, and also have variety in the meats, as long as the appetite for them remains.

Naturally, in the last stages a considerable narrowing of the list, both in quantity and quality, takes place, and one must make the greatest concessions to the individual tastes of the patient. In the morning one will give him either cocoa or tea, with slices of toasted roll spread with meat extract or caviar; then allow a little wine with one soft egg, or egg with cognac and sugar, or a glass of champagne; at noon sweet-bread in soup, smoked ham, pickled meat, smoked meat (which foods are more difficult of decomposition), gruel, rice, mondamin cooked in milk, according to taste. In the afternoon, tea with cognac or cocoa, and in the evening calf's-foot jelly, or meat-extract jelly, or meal soup will be suitable. In addition, the nutritive clysters mentioned above. (A more detailed calculation of the diet at this stage has little value, and is therefore omitted.)

(1) BILL OF FARE FOR CURE OF ULCER (TO BE KEPT UP AT LEAST TEN DAYS).—(*Leube-Penzoldt-Wegele.*)

	Albumin.	Fat.	Carbo-hydrate.
Morning:			
250 gm. of milk,	8.50	9.00	12.0
Two cakes (5 gm. each),	1.10	0.50	7.3
10 o'clock:			
250 gm. of milk or bouillon,	8.50	9.00	12.0
One cake,	0.60	0.25	3.7
12 o'clock:			
150 gm. of bouillon,	0.75	0.45	0.9
50 gm. of meat solution (or one egg),	8.50	3.00	3.5
4 o'clock:			
250 gm. of milk,	8.50	9.00	12.0
Two cakes,	1.10	0.50	7.3
150 gm. of bouillon,	0.75	0.45	0.9
50 gm. of meat solution (or one egg),	8.50	3.00	3.5
Two cakes,	1.10	0.50	7.3
Total,	47.9	35.65	70.4
Calories about,	200	330	330

Total combustion value about 860 calories.

(2) BILL OF FARE FOR CURE OF ULCER (TO BE KEPT UP AT LEAST SEVEN DAYS).—(*Leube-Wegele.*)

	Albumin.	Fat.	Carbo-hydrate.
Morning:			
250 gm. of milk,	8.5	9.00	12.0
Three cakes,	1.8	0.75	11.1
10 o'clock:			
200 gm. of bouillon,	3.2	4.40	3.2
One egg,	6.0	5.00	. . .
Noon:			
One boiled pigeon,	22.0	1.00	0.7
About 200 gm. of rice in bouillon,	5.0	2.00	40.0
4 o'clock:			
250 gm. of milk,	8.5	9.00	12.0
Two cakes,	1.1	0.50	7.3
8 o'clock:			
150 gm. of bouillon,	6.4	6.70	9.0
100 gm. of sweet-bread,	28.0	0.40	. . .
Total,	90.5	38.75	95.3
Calories about,	370	350	390

Total combustion value about 1110 calories.

(3) BILL OF FARE FOR CURE OF ULCER (FOR AT LEAST FIVE DAYS).—(*Wegele.*)

	Albumin.	Fat.	Carbo-hydrate.
Morning:			
Two cups of tea or coffee, with 100 gm. of milk,	3.4	3.60	4.8
20 gm. of sugar,	0.5	. . .	18.2
Three cakes,	1.8	0.75	11.1
10 o'clock:			
200 gm. of bouillon,	3.2	4.40	3.2
One egg,	6.0	5.00	. . .
Noon:			
200 gm. of soup,	3.2	6.00	17.0
150 gm. of beefsteak,	31.0	2.20	. . .
100 gm. of mashed potatoes,	3.1	0.85	21.3
4 o'clock:			
Two cups of tea with 100 gm. of milk,	3.4	3.60	4.8
20 gm. of sugar,	0.5	. . .	18.2
Three cakes,	1.8	0.75	11.1
Evening:			
100 gm. of scraped ham,	25.0	8.10	. . .
200 gm. of soup,	3.2	6.00	17.0
Total,	86.1	41.25	126.7
Calories about,	350	380	520

Total combustion value about 1250 calories.

(4) BILL OF FARE FOR CURE OF ULCER (TO BE KEPT UP AT LEAST ONE WEEK).

	Albumin.	Fat.	Carbo-hydrate.
Morning:			
Two cups of tea or coffee, with 100 gm. of milk,	3.4	3.6	4.8
20 gm. of sugar,	0.5	. . .	18.2
One sweetbread (50 gm.),	4.5	0.5	29.0
10 o'clock:			
200 gm. of bouillon,	3.2	4.4	3.2
One egg,	6.0	5.0	. . .
Noon:			
200 gm. of soup,	3.2	6.0	17.0
150 gm. of roast fowl,	27.6	14.0	1.7
100 gm. of carrots or spinach,	1.0	0.2	8.1
200 gm. of light flour food,	9.0	8.4	45.0
4 o'clock:			
Two cups of tea or coffee, with 100 gm. of milk,	3.4	3.6	4.8
20 gm. of sugar,	0.5	. . .	18.2
One sweetbread,	4.5	0.5	29.0

(4) Bill of Fare for Cure of Ulcer (to be kept up at least one week) (*Continued*).

	Albumin.	Fat.	Carbo-hydrates.
Evening:			
100 gm. of cold roast meat,	38.2	2.8	. .
250 gm. of tapioca,	7.0	5.0	8.0
10 o'clock at night:			
250 gm. of milk,	8.5	9.0	12.0
Total,	120.5	63.0	199.0
Calories about,	495	585	815

Total combustion value about 1900 calories.

Instead of tea or coffee, milk may also be served, by which the nutritive value of this diet is not inconsiderably increased. Concerning the first list it is to be remarked that instead of meat solution eggs may also be given (stirred into the soup).

Further, in the second and third lists it is allowable to give two or three soft eggs instead of meat in the evening.

The fourth list may, after a time, be quantitatively and qualitatively expanded, since the following are allowed: Meats (fillet, roast beef, beefsteak, roast veal "from the leg," spring chicken, pigeons, partridges, venison).

Fish, pike, and perch (boiled) are allowable.

Vegetables,—mashed potatoes, spinach, and golden turnips.

Of the farinaceous foods, the light puff paste of rice, fine oat meal, tapioca, and omelette soufflé come under consideration.

At evening, mushes with whisked eggs; preserved or stewed fruit; stewed apples may be tried gradually.

Salads are entirely to be avoided. Wines can now be permitted in small quantities before meals. By gradual increase in quantity, one must attempt to give the body the necessary increase in material.

Bill of Fare for Chronic Diarrhea (Severe Cases).—(*Wegele.*)

	Albumin.	Fat.	Carbo-hydrate.	Alcohol.
Morning:				
200 gm. of acorn cocoa, boiled in water,	2.3	3.60	12.0	. . .
One soft egg,	6.0	5.00	. . .	. . .

BILL OF FARE FOR CHRONIC DIARRHEA (SEVERE CASES).—(*Wegele*) (*Continued*).

	ALBUMIN.	FAT.	CARBO-HYDRATE.	ALCOHOL.
FORENOON:				
250 gm. of decoction of whortleberries (from 80 gm. of dried berries), . .	0.6	1.30	4.7	. . .
250 gm. of slimy soup, ...	5.5	4.00	7.5	. . .
One egg in the soup, ...	6.0	5.00	. . .	. . .
100 gm. of scraped meat (lean), . .	20.7	1.50	. . .	. . .
50 gm. of rice in bouillon, ...	4.0	0.50	38.0	. . .
AFTERNOON:				
250 gm. of whortleberry decoction, .	0.6	1.30	4.7	. . .
EVENING:				
250 gm. of maltoleguminose soup, . .	6.5	0.25	15.5	. . .
With one egg, ...	6.0	5.00	. . .	. . .
150 gm. of minced chicken, ...	15.0	9.00	12.0	. . .
DURING THE DAY:				
75 gm. of toast, ...	9.0	1.50	42.5	. . .
200 gm. of whortleberry wine, . . .	. . .	. . .	7.0	17.0
10 O'CLOCK AT NIGHT:				
250 gm. of barley mush (20; 250), .	5.0	4.00	25.0	. . .
Total, ...	87.2	42.00	168.9	17.0
Calories about, ...	360	390	690	120

Total combustion value about 1440 calories.

At the beginning of convalescence light flour foods are allowed at noon; afternoon, instead of the whortleberry decoction, acorn cocoa may be substituted; at noon, roast fowl, beefsteak, fillet, roast beef, and gradually pass over to the following list:

DIET LIST FOR CHRONIC DIARRHEA (LESS SEVERE CASES) (*Wegele*).

	ALBUMIN.	FAT.	CARBO-HYDRATE.	ALCOHOL.
MORNING:				
200 gm. of acorn cocoa, ...	2.30	3.6	12.00	. . .
With one egg, ...	6.00	5.0	. . .	. . .
FORENOON:				
250 gm. of kefyr (four days old), . .	8.20	5.7	2.00	3.2
NOON:				
250 gm. of soup, ...	5.50	4.0	7.50	. . .
With one egg, ...	6.00	5.0	. . .	. . .
150 gm. of roast chicken, ...	28.00	10.0	1.80	. . .
200 gm. of mashed potatoes, . . .	6.00	1.7	42.70	. . .
4 O'CLOCK:				
250 gm. of acorn cocoa, ...	2.30	3.6	12.00	. . .

DIET LIST FOR CHRONIC DIARRHEA (LESS SEVERE CASES).—(*Wegele*) (*Continued*).

	ALBUMIN.	FAT.	CARBO-HYDRATE.	ALCOHOL.
6 O'CLOCK:				
250 gm. of kefyr, ...	8.20	5.7	2.00	3.2
8 O'CLOCK:				
200 gm. of soup, ...	3.30	6.0	17.00	. . .
With one egg, ...	6.00	5.0	. . .	. . .
100 gm. of sweetbread, ...	28.00	0.5	. .	. . .
10 O'CLOCK:				
250 gm. of kefyr, ...	8.20	5.7	2.00	3.2
DURING THE DAY:				
75 gm. of toast, or toasted bread, .	9.00	1.5	42.50	. . .
20 gm. of butter, ...	0.15	16.6	0.12	. .
250 gm. of whortleberry wine, . .	. . .	. .	8.75	21.5
Total, ...	127.00	79.6	150.25	31.3
Calories about, ...	520	740	615	210

Total combustion value about 2080 calories.

After convalescence has begun, have the acorn cocoa prepared with milk; add at noon light foods; at night give milk mush for a change; gradually increase the amount of kefyr given and thus gradually a diet of about 2500 calories combustion value is reached, which is to be considered sufficient.

DIET LIST FOR CHRONIC CONSTIPATION.—(*Wegele.*)

	ALBUMIN.	FAT.	CARBO-HYDRATE.	ALCOHOL.
MORNING:				
Before breakfast, Bedford Magnesia Spring Water, ½ liter.				
200 gm. of milk and coffee, . . .	3.20	4.40	3.20	. . .
30 gm. of butter, ...	0.21	24.50	0.15	. . .
30 gm. of honey, ...	0.35	0.03	17.00	. .
100 gm. of Graham bread, . . .	. . .	. . .	. .	. . .
300 gm. of buttermilk, ...	12.15	2.80	11 20	. . .
NOON:				
200 gm. of bouillon, ...	1.00	0.60	1.20	. . .
200 gm. of mutton, ...	23.20	50.50	0.70	. . .
300 gm. of crisped cabbage, ...	4.20	14.40	21.60	. . .
200 gm. of plums, ...	0.80	. . .	11.60	. . .
300 gm. of white wine or apple cider,	. . .	. . .	9.00	24.7
AFTERNOON:				
300 gm. of buttermilk, ...	12.15	2.80	11.20	. . .

DIET LIST FOR CHRONIC CONSTIPATION.—(*Wegele*) (*Continued*).

	ALBUMIN.	FAT.	CARBO-HYDRATES.	ALCOHOL.
EVENING:				
150 gm. of meat,	28.20	11.00	0.10	. . .
30 gm. of butter,	0.21	24.50	0.15	. . .
300 gm. of stewed apples,	1.00	. . .	39.00	. . .
For the several meals, 250 gm. of Graham bread,	22.50	2.50	125.00	. . .
After evening meal, 750 gm. of beer,	42.60	6.50	4.70	28.8
Total,	145.77	194.50	245.80	53.5
Calories about,	600	1800	1000	375

Total combustion value about 3800 calories.

This list is easily varied in accordance with above statements, and eventually it may be diminished along the entire scale, or it may be changed with regard to co-existent stomach troubles. For the rest it is to be noted that with the difficult solubility of many of the foods mentioned, and with an acceleration of digestion brought about by the diet prescribed, a considerable part of the nutriment introduced with the "ingesta" will be only partially turned to the best advantage. Naturally, if the chronic constipation is due to real catarrh, one must prescribe less irritating food and give the softer vegetables, such as cauliflower, spinach, asparagus, carrots; also the legumes and preserves more in form of purées.

DIET LIST FOR HYPERACIDITY.—(*Boas-Wegele-Fleischer.*)

	ALBUMIN.	FAT.	CARBO-HYDRATE.	ALCOHOL.
MORNING:				
100 gm. of tea with milk,	3.4	3.6	4.8	. . .
2 soft eggs,	12.0	10.0	. . .	. . .
100 gm. of raw ham,	25.0	8.0	. . .	. . .
50 gm. of cream,	2.0	13.5	1.7	. . .
NOON:				
200 gm. of aleuronat meal soup (10 al.; 20 oatmeal; 250 soup),	10.2	1.7	8.0	. . .
150 gm. of beefsteak,	58.0	3.0	. . .	. . .
200 gm. of mashed potatoes,	6.2	1.7	42.6	. . .
100 gm. of white wine, mixed with Saratoga Vichy or Biliner water,	. . .	. . .	3.5	8.0
AFTERNOON:				
100 gm. of tea,	3.4	3.6	4.8	. . .
150 gr. of cream,	2.0	13.5	1.7	. . .

DIET LIST FOR HYPERACIDITY.—(*Boas-Wegele-Fleischer*) (*Continued*).

	ALBUMIN.	FAT.	CARBO-HYDRATES	ALCOHOL.
EVENING:				
50 gm. of cold roast meat,	60.2	4.0	. . .	. . .
2 scrambled eggs,	12.0	12.0	. . .	. . .
100 gm. of wine,	. . .	. . .	3.5	8.0
For the several meals, 100 gm. of aleuronat toast,	28.3	1.5	66.7	. . .
10 O'CLOCK AT NIGHT:				
250 gm. of milk,	8.5	9.0	12.0	. . .
Total,	229.2	85.1	149.4	16.0
Calories about,	940	790	600	112

Total combustion value about 2500 calories.

With the tea a little sugar is to be allowed, and the white wine is usually to be mixed with an alkaline acidulous water not containing too much carbonic acid (such as Biliner water). When convalescence sets in, the daily amount of milk is to be increased.

DIET LIST FOR HYPERSECRETION.—(*Wegele.*)

	ALBUMIN.	FAT.	CARBO-HYDRATE.
MORNING:			
Tea with 100 gm. of milk,	3.4	3.0	4.8
2 soft eggs,	12.0	10.0	. . .
FORENOON:			
150 gm. of calf's-foot jelly,	35.0	17.0	1.0
NOON:			
150 gm. of sweetbread in bouillon,	32.0	. . .	. . .
250 gm. of tapioca mush,	12.0	8.0	11.0
50 gm. of cream,	2.0	13.5	1.7
AFTERNOON:			
200 gm. of milk,	6.8	6.0	9.6
EVENING:			
200 gm of ham,	48.0	70.0	. . .
2 scrambled eggs,	12.0	12.0	. . .
For the several meals, 100 gm. of aleuronat toast,	28.3	1.5	66.7
10 P. M.:			
100 gm. of milk,	6.5	6.0	10.0
DURING NIGHT:			
100 gm. of milk,	6.5	6.0	10.0
Total,	218.0	147.0	104.8
Calories about,	900	1360	430

Total combustion value about 2700 calories.

Of course, other meats than those mentioned above may be chosen, only the glutinous are particularly to be selected; eventually also scraped meat, ham, etc.

With convalescence go over to the preceding list. With nightly complaints in consequence of acid formation, there is to be recommended, besides milk or glair water, especially raw or hard grated eggs and drinking afterward of alkaline waters. Penzoldt recommends the addition of one-fourth to one-third lime-water to the milk.

As concerns the power of the various foods to combine with HCl, the following table is based upon results obtained experimentally by Fleischer ("Krankh. d. Speiseröhre, d. Mag. u. d. Darms," Wiesbaden, 1896, p. 932):

	Pure HCl.	25 Per Cent. HCl.	12½ Per Cent. HCl.
Meats, 100 gm., Combine With:			
Calf's brain, boiled,	0.65	2.6	55.2
Liver pudding,	0.80	3.2	6.4
Sweetbread, boiled,	0.90	3.6	7.2
Mettwurst,	1.00	4.0	8.0
Saveloy,	1 10	4.4	8.8
Black pudding,	1.30	5.2	10.4
Pork, boiled,	1.60	6.4	12.8
Ham, boiled,	1.80	7.2	14.4
Ham, raw,	1.90	7.6	15.2
Mutton, boiled,	1.90	7.6	15.2
Beef, boiled,	2.00	8.0	16.0
Veal, boiled,	2.20	8.8	17.6
Leube-Rosenthal's meat solution,	2.20	8.8	17.6
Other Foods.			
Beer,	0.10	.40	.80
Milk,	0.36	1.44	2.88
Wheatbread,	0.30	1.20	2.40
Graham bread,	0.30	1.20	2.40
Black (gray) bread,	0.50	2.00	4.00
Pumpernickel,	0.70	2.80	5.60
Hand cheese,	1.00	4.00	8.00
Fromage de Brie,	1.30	5.20	10.40
Edam cheese,	1.40	5.60	11.20
Brick cheese,	1.70	6.80	13.60
Pease sausage,	1.70	6.80	13.60
Roquefort cheese,	2.10	8.40	16.80
Swiss cheese,	2.60	10.40	20.80
Cocoa,	4.10	16.40	32.80

The author's personal views on the dietetic treatment of hyperacidity and hypersecretion have been clearly stated in pages 188 and

189, on the basis of a very large number of quantitative analyses of the gastric contents of 42 normal healthy adults, he has become convinced that proteids, such as beef, eggs, fish, etc., cause a stronger secretion of HCl than amylaceous foods, such as rice, sago, farina, cerealin. When the glandular layer is in a state of increased excitation it is logical to avoid proteid and albuminous food as much as possible. We have made numerous prolonged observations showing that amylaceous foods and fats can maintain the nitrogen equilibrium, and even add to body weight when proteids are excluded. We do not wish to defend the standpoint of the vegetarian, as we generally allow a small quantity of easily digestible meat for dinner, and advise about 1½ liters of milk if it agrees well. The views of v. Sohlern on this question merit careful investigation.

SCHEDULE FOR INTESTINAL ANTISEPSIS BY MILK DIET.

ALSO AVAILABLE IN NEURASTHENIA, SENSORY AND SECRETORY NEUROSES.—(*Burkart.*)

FIRST DAY:

7.30 A. M.—One-half of a liter of milk and two pieces of toast (the milk is to be taken a mouthful or a spoonful at a time, ½ of a liter in one-half hour).

10 A. M.—One-third of a liter of milk and one toast.

12.30 P. M.—One plate of soup with one egg, 50 gm. of roast meat. Potato purée.

3.30 P. M.—One-third of a liter of milk and one toast.

5.30 P. M.—One-half of a liter of milk and two toasts.

8 P. M.—One-half of a liter of milk, 50 gm. of roast meat, wheat bread and butter.

On the ninth day the following list is reached:

7.30 A. M.—One-half of a liter of milk and two toasts.

8.30 A. M.—Coffee and cream, wheat bread and butter.

10 A M.—One-third of a liter of milk and two toasts.

12 M.—One-half of a liter of milk.

1 P. M.—Soup with one egg, 100 gm. of meat, mashed potatoes, 75 gm. of stewed prunes.

3.30 P. M.—One-half of a liter of milk.

5.30 P. M.—One-third of a liter of milk and two toasts.

8 P. M.—One-half of a liter of milk, 60 gm. of meat, wheat bread and butter.

9.30 P. M.—One-third of a liter of milk and two toasts.

On the fifteenth day Burkart (in a severe case of dyspepsia on hysterical basis) reached the following most respectable diet list:

	Albumin.	Fat.	Carbo-Hydrate.
7 A. M.			
500 gm. of milk,	17.0	18.2	24.0
8 A. M.			
One small cup of coffee or tea, with 20 gm. of cream,	0.7	5.0	0.7
80 gm. of cold meat,	30.8	2.0	. .
One French roll,	4.5	0.5	29.0
20 gm. of butter,	0.3	16.6	0.1
100 gm. of roast potatoes,	1.8	10.0	25.0
10 A. M.			
300 gm. of milk,	10.2	10.9	14.4
12 M.			
300 gm. of milk,	10.2	10.9	14.4
1 P. M.			
200 gm. of soup,	2.2	3.0	11.4
200 gm. of roast meat,	76.4	5.4	. . .
200 gm. of mashed potatoes,	6.2	1.7	42.6
125 gm. of prunes,	0.4	. .	8.3
200 gm. of flour food,	12.8	21.2	45.0
3.30 P. M.			
500 gm. of milk,	17.0	18.2	24.0
5.30 P. M.			
300 gm. of milk,	10.2	10.9	14.4
80 gm. of cold meat,	30 8	2.0	. . .
One French roll,	4.5	0 5	29.0
20 gm. of butter,	0.3	16.6	0.1
8 P. M.			
80 gm. of roast meat,	30.8	2.0	. . .
40 gm. of toast,	0.6	5.2	33.2
500 gm. of milk,	17.0	18.2	24.0
9.30 P. M.			
500 gm. of milk,	17.0	18.2	24.0
20 gm. of toast,	0.3	2.6	16.6
Total,	295.0	199.8	380.2
Calories about,	1200	1850	1550

Total combustion value about 4600 calories.

Effects of Cooking on Food.—The practice of cooking is not equally necessary in regard to all articles of food. There are important differences in this respect, and it is interesting to note how correctly the experience of mankind has guided them in this matter. The articles of food which we still use in the uncooked state are comparatively few; and it is not difficult in each case to

indicate the reason of the exemption. Fruits which we consume largely in the raw state, owe their dietetic value chiefly to the sugar which they contain; but sugar is not altered by cooking. Salads may be regarded more as a relish for other food, and as having a quasi-medicinal purpose, rather than as a substantial source of nutriment. Milk is consumed by us, both cooked and uncooked, indifferently, and experience justifies this indifference; for Sir William Roberts found on trial that the digestion of milk by pancreatic extract was not appreciably hastened by previously boiling the milk.

This eminent writer characterizes our practice in regard to the oyster as being exceptional and furnishing a striking example of the general correctness of the popular judgment on dietetic questions. The oyster is almost the only animal substance which we habitually, and by preference, eat in the raw or uncooked state; and it is interesting to know that there is a sound physiological reason at the bottom of this preference. The fawn-colored mass which constitutes the dainty of the oyster is its liver, and this is little else than a heap of glycogen, or animal starch. Associated with the glycogen, but withheld from actual contact with it during life, is its appropriate digestive ferment—the hepatic diastase. The mere crushing of the dainty between the teeth brings these two bodies together, and the glycogen is at once digested, without other help, by its own diastase. The oyster in the uncooked state, or merely warmed, is, in fact, self-digestive. But the advantage of this provision is wholly lost by cooking, for the heat employed immediately destroys the associated ferment, and a cooked oyster has to be digested, like any other food, by the eater's own digestive powers.

With regard, however, to the staple articles of our food, the practice of cooking them beforehand is universal. In the case of the farinaceous articles cooking is actually indispensable. When men under the stress of circumstances have been compelled to subsist on the uncooked grain of the cereals, they have soon fallen into a state of inanition and disease. By the process of cooking, the starch of the grain is not only liberated from its protecting envelopes, but it undergoes a chemical change by which it is transformed into the gelatinous condition, and this immensely facilitates the attack of the diastatic ferments. A change of equal importance seems to be induced in the proteid matter of the grain. Sir

William Roberts found that the gluten of wheat was much more digestible by both artificial gastric juice and by pancreatic extract in the cooked than in the uncooked state. In regard to meat the advantage of cooking consists chiefly in its effects on the connective tissue and the tendinous and aponeurotic structures associated with muscular fiber. These are not merely softened and disintegrated by cooking, but are chemically converted into the soluble and easily digested form of gelatin. Sir William Roberts made instructive observations on the effects of cooking on the contents of the egg. The change induced on egg-albumen by cooking is very striking. For the purpose of testing this point, he employed a solution of egg-albumen made by mixing white-of-egg with nine times its volume of water. This solution when heated in the water-bath does not coagulate nor sensibly change its appearance, but its behavior with the digestive ferments is completely altered. In the raw state this solution is attacked very slowly by pepsin and acid, and pancreatic extract has almost no effect on it; but after being cooked in the water-bath, the albumen is rapidly and entirely digested by artificial gastric juice, and a moiety of it is rapidly digested by pancreatic extract.*

Indications of the Palate.—"The indications of the palate are of great importance in the regulation of diet, and should always be inquired into and carefully considered. The palate is placed like a dietetic conscience at the entrance gate of food, and its appointed function is to pass summary judgment on the wholesomeness or unwholesomeness of the articles presented to it. It acts under the influence of a natural instinct, which is rarely at fault. This instinct represents an immense accumulation of experience, partly acquired and partly inherited. It is, of course, not infallible—no instinct is; but so close and true are the sympathies of the palate with the stomach and the rest of the organism, that its dictates are entitled to the utmost deference as those of the rightful authority in the choice of food. I am, of course, aware that the palate—or, rather, the civilized palate—is not always credited with these solid, good qualities. Some persons there are, not medical authorities, who distrust its office, and regard its indications with suspicion, as if they were the suggestions of some frivolous and wanton agency, tempting men to a vain gratification of the senses, rather than as

*The fact was probably overlooked that raw egg-albumen does not require digestion and can be absorbed from the intestine as such.

those of an honest and skilful guide in the choice of food. This puritanical view of the palate is wholly unscientific; it moreover implies, to speak figuratively, a gross slander on a responsible and rarely endowed organ, which has performed in the past, and still performs, most difficult and most complicated functions with conspicuous success; for who shall venture to say, that in the evolution of the human animal from the short-lived, immoral, and stupid savage, with his diet of wild fruit, roots, raw flesh, and unfiltered water, to the status of civilized man the promptings of the palate have not played an important and even indispensable part? We are apt to forget that there is no such a thing as an absolutely good or an absolutely bad flavor to the animal palate. Sweet things are indifferent to the palate of the carnivora; and, conversely, the taste of flesh has no attraction to the herbivora. Each animal has its own gustatory standard, which is accurately adjusted to the wants of its particular economy." (Sir William Roberts, "Digestion and Diet.")

DIETETICAL COOKING.*

General Remarks.—It is evident that the subtle art of cooking can be practised with advantage to those suffering from indigestion only by those who understand thoroughly the general fundamental principles of the art, and have in addition some experience therein. But if diligence, care, and cleanliness are very desirable qualities in cooking for people in good health, they become an absolute necessity for those who undertake the preparation of food for digestive organs which are not in good health. By no means should the attention be taken from the work by other matters, for in that case the care which is necessary will suffer, and the most scrupulous cleanliness must be applied (Wegele).

It should be understood here that we have confined ourselves to the most necessary things, and have not considered the details concerning the arrangement of the kitchen, construction of the fireplace, the cooking utensils and fuel, food-stuffs or their adulteration; and, further, we have not undertaken the description of

* We have availed ourselves of a large number of works in compiling this particular chapter. The name of the originator of any particular article of diet has been added to the directions given whenever it was obtainable. Of the larger works used we mention Sir Wm. Roberts, Munk and Uffelmann, Wegele, Biedert and Langermann, Gilman Thompson, Boas, Penzoldt and Stintzing's "Handbuch d. Therapie," etc.

complicated dishes, but have given directions for the preparation of only the simplest every-day dishes in such a way as to serve dietetical purposes. In this respect the advice of Penzoldt deserves consideration, to use vessels with protecting lids for the keeping of foods which are to be eaten later when cold. Naturally all food-stuffs must be of the best quality, for the best is just good enough for the sick. Aside from this, nothing in the slightest degree spoiled may be used in cooking for the sick. In the eulogy of the palate we have already emphasized appetizing preparation, for, as is well known, dyspeptics are easily seized by nausea, while on the other hand a suitable way of preparing food may stimulate the appetite. For the same reason, every after-taste due to the character of the cooking utensils or their uncleanliness is to be avoided as far as possible, and the utensils should be, wherever possible, earthen, enameled, or nickel-plated. Food must never be brought to the table too hot, for the patients are thus tempted to eat them in this state in spite of the directions of the physician, and on this account it is best to put the food on a second plate or cup. The contrary is just as injurious, and it is therefore well to prepare foods, which are subject to rapid cooling, in vessels with double bottoms, filled with hot water. Concerning the seasoning of the dishes, only a moderate use of cooking salt is allowed, and other spices are not to be used without the permission of the physician; Wegele strictly forbids the use of citron or pomegranate skins in dietetical cooking. Water which is to be used for cool drinks should best be boiled and then be allowed to cool.

Concerning the measures used in the following chapter,—

1 teaspoon equals about five gm.,
1 tablespoon equals about 15 gm.,
1 soup plate equals about 250 gm.,
1 cup equals about 200 to 250 gm.,
1 wineglass equals about 150 gm.,

in which calculation naturally no attention has been paid to the specific gravity of the different substances.

I. Drinks and Liquid Foods.

Barley Soup (*Ringer*).—To a tablespoonful of pearl barley washed in cold water add two or three lumps of sugar, the rind of one lemon, and the juice of half a lemon. On these pour a quart

of boiling water, and let the mixture stand for seven or eight hours. Strain. The barley water should never be used a second time. Half an ounce of isinglass may be boiled in the water. If not needed at once, these barley preparations should be kept in the refrigerator, and warmed when required.

Rice-water, or Mucilage of Rice (*Pavy*).—Thoroughly wash one ounce of rice with cold water. Then macerate for three hours in a quart of water kept at a tepid heat, and afterward boil slowly for an hour, and strain. A useful drink in dysentery, diarrhea, and irritable states of the alimentary canal. It may be sweetened and flavored in the same way as barley water.

Lemonade (*Pavy*).—Pare the rind from a lemon thinly, and cut the lemon into slices. Put the peel and sliced lemon into a pitcher with one ounce of white sugar, and pour over them one pint of boiling water. Cover the pitcher closely, and digest until cold. Strain or pour off the liquid.

Beef-essence (*Yeo*).—Cut the lean of beef into small pieces, and place them in a wide-mouthed bottle securely corked, and then allow it to stand for several hours in a vessel of boiling water. This may be given in teaspoonful doses to infants who can not take milk, and in larger quantities to adults.

Beef-tea (*Germain-Sée*).—Meat cut into small pieces, cold water added, and then gradually heated to 140° or 160° F. Press, strain, and flavor with salt and pepper. This is much inferior to the preparations made with hydrochloric acid.

Chicken Broth (*Bartholomew*).—Skin and finely mince a small chicken or half of a large fowl, and boil it, bones and all, with a blade of mace, a sprig of parsley, and a crust of bread, in a quart of water for an hour, skimming it from time to time. Strain through a coarse colander.

Chicken, Veal, or Mutton Broth (*Yeo*).—Chicken, veal, or mutton broth may be made like beef-tea, substituting chicken, veal, or mutton for beef, boiling in a saucepan for two hours, and straining. For chicken broth the bones should be crushed and added. For veal broth the fleshy part of the knuckles should be used. Either may be thickened and their nutritive value increased by the addition of pearl barley, rice, vermicelli, or semolina.

Mutton and Chicken Broths (*Osler*).—Mince a pound of either chicken or mutton, freed from fat, put into a pint of cold water, and let stand in a cold jar on ice two or three hours. Then cook three

hours over a slow fire, strain, cool, skim fat off, add salt, and serve hot or cold. Such broth is much better than any manufactured meat preparations. Good mutton broth is difficult to make on account of the meat containing so much fat.

Raw Meat Diet (*Ringer*).—Use two ounces of rump steak; take away all fat, cut into small squares without entirely separating the meat, place in a mortar, and pound for five or ten minutes; then add three or four tablespoonfuls of water and pound again for a short time, afterward removing all sinews or fiber; add salt to taste. Before using, place the cup or jar containing the pounded meat in hot water until just warm.

Or scrape the beefsteak with a sharp knife, and after removing all fat and tendon, if not already in a complete pulp, pound in a mortar. Flavor with salt and pepper. This may be taken in the form of a sandwich, between thin bread and butter or mixed with water to the consistency of a cream. If preferred, the meat may be rolled into balls with a little white-of-egg, and boiled for two or three minutes, or until the outside turns gray, just long enough to remove the raw taste.

Chicken Jelly (*Adams*).—Clean a fowl that is about a year old, remove skin and fat; chop fine, bones and flesh, in a pan with two quarts of water; heat slowly, skim thoroughly, simmer five to six hours; add salt, mace, or parsley to taste; strain; cool. When cool, skim off the fat.

The jelly is usually relished cold, but may be heated. Give often in small quantities.

Milk-punch.—Make by adding brandy or whiskey or rum to milk in the proportion of about one to four or six parts of milk; flavor with sugar and nutmeg; shake well.

Sherry or Brandy and Milk (*Ringer*).—To one tablespoonful of brandy or one wineglassful of sherry, in a bowl or cup, add powdered sugar and a very little nutmeg to taste. Warm a breakfast cupful of new milk and pour into a pitcher. Pour the contents from a height over the wine, sugar, etc. *The milk must not boil.*

Junket (*Anderson*).—Sweeten with white sugar one pint of good milk. If wine is allowed, a dessertspoonful of sherry is an improvement. Heat to new milk warmth, pour into a shallow dish, and stir in two teaspoonfuls of essence of rennet. This will form a slight curd. Grate a little nutmeg over it, or add a pinch of powdered cinnamon. Serve when quite cold. In cold weather the

milk should be placed in a warm room to set. An excellent food and good substitute for milk in typhoid fever, etc.

Egg-nog.—Egg-nog is made by adding the beaten yolk of egg and a little spirits to a tumblerful of milk, stirring well, adding sugar and white of the egg, separately beaten. The digestibility of both of these highly nourishing and stimulating preparations is enhanced by the addition of ½ of an ounce of lime-water, which does not affect the taste.

Egg and Wine (*Ringer*).—Take one egg, ½ of a glass of cold water, one glass of sherry, sugar, and a very little nutmeg, grated. Beat the egg to a froth with a tablespoonful of cold water. Make the wine and water hot, but not boiling; pour on the egg, stirring all the time. Add sufficient sugar to sweeten, and a very little nutmeg. Put all into a porcelain-lined saucepan over a gentle fire, and stir one way till it thickens, but do not let it boil. Serve in a glass, with crisp biscuits or sippets of toast.

Milk for Pudding or Stewed Fruit (*Ringer*).—Boil a strip of lemon and two cloves in a pint of milk; mix ½ of a teaspoonful of arrowroot in a little cold milk and add it to the boiling milk; stir it until about the consistency of cream. Have ready the yolks of three eggs beaten up well in a little milk. Take the hot milk off the fire, and as it cools add the eggs and a teaspoonful of orange-flour water, stirring it constantly till quite cool. Keep it in a very cool place until required for use.

Arrowroot Blancmange (*Ringer*).—Take two tablespoonfuls of arrowroot, ¾ of a pint of milk, lemon, and sugar to taste.

Mix the arrowroot with a little milk to a smooth batter; put the rest of the milk on the fire and let it boil, sweeten and flavor it, stirring all the time till it thickens sufficiently. Put into a mold until quite cold.

Arrowroot (*Pavy*).—Mix thoroughly two teaspoonfuls of arrowroot with three tablespoonfuls of cold water, and pour on them ½ of a pint of boiling water, stirring well meanwhile. If the water is quite boiling, the arrowroot thickens as it is poured on, and nothing more is necessary. If only warm water is used, the arrowroot must be boiled afterward until it thickens. Sweeten with loaf sugar, and flavor with lemon-peel or nutmeg, or add sherry, port wine, or brandy if required. Boiling milk may be employed instead of water, and when this is done no wine must be added, as it would otherwise curdle.

Oatmeal Gruel (Plain).—Two tablespoonfuls of oatmeal, one saltspoonful of salt, one scant teaspoonful of sugar, one cup of boiling water. Cook for thirty minutes; then strain through a fine wire strainer to remove the hulls, place again on the stove, add the milk, and heat just to the boiling point. Serve hot.

Farina Pudding (U. S. Army Hospital Recipe for 12 Men).—Farina, ½ of a pound; milk, two pints; water, one pint; sugar, 2½ ounces; eggs, four ounces; nutmeg, ½ of an ounce.

Directions.—Put the water into a stewpan with a little salt. When it boils stir in the farina. Let it boil twenty minutes. Stir in the milk, which must be hot. Beat the eggs until they are very light; mix the sugar with them. Stir in the eggs and sugar with the farina. Add the spice. Put it into a moderate oven and bake a half or three-quarters of an hour.

Port-wine Jelly (*Ringer*).—Put into a jar one pint of port-wine, two ounces of gum arabic, two ounces of isinglass, two ounces of powdered white sugar-candy, ¼ of a nutmeg, grated fine, and a small piece of cinnamon. Let this stand closely covered all night. The next day put the jar into boiling water and let it simmer until the contents are dissolved, then strain, let stand till cold, and then cut into small pieces for use.

Nutritious Coffee (*Ringer*).—Dissolve a little isinglass in water, and then put ½ of an ounce of freshly ground coffee into a saucepan with one pint of new milk, which should be nearly boiling before the coffee is added; boil both together for three minutes. Clear it by pouring some of it into a cup and dashing it back again, add the isinglass, and let it settle on the hob for a few minutes. Beat up an egg in a breakfast cup and pour the coffee upon it; if preferred, drink it without the egg.

Glair-water.—Into 200 c.c. of cold water which has previously been boiled, put with constant stirring the white of one egg, and add, according to prescription, three teaspoonfuls of powdered sugar, or grape-sugar, or ten gm. of cognac. The white of an egg equals about 12 calories; 15 gm. of sugar equals about 50 calories; ten gm. of cognac equals 50 calories.

Kefyr.—It is best to procure moist kefyr mushrooms (not the dried grains) prepared for immediate use. They can be procured from the Caucasian Kefyranstalt in Breslau, or from Dr. M. Lehmann, Berlin C. (43 and 44 Heiligegeist Strasse). Pour away the liquid contained in the bottle, wash the mushrooms in a luke-

warm (about 15° R. or 18.7° C.) soda solution of 5 : 1000, rinse with clean lukewarm water, and after pouring away the water place the mushrooms in a vessel of porcelain or Bunglan clay of two liters' capacity. Previously two liters of milk should have been boiled and allowed to cool again. Now pour the milk, whose temperature should be about 15° R. or 18.7° C., upon the mushrooms, close the vessel tightly, and let it stand twenty-four hours in a place whose temperature is 13° to 15° R. or 18.7° C. (in summer in the cellar), during which time it is expedient to often stir the milk carefully. At the expiration of this time it should be stirred again, and the milk is then poured through a moderately fine wire sieve into thoroughly cleaned bottles with patent stoppers. These bottles are again to be kept twenty-four, thirty-six, forty-eight, or at the highest fifty-four hours (according as kefyr one, two, three, or four days old has been prescribed) in a place whose temperature is kept at about 15° R. or 18.7° C., lying, not standing up, and are then ready for use.

By frequent shaking, the process of fermentation may be hastened, likewise through heat, on which account the fermentation takes place more quickly in midsummer, and the kefyr consequently will be finished sooner. The mushrooms which remained in the sieves after pouring off the milk into the patent flasks must each time be rinsed with lukewarm water and freed from particles of cheese, and afterward placed again in the thoroughly clean porcelain vessel, and milk is then again poured upon them. After two or three days the preparation is so regulated that each day two bottles (of one liter each) become ready for use, for which reason four patent-stoppered bottles are necessary. Once a week the bottles must be rinsed with a lukewarm soda solution of 5 : 1000 instead of with lukewarm water, in order to free them from acid. At first let the patient drink one wineglassful two or three times a day, then ⅓ of a liter, and constantly increase the quantity until the prescribed dose has been reached. One hundred gm. of kefyr equal about 45 calories.

Almond Milk.—Thirty grs. of sweet almonds and two bitter almonds are blanched after they have lain twenty-four hours in cold water. One can also scald the almonds with boiling water; then they can be easily pressed out of their hulls after a few minutes. The almonds are either ground in a mill or pounded in a mortar, then mixed with ¼ of a liter of warm water or warm milk,

and the mixture is allowed to stand two hours, after which it is strained through a cloth and the juice well pressed out.

Thirty grs. of almonds equal 200 calories; 250 gm. of milk equal 170 calories.

Extract of Meat (*according to Wiel*), "Succus carnis recenter expressus."—Meat free from fat is chopped fine, arranged in several layers, which are separated by coarse (filter) linen, and subjected to pressure in a colander; the juice is given pure (as medicine) by the teaspoonful, or also diluted with beef-tea, but must not be subjected to a temperature higher than 50° R. or 62.5° C., for otherwise the albuminous parts contained in it would coagulate. "Valentine's meat juice" (extract) may serve as a good substitute for the fresh extract of meat particularly prepared. A teaspoonful of this preparation is diluted with one to two tablespoonfuls of cold, or, at the most, lukewarm, water; the yolk of one egg may also be added.

Meat Extract Ice (*according to v. Ziemssen*).—One k. of fresh beef is cut into pieces the size of a hand, and is wrapped in coarse lattice-like linen, put under a lever-press and slowly pressed; this is best done by an apothecary. The juice is caught in a porcelain dish. In this way one gets about 500 gm. This is mixed with 250 gm. of sugar and 20 gm. of freshly pressed lemon juice (though this had better be omitted for dyspeptics), and 20 gm. of cognac, containing extract of vanilla, which has been well stirred with the yolks of three eggs, is added, and the whole is placed in a freezer.

Bottled Bouillon (*according to Uffelmann*).—Three hundred gm. of fresh, lean meat are cut into small blocks, and without any addition is put into a clean bottle with wide mouth. This is closed, if there be no suitable stopper, with a stopper of pure, sterilized cotton, and placed in a vessel of warm water, slowly heated, and the water should be allowed to boil one-half hour. The bottle which is now to be taken out contains about 100 gm. of a turbid brown broth, which is poured off without straining.

Simple Bouillon, or Beef-tea.—One-half of a k. of lean beef is cut into small pieces, put into a pot holding about three liters, with a well-fitting cover, or into a steam cooking apparatus. This is to be filled with cold water, and the meat to be boiled three to four hours. According as the bouillon is desired concentrated or dilute, the liquid which evaporates must be replaced by the addition of boiling water. Finally, one obtains about two liters of bouil-

lon, and the meat which remains is of no further use. To obtain greater palatability and a prettier color, the meat may be first browned in a little hot, pure lard before cooking, fresh soup herbs or a handful of dried Knorr's julienne added, then finally add the three liters of cold water.

Meat Jelly (*according to Hepp*).—Good beef, free from fat and bones, is cooked on the waterbath with a little water for sixteen hours, until it congeals into jelly. Often one is compelled to use artificial preparations in the making of bouillon or in strengthening weak bouillon. The most reliable in this respect is Liebig's extract of meat (about ten c.c. to 250 gm.), or Cibil's bouillon (one tablespoonful to 250 gm.); very convenient also are Quaglio's bouillon capsules. If at the same time one wishes to give to the bouillon an increased nutritive value, one can add one teaspoonful of meat peptone; or either Mosquera Julia beef meal, Armour's vigoral, or Valentine's meat juice may be used.

A preparation which is often of service is Leube-Rosenthal's meat solution. One k. of beef is chopped fine, put into a vessel with one liter of water and 20 gm. of pure hydrochloric acid, which vessel is put in a Papin's steam cooking apparatus, in which it should boil ten to fifteen hours (with frequent stirring). After this the mass is put into a mortar and ground to an emulsion. After a further cooking of fifteen hours with bicarbonate of soda, it becomes neutral, and is then steamed to a consistency of mush, and put into four cans, which are to be soldered. As the making of this preparation requires much time and particular care, it is advisable to procure it from one of the following firms, who put it upon the market in cases of ¼ k. (enough for an adult for one day): Armour & Co.,* Parke, Davis, & Co.,† Dr. Mirus'sche Hof-apotheke (R. Stutz),‡ Huffner's Hof- und Ratsapotheke (R. Wahrburg),‡ C. Reinhardt (formerly Charrier). §

Soups with Fillers.

(*a*) *Soups with Fillers from the Cereal Kingdom.*—The grains in question (such as barley, or peeled barley, oats, green corn, rice) should be softened the night before in cold water, in which they are to remain until the following forenoon. Then the water is poured off and the grains are put on the fire with weak, cold bouillon, where they should be kept boiling at least three hours;

* Chicago. † Detroit, Mich. ‡ Jena. § Berlin W., 27 Behren Strasse.

one-half hour before serving, the soup is strained through a fine hair sieve, and, after the addition of a little meat extract, is made to boil again; salt is then added as required, and to one plate of soup the yolk of one egg may be added. If one is to prepare a single plate of such soups, the soup meals of Knorr in Heilbronn are very serviceable, although they do not become gelatinous like the soups prepared from whole grains, and are not as appetizing. These meals must be stirred with cold bouillon to a thin liquid mass, and allowed to run into boiling beef-tea, which after that must boil at least one to two hours longer. Twenty gm. of meal is calculated for one plate of soup. In serving, one can add also the yolk of an egg. The nutritive value of these soups may be considerably increased by the addition of aleuronat flour. It is best to take eight gm. of aleuronat flour and 16 gm. of oat- or green corn meal for one plate of soup.* The aleuronat meal is mixed with cold water (or beef-tea), and is only added to the soup after the latter has boiled one-half hour. The meal swells hardly at all, and for that reason more of the two flours is to be taken than is necessary, ordinarily, in making of soup. Soups prepared with 20 gm. of oat meal, or leguminose meal, barley meal, tapioca, rice, etc., have a combustion value of about 70 to 75 calories, which is increased about 60 calories by the addition of the yolk of one egg.

(*b*) *Tapioca Soup.*—For this soup the French tapioca of N. & J. Bloch in Paris, and Knorr in Heilbronn, had best be used, which can be had in most of the larger fancy groceries in packages of 250 gm. For one plate of soup a heaped teaspoonful of these grains is boiled for half of an hour with beef-tea, which has been boiling for some time previous, and to this, after a quarter of an hour, a little extract of meat, sufficient to cover the point of a knife, is added; if this be added later, just before serving, the taste of the extract is easily distinguished, which is disagreeable to many patients.

(*c*) *Sweetbread Soup.*—The sweetbread is soaked for one hour in cold water, which is during this time often to be renewed; then it is boiled in slightly salted beef-tea or salt water for one hour, to which one may add one teaspoonful of julienne for improving the flavor. After it is cooked completely soft, it is taken out of the beef-tea and freed from all skins, blood-vessels, etc. Now it can be cut

*Can be procured from Dr. Hundhausen's Stärkefabrik, Hamm in Westfalen; 4½ k. cost seven marks, C. O. D.

either in pieces the size of a walnut, which one lays on the soup plate and then pours over the beef-tea, or the sweetbread can be forced through a fine sieve; beef-tea is poured over the mass and the whole is again put on the fire until it boils, after which the soup may be served. The latter proceeding is rather to be recommended in the case of dyspeptics. One hundred gm. of sweetbread (raw) is equivalent to about 90 calories.

(*d*) *Brain Soup.*—A calf's brain is allowed to lie in water (cold) for one hour, in order to draw out the blood contained in it; then the water is poured off, the brain is once more thoroughly washed and cooked in weakly salted beef-tea or salt water with the addition of one teaspoonful of julienne, for one hour. Then immediately force it through a fine sieve, dilute the mush with beef-tea and cook it again. In serving, the yolk of an egg may be added. One hundred gm. of calf's brain equal 140 calories.

(*e*) *Soup Containing Meat* (*according to Professor M. Rosenthal*).—Scraped raw beefsteak is chopped fine, forced through a sieve, the mass, soft as butter, is thoroughly mixed with the yolk of an egg, and mixed in minute particles to a greater or less degree with boiling soup.

(*f*) *Meat Purée Soup* (*according to Hedwig Heyl*).—Twenty gm. of grated rolls are cooked for one-quarter of an hour with ⅜ of a liter of bouillon. Stewed chicken meat is pounded fine, passed through a hair sieve, and 25 gm. of it are stirred together with one tablespoonful of cream or one teaspoonful of meat peptone, several spoonfuls of soup are added, and now beaten up with the entire mass, and served without further cooking.

(*g*) *Roll Soup* (*according to Hedwig Heyl*).—Thirty gm. of grated rolls are roasted with ten gm. of butter, without coloring the latter, ⅜ of a liter of bouillon is poured over and slowly boiled for half an hour. The yolk of an egg is beaten up with a tablespoonful of sweet or sour cream, and then put into the soup, and the latter is passed through a sieve upon the previously warmed plate (equal to about 240 calories).

(*h*) *Soup Biscuit.*—Forty gm. of butter are stirred for one-quarter of an hour, afterward mixed with two whole eggs, a little salt is added, and at last 40 gm. of flour. In order to make the mass rise more easily, one can add three gm. of baking powder (consisting of bicarbonate of soda and tartaric acid, which can be had in most drug stores in packages of 30 gm.). A long square sheet

iron mold is rubbed with butter, the mass is put into it and baked in the oven with moderate heat for half an hour. When the biscuit has cooled off, it is taken out, cut into blocks and can then be added to the various soups (such as sweetbread, brain, or pea soup). The whole mass corresponds to about 630 calories.

(*i*) *Noodle Soup* (*Vermicelli Soup*).—The noodles (only the best quality) must be boiled half an hour in very good bouillon. A soup of about ten gm. of vermicelli equals about 50 calories.

(*k*) *Butter Dumpling Soup*.—Thirty gm. of butter is stirred one-quarter of an hour, one whole egg and a little salt being added; stir the same and mix well with the butter, and then add 30 gm. of flour. With a teaspoon rather long lumps are cut out of the dough and put into boiling beef-tea, in which they must boil twenty minutes more on a fire not too strong. The whole mass equals about 420 calories.

(*l*) *Green Pea Soup* (*Mashed*).—Fresh green peas are boiled in salt water until thoroughly soft; in advanced seasons when they are no longer very young, add ¼ of a gm. of carbonate of soda; canned peas are also very good at any time for making this soup. Let the water run off through a strainer, force the peas through a fine sieve, mix with a teaspoonful of flour (aleuronat flour), pour beef-tea over the mass and cook again; 100 gm. of peas equal 75 calories; ½ of a liter (420 gm.) of peas gives 280 gm. of mashed, equal to 300 calories.

II. Fish.

Fish for the table of a sick person should never be boiled or fried in fat, but only boiled in water. Of the fresh-water fish the trout, the perch-pike (Lucioperca sandra), pike, carp, grayling, and salmon come under consideration here. Of salt-water fish the black- or sea-bass, sea-trout, the bluefish (Pomatomus saltatrix), the mackerel, cod, rockfish, and haddock are suitable. The fish is carefully freed from scales, rubbed inside with salt, and boiled in very strongly salted boiling water, in which it is allowed to remain, according to size, from one-quarter to one-half hour; all spices are to be omitted, only a handful of dried julienne may be put in the boiling water, by which the flavor is considerably increased. All fat and pungent sauces are to be avoided, and even hot butter will generally not agree with the dyspeptic, so that it is best to put only a little fresh butter on the fish when serving. Of the sea fish, the cod,

rose-fish, and haddock are to be recommended. Their preparation is the same, except that they are soaked one-quarter of an hour previously in fresh water (not in boiling, but cold water), in which is put a large quantity of salt and also some julienne. The vessel must be large enough to allow the fish to be surrounded on all sides by the water. A two- or three-pound haddock must remain on the fire thirty to forty minutes to be thoroughly done; sea fish also are to be served with fresh butter.

III. Meats.

1. *Sirloin* (*Fillet*).—For the tenderness of beef it is of importance that it be allowed to hang long enough; for this two to four days are necessary in summer, in winter as many as eight days; only in the coldest season it must be protected from frost, through which it becomes very dry. The meat is freed from all fat and membranous parts, well beaten, washed and salted, and thus put into a stewpan with hot lard, in which it is quickly turned over several times. The meat loses, in roasting in the English style, ten per cent. in weight, and in slow roasting, 30 per cent. in weight. To prepare a fillet in English style, so that it is still red inside, one calculates for each pound of meat one-quarter of an hour, so that a four-pound roast requires one hour's roasting. It is entirely unsuitable to try, by means of sticking with a fork, how far the roast is done, for much juice is lost by this, and the cook must learn by practice, by the nature of the pan, the thickness of the roast, the strength of the fire, to calculate the period of time necessary for the completion of the roast. During roasting frequently add spoonfuls of beef-tea, so that the butter does not become too dark, but the bouillon must never be poured upon the meat itself. One-quarter of an hour before serving, the roast is taken out of the pan, all fat is carefully skimmed from the sauce, a tablespoonful of white flour and a teaspoonful of aleuronat flour are mixed with a little cold bouillon, a little extract of meat is added, and this thin mixture is then added to the sauce of the roast, which is again made to boil, and the roast is again laid into it until serving. One hundred gm. of beef roasted in English style equal about 210 calories.

2. *Roast Beef.*—This roast is only good and juicy when in large, thick pieces; the preparation is exactly the same as the preceding. It is juicier when roasted on the spit, though in most households

the necessary equipments are wanting. With this meat, which has a tolerably coarse grain, a sufficient time for hanging is absolutely necessary.

3. *Raw Beefsteak* (*according to Leube*).—From the loin, which has hung a sufficient time, as much meat is scraped off with a dull spoon-handle as can be separated without violence, until one has a mass of about 150 gm. The mass thus scraped off is slightly salted, made into a very small cake, and eaten either entirely raw or just roasted on the surface in fresh butter. One hundred gm. equal about 120 calories.

4. *Beefsteak* (*according to Wiel*).—Take some of the best sirloin and cut across a piece as thick as a thumb; after this has been well pounded and slightly salted on one side, it is put into an iron or enameled pan, fried for one minute on one side in fresh butter, then turned, gravy poured over, and is fried on the other side only one-half of a minute, after which it is immediately served on a warmed plate. One hundred gm. equal about 130 calories.

5. *Beefsteak in Oil.*—From a well-hung fillet a piece as thick as a thumb is cut, all skins and fat removed, the same well pounded, and salted. Then spread on both sides with the finest olive oil, cover up well and allow it to remain thus two hours. Thereafter put into the pan and fry without any further grease (except the oil previously spread over it) till it is brown on both sides. The time necessary for frying varies from five to ten minutes, according to the degree one wishes it done inside.

6. *Roast Veal.*—The leg of veal, after it has hung a sufficient time, is freed from the thick outside skin and laid in sweet milk for one or two days in summer, two or four days in winter, by which it becomes tender and soft. Before using, it is carefully washed, thoroughly skinned, and well salted; thereupon it is larded with fresh lard and roasted in tolerably hot butter or white beef fat, of which about 200 gm. are necessary. For the rest it is treated like any other roast, except that it is best in the case of veal to roast until well done, which for a small roast takes two hours, for a large one, three hours. In the English way one and one-quarter to two hours are sufficient. Roast veal, when the bone is not previously taken out, gives a very good, thick sauce, so that in most cases it is necessary to add only a little bouillon after the fat has been skimmed off. Its value in calories is about the same as that of lean beef.

7. *Veal fricandeau* is also laid in milk a few days before using, which milk it is best to let sour, for the flavor is thus increased; it must be done, however, in such a manner that the milk covers the meat completely. For the rest, the meat is treated as any other veal roast, except that one and a half hours' roasting with a good fire will suffice. The sauce is to be mixed with flour, and it can be given a piquant flavor by the addition of some cream.

8. *Veal Cutlets* (*Chops*).—The ribs of the calf are separated from the backbone; the single cutlets separated from each other are washed and freed from skins, pounded, salted, and fried in a pan with hot butter; they will be more tender if they have lain one day previously in milk; in this case they need be fried only eight or ten minutes, but otherwise it is preferable to fry them from one-half to one hour, not leaving them long in one place, often shoving them to and fro, during which time a piece of fresh butter is also added, and the melted butter is constantly poured over the cutlets; before serving, some good liquor from a roast is added. To cover cutlets with bread crumbs is not advisable in dietetic cooking. One hundred gm. of fried veal cutlets (also the following veal dishes) equal 230 calories.

9. *Scotched Collop.*—From the leg of veal, which has lain in milk two or three days, cut slices as thick as your thumb, wash, beat, and salt them, and put them in a pan with hot butter, where they must be allowed to brown slightly on both sides. Then pour in one glass of white wine and some bouillon, cover up tightly, and let them steam altogether for about one and a quarter hours, pouring in some bouillon from time to time. The addition of sour cream improves the flavor. But the digestibility is decreased by the sour cream. Then skim off all fat and with flour prepare a sauce as directed above.

10. *Fillet of Veal.*—From the fricandeau piece cut strips one cm. thick and six cm. wide, and prepare them exactly as in No. 9; in the middle lay a few pieces of middling, roll them up and tie with cord. For the rest proceed exactly as in the case of scotched collop.

11. *Veal Steak.*—Cut from the leg pieces as thick as your thumb, weighing about 100 gm., pound them well, wash, salt, and lay them in a pan with hot butter, and fry them, with frequent turning, for ten minutes. Either add some sauce from a roast, or prepare one from bouillon, flour, and meat extract, which is put into the pan, and then let the steaks fry in it for two minutes longer.

12. *Lamb's Saddle.*—The saddle of a young animal is laid in milk for two days, or the milk is allowed to sour, through which a venison-like flavor is obtained. Before using, the roast is washed, freed from fat and skins, and larded with fresh, unsmoked bacon; then it is put into a pan with previously heated beef fat or good butter, in which it must immediately be turned several times. It is roasted one and a half hours, during which time it is to be diligently basted by the addition of beef-tea. In the last hour pour in one glass of white wine and as much bouillon as the sauce has boiled down. With sour cream the roast becomes particularly well flavored, but not every patient can stand it. The sauce is prepared as in other roasts with flour.

13. *Roast Fowl.*—Fowl destined for roasting must be picked and cleaned immediately after killing, and then it is allowed to hang in a cool place at least one day; in winter, two to four days, for which reason one should always inquire, in buying dressed poultry, how long it had been killed.

(*a*) Young Cockerels.—Must be scalded before picking. Before roasting, the hair must be singed off, and they must be carefully washed and rubbed with salt inside and outside; afterward they are put in a pot with plenty of hot butter, roasted brown on both sides, with frequent basting, for which three-quarters to one hour is necessary. The sauce is made as above, with a little flour. One hundred gm. of raw chicken equal 100 calories.

(*b*) Capons and Pullets.—Should be roasted with little butter, since they are generally fat enough. According to their size they must be roasted, with frequent basting, from one and one-half to two hours.

(*c*) Young pigeons are treated just as young cockerels. Time of roasting about three-quarters of an hour.

(*d*) The Pheasant.—Yields a fine roast after it has hung about eight to fourteen days. Roast it from two to three hours with plenty of butter and frequent basting.

(*e*) The Partridge.—The same must be young and must have been killed several days before using, in order to furnish a tender roast. After it has been picked, cleaned, and washed, it is put in a tolerable quantity of hot butter, and a piece of fresh butter is also put inside the partridge. On the other hand, wrapping with bacon is less to be recommended for those having stomach trouble, and a roast just as juicy can be obtained by diligent basting; the

palatability can also be increased by the addition of white wine and sour cream. Time of roasting, one and one-quarter hours.

(*f*) Boiled Cockerels and Pigeons.—They are prepared just as for roasting, then laid in boiling, slightly salted bouillon, to which a little julienne has been added, and boil one to one and one-quarter hours. Very young pigeons are cooked soft in three-quarters of an hour; likewise very young cockerels.

14. *Roast Game.*

(*a*) Roast Hare.—The hare is skinned and then cleaned, but the liver, heart, head, etc., are not to be used in cooking for the sick. After the roast has been thoroughly washed within and without, it is well salted, and larded with fresh (not smoked) bacon, and treated exactly as the roast lamb, so that it is done in about one and one-half hours. By the addition of sour cream the roast hare becomes very good, but in this way it does not agree with every one. The sauce is prepared in the same way as in the case of fillet roast, with flour and beef-tea.

(*b*) Roast Venison (Doe).—The venison saddle is the most beneficial game for those who have stomach troubles. It is to be treated exactly as the roast hare, only it must be roasted about two and one-half hours, on account of its size. The joint of venison will gain considerably in tenderness and flavor if it is laid in light red wine a few days before using; for the rest it is to be treated exactly as the venison saddle, only it must be roasted two and one-half, three, or four hours, according to size. The sauce is the same as with roast hare. But game can also be treated in the English fashion, by roasting it only a short time, as in the case of fillet and roast beef. A venison joint thus requires one and one-quarter hours, approximately, with strong heat, and if very heavy, one and one-half hours. A venison saddle, if young and tender, requires three-quarters of an hour; if older, one and one-quarter hours. In this way the meat remains juicier and stronger.

(*c*) Venison Saddle (Stag).—Is to be treated in the same way, except that it must be roasted a correspondingly longer time; but generally the meat is not as tender and palatable as that of the doe. One hundred gm. of game (roast) equal about 215 calories (when thoroughly done).

15. *Stewed Meats.*

(*a*) Preserved Veal.—The meat from a leg or breast which has hung sufficiently is cut into pieces the size of a walnut; the latter

are put into a small stewpan with hot butter, and a little salt sprinkled over; immediately after they have been once turned in the butter, ½ of a glass of white wine, about 75 gm., is poured in and the whole covered up well and stewed for one and a quarter hours with moderate heat, some good bouillon being added from time to time; one quarter of an hour before serving, the sauce is prepared in the way before indicated, and immediately before serving, the yolk of an egg is mixed with water and put into the sauce.

(*b*) Preserved Sweetbread.—The sweetbread is cooked till it is soft as in the case of soup, is skimmed, cut into two halves and ten minutes before serving is laid in butter sauce, to be prepared in the following way: A little piece of butter is melted in a small dish, without being allowed to brown; then one tablespoonful of flour is added, well mixed with the butter; then pour in cold bouillon and a little white wine, so that, after the sauce has boiled, the whole forms a tolerably thick liquid. The amount of the ingredients must be determined by the amount of sauce desired. Before serving, the yolk of an egg is added to the sauce.

(*c*) Stewed Cockerels or Pigeons.—A young cockerel or pigeon is dressed as for roasting, quartered into equal parts, slightly salted, and laid in a stewpan in which a small piece of butter has been previously melted without being browned. The stewpan is covered tightly and the poultry stewed slowly for a quarter of an hour. Then ½ of a glass of white wine, about 75 gm., and some good bouillon are added, and it is again allowed to stew for about three-quarters of an hour longer, a little beef-tea being added from time to time. The sauce is the same as in the case of stewed veal. One hundred gm. of meat equal about 120 calories.

16. *Dishes from Chopped Fresh Meat.*—Be warned against allowing the butcher to chop the meat, as in some cases less desirable or less appetizing meat may be mixed in. Every household should possess a machine for chopping meats; in cases where there is none, do not mind the trouble of chopping it yourself.

(*a*) Roast Chopped Meat.—One-half of a pound of veal, ½ of a pound of beef, and ½ of a pound of pork, not entirely lean, are put through the chopping machine; the whole mass is then mixed in a dish with three whole eggs, ⅛ of a liter of milk, 1½ grated rolls, and a tolerable amount of salt; if the dough then seems too stiff, a little more milk may be added. The mass is made into a longish cake and roasted in hot lard or good butter (100 gm.) first on one

side and then on the other, until it is light brown. Time, one hour and a quarter. From this hardly any sauce will be obtained, hence one must be prepared from flour, bouillon, extract of meat, and a little white wine, which is to be poured over a quarter of an hour before serving. One hundred gm. of this roast equal about 250 calories.

(*b*) Cutlets from Chopped Meat.—The same mixture as in the preceding is made into little cutlets, allowed to fry on both sides in hot butter until light brown; then skim off all fat, prepare a butter sauce, pour it over, and let it fry with this for another half hour.

(*c*) Meat Balls (Veal).—One pound of meat from the leg is chopped up fine in the machine; 40 gm. of butter are stirred to foam, two whole eggs, and one roll, grated fine, are added; also a little salt, and according to taste of the individual a little finely chopped parsley. Of this mass flat cakes are made and cooked for one-quarter of an hour in salt water; butter sauce, or when allowed, anchovy sauce, is added, which is to be poured over the cakes one-quarter of an hour before serving. One hundred gm. equal 250 calories.

17. *Dishes from Chopped Roast Meat.*

(*a*) Hash.—In a little butter or lard put some finely chopped roast meat (veal, fowl, or game), stew for five minutes with frequent stirring and pour over any sauce remaining from the roast, or make a special sauce as follows: Sprinkle some fine flour upon the stewed meat, mix well, pour in a little white wine and enough bouillon so as to produce a rather thick gruel. Then stew for one-quarter of an hour longer with moderate heat, keeping the vessel well covered. The hash is now done. A little extract of meat added will improve the flavor. One hundred gm. equal about 225 calories.

(*b*) Meat Pudding.—Sixty gm. of butter are stirred until foamy, four yolks of eggs, salt, and a little fine-cut parsley added. Two French rolls are grated fine, the inside cut into small pieces, and soaked in milk, in which it remains one hour; 170 gm. of roast meat is cut fine or chopped in a machine; the grated rolls are taken out of the milk, pressed, and with the chopped roast meat mixed with the other mass (butter and eggs). If allowed, two tablespoonfuls of sour cream may also be added. At last the whipped whites of four eggs are mixed in, and the whole dough is put in a mold rubbed with butter, and stewed with dust from the rolls. In this the pudding is cooked for one and one-quarter hours in a waterbath. Any

sauce remaining from a roast is added (or anchovy sauce). One hundred gm. equal about 220 calories.

(*c*) Omelette Soufflé from Remnants of Roasts.—Forty gm. of finely cut roast meat are mixed with one tablespoonful of sweet or sour cream; a little salt and the yolk of an egg are added; the whipped white of an egg is mixed in; the mass is put into a small porcelain mold and baked in a well-heated oven for twenty minutes; sauce from a roast is added. The whole mass equals 215 calories.

(*d*) Sweetbread Pudding (according to Hedwig Hehl).—Twenty-five gm. of French rolls are grated and laid in milk. The sweetbread is cooked, until soft, in bouillon or salt water, skinned, and cut into small blocks. Thirty gm. of butter are stirred until foamy, and two yolks of eggs, the roll which has been pressed out, a little salt, parsley, and the blocks of sweetbread are put into the butter, with which the whipped white of an egg is mixed; the whole is put into a cup well rubbed with butter, covered, and cooked for three-quarters of an hour in the waterbath. Anchovy sauce or meat gravy is added. One hundred gm. equal about 150 calories.

IV. Jellies.

1. *Wiel's Jelly, for Dyspeptics.*—Take off the skin and meat from a calf's foot, mash the bones, and put on the stove with some cold water until it is heated to foaming, when all refuse will be separated. After rinsing off the scum with cold water, put the bones with ¼ of a k. of beef, or ½ of an old hen, and 1¼ liters of water, and five gm. of salt, and boil slowly from four to five hours. Pour the jelly thus formed through a fine sieve, and place overnight in the cellar. Next morning take off the layer of fat, and to clarify the cold jelly add one egg with the mashed shell, and mix with steady beating and stirring. Then subject the whole with constant beating and stirring to a temperature of not over 60° R. (or else the white of the egg will curdle). If the jelly begins to show grains, cover and let cool, until the white of egg becomes flaky and separates itself. Hereupon strain a few times more until it becomes perfectly clear, add five gm. of extract of meat, and pour the jelly into a mold and let cool again. An addition of gravy from a roast is very palatable. It must be mixed in while the mass is still warm and liquid. The dish is very palatable with cold fowl, but does not keep well in summer, and had, therefore, best be put on ice.

2. *Ichthyocol Jelly.*—Cut 15 gm. of ichthyocol into small pieces and let soften in ½ of a liter of cold water for eight to ten hours; boil for one-quarter of an hour and add gravy from a roast and extract of meat. Pour the mass when hot through a fine cloth, or, better, through filter-paper. One can add to 100 gm. of the liquid also 0.5 gm. of hydrochloric acid, or ten gm. of white wine.

3. *Milk Jelly.*—Boil two liters of milk for five to ten minutes with 250 gm. of sugar. To the well-cooled mixture add, while slowly stirring, a solution of 30 gm. of white gelatin in 250 gm. of water, and also add three wineglassfuls (400 gm.) of good Rhine wine, or 30 gm. of cognac; afterward pour the mass into a form and let cool. One hundred gm. equal about 250 calories.

V. Vegetables.

1. *Asparagus.*—The asparagus stems are washed, peeled from the top downward, and the lower woody ends cut off; then they are bound in a small bundle, and cooked until soft in salt water, which requires, according to the thickness of the stems, one-half to one hour; a large quantity of water must be used in cooking, otherwise the asparagus easily takes on an ugly color. Make a butter sauce with yolk of egg. Dyspeptics can take only the soft heads without sauce. One hundred gm. equal about 20 calories.

2. *Spinach.*—The spinach leaves are carefully picked, washed, and laid in boiling salt water, in which they are to be cooked slowly, without being covered, for otherwise they lose their color easily. After twenty minutes put them on a sieve, pour cold water over them and press them. Then cut the spinach very fine or pass through a hair sieve, lay in a little melted butter, dust flour over it several times, and add strong bouillon. At last, mix in the yolk of an egg with cold bouillon. One hundred gm. equal 165 calories (prepared from 250 gm. of spinach leaves).

3. *Comfrey or Bruisewort.*—Wash, clean carefully, cut in pieces two inches long, and also split the thicker pieces lengthwise. Mix one tablespoonful of flour with one liter of water and one tablespoonful of vinegar, and lay each cleaned piece of root in the mixture. Afterward they are again rinsed on a sieve with clean water, laid in melted butter, salted, covered tightly and stewed, adding strong bouillon from time to time. According to size and age the roots require from three-quarters to one and one-half hours in order to become soft. One hundred gm. equal about 120 calories.

4. *Green Peas.*—The peas (½) are hulled and stewed in 15 gm. of butter and bouillon as the preceding ; time, one to one and a half hours. Or, take canned peas and put the opened can in hot water, or cook them with the same amount of butter and some salt. For the sick it is advisable to pass the peas through a sieve and serve them as a purée. One-half of a liter of peas yield 280 gm. of pea purée; of this, 100 gm. equal 160 calories.

5. *Carrots.*—Carrots are serviceable in the dietetic kitchen only when very young and tender. They are cleaned, washed, cut into pieces and then stewed similarly to peas. The time is also the same. If it is desired to serve them as a purée, they are passed through a hair sieve after they are cooked ; a little flour dusted over them and cooked to a thick mush. One hundred gm. of purée equal 120 calories.

6. *Beans (Green).*—Young beans are cleaned, washed, cut fine, and, like the peas, stewed in butter and bouillon. In a season when there are no young, fresh vegetables, one can use to advantage canned beans, of which Prince beans (Flagiolettes) are the tenderest. One hundred gm. equal about 40 calories.

7. *Cauliflower.*—The cauliflower is cleaned, washed, and treated like the asparagus. Time of cooking, one-half hour. One hundred gm. equal about 60 calories.

8. *Rice in Bouillon.*—Thirty gm. of rice are washed twice on the previous evening, and then water in which a little carbonate of soda has been dissolved is poured over it, so that the rice may swell during the night; then the water is drained off, and the rice with a piece of butter and some strong bouillon is put in a stewpan and stewed for one and one-quarter hours, tightly covered, except the last quarter of an hour ; finally the beaten-up yolk of an egg is added. Now rinse out a small porcelain dish with cold water, without drying it, and press the rice into it, let stand five minutes and then turn the mold. The amount is calculated for one person, and is best suited for a side dish to meats. The whole equals about 225 calories.

9. *Chestnut Purée.*—One-half kilo of chestnuts are peeled and boiled in water as long as to get the second (inside) skin off easily. The chestnuts are laid upon a sieve until all the water has drained off. Then they are mashed in a dish and afterward pressed through a hair sieve. One hundred gm. of butter are melted in a stewpan on the fire ; a little salt and sugar, enough to cover the

point of a knife, are added (to the butter), and then the chestnuts are put in. Stew them, with frequent stirring, for one-half hour, and pour in enough bouillon to get a mush not too thick.

VI. Side Dishes from Eggs and Flour.

1. *Scrambled Eggs.*—Two eggs are thoroughly beaten with a little salt until yolk and white are completely mixed. Then melt five gm. of butter in a small enameled vessel, add the egg mixture, and heat, with continued stirring, until a rather thick mush is formed. Serve in a well-warmed dish. This dish is suitable with cold roast, ham, smoked meat, etc. Two scrambled eggs equal about 200 calories.

2. *Potato Purée.*—Peel ¾ of a pound of very mealy potatoes, cut into quarters, wash, and cook until soft in a steam-cooking apparatus; then pass through a coarse hair sieve; add 20 gm. of fresh butter, a little salt, and 60 to 70 gm. of warm milk, and beat thoroughly for five minutes while the mixture is on the fire, until it becomes very foamy. This must only be prepared just before serving, as it loses flavor in standing. One hundred gm. equal about 125 calories.

3. *Suabian Dumplings.*—One hundred gm. of flour, two eggs, two tablespoonfuls of milk, and a little salt are thoroughly stirred together; the dough is put in a special sieve (coarse), through which it is forced and allowed to drop into strongly salted boiling water. One must take a large pot with plenty of water, so that the dumplings may rise better; they are allowed to boil for half an hour. When done they are poured on a large sieve, and remain until all the water has drained off. Meanwhile melt in a stewpan ten gm. of fresh butter, put the dumplings into it, shake them around well, and serve. The sieve necessary is known only in South Germany, but it can be made by any tinner, for it is like an ordinary strainer, the holes having a diameter of one cm. (about ⅔ of an inch). One hundred gm. equal 175 calories.

4. *Roll Dumplings.*—Rolls from the day before are grated (that is, the crust), the inside is cut into slices and cold milk poured over until the bread is thoroughly soft, for which at least an hour is necessary. Meanwhile stir 60 gm. of butter for one-quarter of an hour, and add slowly four eggs and a little salt. Then squeeze the milk out of the slices and stir them with the butter and eggs until finely divided. In order to test whether the mass be of the right consistency, make a lump as large as a walnut and boil in salt water.

If it breaks, a little dust from grated rolls must be added. When the dough has acquired the necessary firmness, make dumplings the size of an apple (about seven from the given quantity of materials). After they have boiled well for one-quarter of an hour in salt water, take them out with a sieve spoon, cut in half, and serve. One hundred gm. equal 250 calories.

5. *Vermicelli (Water Noodles or Vegetable Noodles).*—For the dough take 180 gm. of flour, and three eggs, which are to be mixed with the flour in a dish; then put the dough on a board, and knead well with the hands until it is tender. Then form it in the shape of a long sausage and cut into four equal parts. First take one part: knead into a flat, round cake; weigh off, in addition, 20 gm. of flour; dust the board and rolling-pin with this, and roll out thin (20 gm. of flour will suffice for all the four parts of the dough). At each turning of the dough, dust the board again with flour, so that the dough may not stick and tear. The necessary thinness is reached when one can distinguish through the dough the pattern of a piece of calico, etc., laid underneath. When thin, lay the four parts on a clean white cloth near the fire; let them become half dry, and cut into strips one cm. broad, which are to be separated and hung up in the kitchen to dry for twelve hours. They can be kept for some time in a tureen. When using, lay them for ten minutes in boiling salt water; pour off the water through a strainer, and put the noodles in a dish. Vegetable noodles of very good quality are now also made by factories. One hundred gm. of boiled noodles equal about 190 calories.

6. *Macaroni.*—Buy only the best quality. Put in a vessel with much boiling water, and after they have boiled ten minutes pour off the water; pour over some more boiling salt water and let boil for half an hour. Drain, put in a stewpan with a little butter (which is on the fire), mix, and serve immediately. One hundred gm. equal about 150 calories. One can also, instead of putting hot butter over the macaroni, add a butter sauce, described under "preserved sweetbread." When the macaroni has been drained, put in a porcelain dish, in which it is served, pour the thickish sauce over and put the dish for ten minutes in the oven.

VII. Flour, Milk, and Egg Dishes.

1. *Rice Mush.*—Thirty gm. of rice (Caroline rice is the best) is washed the night before twice, thoroughly; then cold water, in which a little carbonate of soda has been dissolved, is poured over,

and allowed to stand until the next day. Before using, the water is poured off; ½ of a liter of milk is boiled and the rice then added and boiled, well covered up, for one and one-quarter hours on a moderate fire, with frequent shaking. If the milk becomes too thick from boiling before the rice has been thoroughly softened, add a little more hot milk. Whip the whites of two eggs, and just before serving mix lightly with the rice; if it is desired to make it more nourishing, the yolks of the two eggs can also be added before the whites (this quantity, for one person, equals 700 calories). One hundred gm. equal about 160 calories.

2. *Tapioca.*—Boil ¼ of a liter of milk; mix 20 gm. of best imported tapioca and boil for one-quarter of an hour longer, with constant stirring. Further procedure same as with rice (in the same way oat-meal may also be treated). Value in calories of above quantity, about 250.

3. *White Pot.*—Moisten in a small, well-enameled pan 65 gm. of fine sugar with one tablespoonful of water, and burn to caramel sugar. This requires great care, for the sugar easily becomes too dark and then takes on a bitter taste. On a hot stove, not over an open fire, one must constantly stir the sugar with a tin spoon until it gets a fine brown color. During this process heat a tin form, such as are usually used for sweet dishes, jelly, etc., and pour into it the sugar as soon as it has browned, and let it spread on all sides until the surface of the plate is covered. Then let cool. Now beat up three whole eggs in a dish, add ¼ of a liter of unboiled milk, the contents of a package of vanilla, or ¼ of a stick of vanilla boiled in milk, powdered sugar to taste, mix the whole thoroughly, and pour into the form with the sugar, which is now cold. Put on a waterbath, cover, and boil until the mass has amalgamated, which can be tried by thrusting in a teaspoon. Take out the form, allow it to cool, and upset on a plate. This is a pleasant, cooling, well-tasting dish, nourishing as well as easily digestible. One hundred gm. equal about 30 calories.

4. *Egg Crême (according to Mrs. Dr. Pariser).*—For this one reckons, for one person, one yolk of egg, two tablespoonfuls of beaten cream flavored with vanilla, sugar according to taste, a few drops of arrack or cognac. The yolk of egg is first beaten with sugar, to foam. Then the whipped cream is added and well mixed in; at last a few drops of arrack or cognac are added, and the whole served in wineglasses.

5. *Vanilla Crême* (*according to Mrs. Dr. Hughes*).—Stir four yolks of eggs to foam, with ¼ of a pound of fine sugar; boil ¼ of a liter of milk with some vanilla and add immediately to the eggs; mix with an egg-beater and again put on the fire, with continual stirring. Six pieces of white gelatin are dissolved in a little hot water and poured into the mass while the latter is still on the fire. As soon as it is risen, take quickly from the fire, pour through a strainer and nearly allow it to cool, with constant stirring. Then the whipped whites of four eggs are added and the mass poured into a porcelain dish which has been rinsed with cold water; allow it to cool, and turn over just before using. A fruit sauce may be served with the *crême*.

6. *Roll Pudding*.—Stir 30 gm. of butter until foamy, add yolks of two eggs, with one tablespoonful of fine sugar, 15 gm. of grapes, 15 gm. of raisins, and 20 gm. of finely grated almonds. The outsides of two French rolls are grated off, the insides cut in pieces, soaked one hour in milk, and then squeezed thoroughly and mixed with the rest of the mass. Now the whole is thoroughly mixed, the whipped whites of two eggs stirred in, and the dough put into a form rubbed with butter and dusted with roll dust. Either bake the pudding for three-quarters of an hour in the waterbath or bake in a small porcelain dish for one-half of an hour in an oven. Add vanilla or wine sauce. If necessary, the almonds, raisins, and grapes may be omitted. One hundred gm. equal about 250 calories.

7. *Tapioca Pudding*.—Thirty-five gm. of tapioca are cooked for five to seven minutes with ¼ of a liter of milk until it turns to a thick mush. Meanwhile stir to foam 25 gm. of butter; add yolks of two eggs and one small tablespoonful of fine sugar, and stir this mass into the no longer hot, but still warm, mush. After rubbing a small porcelain form with butter, whip the whites of two eggs, add, and mix with the mass. Put into the form and bake the pudding in a well-heated oven for three-quarters of an hour. One hundred gm. equal 175 calories.

8. *Flour Mush Pudding*.—Melt 20 gm. of butter in a saucepan; mix in smoothly 50 gm. of flour and ⅛ of a liter of milk, and cook the mush until it separates from the pan. Then let cool a little, and add afterward one yolk of egg. Now stir until foamy 20 gm. of butter, to which add yolks of two eggs, 1½ tablespoonfuls of sugar and one teaspoonful of arrack, and with this mass mix the cooked mush; then whip the whites of the three eggs, mix lightly

with the mass, fill into a form rubbed with butter and dusted with roll dust, and let cook for one hour in the waterbath. One hundred gm. equal about 220 calories.

9. *Rice Pudding.*—Thirty-five gm. of finest rice is soaked the night before, as in the case of rice mush. Heat ½ of a liter of milk, add the rice; cover and cook slowly until entirely soft; stir 25 gm. of butter to foam, add two yolks of eggs and one tablespoonful of sugar. When the rice has become lukewarm, mix in the other mass; whip two whites of eggs, put the dough in a porcelain form rubbed with butter, and bake in an oven for half an hour. One hundred gm. equal about 150 calories.

10. *Biscuit Pudding.*—Five yolks of eggs are stirred for half an hour with ¼ of a pound of fine sugar; then add a small tablespoonful of fine flour and a little vanilla; also a little arrack and five whipped whites of eggs. Rub a pudding form with butter and dust with fine roll crumbs; fill in the mass and cook for one hour in the waterbath. One hundred gm. equal 215 calories.

11. *Noodle Pudding.*—Of the best egg noodles (fine) take 70 gm., crumble to pieces, throw into ½ of a liter of boiling milk, and boil for half an hour. Meanwhile stir 50 gm. of butter until foamy, add three yolks of eggs and about one tablespoonful of fine sugar, and mix this mass with the half-cooled mush. At last whip the whites of three eggs, mix with the rest, put the whole in a form rubbed with butter, and bake the pudding for one hour in the oven.

12. *Omelette Soufflé.*—Stir the yolk of an egg, with one tablespoonful of fine sugar, for a quarter of an hour; add on the point of a knife a little of the finest flour, one tablespoonful of arrack, and the whipped whites of 1½ eggs. In an omelette pan, melt five gm. of butter, and meanwhile put on the hearth a porcelain soup plate, which must fit the pan exactly, and heat the plate well. Then put the dough in the pan, and cover this immediately with the hot plate. Now bake the omelette with a moderate fire until the surface has become solid, which requires four to five minutes; then turn out on another warmed flat plate; then fold in the middle, strew sugar over, and serve at once. The whole, about 240 calories.

13. *Soufflé Baked in the Oven.*—Stir the yolks of two eggs, with 35 gm. of sugar, for a quarter of an hour; add on the point of a knife a little fine flour, and one tablespoonful of arrack and the whipped whites of two eggs; then at once fill the dough into a

porcelain form rubbed with butter, and bake eight to ten minutes in the oven. The whole, about 370 calories.

14. *Snowballs in Vanilla Crême.*—One liter of milk, with one tablespoonful of fine sugar, mixed, is used in the cooking. The whites of four eggs, with one tablespoonful of sugar, are whipped until stiff. Then from the whipped eggs longish lumps are cut out with a tin spoon and these put into the boiling milk. The milk must be put on the fire in a large, wide can so that the snowballs may expand. One must never put more than six in the pan at one time. When they have lain one minute in the milk, turn them; let them lie another minute on the other side, take them out carefully, and lay on a large platter. After the whole has thus been treated, take the four yolks of eggs, mix with a teaspoonful of fine flour, ¼ of a liter of milk, and a package of vanilla, and make of them a *crême*. When this begins to boil take it from the fire; let it cool, and just before serving place the snowballs upon the *crême* in a porcelain dish.

VIII. Miscellaneous.

1. *Stewed Apples.*—Peel good apples, cut them and stew with a little water and sugar according to taste; then pass through a coarse hair sieve. One hundred gm. equal about 75 calories.

2. *Pears.*—Peel good pears, cut in halves, but do not take out seeds, put on the fire with plenty of water and a little sugar, and boil until soft; a little wine added will improve the palatability.

3. *Wine Sauce or Chandeau.*—Two yolks of eggs are beaten up in a small pan, with ½ of a teaspoonful of the finest potato flour; then slowly stir in ¼ of a liter of good wine and add two to three tablespoonfuls of fine sugar. Put on the fire and stir until the sauce has gained a thick consistency; then immediately take from the fire and cover; now whip two whites of eggs, and pour the sauce into this slowly, with vigorous stirring, and serve at once. Reckoning the value in calories of the alcohol, 100 gm. equal about 110 calories.

4. *Vanilla Sauce.*—Mix two yolks of eggs with one tablespoonful of fine sugar, add ¼ of a liter of cold milk, a little vanilla or ½ of a package of vanillin. The sauce is put on the fire and stirred until it begins to thicken. Then take from the fire immediately and serve. One hundred gm. equal about 125 calories.

5. *Aleuronat Bread (according to Dr. Huth, Aerztl. Centralbl.,*

August, 1894, No. 46).—Mix 500 gm. of aleuronat flour and 1500 gm. of rye flour; mix one-half of this mass with one liter of warm water, two good tablespoonfuls of salt, and 180 gm. of yeast finely divided in a little water; set this dough, sprinkled with a little flour, to rise. After the usual rise, the dough is worked up with the remaining flour into two loaves. These are baked in square pans (10, 15, 20 cm.) rubbed with butter; after letting them rise well once more, they are baked for two hours with strong heat.

6. *Nutritive Enemata:*

(*a*) Meat Pancreas Clyster (according to Leube).—One hundred and fifty gm. of good beef are scraped and then chopped fine; 50 gm. of fresh pancreatic gland, free from fat (either of a cow or of a hog), are mixed with this and stirred carefully, with the addition of not more than 150 gm. of lukewarm water. Injections of from 50 to not more than 100 gm. at a time in a lukewarm state, by means of a simple funnel, ending in a nozzle which must have a wide opening. The mixture will keep only a short while. One hundred gm. equal about 120 calories.

(*b*) Nutritive Enema (according to Ewald).—Two or three eggs are beaten smooth with one tablespoonful of cold water and a little salt, as much as can be held on the point of a knife. Wheaten starch, as much as can be held on the point of a knife, is boiled with ½ of a cup (100 gm.) of a 20 per cent. solution of grape sugar, and one wineglass (150 gm.) of red wine added. Then the solution is cooled to 30° R., and the eggs are stirred in slowly. One can add also one teaspoonful of meat peptone, but this is not absolutely necessary. Nutritive clysters are to be injected while at blood heat, and in quantities of 250 gm. at a time. Previously the rectum must have been cleansed by a purgative clyster. The addition of grape sugar had better be omitted, since through it decomposition and irritations of the intestines arise (Wegele). It contains about 400 calories.*

(*c*) Nutritive Clyster (according to Boas).—Warm 250 gm. of milk, and stir with two yolks of eggs, one teaspoonful of common

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* In calculating the value in calories of the nutritive clysters it is to be noted that the amount of resorption is difficult to determine, since it depends upon the state of the intestines, the skill of the patient in retaining the enema, etc. It is therefore well to assume only one-half as resorbed.

salt, and one tablespoonful of wheaten starch, and afterward add one tablespoonful of red wine. If the mucous membrane of the rectum is easily irritated one may add four to five drops of tincture of opium. Such clysters may be administered from one to four times in twenty-four hours (heated to blood heat), with a long, soft, rectal tube and a Heger's funnel and tubing. Contains about 400 calories.

(*d*) Meat Bouillon-wine Clyster (according to Fleiner).—Consists of 80 gm. of beef-tea and 40 gm. of mild white wine; to be injected two or three times a day at body heat. (According to Fleiner, they bring sleep to weakened patients.)

7. *Alcoholic Pancreas Extract* (*according to Dr. Reichmart.*)—A fresh ox pancreas is freed from fat and skin immediately after killing, chopped up, and ½ of a liter of 12 to 15 per cent. alcohol is poured over. Let stand two to three days in a cool place, and filter. One wineglass for each meal.

The following is a meat food recommended in the absence of secretion of hydrochloric acid:

Meat Dumplings with Sardelle Dressing (*according to Mrs. Dr. J. C. Hemmeter*).—Take ½ of a cup of finely scraped beef, ½ of a cup of lean pork ground through the meat-chopper. Add salt and a small amount of nutmeg; 2⅓ ounces of butter creamed, yolks of two eggs creamed; two ounces of stale bread soaked in cold water; after it is softened press it dry and add to the meat; then add the beaten white of one egg and mix all thoroughly. Turn into 30 dumplings and boil for five minutes.

Dressing.—Take one cup of beef bouillon, add four sardelles scraped fine, the juice of ½ of a lemon, and boil this for ten minutes. Thereafter add ½ of a glass of white wine, one teaspoonful of corn-starch, lastly the yolks of two eggs stirred in a little water; then strain and pour over the dumplings. Serve only in a covered tureen.

Gelatin Cream (*according to Mrs. Dr. J. C. Hemmeter*) *for Anacidity Without Symptoms of Stagnation.*—Juice of two oranges and one lemon, slight flavor of vanilla extract, ¼ of a pound of sugar; stir well and then add one pint of cream and beat until thick. Dissolve ½ of a box of gelatin in ½ of a pint of cold water; heat very gradually until all is thin and dissolved, but not to solidity. When cool add the cream, and beat until it is stiff. Can be poured into a mold and given any shape.

CHAPTER III.

THE DIETETICS OF ALCOHOL AND ALCOHOLIC BEVERAGES.

The literature on the subject of the physiological action and the metabolic and dietetic influences of alcohol is very extensive; its abnormal growth appears to those who make an effort to keep abreast of the progress and advancement of experimental therapeutics, out of all proportion to any real increase in our knowledge of the subject. We are directly concerned only with the (1) value (if any) of alcohol as a food; (2) as a tonic and stimulant; (3) its effects upon the digestive functions. The use of alcohol in any shape is wholly unnecessary for the use of the human organism in health. A large number of persons are undoubtedly wiser and prolong their lives by total abstinence. This may, of course, with certain limitations, be said of all food and drink; but of alcohol it should be published with emphasis, since there are so many who imagine it is indispensable, when in reality they are injured by it. The effects of alcohol on other organs than the stomach are very important; but we must refer to the literature on the special experiments: For the influence of C_2H_6O on the heart see J. C. Hemmeter, "The Comparative Physiological Effects of the Ethylic Alcohol Series on the Isolated Mammalian Heart" (in "Studies from the Biological Laboratory of the Johns Hopkins University," vol. IV, No. 5). On the value of alcohol on various body functions, see Gilman Thompson, "Dietetics," pages 205 to 232; Binz, "Pharmakologie"; Schmiedeberg, "Arzneimittellehre." The literature on the effect of alcohol on the functions of the stomach can be found in the text-books of Riegel, Boas, Ewald, Wegele, Penzoldt (vol. IV of "Handbuch d. Therapie"), Munk, and Uffelmann. The literature is too great and the results are too uncertain to permit any résumé to be given here. The question arises, "Why do we give alcohol in gastric therapeutics? Is it a food or merely a stimulant? In doses taken ordinarily with the more common beverages does it facilitate or retard digestion?" Most of the text-books mentioned take the stand that as

alcohol is oxidized in the body it furnishes a considerable amount of energy.

The question whether alcohol is a true food-stuff, capable of serving as a direct source of energy and of replacing a corresponding amount of fats or of carbohydrates in the daily diet, is a matter of controversy. Reichert (*Therapeutic Gazette*, Feb. 15, 1890) concludes that moderate doses of alcohol do not affect the total amount of heat produced in the body of a dog. As it is nearly completely oxidized in the body, and gives off considerable heat in the process, the fact that the total heat production remains unaltered indicates that the oxidation of alcohol protects an isodynamic amount of food-materials in the body from consumption, thus acting as a food-stuff capable of replacing other elements of the food. Opposed diametrically to these results are those of Miura (*Zeitschr. f. klin. Medicin*, 1892, vol. xx, p. 137), whose observations were made on his own metabolism, after he had brought himself into a condition of nitrogen equilibrium upon a mixed diet. Then for a time a portion of the carbohydrates was omitted, and its place substituted by an isodynamic amount of alcohol. The result was a loss of proteid from the body, proving that the alcohol had not protected the proteid tissue as it should have done if it acts as a food. In a third period the old diet was resumed, and after nitrogen equilibrium had again been established, the same proportion of carbohydrates was omitted from the diet, but alcohol was not substituted.

When the diet was poor in proteid, it was found that less proteid was lost from the body when alcohol was omitted than when it was used, indicating that so far from protecting the tissues of the body by its oxidation, the alcohol exercised a directly injurious effect upon proteid consumption. In our opinion, the effect of moderate quantities of alcohol upon metabolism is not yet satisfactorily understood, and positive statements can not be made until reliable experimental results have accumulated.

Concerning the effects of moderate amounts of alcohol on digestion by pepsin-hydrochloric acid, and on salivary and pancreatic digestion, we believe the following abstract of Chittenden's experiments (*American Journal of Medical Sciences*, Jan. to April, 1896) to be a clear representation of this matter.

A careful study of Chittenden and Mendel's results (Chittenden and Mendel's paper upon "The Influence of Alcohol upon the Chemical Processes of Digestion") makes it evident: That we

can not define with mathematical exactness the action of a given percentage of absolute alcohol on pepsin proteolysis, since variation in the attendant conditions, *i. e.*, the relative amounts of pepsin, acid, and proteid, together with the period of digestion, the digestibility of the particular proteid, etc., are prone to modify the final result. Thus, with a weak gastric juice, where the amount of ferment present is small, and digestive action consequently slow, or where the proteid material used is difficult of digestion, the retarding effect of a given percentage of alcohol is far greater than when the digestive fluid is more active; that is, when it contains more pepsin. Further, this difference of action is more pronounced the larger the percentage of alcohol present. The following general conclusions were drawn from artificial digestive mixtures.

First. It is plainly manifest that in the presence of small amounts of alcohol (one to two per cent. of absolute alcohol), gastric digestion may proceed as well or even better than under normal circumstances. In fact, many of their experiments show a slight increase in digestive power when the mixture contained one or two per cent. of absolute alcohol. This increased digestive action, though slight, occurred too frequently to be the result of mere accident, and apparently indicates a tendency for alcohol, when present in small quantity, to increase slightly the digestive action of pepsin-hydrochloric acid; or, in other words, to stimulate the ferment so that it can accomplish somewhat more, under given conditions, than it otherwise could do. As the percentage of alcohol is raised, retardation or inhibition becomes more noticeable, although ordinarily it is not very pronounced until the digestive mixture contains five to ten per cent. or more of absolute alcohol. With 15 to 18 per cent. of absolute alcohol digestive action may be reduced one-quarter, or even one-third; the exact amount of retardation, however, being especially dependent upon the strength or activity of the gastric juice and upon the natural digestibility of the proteid material. It is to be remembered, however, that 18 per cent. of absolute alcohol would be equivalent to 36 per cent. of proof-spirit, so that if we could assume the contents of the human stomach at a given period to be one-third proof-spirit, it might perhaps be considered that digestive action would be retarded to the extent of 25 to 35 per cent., provided the gastric juice present in the stomach was of fair strength and the proteid matter of ordinary digestibility. Such percentages of proof-spirit, however, are not likely to be

long present in the stomach, and it is perhaps idle to speculate on such hypothetical cases. We may in this connection, however, again emphasize the fact that the stronger the gastric juice, and the more digestible the proteid food undergoing digestion, the less retardation will a given percentage of alcohol produce; while, on the other hand, the weaker the gastric juice and the more indigestible the proteid, the greater will be the inhibition caused by a given percentage of alcohol. In other words, those variations which must naturally exist in the stomach contents of different individuals, both in health and disease, will lead to different degrees of retardation in the presence of given percentages of absolute alcohol. It would, therefore, be unwise to make a general specific statement regarding the action of a given percentage of alcohol. Under definite conditions, however, as Chittenden's experiments plainly show, the presence of a definite amount of alcohol always leads to essentially the same results.

In order to prevent any misinterpretation of these results, we would again call attention to the fact that we are dealing here with only one of the four questions that need to be answered before we can hope to fully understand the influence of alcohol on gastric digestion as a whole. Thus, the results afford plain evidence of the influence of alcohol on the digestive or solvent power of the gastric juice; but we should not be justified in arguing that exactly the same results would follow from the introduction of alcohol into the living stomach. The action of a given percentage of alcohol on proteolysis alone would be essentially the same in the stomach as in a beaker, provided the alcohol was not absorbed into the blood and thus removed from contact with the digestive mixture, and provided it did not exert any influence on the character of the gastric juice secreted. But it is easily conceivable that a percentage of alcohol which does not interfere with solution of the proteid food-stuffs may so modify the amount or character of the secretion that digestion might be greatly stimulated or greatly retarded. Further, as already stated, the presence of alcohol in the stomach may so affect absorption and peristalsis that the rate of digestion may be modified from this cause; hence, the results above recorded are to be used only in drawing conclusions as to the effect of various percentages of alcohol on the purely chemical process of gastric digestion, *i. e.*, on pepsin-proteolysis.

In conclusion, it is to be noted that Chittenden's results are more

or less in accord with what has been previously published concerning the action of alcohol on gastric digestion. Thus, Bikfalvi found, in artificial digestive experiments, that alcohol, even in small quantities, retards normal gastric digestion. Klikowicz found that the presence of five per cent. of alcohol in the digestion of egg- and serum-albumin led to somewhat variable results, although, as a rule, there was an indication of a slight stimulation of proteolytic action. In the presence of ten per cent. of alcohol there was always marked retardation, while 15, 20, and 30 per cent. of alcohol checked digestion to a marked degree.

Roberts found by artificial-digestion experiments that in the presence of less than ten per cent. of proof-spirit there was no appreciable retardation. With ten per cent., retardation was only barely detectable. With 20 per cent. there was quite distinct, but still only a slight, retardation. Above this point, however, the inhibitory effect of alcohol increased rapidly. (Refer to the tables of Roberts at the end of this chapter.) That the action of a digestive ferment may be both stimulated and retarded by the same substance, according to the quantity present, has been already demonstrated; hence there is no inconsistency in the above results with alcohol. The same action has likewise been observed with yeast-cells.

Action of Alcohol on Pancreatic Digestion.—In view of the position which pancreatic digestion occupies in the digestive process, it is readily seen that it is more desirable to ascertain the influence of small quantities of alcoholic liquors than large amounts; since absorption must naturally lead to a decided diminution of alcohol, etc., before these fluids can normally become mixed with the pancreatic juice and partially digested food-material in the small intestine. Hence, more stress was, as a rule, laid upon the influence of small percentages of the various fluids experimented with, and only occasionally the action of large quantities was tried.

The results with absolute alcohol indicate that the proteolytic ferment of the pancreatic juice is more sensitive to absolute alcohol than the ferment of the gastric juice. Retardation of digestive action is more pronounced, even with small amounts of alcohol. Further, as in the case with pepsin, the weaker the digestive powers of the pancreatic juice, the greater the retarding action of absolute alcohol. When the amount of alcohol present in the

digesting mixture is less than one per cent., the retardation of the digestive action is very slight, provided the ferment is fairly vigorous in its action.

Action of Alcohol on Salivary Digestion.—(*Methods Employed*).—In the first set of experiments on salivary digestion Chittenden determined the time it took to reach the achromic point. By this method he found it to be manifest that absolute alcohol has very little influence upon the amylolytic or starch-digesting power of neutral saliva. Only when the saliva, added to the digestive mixture, is diluted in the proportion of 1 : 30, does the presence of even ten per cent. of alcohol have any measurable influence, and then only to retard the appearance of the achromic point two minutes. As this percentage of absolute alcohol is equal to at least 20 per cent. of proof-spirit, it follows that pure alcohol, free from admixture, is practically without influence upon the digestion of farinaceous food by the saliva.

By the second method, which was to determine the amount of maltose formed, he found that small amounts of absolute alcohol may actually cause an increased formation of maltose. On the other hand, the presence of 10 or 15 per cent. of absolute alcohol leads to a distinct retardation in the formation of sugar, although the inhibition is not very pronounced considering the amount of alcohol present. This retardation of the secondary action of the ferment is perhaps suggested by the slight delay in the appearance of the achromic point in the presence of ten per cent. of absolute alcohol.

Concerning the effect of alcohol on the *gastric motility*, we have personally made a number of observations on three healthy students with normal stomachs by means of our method of graphically registering the gastric peristalsis on the kymographion (*New York Medical Journal*, June 22, 1895). These students were teetotalers, and to exclude the influence of suggestion the alcohol was poured into the stomach, diluted, through a tube, and sometimes water was used in place of alcohol. The subject was at no time aware of what was being used. It was found that alcohol, when contained in gastric contents up to six per cent., exerts no appreciable effect on the motility one way or the other; but beyond this the peristalsis begins to be interfered with.

The presence of 20 to 25 per cent. of alcohol leads to a very distinct retardation and reduction of the tonicity in the gastric

movements, which seems to last in its effects from two to three hours. Even after the alcohol is thoroughly washed out of the stomach the peristalsis continues to show itself embarrassed if the amounts mentioned have been used.

In dogs the identical results were obtained, except that at five to six per cent. a short period of peristaltic unrest is noticed before the marked inhibition develops. The inhibition of the peristalsis when the gastric contents contain 20 to 25 per cent. of alcohol occurs quite regularly, and is not the result of mere accident. It is probably due to a direct poisoning effect on the muscularis, similar to the poisoning effect on the heart muscle observed by us (Hemmeter, "Studies from the Biological Laboratory," *loc. cit.*). This amount of alcohol must be in the organ at least ten minutes before the peristaltic inhibition sets in.

The effect of alcohol on the *rate of absorption* from the stomach is a different question from the absorbability of the substance itself. There is a general unanimity that, owing to its rapid diffusibility, alcohol is promptly absorbed from mucous surfaces. At the same time the experiments of von Mehring suggest that the absorption of substances soluble in alcohol may be facilitated by the latter; for example, that peptone or maltose taken in alcohol may be absorbed more rapidly than when taken in water. Exact experimental facts concerning this matter are wanting.

In large doses alcohol hinders absorption by the direct damage it does to the cylindrical surface epithelium.

Among gastro-enterologists the impression prevails that alcohol and alcoholic beverages are capable of promoting the appetite; and probably for this purpose and for its stimulating effect we are justified in giving it. Summing up the physiological action, as far as we are concerned, it may be said: (1) The effect of moderate doses of alcohol on metabolism is uncertain. (2) That on pepsin hydrochloric acid it acts favorably in quantities equal to one to two per cent. of absolute C_2H_6O, but beyond that it gradually inhibits this action. (3) On pancreatic digestion it acts unfavorably. (4) On salivary digestion it acts favorably, increasing the formation of maltose when present in amounts not exceeding five per cent. (5) On the peristalsis it has no influence one way or the other until the amounts exceed six per cent., when it begins to inhibit the motility. (6) Its effect on the rate of absorption is unknown.

In pathological conditions the effects of alcohol are undeniably

different, its stimulating and temperature-depressing influence making it of value in continued fevers. In pathological cases, wherever the amount of free HCl is altered, either in hyperchylia or achylia gastrica, Chittenden's deductions do not hold good. In hypochylia or subacidity, alcohol may be of some service in stimulating the mucosa to more prolific secretion, but in hyperchylia it irritates the already very much excited gland-cells still further. In achylia with entire absence of secretion, digestion is considerably reduced by alcohol. Speaking generally, alcohol might be dispensed with as a therapeutic and dietetic agent if it were not for its appetizing and stimulating qualities. In hyperacidity and hypersecretion, in ulcer and all chronic affections with augmented secretion, alcohol is contra-indicated. Healthy stomachs not rarely exhibit a certain adaptation to alcohol, and, naturally, upon such organs the agent has a different effect than upon the stomach of a teetotaler.

Beer has very little therapeutic utility; by reason of its weight it is contra-indicated in all conditions weakening the gastric wall. Riegel holds that it is well to permit its use in simple hyperacidity. It contains a certain amount of nutritious matter, and should, therefore, not be forbidden if the patient craves it, provided the motor power is good. Even the absorption of less bulky wines is attended by an excretion of water into the stomach (von Mehring, *loc. cit.*), which may favor stasis of liquids and furtherance of existing dilatation.

The following is a table by Roberts ("Lectures on Dietetics and Dyspepsia"), showing the effects of various percentages of malt liquors on gastric digestion:

Proportion of Malt Liquors in the Digesting Mixture.	Time in which Digestion was Completed. (Normal, One Hundred Minutes.)		
	Ale, Burton.	Light English Table Beer.	Lager Beer.
Ten per cent.,	115 minutes.	100 minutes.	100 minutes
Twenty per cent.,	140 "	115 "	115 "
Forty per cent.,	200 "	140 "	140 "
Sixty per cent.,	Embarrassed	180 "	180 "

The digesting mixture contained two gm. of dried beef fiber, 0.15 per cent. of hydrochloric acid (HCl), and one c.c. of glycerin, extract of pepsin, and varying quantities either of wines or malt liquors, and filling up to 100 c.c. with water.

The following table gives the effects of various percentages of hock, claret, and champagne upon peptic digestion (Roberts):

Proportion of Hock, Claret, or Champagne in the Digesting Mixture.	Time in which Digestion was Completed. (Normal, One Hundred Minutes).		
	Hock.	Claret.	Champagne.
Ten per cent.,	100 minutes	100 minutes	90 minutes
Twenty per cent.,	115 "	140 "	100 "
Forty per cent.,	150 "	180 "	130 "
Sixty per cent.,	Embarrassed	Embarrassed	180 "

In cases of gastric disease where great general debility commands liberal alcoholic stimulation, particularly if the gastric motor function be impaired, it is best to administer the stimulant by rectal enema. It may have been observed that most of the enemata have provided for this emergency, and contain more or less wine.

Sir William Roberts has a very interesting theory concerning the probable utility of retarding digestion by alcohol:

"We must bear in mind that among civilized races the preparation of food for the table is carried to a high degree. The cereal grains which are employed to make bread are first finely ground and sifted from the bran by the miller; the flour is then subjected, with the aid of moisture and artificial heat, to a cooking process; the meats and fish we eat are boiled or roasted; the vegetables we use are carefully deprived of their coarser parts, and are then boiled. All this preliminary preparation and cooking renders our food highly digestible and easy of attack by the gastric juices. But this is not, I apprehend, the sole object in view. The preliminary preparation and cooking not only renders our food more digestible, but makes it also more capable of being more thoroughly exhausted of its nutritive qualities. These two objects are not quite the same. Even as it is, and with all this careful preparation, some waste occurs; and the feces always contain considerable remnants of undigested food. But it is obvious that, if food be rendered too easy of digestion, there arises a risk that the meal will pass too quickly and wastefully into the blood, and on through the tissues into the excretory organs, and so out of the body before it has been made fully and economically available for the sustenance of the slow nutritive processes. Moreover, a sudden irruption into the blood of large quantities of newly digested aliment would tend to disturb

the chemical equilibrium of that fluid, and so interfere with the tranquil performance of its functions. It would also tend to produce hepatic and other congestions, to the general disadvantage and discomfort of the economy. A too rapid digestion and absorption of food may be compared to feeding a fire with straw instead of with slower burning coal. In the former case it would be necessary to feed oftener and oftener, and the process would be wasteful of the fuel; for the short-lived blaze would carry most of the heat up the chimney. To burn fuel economically, and to utilize the heat to the utmost, the fire must be damped down, so as to insure slow as well as complete combustion. So with human digestion our highly prepared and highly cooked food requires, in the healthy and vigorous, that the digestive fires should be damped down in order to insure the economical use of food.

"In the plan of the dietary of the civilized races, arrived at slowly as the result of an immense experience, we seem, therefore, to detect two apparently contradictory aims,—namely, on the one hand, to render food, by preparation and cooking, as digestible as possible; and, on the other hand, to control the rate of digestion by the use of certain accessory articles with food. In reality these objects are not contradictory but co-operative to a beneficial end. For, to express the problem in another way, it may be said that we render food, by preparation, as capable as possible of being completely exhausted of its nutrient properties; and, on the other hand, to prevent this nutrient matter from being wastefully hurried through the body we make use of agents which abate the speed of digestion. This combination of appliances renders our plan of feeding more elastic, more adaptable to variety of individual health and constitution, and to variety of external conditions.

"During the early periods of life retardation of digestion is less required than in the adult state, because the growing organism can more fully utilize, in the work of the building up of the framework, any excess of food which is poured into the blood. Accordingly, we observe that retarding agents (tea, coffee, and alcoholic beverages) are not used at all, or only used sparingly, in the diet of infants and children.

"If this view of digestive retardation in the stomach be well founded, the stomach becomes in some degree a storage organ for food,—like the crop of birds, the paunch of ruminants, the dilatable cheeks of monkeys, and the pouch of the pelican."

This classical writer on dietetics expresses himself similarly on the importance of preparing the food in such a way that it tastes good (Sir William Roberts, *loc. cit.*, "The Eulogium of the Palate"). Even Bunge, the well-known physiologist, who is a pronounced teetotaler, declares that we are justified in the use of any food or drink if for no other reason but that it tastes good, provided it does no harm.

CHAPTER IV.

LAVAGE AND THE GASTRIC DOUCHE.

The technics of lavage—the indications for and against it—have been treated in the section on the Stomach-tube. In brief, lavage is indicated, (*a*) where the exit of the chyme from the stomach is hindered by a mechanical obstruction, giving rise to decompositions. To this class belong all forms of dilatation except those depending on simple atony, for here we are not dealing with any obstruction to the outflow, but with a lowering of the peristalsis, which is not markedly benefited by lavage. Dilatations that indicate lavage are those due to cicatricial stenosis, or neoplasm of the pylorus and duodenum, and impairment of motor function in consequence of carcinoma, sarcoma, syphilitic and tuberculous gastritis, atrophic gastritis, myasthenia, corrosive contractions caused by acids and alkalies. The benefit derived from lavage must vary with the stage at which the treatment is undertaken. In cases of cicatricial stenosis of mild and incipient character, the dilatations have been cured by lavage, probably because a compensatory hypertrophy of the musculature, developing gradually, enabled the organ to expel the chyme. In these gastrectasias, the stomach, after the systematic lavage treatment, no longer contained food and HCl in large quantities in the morning. The stools and the quantity of the urine became normal, and the patients could tolerate an ordinary diet. But such cases must, even after recovery, avoid overloading the stomach, as this has been known to bring about relapse.

(*b*) The second main indication is where foreign or irritating collections are mixed with the gastric contents, which sooner or later interfere with digestion. These collections may consist of abnormally augmented gastric juice, of gastric, pharyngeal, and esophageal mucus, and of bile. In hypersecretion lavage is best carried out with sodium bicarbonate, and thereafter with argentic nitrate or bismuth subnitrate. The pyrosis, constipation, and distention are much relieved thereby. In cases of much accumulation of mucus, warm alkaline and saline solutions are preferable; for in

the gastritis mucosa the HCl secretion is lost, and common salt is a stimulation to that secretion—if there be any secreting glands left. Toxic products of complex nature may accumulate in the organ, in consequence of carcinoma, uremia, and diabetes mellitus; here lavage is also indicated. During the lavage one should always have a second glass vessel holding one liter, into which the outflow discharges, so that it may be ascertained each time how much is regained. It is very dangerous to wash out the stomach with medicated and antiseptic solutions without ascertaining whether all the solution flows out again. Even with simple water, overloading of the stomach can not be avoided except by measuring the outflow. In a paper published in the *Practitioner* (1892, No. 4), W. Soltau Fenwick reports three cases of poisoning from leaving antiseptic solutions in the stomach. In one case (*Schmidt's Jahrbücher*, 1883, Bd. CXCVIII, p. 28), death was caused in six days by leaving a two to three per cent. solution of boric acid in the stomach. Fenwick also makes a strong plea against the indiscriminate use of antiseptics within the stomach, for the alimentary canal is "endowed with the power of absorbing not only the poisonous products of the bacteria, but also most of the substances which are introduced to destroy them" (W. S. Fenwick, "Disorders of Digestion in Infancy," etc., p. 141).

Tetany has been observed after lavage by Bouveret and Devic, who collected 21 cases (*Revue de Méd.*, February, 1892). In all, 34 such cases of tetany of gastric origin have been reported, though not all due to lavage. Ewald reported two cases that are of interest in this connection,—one a male, aged forty-five, who died from a sudden, copious, esophageal hemorrhage two days after he had sought relief by introduction of the esophageal sound. There were symptoms of mediastinal tumor or aneurism. The second patient died suddenly while he was introducing the tube himself; the autopsy showed a dissecting aneurism at the beginning of the ascending aorta, still within the pericardium. Frerichs and Penzoldt have reported similar cases. Moral: Examine every new patient carefully for abnormal conditions within the thorax before lavage.

The Gastric Douche.—By douching the stomach is meant an internal irrigation with water under high pressure. It was first practised at Kussmaul's clinic, and described later by Malbranc ("On the Treatment of Gastralgias by the Internal Gastric

Douche," etc., *Berlin. klin. Wochenschr.*, 1878, No. 4). It does not differ essentially from ordinary lavage except in the fact that the funnel or vessel into which the water is poured is held at least one meter above the cardia. Rosenheim improved and revived the method, after it had been disregarded for twelve years, by devising a special douching tube with numerous very small, lower openings instead of one or two large ones. Water that is allowed to run into the stomach through such a tube under high pressure, strikes the walls in the form of a drizzling rain, with many currents of considerable impetus. The central or terminal opening in Rosenheim's douche tube is larger than the lateral ones, and permits of an easy outflow. Dr. F. B. Turck, of Chicago, has devised a stomach needle douche with a separate outflow tube; it also produces an intragastric shower (Fig. 26).

FIG. 26.—RECURRENT GASTRIC NEEDLE SPRAY OR DOUCHE.

Rosenheim recommends the douche for nervous dyspepsia and chronic gastritis, with or without impaired motility. If the douching was done with solution of sodium chlorid an increase in the HCl production could be ascertained; whereas nitrate of silver caused a reduction of the secretion (*Berlin. Klinik*, 1894, Heft 71). Riegel speaks well of argentic nitrate applied in this manner for all irritative states of secretion (*loc. cit.*, p. 300).

Fleiner has called attention to the fact that he and Kussmaul could incite a feeling of hunger by these douches. These clinicians increased the effect by irrigating the gastric mucosa with solutions of bitter tonics, of which extracts or solutions of hops and quassia were experimented with.

In severe cases of anorexia we have tried this method with infusions of gentian and cinchona, and were pleased with the effects. Einhorn has invented an intragastric spray (Fig. 27), which is recommended for disinfection of the mucosa, to produce an astringent or an anesthetic effect. Upon Einhorn's solicitation we experimented with a spray presented by him, and have never since been without one. It is surprising what a trifling amount of cocain is necessary to relieve a gastralgia when used in this manner. For

gastric erosions the nitrate of silver spray (1 : 1000) is frequently curative. The gastric douche and spray should only be applied in an empty stomach. Motor impairment of nervous origin is occasionally much improved by alternately douching with warm (100° C.) and cold water.

Electricity in the Treatment of Gastric Diseases.—The effect of electricity on the various functions of the stomach have been already referred to under the consideration of the motor function and will be further described under the various diseases in which it is recommended. The results of physiological experiment and of clinical experience are largely contradictory. Physiological experiments, when conducted by medical men, are frequently inexact and misleading. It requires special physiological laboratory training of years to control the technics of vivisection. In the experiments of medical

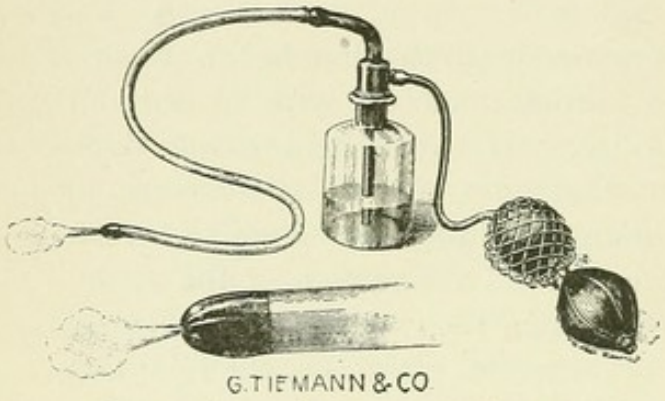

Fig. 27.—Einhorn's Intragastric Spray.

men on the physiological effects of electricity it is not difficult to find numerous defects in the physics and physiology of the methods used, the conduct and the execution of the experiment, etc., which render their results invalid from the outset; so that it is useless to go into the literature of the history of gastric electrotherapy exhaustively. Many experimenters fail to give the details and conditions, the kind of cell used, the number of milliampères, the number of faradic stimulations to the minute, the kind of electrode, the distance of primary from secondary coil. Control experiments are wanting to ascertain whether, in the same animal, peristalsis could not be observed *per se* without stimulation by electricity, or whether the stimulation may not have been purely mechanical, not electrical.

From a clinical standpoint it is not necessary to demonstrate that electricity can produce changes in the chemistry, resorption,

and motility of the stomach in order to justify its employment; for there may be, and probably are, influences exerted by electricity upon the nutrition of living cells which as yet escape our methods of analysis. The effect of electrical stimulation of the cells of spinal ganglia, as seen and determined by micrometric measurement, and consisting in a loss of bulk mainly in the nuclei, was first described by C. F. Hodge, in the *American Jour. of Psychology* for May, 1888, and May, 1889. Judging from these experiments, which were conducted with exemplary accuracy and regard for physiological detail, it is reasonable to presume that, in some way or other, the metabolism of muscle-, gland-, and nerve-cells of the stomach may be influenced by electricity. The demonstration of this is a future prospect; at present the main reason we employ this agent is simply because we know that in certain diseases it does good. Physiology may come to our aid later on, and tell us why it is that these results are produced. From the work done by medical physiologists so far, no clear deductions are possible.

The electrical stimulation of the vagus in a subject, forty-five minutes after execution, which was carried out by Beynard and Loye (*Progrès Médicale*, 1885, No. 29), and produced a secretion of gastric juice, has strengthened the belief that the vagus contains gastric secretory fibers. Ziemssen (*Klinische Vorträge*, No. 12, 1887), Rossi (*Lo Sperim.*, 1881), and Hoffmann, who experimented at Riegel's clinic (*Berlin. klin. Wochenschr.*, 1889, No. 12), all arrived at the conclusion that electricity promoted the secretion of gastric juice.

The results of investigations concerning the influence of the two kinds of currents—the constant and the interrupted—differ widely. Einhorn was of the opinion that the faradic current promoted secretion and the galvanic impeded it (*Deutsche med. Wochenschr.*, 1893; also *Zeitschr. f. klin. Med.*, Bd. XXIII). The experiments of Hoffmann (*loc. cit.*) suggested that the galvanic current favored an increased secretion, while Bocci's results with intragastric faradization would have us believe that in animals the interrupted current can augment both peristalsis and secretion (*Lo Sperimèntale*, Giugno, 1881).

Concerning the effect of electricity on the motor function we might quote a few experimenters. Schillbach (*Virchow's Arch.*, Bd. CIX, p. 284) produced strong contractions at the site of the anode by applying the galvanic current to the intestines of a rabbit.

Von Ziemssen (*loc. cit.*), Bocci (*loc. cit.*), Ludwig, and Weber have stated that the faradic as well as the galvanic current applied directly to the stomach cause contraction of the same in animals. Fubini (*Centralbl. f. med. Wiss.*, 1882, No. 33) concluded that electricity accelerates intestinal peristalsis; he experimented on Vella's double intestinal fistula. Two Americans, Rockwell and Beard (*Philadelphia Medical and Surgical Report.*, 1868, No. 20), were among the first to employ electricity in the treatment of nervous dyspepsia.

In Pepper's case of spontaneous, visible, gastric peristalsis (*Phila. Med. Times*, 1871, p. 274) no peristaltic movements could be produced by applying electricity percutaneously. Kussmaul, in 1877, stated that "the therapeutic results obtained by Fuerstner in gastrectasias did not prove that an actual peristalsis was produced by the current, but they were probably due to contraction of the abdominal muscles" (*Archiv f. Psychiatrie u. Nervenkrankh.*, 1878, p. 205). Canstatt first suggested treating dilatations by placing one electrode in the esophagus, the other on the stomach; and Duchenne first actually applied this method (both the latter quoted from Kussmaul (*loc. cit.*). In 1877, Kussmaul (*loc. cit.*) began introducing intragastric electrodes, made by inserting a copper wire in a stomach-tube, the wire terminating in a little exposed knob which came in direct contact with the inner gastric surface.

Bardet improved this method by a similar electrode, which, however, did not come in contact with the mucosa in one spot only, but by a quantity of water previously taken it was distributed over the entire surface (Bardet, *Bull. Gén. de Therap.*, 1884). Ziemssen then employed a similar device, but Einhorn completed the evolution of the intragastric electrode by originating a soft, very plastic, deglutable instrument, the end of which is inclosed in an ovoid, perforated, hard-rubber cap. Ewald prefers Einhorn's electrode a little more rigid, so that it can be pushed into the stomach and need not necessarily be swallowed. The thickness of the rubber tube in Ewald's modification is 1½ mm. Rosenheim (*loc. cit.*), Wegele (*Therap. Monatshefte*, April, 1895), Charles G. Stockton ("A New Gastric Electrode," *Medical Record*, Nov. 9, 1889), and F. B. Turck have later devised electrodes for this purpose which represent no advance over those mentioned. The thin-wired electrode of Einhorn (Fig. 29) possesses the advantage that it

can be swallowed by those not used to the stomach-tube, to which they must become accustomed in case Wegele's, Stockton's, or Rosenheim's electrode is employed. The inclosing tube of Einhorn's instrument is really too thin, however, for in our experience it rapidly wears through near the connection with the hard-rubber end-cap, and we consider Ewald's modification safer and more durable.

For more complete literature of the subject the reader is referred to the writings of Kussmaul (*loc. cit.*), Einhorn (*loc. cit.*, and Einhorn, *Berlin. klin. Wochenschr.*, 1891, No. 23; also *Zeitschr. f. klin. Med.*, 1893, XXIII, p. 369), and Goldschmidt (*Deutsches Archiv f.*

FIG. 28.—RECTAL ELECTRODE.

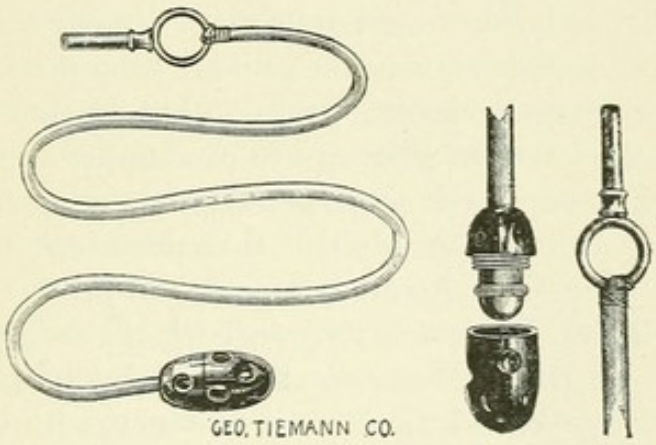

FIG. 29.—EINHORN'S INTRAGASTRIC ELECTRODE.

klin. Med., Bd. XV, p. 295). The latter investigator worked under Moritz, whose capital experiments on the motility we have already abstracted. Goldschmidt concludes that the "direct faradization and galvanization of the stomach (distance of primary from secondary coil = zero, duration fifteen to twenty-five minutes) has only an unimportant and inconstant influence on the peristalsis, and on secretion it has no influence whatever."

These results correspond in the main with those previously published by Meltzer (*New York Med. Jour.*, June 15, 1895), whose experiments were conducted with great care, and from a physiological aspect are beyond reproach. We would have pre-

ferred knowing how many vibrations to the second Meltzer used, since we have assured ourselves that when too many stimulations to the second are thrown into a muscle, particularly an involuntary muscle, it will not contract at all; whereas the same muscle will contract if a smaller number of stimulations be used (judged by the Kronecker interrupter and a Jacquet chronograph). These facts were first stated in an article in the *New York Med. Jour.* (Hemmeter "Recording Motor Functions of the Stomach," *New York Med. Jour.*, June 22, 1895, p. 772). We used an intragastric deglutable rubber bag (see illustrations, Plate IV), which had small brass knobs extended at any desirable location, and when the bag was distended by blowing it up within the stomach, the end electrodes pressed directly against the mucosa,—usually one at the pylorus and one in the fundus; the bag was in connection with a tambour or manometer recording on the Ludwig kymographion. We have already briefly stated that we were unable to produce any contraction of the human or animal stomach with the strongest currents to be obtained from one Grove's cell prepared anew for each experiment, and the distance of the primary from the secondary coil equal to zero when both electrodes were within the stomach touching the mucosa. We have elsewhere given our studies on the resistance which fresh human gastric mucosa offers to the constant current, and in the main can confirm Meltzer that percutaneous and direct faradization of the stomach and intestines can produce no contraction of these parts. Not every current which, according to magnetic needles or the milliampère meter, actually penetrates the gastric wall, causes contraction. For instance, with one electrode within the stomach of man or dog, and another on the gastrocnemius, the skeletal muscle may contract vigorously and the stomach remain passive. Again, in human subjects, the factors of natural peristalsis occurring under the nervous tension due to the experiment, and of suggestion, can not be satisfactorily eliminated.

In a recent publication Einhorn has attempted to disprove the experiments of Meltzer, but as far as can be judged from the report of the former (in the *Archiv f. Verdauungskrankheiten*, von Boas, Bd. II, p. 454), the experiments were not conducted along the same lines nor with the same regard for physiological detail as those of Meltzer. Einhorn gives brief synopses of 18 experiments, 12 of which were made with frogs, which Meltzer did not work with, and from the results of which conclusions regarding the mamma-

lian stomach are not applicable. Three animals were rabbits; in these the stomach is always full of ingesta, unless starved. Two were rats; one only was a dog; this last was the animal with which Meltzer mainly worked. Nor is it evident that Einhorn's results, as stated by him, contradict those of Meltzer in salient points. Taking, for instance, the last experiment—No. 18—with the dog; here Einhorn made three kinds of stimulations with the double electrode: (1) on the serous (peritoneal) surface, near fundus—contraction; (2) on the peritoneal surface over the pylorus —strong contraction; (3) opening of the stomach,—one electrode against the mucosa, the other on the peritoneal layer outside; a weak current causes slight peristaltic contractions.

Meltzer does not, in his original paper, deny any of these possibilities; even the contraction of the third stimulation experiment was witnessed by him when the inner electrode on the mucosa was placed near the outer one on the serosa. But with bipolar internal stimulation—*i. e.*, with both electrodes on the mucosa—even Einhorn does not claim to have obtained any peristalsis of considerable tonicity. *We incline to the opinion* that *satisfactory* evidence has *not* yet been furnished that internal electric stimulation can influence secretion or motility either way. This conclusion has been reached after years of experimenting on both functions in the Biological Laboratory of the Johns Hopkins University.

In a recent reply to Einhorn's criticism, Meltzer accepts the explanation of the former, concerning the difficulty of penetration of the electric current to the muscular layer (*Boas' Archiv f. Verdauungskrankh.*, Bd. III, Heft 2, S. 133). This may be caused, according to Einhorn, by the mucosa being a bad conductor as well as by its being a very good conductor—leading the current away from the point of contact. We have shown conclusively that the fresh normal human mucosa is a poor electric conductor. Einhorn, however, assumes that the mucosa conducts so well that the currents does not reach the muscularis, because it moves in the direction of least resistance—in the glandular layer itself. Tests made with the mucosa peeled off from the other layers of the stomach of a dog under narcosis, with the milliampèremeter in the circuit, show that fresh mucosa is practically a non-conductor.

Indications for the Employment of Electricity and Manner of Application.—Direct gastric faradization is recommended for dilatations

due to relaxation of the musculature, but not to stenosis, whether these cases are associated with reduced secretion or not. Relaxations of the cardia or pylorus are benefited by the faradic current. Sensory disorders (gastralgia) are successfully treated with direct galvanization. To these statements we can give our approval, although other clinicians differ slightly from Einhorn. Thus, Rosenheim (*loc. cit.*) believes that the galvanic current is more effective in debility of the peristalsis. In all symptoms of sensory irritation he prefers the constant current also, but in secretory disturbances he has ceased to use electricity; and we agree with him that, in the latter class, more can be accomplished by medicated douches, and adapted acid or alkaline and bitter tonic medicines, than with electricity. Brock confirms the good effect of galvanism on the course of gastric neuroses (*Therap. Monatshefte*, June, 1895), though he is not so enthusiastic as Einhorn.

According to Goldschmidt, there are no distinct differences between the effects of direct galvanization and direct faradization; but nevertheless he recommends the former for the painful, the latter for the functional disturbances of the stomach. In contrast with those mentioned, von Ziemssen prefers the percutaneous to the direct intragastric application; his reasons are not very convincing, in the light of Meltzer's and Goldschmidt's experiments. The electric brushing of the skin of the abdomen, breast, and back, urged by von Ziemssen, seems to be a great stimulus for nervous energy in neuropathic cases.

In this country, Allen A. Jones (*Med. Rec.*, June 13, 1891), Charles G. Stockton (*Med. Rec.*, 1889, p. 530), and D. D. Stewart (*Therap. Gazette*, 1893, p. 744), have published clinical observations on the intragastric employment of electricity, and there is a fairly uniform agreement that the class of gastric neuroses, particularly the sensory neuroses, nervous vomiting, and anorexia, are special indications for electricity in the form of the constant current, and that the direct intragastric method is to be preferred to the percutaneous.

In simple atony and atonic dilatations (but not in those dependent upon pyloric obstruction) the preference is to be given to the direct faradic current. The manner of application is simple; the anode is, as a rule, swallowed, and forms the intragastric pole. The cathode must have the shape of a conveniently broad and long, felt-covered plate (Fig. 30), which, after it is dipped in warm water, is

placed for ten minutes on the epigastrium; thereafter passed slowly up and down over the spinal column from the cervical to the sacral region. The meter showing about 25 milliampères should always be in the circuit, in case the galvanic current is used. For the faradic current we employ the full strength of three dry cells (chlorid of silver), and gradually increase to six cells. The electric bath, or electricity applied when the body is immersed in a saline bath, has its advantages in gastric neuroses.

Hydrotherapeutic and Orthopedic Methods.—Hydriatic procedures are much lauded by German and French gastro-enterologists. Most of these methods can only influence the stomach indirectly and secondarily, and therefore are of greater utility in functional disturbances than in the organic gastric diseases. These methods may be divided into (1) general or systemic, (2) special or local hydrotherapic treatments. The method to employ depends more upon the state of general health and the condition of the nervous system than upon any local condition. Wherever hydriatic treatment becomes necessary, it is imperative to send the patient to some well-managed institution, as most of the procedures can not be executed at home. Among the methods most employed are the various local and general douches, viz., fan douche, cold, hot, graduated, Scotch, and French douches. Among these we have tried the Scotch douche most frequently in gastric myasthenia. It consists of a stream of moderate intensity directed against the epigastrium for three or four minutes. But during this time the temperature of the water is changed every ten or twenty seconds from 28° to 8° R., or vice versâ; it is much lauded by von Ziemssen and Rosenthal ("Magenneurosen," etc., Wien, 1886). There are, besides, many kinds of fine sprays, and the sponge, sea salt, pour, dash, shower, shallow, vapor and sitz-baths; also the various packs, fomentations, and compresses. A local application which is very soothing in gastralgic affections is the so-called Priessnitz pack, which consists simply of a towel folded together to the size of six by ten inches, and dipped into hot or cold water, and then wrung out

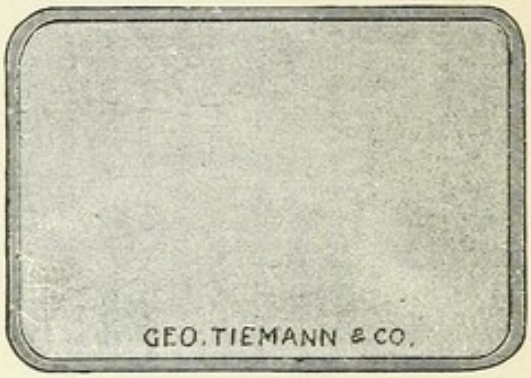

FIG. 30.—ABDOMINAL ELECTRODE.

so that there is no dripping from it, and then applied to the epigastrium; a layer of oiled silk or gutta-percha paper is laid over it and the whole is snugly secured and held in place by a broad flannel bandage passed around the body. As a matter of fact, most patients feel relieved and free from pain, but how the quieting effect is produced is a matter of conjecture.

In the treatment of gastric ulcer hot cataplasms are very serviceable. We are in the habit of using spongiopiline dipped into hot water and applied to the epigastrium after the excess of water is pressed out.

In the first volume of Hare's "System of Practical Therapeutics," Dr. Simon Baruch has an interesting article on "Hydrotherapy and Mineral Waters," in which the rationale of the action of water, peripheral irritation on temperature changes, and the technic of hydrotherapy are instructively described.

The *orthopedic appliances* used in the treatment of gastric diseases are often only imperfect substitutes for the more lasting effects of proper operations. They consist of contrivances to support the stomach in gastroptosis, or to keep up a floating kidney and prevent its interfering with the gastric or intestinal peristalsis. Where the abdominal muscles have become so relaxed that they seem to drag the viscera downward instead of supporting them, a condition found in obesity (Hängebauch), the Landau abdominal corset, for both sexes, is recommended by Riegel. Abdominal gymnastics are the most effective prophylaxis against these conditions.

Gastric Massage.—Von Ziemssen ("Ueber d. physik. Behandl. chronisch. Magen- u. Darmleiden," Leipzig, 1888, p. 29) and Rosenheim (*loc. cit.*, p. 146) consider massage a very subordinate means of treatment for stomach diseases. Ewald (*loc. cit.*), Boas (*loc. cit.*), and Riegel (*loc. cit.*), however, believe that there is something of value in the treatment. The best study of gastric massage was published by Zabludowsky (*Berlin. klin. Wochenschr.*, 1886, No. 26) and Csèri (*Wien. med. Wochenschr.*, 1889). The technic of massage differs according to the object to be accomplished. If it is intended as a passive exercise to strengthen the musculature, it is best done on an empty stomach, in bed before breakfast. But if the massage is expected to assist in the expulsion of chyme, it should be undertaken three to four hours after the principal meals. Massage can not be properly performed except on the uncovered skin. Indica-

tions for massage are given in the disturbances of the motor functions, viz.: (1) Those depending on myasthenia or atony; (2) depending on a stenosis of moderate degree; (3) cases of reduced secretion and chronic gastritis; (4) gastroptosis or prolapse of the stomach; and (5) certain cases of nervous inhibition of peristalsis. In cancer, ulcer, hematemesis, all acute inflammations in or around the organ, in excessive dilatation, distention, or contraction, and in all cases of intragastric putrefaction, massage is contra-indicated.

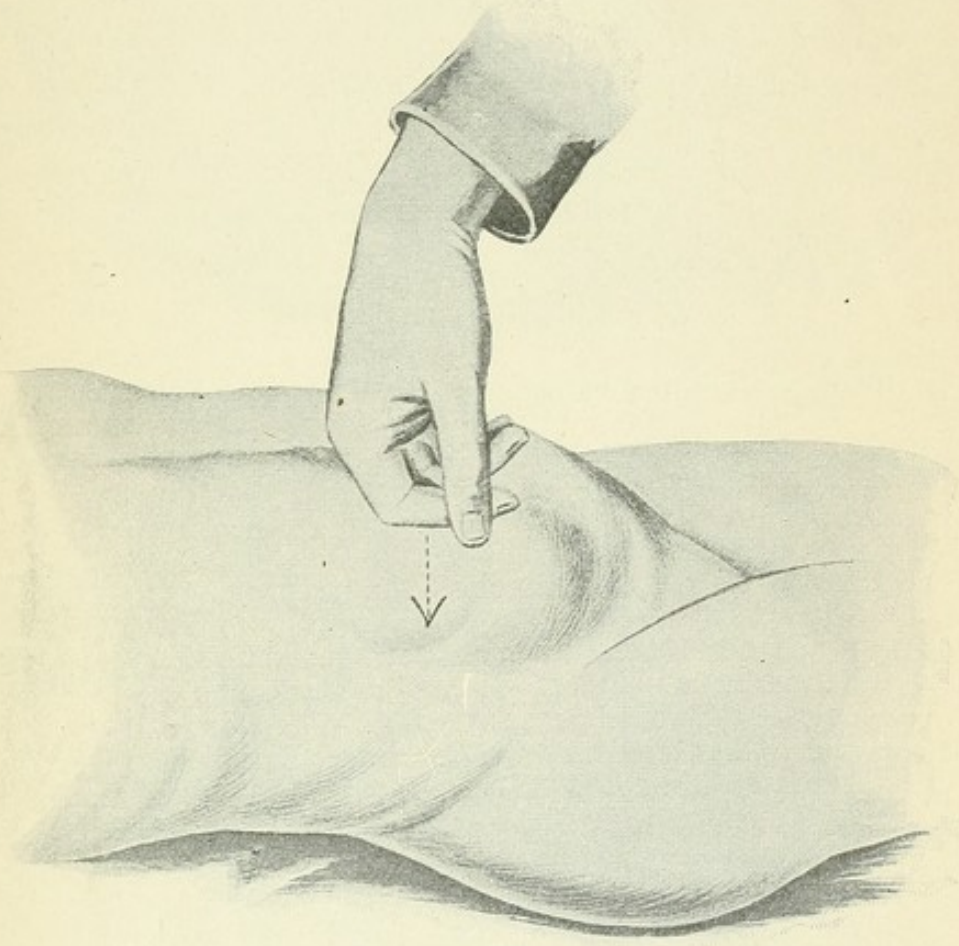

FIG. 31.—MASSAGE OF THE STOMACH IN DILATATION OR GASTROPTOSIS. (*Penzoldt and Stintzing, "Handbuch d. Therapie," etc.*)

The technic varies with the indication. For improving the muscular tone of the empty stomach, the masseur places himself to the right of the patient, who must lie on his back with knees slightly flexed. First movement (*a*): Insert the left hand deeply under the left arch of false ribs, under and past the edge. To increase the pressure, gently press the right hand firmly on the left. Second

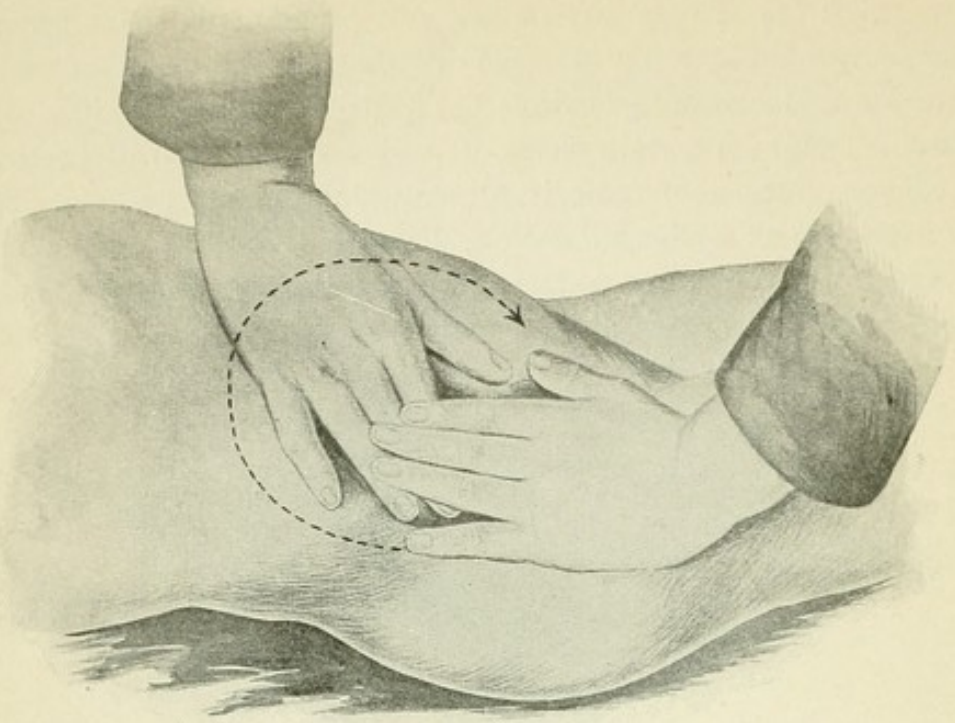

FIG. 32.—MASSAGE FOR IMPROVING GASTRIC TONICITY. (*Penzoldt and Stintzing, "Handbuch d. Therapie, etc."*)

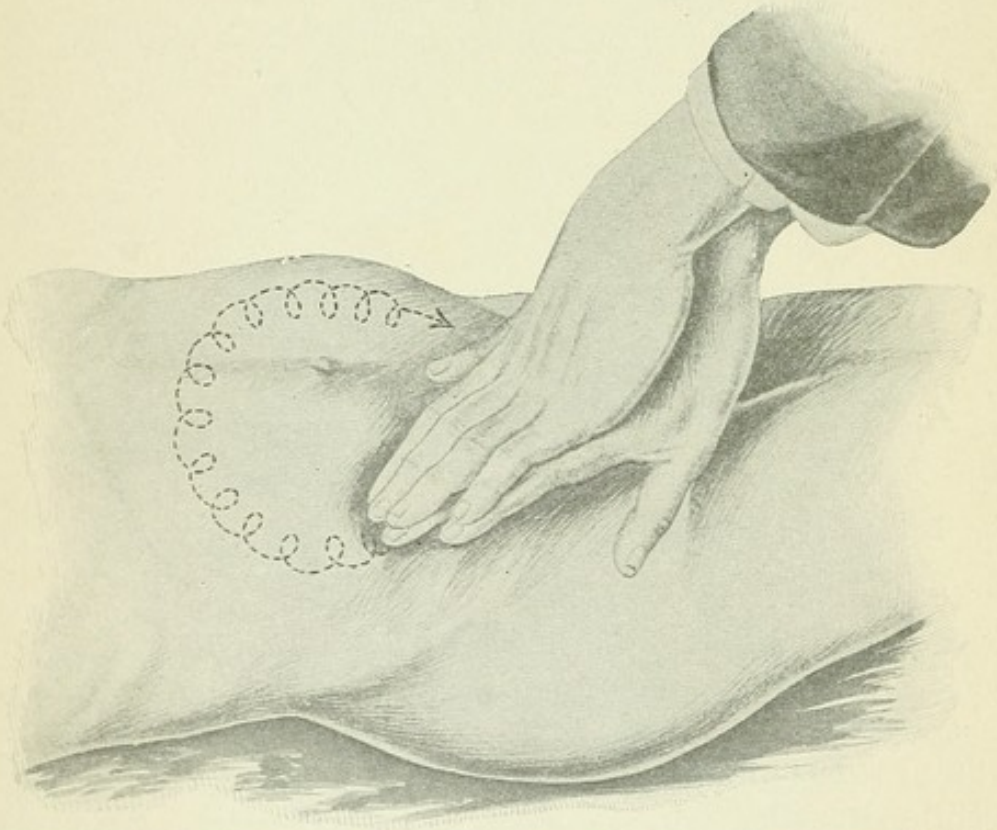

FIG. 33.—MASSAGE OF THE STOMACH AND OF THE COLON. (*Penzoldt and Stintzing, "Handbuch d. Therapie," etc.*)

movement (*b*): Now describe small circles with the hands thus arranged, progressing slowly from the pylorus to the fundus. Third movement (*c*): Perform strong vibratory movements toward the depth with the finger tips while *a* and *b* are being executed. Fourth movement (*d*): Knead the stomach between thumb and four fingers, and in conclusion execute stroking passes, with extended four fingers, from left to right.

Massage of the full stomach is undertaken with a view to mix its contents thoroughly or to aid in forcing them into the duodenum. Zabludowski (*loc. cit.*) advises the pressing of the stomach against the spinal column, dividing it into two halves; by compressing the half nearest the pylorus, he widens the latter by wedging the chyme through it into the duodenum. This is justifiable only where it is sure that the chyme is comparatively fresh and not in a state of putrefaction.

CHAPTER V.

MINERAL SPRINGS.

*The Uses and Abuses of Natural Mineral Waters in Diseases of the Digestive Organs.**

With such a wealth of valuable mineral springs in this country, it is difficult to understand the large annual exodus of Americans to foreign water resorts. As a fact, we fear the fault rests with the American physician, not the American waters. Few native physicians give to the selection and adaptation of proper mineral waters the consideration it deserves; whereas, in German, French, and English practice, this forms a common and important factor. According to Baruch, even American doctors resident at the springs do not insist upon precision in proper drinking, diet, hydrotherapy, or exercise.

The surpassing virtues of our American mineral waters can only be attested by making an individualizing selection of the waters to each case, after establishing the diagnosis.

Too much empiricism, too much fashion and sport, too much abuse of alcohol, and not sufficient peace and quiet of mind exhibit themselves at our American springs.

Without a diagnosis—not to speak of test-meals—we have known of numerous instances where springs were ordered. Systematic mineral-water treatment should be recommended only after the institution of careful chemical and physical examinations.

In reference to the abuse of mineral waters, we will limit ourselves to their misuse in gastric diseases. We would exclude, first, all cases of motor insufficiency of any kind, whether of the simple atonic or the stenotic form, whether with pronounced dilata-

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* In the preparation of this chapter we have availed ourselves of the Analyses of the Mineral Springs of the U. S. as given in the records of the Department of the Interior (Agriculture), Washington, D. C.; and in the description of the physiological effects of the various springs, we have followed the works of Flechsig, S. Baruch, Ludwig, and G. Thompson in reference to waters with which we have no personal experience.

tion or not, because we know, upon very good authority (von Mehring, *loc. cit.*), that water is not absorbed from the stomach, and hence can only aggravate (by its weight) the myasthenia and dilatation. Where, however, the various saline and alkaline waters can be readily obtained, they serve admirably for lavage. The sodium chlorid spring water is beneficial in sub- or anacidity, and the alkaline waters whenever hyperchylia is associated with dilatation.

In neoplasms of the stomach, particularly in carcinoma, mineral-water treatment is harmful. For ulcer, the Carlsbad springs have been much lauded by Leube and others; but we coincide with Ewald's opinion, that the same or perhaps more rapid effects would have been obtained in such cases had patients taken the rest-cure at home. Rest, diet, and effective local treatment are the things most needed, and these can be obtained much more readily at home than elsewhere. For, after all, as far as gastric sufferers are concerned, the most important things, even at the springs, are diet, suitable food, good cooking, etc.

In acute gastritis, mineral waters are useless. There remain still to be considered the neuroses of secretion and motility. All secretory neuroses are, more or less, indications for mineral-water treatment, particularly those in which an excessive amount of HCl is formed, with which the alkaline waters combine, at the same time executing a very desirable astringent effect on the mucosa. In achylia of nervous origin, the saline waters might rationally be tried; but where the glandular elements are destroyed they can not restore the secretion, although they may aid in dissolving mucus and keeping the membrane cleaner than otherwise. In the motor neuroses, if dependent on hyperchylia or hypersecretion, the alkaline waters may benefit by removing the causes, as stated above. But in insufficiency of the pylorus and cardia we have neither heard of nor seen improvement.

The proper field for these waters is undoubtedly chronic gastritis. With their judicious use much good can be effected. It should not be overlooked, however, that there may be a chronic gastritis with normal or excessive acidity; here the alkaline waters are to be preferred to the salines—sodium chlorid. In chronic gastritis with achylia, only salines of a mild concentration are useful; for the powerful saline (NaCl) waters, such as Carlsbad (Mühlbrunnen), may undoubtedly cause an injurious, alkaline, irritative transudate from the mucosa if retained in the stomach.

Ewald (*loc. cit.*) says: "The saline waters are indicated in all cases of catarrh with lessening of secretion, either with or without the production of mucus. Here we may use the simple sodium chlorid waters where the patient is otherwise well, and only the gastric and intestinal secretions are to be augmented; the sparkling sodium chlorid waters are useful where we desire the stimulating effects of the carbonic acid gas, and where, by moderate catharsis and the use of the brine as such, the metabolism may be increased. Finally, all waters which are to act on the stomach are borne better warm than cold.

"On the other hand, experience has shown that the alkaline-saline and the alkaline springs are very beneficial in conditions of hyperacidity or hypersecretion. The successful use of Carlsbad water in ulcer of the stomach is now much more readily understood, since we know that the ulcer is in many cases accompanied by hyperacidity, and that the mineral water not alone momentarily neutralizes this (just as in cases of hypersecretion), but also that it may actually lessen the activity of the secretion. A similar effect might also be produced by the purely alkaline waters, but they have not yet been much used for this purpose. Finally, the sodium sulphate waters are to be used in those cases in which the stomach is only secondarily involved from disturbances of the liver and intestines."

According to Stillé and Maisch, there are, indeed, two classes of patients who require the use of very different mineral waters. The first is composed of that large body of invalids in whom there exists no organic change of structure, but whose functions are merely weakened or clogged by the strain of business, the exhaustion of pleasure, sensual excesses in eating or drinking, or, in this country especially, by the manifold errors committed in the preparation and consumption of food and the disregard of hygienic rules in their habits of living. The second consists of that smaller but still numerous class of persons who, besides being more or less injured by the causes of ill health just enumerated, have been affected with definite diseases, and especially rheumatism, gout, calculous disorders, cutaneous eruptions, scrofula, syphilis, diabetes, paralysis, uterine disorders, etc. Of these two classes, the former is benefited most by a visit to the less mineralized springs, while the latter requires a course of active medicinal treatment such as the stronger mineral waters afford. In both classes of

patients, but particularly in the first, the action of the waters is only one out of many influences that combine to restore their health. Toward that end a total change of habits is one of the most influential agencies in very many cases. Escape from the anxieties and fatigue of business, from the excitement of fashionable life, the mental tension of political and professional pursuits, the worrying annoyances of domestic affairs, endured, perhaps, in a large city, with all its enervating social duties, its Babel-like sounds, and its polluted atmosphere,—escape from these alone ought to suffice to restore the disturbed balance of health. When we consider how much more probable must this result become when fatigue, anxiety, contention, wearisome routine, and foul air are exchanged for repose and peace in the midst of novel scenes and new associates, and freedom from the onerous conventionalities of fashionable life, different apartments, food, and occupation, it may even seem doubtful whether, after all, some other new residence would not profit the invalid as much as the frequented springs. But there are two reasons against this conclusion: the one is that, with many persons, relief would be impossible without an exercise of the faith which gives potency to waters as well as to other remedial agents; and the other is that even the purest of these waters, systematically used, especially in conjunction with bathing and regular exercise, do, in a greater or less degree, depurate the system through the kidneys, bowels, and skin, and by a gentle but sustained action gradually remove effete products of tissue change from the system and free the organs from the poisons that tainted them. Judiciously used under the advice of a competent physician, these almost neutral waters and the milder saline springs are capable, in a few weeks, of changing the languid, indifferent, pale, and feeble invalid into the lively and energetic leader of the gay crowd. Such rapid transformations are frequently witnessed, especially at the hot springs of Virginia, the Bedford Springs, Pa., at some of the Saratoga Springs (though in the last-mentioned place routine hygienic treatment becomes more difficult because of numerous side temptations), and certain European springs, such as Wildbad, Gastein, and Pfeffers, none of which contains any considerable proportion of mineral ingredients. But these waters, whether drunk warm or cold, if they are largely used, act as organic purgatives, and increase materially the total amounts of solids, and especially of urea, excreted with the urine,

without causing the debility which an equal discharge from the bowels would occasion.

Alkaline Waters.—The chief ingredients of these waters are the alkaline carbonates, especially the carbonate of sodium. They also contain varying amounts of the carbonates of lime, magnesium, lithium, sodium chlorid, etc., and many of them are strongly charged with carbonic acid gas. Although it is probable that the other saline constituents may contribute to the total physiological effects of these waters, they owe their main therapeutic activity to the alkaline salts they contain. The temperature of these springs is also a point worthy of consideration. In a general way, it may be said that the physiological action of these waters is like that of any alkaline salt, plus the effect produced by the circulation of large quantities of water in the system. The carbonate of sodium neutralizes free acids or fermentation products in the stomach, whether taken during or after meals. According to Brunton and Sidney Ringer, the stronger alkaline waters, if taken before meals, increase the secretion of gastric juice. The fact is, plain, distilled water will also cause a secretion of gastric juice in the normal stomach. The carbonic acid set free by the decomposition of the carbonates in the stomach, and the sodium chlorid usually present in these waters, act as a stimulant to the gastric mucous membrane, promoting secretion and counteracting any disturbing influence exerted by the carbonate. The free carbonic acid frequently contained in waters of this class, by its stimulating effects on gastric peristalsis, accelerates digestion, and thereby increases the desire for food.

It would appear, therefore, that the alkaline waters have a wide range of usefulness. They seem to be especially indicated in gastric affections in which there is an excessive production of hydrochloric acid, as in acid dyspepsia, atony of the gastric mucous membrane, and gastric ulcer. In all catarrhal conditions of the stomach they are most serviceable, but a free and prolonged use lowers the nutrition, except in case of waters containing chlorid of sodium.

The names of a few of the more important ALKALINE WATERS are here appended:

Vichy, in France; Ems and Fachingen, in Germany; Saratoga Vichy (rich in CO_2), New York; St. Louis Springs, Michigan (poor in CO_2); Bethesda Springs, Wisconsin. Other sodium

chlorid waters, containing also some carbonates and CO_2, are: Hathorn, Congress, and Kissengen Springs, in Saratoga, New York; Homburg, Wiesbaden, Kissingen, and Selters, in Germany; Bourbonne, in France.

All alkaline waters contain more or less carbon dioxid, and their most important ingredients are the alkaline carbonates. They also contain sodium chlorid, and sometimes sodium sulphate. In some, one variety of salts, in others, another preponderates. Generally speaking, the European waters are richer in alkalies than are the American.

Alkaline waters are useful in uric acid diathesis and lithemic conditions, gout, chronic rheumatism, obesity, hepatic engorgement, gall-stones, hyperacidity, gastric ulcer, and catarrhs of the mucous membranes, especially of the stomach, respiratory tract, and bladder.

Alkaline Sulphur Waters.—Richfield Springs, Sharon Springs, and Avon Springs, in New York; Greenbrier White Sulphur Springs, in West Virginia; Harrogate, in England; Neuendorf and Meinberg, in Germany; Aix-la-Chapelle, in Rhenish Prussia.

Those waters containing sulphuretted hydrogen in addition to other ingredients are used moderately in gout, chronic rheumatism, obesity, and chronic eczema. They are often supplemented by a course of chalybeate waters.

Alkaline and saline purges contain a high percentage of sodium and magnesium sulphates. These waters are often called "bitter waters." Such are Püllna, in Bohemia (the strongest of all and one of the oldest known); Carlsbad (Sprudel), in Bohemia; Marienbad (Kreuzbrunnen), in Bohemia; Friedrichshall, in Germany; Franz Josef, in Austria; Kissingen Bitterwater, in Bavaria; Hunyadi Janos, in Hungary; Rubinat Condal Spring and Villacabras, in Spain; Crab Orchard and Estill Springs, in Kentucky; Bedford Springs, in Pennsylvania; Epsom, in England; some of the Saratoga waters. These waters are useful to counteract indiscretions in diet and congestion of the liver. The Rubinat water is effective and possesses the advantage of being less disagreeable than many of the others. Villacabras water is a Spanish sodium sulphate, strongly purgative water, obtained not far from Madrid.

These waters should be taken either very cold or in a half-pint of very hot water. If drunk lukewarm their taste is nauseous and may

excite emesis. We advise that these powerful waters be entirely avoided where there is any distinct organic disease of the stomach.

Chalybeate Waters.—Schwalbach (Stahlbrunnen), Pyrmont (Neubrunnen), Spa, Belgium; St. Moritz, Switzerland; Rock Enon, Virginia. These waters are claimed to serve as tonics for the blood and nerves, but if used too long they cause dyspepsia and anemia. There is no evidence that there is any increase of red blood-corpuscles by the use of these waters exclusively; whatever improvement follows is due to the diet and the hygienic environment.

Acidulous Waters.—These waters contain CO_2 in excess, and but very little salts of any kind. Such are Clysmic Spring, in Wisconsin; Blue Lick, in Kentucky; Carlsbad (Dorotheenquelle), in Bohemia.

Various other waters are the Alum Springs, in Virginia; Oak Orchard Acid Spring, in New York; Bourboule, in France, which contains arsenic. Roncegno water is a ferruginous arsenical water from the Tyrolean province of Trent.

COMPARATIVE CHART ILLUSTRATIVE OF ALKALINE WATERS.—(*Baruch.*)

American.	European.	One Pint Contains:			
		Sodium Carbonate.	Carbonic Acid Gas.	Temperature.	Other Prominent Constituents.
		Grs.	Cub. in.	Fahr.	
	Vichy (Grand Grible Spring), France, . . .	26	14	105.8°	Sodium chlorid, 4 grs.; sodium sulphate, 2 grs.; potassium carbonate, 2 grs.
Ojo Caliente Spring, New Mexico, .	. . . . .	14	. .	100°	Sodium chlorid, 4 grs.; sodium sulphate, 1 gr.
	Fachingen Spring, Germany, . .	19	32	50°	Sodium chlorid, 4 grs.; calcium carbonate, 2 grs.
Saratoga Vichy Spring, NewYork,	. . . . .	11	48	50°	Calcium and magnesium carbonates, 17 grs.; sodium and potassium chlorids, 18 grs.
	Ems (Kesselbrunnen Spring), Germany, . .	10	6	115°	Sodium chlorid, 7 grs.; calcium carbonate, 1 gr.
St. Louis Spring, Michigan, . . .	. . . . .	7	1	50°	Calcium and magnesium carbonates, 6 grs.; calcium sulphate, 7 grs.

Saline Waters.—This class may be conveniently subdivided into, first, waters containing chiefly the chlorid of sodium; and, second, waters containing large quantities of the sulphates of sodium and magnesium—the so-called "bitter waters" of German authors.

The Sodium Chlorid Waters.—These waters contain, besides large quantities of sodium chlorid, a certain proportion of other chlorids, especially those of lime and magnesium, and small amounts of alkaline and earthy sulphates and carbonates, iodids and bromids. Carbonate of iron is sometimes present in considerable quantity. The gases consist for the most part of carbonic acid, which renders the water more agreeable to the palate and more readily absorbed. Some of these waters are heavily charged with sulphuretted hydrogen. They occur both as cold and thermal springs, and may be utilized both for drinking and bathing purposes.

The physiological action of these waters is chiefly attributable to the presence of sodium chlorid. This salt, as is well known, has a stimulating effect upon all the mucous membranes of the body, especially that of the gastro-intestinal tract. In the stomach it dissolves the mucus, increases the secretion of gastric juice, promotes the digestion of albuminous substances, and excites peristalsis. In the intestines it stimulates the flow of pancreatic juice and bile, and, owing to its well-known influence on the process of osmosis, promotes the absorption of food. Intestinal peristalsis is also increased, and, if the sodium chlorid is present in large quantity, the water may, in its effects, be laxative and even purgative. Some authors have regarded this purgative action as representing the chief therapeutic virtues of these waters, but, according to Flechsig, it is subordinate in importance to the effect of the sodium chlorid on the blood. He states that this salt exerts considerable influence on the process of tissue metabolism, augmenting the metamorphosis of nitrogenous matters and increasing the oxidation of albuminous substances, as is shown by the increased quantity of solids in the urine. The iodids and bromids contained in some of these waters are usually present in such very minute amounts that it is doubtful whether they contribute to their therapeutic action; at any rate, it is impossible to separate their effects from those of the sodium chlorid.

The therapeutic indications of sodium chlorid waters, as based upon their physiological action, are sufficiently obvious. Their

stimulating effects upon the mucous membranes have been utilized in the treatment of catarrhal processes, especially in the stomach, duodenum, and bile-ducts; and in chronic intestinal catarrh associated with constipation, their use has been highly commended.

COMPARATIVE CHART ILLUSTRATIVE OF SALINE WATERS.—(*Baruch.*)

American.	European.	One Pint Contains:			
		Sodium Chlorid.	Carbonic Acid Gas.	Temperature.	Other Prominent Constituents.
		Grs.	Cub. in.	Fahr.	Grains.
	Homburg (Elizabethbrunnen), Germany, ...	79	48	50°	Chlorids of calcium and magnesium, 15; calcium carbonate, 11.
Ballston Artesian Lithia Well, New York, ...	. . . . .	93	53	. .	Magnesium and calcium carbonate, 34; potassium chlorid, 4; lithium carbonate, 0.7.
	Wiesbaden (Kochbrunnen), Germany, ...	52	17	155°	Chlorid of potassium, 1; calcium carbonate, 3.
Hathorn Spring, Saratoga, New York,	. . . . .	64	47	47°	Calcium and magnesium carbonates, 28.
	Bourbonne (Fontaine Chaude), France,	46	. .	149°	Calcium chlorid, 5; calcium sulphate, 6.
Congress Spring, Saratoga, New York, ...	. . . . .	50	49	52°	Calcium and magnesium carbonates, 21; sodium bromid, 1 06.
	Kissingen (Rakoczi), Germany, . . .	44	42	51°	Potassium chlorid, 2; calcium carbonate, 8.
Kissengen Spring, Saratoga, New York, ...	. . . . .	42	45	40°	Calcium and magnesium carbonates, 26; sodium carbonate, 8; lithium carbonate, 0 64.
	Selters, Germany, .	17	30	62°	Sodium carbonate, 6.
Saratoga Seltzer Spring, New York,	. . . . .	17	. .	50°	Sodium carbonate, 2; calcium and magnesium carbonates, 10.

Bitter or Purgative Waters.—This name has been applied to waters characterized by a high percentage of the sulphates of sodium and magnesium. They also contain considerable quantities of the sulphates of lime, and the carbonates of lime and magnesium, though rarely small amounts of carbonic acid gas. Carbonate of sodium, however, is seldom, if ever, found in them.

The chief physiological action of these waters is comprised in the stimulating effect which they exert upon the mucous membranes of the gastro-intestinal tract. They give rise to a profuse watery secretion of a serous, or even mucous, character, and thus act as purgatives. If taken in large quantities, they frequently produce gastric and intestinal disturbances, and their

COMPARATIVE CHART ILLUSTRATIVE OF BITTER WATERS.—(*Baruch*)

American.	European.	One Pint Contains:				
		Sodium Sulphate.	Magnesium Sulphate.	Carbonic Acid Gas.	Temperature.	Other Prominent Constituents.
		Grs.	Grs.	Cub. in.	Fahr.	
	Püllna, Bohemia,	124	93	. . .	. . .	Chlorid of magnesium, 16 grs.; magnesium carbonate, 6 grs.
Crab Orchard, Foley's Spring, Kentucky, . .	. . . . .	7	25	. . .	. . .	Calcic carbonate, 7 grs.; potassium sulphate, 1 gr.
	Friedrichshall, Germany, . .	41	39	5	46°	Sodium chlorid, 67 grs.; magnesium chlorid, 31 grs.; calcic sulphate, 11 grs.
Estill's Springs, Irvine Springs, Kentucky, . .	. . . . .	. . .	32	. . .	. . .	Calcic carbonate, 4 grs.; sodium chlorid, 2 grs.
	Carlsbad (Sprudel), Bohemia,	19	. . .	8	162°	Sodium carbonate, 9 grs.; sodium chlorid, 8 grs.
Bedford Springs, Pennsylvania,	. . . . .	. . .	10	9	58°	Chlorid of sodium, 1 gr.; calcium sulphate, 2 grs.
	Marienbad (Kreutzbrunnen),					
Harrodsburg Spring, Saloon Spring, Kentucky, ...	Bohemia, . .	36	. . .	15	53°	Sodium carbonate, 8 grs.; sodium chlorid, 11 grs.
	. . . . .	. . .	28	. . .	. . .	Calcic sulphate, 10 grs.; sodium chlorid, 1 gr.

protracted use is apt to be followed by atony of the intestines and intestinal catarrh. It is as yet a matter of speculation whether this purgative action is due to the increased exudation of fluids, or whether it results from the stimulation of intestinal peristalsis, as is assumed by Flechsig and others. Owing to the increased peristalsis, the passage of food through the intestines is accelerated;

and in consequence of the diminished absorption of nutriments engendered by this, a loss of weight and disappearance of the fatty tissue results.

It follows from the above considerations that the use of these waters is restricted to cases in which we desire to stimulate the intestinal secretions, as in chronic constipation occurring in plethoric persons, engorgements of the abdominal and pelvic viscera, hemorrhoids, etc. They also prove serviceable in cases of obesity, as part of a treatment of denutrition. On the other hand, their use is contra-indicated in anemic persons, and where there is great irritability of the stomach and intestines, with a tendency to diarrhea.

American waters of this class are somewhat weaker in sulphates of sodium and magnesium than the European, but the quantity of purgative salts present in the former is quite sufficient to produce active therapeutic effects. All these waters contain a considerable amount of sodium chlorid, which contributes essentially to their physiological action.

The Bedford Spring (Pa.) water is especially to be recommended on account of its mildness. It is, in our opinion, of no advantage when spring waters possess an excessively large percentage of drastic salts. In a concentrated solution magnesium chlorid acts as a cellular poison on the superficial gastric and intestinal epithelium when used for weeks. Bedford Mineral Magnesia Spring has also a mildly diuretic effect; its laxative effect is not experienced until at least 500 c.c. are taken in twelve hours.

Sulphuretted Waters.—The constituent imparting to these waters their distinguishing characteristic is the sulphuretted hydrogen which they contain in greater or lesser amount. With this gas we find associated a varying quantity of sulphur combinations, such as the sulphids of potassium, sodium, calcium, and magnesium. They also contain the alkaline and earthy sulphates and carbonates, the chlorid of sodium, and the sulphates and carbonates of iron; and these are frequently present in large quantities, and certainly play a not unimportant part in the therapeutic action of these waters. According to Daland, "a sulphur spring of moderate strength contains not less than 12 cubic inches of sulphuretted hydrogen in the gallon, though many springs contain so small an amount that therapeutically they are inert, and the good effects observed are due to the influence of the increased use of water,

change of scene and climate, cessation of work, regular meals, good hygiene, and hope,—all of which contribute strongly to restore health at all springs." Many of the sulphur waters are thermal, and are chiefly employed in baths.

Regarding the physiological action of sulphur waters on the system, nothing positive can be said. Various plausible theories have been proposed to account for their curative effects in the diseases for which they are employed. It is claimed that their chief action is exerted on the intestinal canal, where they stimulate the functions of the glands, augmenting secretion and producing laxative effects. When administered for prolonged periods, they give rise to gastro-intestinal disorders and exert a debilitating influence upon the blood, heart, and lungs, as evidenced by anemia, cardiac weakness, etc. According to Leichtenstern, the sulphuretted hydrogen absorbed into the blood is rapidly converted into sulphuric acid, and is therefore devoid of any specific effect, unless present in very large amounts. On the other hand, Stifft concludes that the sulphuretted hydrogen has a specific excitant action upon the sensitive fibers of the pulmonary branches of the pneumogastric and upon the respiratory, cardiac, and vasomotor centers, its prolonged use giving rise to paralysis from overstimulation. In this way he explains the action of the sulphur waters upon the respiratory and circulatory systems, upon tissue-metabolism, and upon the secretory and excretory functions.

These waters have been administered internally in passive congestion of the abdominal and pelvic viscera, especially in plethoric persons; in enlargements of the liver and spleen; hemorrhoids; chronic intestinal catarrh; and chronic poisoning by metals. In the form of baths they have been recommended in gout and chronic rheumatism, but their curative effect in these cases is attributable to the elevated temperature of the waters rather than to any specific action of the sulphuretted hydrogen or other constituents. At many baths the internal or local use of the waters is combined with inhalation of the gases or of the nebulized waters; and this method has been found useful in the treatment of chronic catarrhs of the pharynx, larynx, and bronchi. Sulphuretted waters are, in our estimation, worthless as a therapeutic agent in gastro-intestinal diseases.

The following chart illustrates the superiority of the sulphur waters of America:

COMPARATIVE CHART ILLUSTRATIVE OF SULPHURETTED WATERS.—(*Baruch.*)

American.	European.	One Pint Contains:			
		Sulphuretted Hydrogen.	Sulphids.	Temperature.	Other Prominent Constituents.
		Cub. in.	Grs.	Fahr.	
	Neundorf, Germany,	1.28	0.55	53°	Calcium and magnesium sulphates, 10 grs.
Sandwich Spring, Ontario, Canada, . .	. . . . .	4.72	. .	52°	Chlorid of magnesium, 19 grs. ; calcium sulphate, 15 grs.
	Aix-le-Bains, France, ...	0.82	. .	108°	Calcium carbonate, 1 gr.
Sharon Spring, White Sulphur Spring, New York, . . .	. . . . .	2.00	0.28	48°	Calcium and magnesium sulphates, 24 grs.
	Harrogate, England, ...	0.53	1.54	. .	Sodium chlorid, 86 grs. ; potassium and magnesium chlorids, 10 grs.
Paroquet Spring, Kentucky, . . .	. . . . .	3.75	. .	. .	Sodium chlorid, 39 grs.
	Meinberg, Germany,	0.61	0.67	48°	Sodium sulphate, 6 grs.
Salt Sulphur Spring, Iodin Spring, W. Virginia, . . .	. . . . .	2.39		. .	Sodium sulphate, 3 grs. ; calcium sulphate, 8 grs.

Chalybeate Waters.—A large number of mineral springs contain the salts of iron, but the quantity present is frequently so small as to be practically devoid of therapeutic effects. In the class under consideration only waters containing a sufficient quantity of chalybeates to be of value in the treatment of disease will be mentioned.

Iron salts usually occur in the form of the carbonate or sulphate. Other constituents, which are sometimes present in large amounts, are the alkaline carbonates and sulphates, the earthy carbonates, sodium chlorid, alum, and sulphuric acid. Alum often exists in considerable quantities, especially in the chalybeate springs of Virginia.

Chalybeate waters containing the carbonate of iron are clear, odorless, have a slight inky taste, and are highly charged with carbonic acid gas, which renders them palatable. They are chiefly employed for drinking purposes. The sulphate-of-iron waters

have a marked astringent taste, which sometimes proves an objection to their use. This astringency may be decidedly increased by the presence of alum.

The following chart illustrates the superior quality of some American chalybeate springs:

CHALYBEATE WATERS.—(*Baruch.*)

American.	European.	One Pint Contains:			
		Sulphates of Iron.	Carbonates of Iron.	Carbonic Acid Gas.	Other Prominent Constituents.
	Brighton, England,	1.80	. .	. .	Calcium sulphate, 4 grs.
Church Hill Alum Springs, Virginia,	. . . . .	19.8	. .	. .	Magnesium and calcium sulphates, 22 grs.; aluminium sulphate, 9 grs.
	Spa (Buhon), Belgium, ...	. .	0.67	71.6	Small amounts of calcium carbonate and alumina.
Rock Enon Springs, Virginia, ...	. . . . .	. .	1.78	. .	Calcium and magnesium sulphates, 2 grs.; calcium and sodium carbonates, alumina.
	Schwalbach (Stahlbrunnen), Germany, ...	. .	0.46	50.2	Calcium carbonate, 1 gr.; manganese carbonate 0.10 gr.
Vichy Springs, New Almaden, Cal., .	. . . . .	. .	0.60	29.8	Sodium carbonate, 17 grs.; calcium carbonate, 3 grs.; magnesium sulphate, 1 gr.; sodium chlorid, 4 grs.
	St. Moritz (Grande Source), Switzerland, ...	. .	0.17	39.2	Sodium carbonate, 1 gr.; calcium carbonate, 6 grs.; sodium sulphate, 1 gr.
Estill Springs, Kentucky, ...	. . . . .	. .	0.23	4.15	Calcium carbonate, 1 gr.; magnesium sulphate, 1 gr.

The physiological action of chalybeate waters is essentially similar to that of all iron compounds; they promote constructive metamorphosis, increasing the number of red corpuscles in the blood and stimulating all the body functions. For internal use the waters containing the carbonate of iron are preferable, since

they are less apt to disturb the stomach and are more easily assimilated, owing to the carbonic acid gas present. According to the character of the case, it may be necessary to select an iron water containing alkalies, sodium chlorid, sulphate of sodium and magnesia, or alum.

The chalybeate waters have been recommended in anemia, chlorosis, and all conditions attended with anemia, such as hysteria and neurasthenia; chronic endometritis, dysmenorrhea, amenorrhea, chronic gonorrhea, and spermatorrhea; chronic affections of the kidneys, diabetes mellitus, chronic gastritis, nervous dyspepsia, chronic diarrhea, etc.

Among the iron and alum springs, Bedford Alum Spring has been found remarkably efficacious by Baruch in chronic diarrhea, which had resisted both private and hospital treatment.

The contraindications to their use, as given by Flechsig, comprise all febrile and congestive conditions and advanced organic diseases of the lungs, liver, and kidneys. The sulphate-of-iron waters are excellent astringents and disinfectants, and have been highly recommended in chronic diarrhea, gastric ulcer, etc.

Acidulous Waters.—These waters owe their therapeutic properties to the large quantity of carbonic acid gas they contain, the solid constituents being present only in small amounts. As has been stated, many alkaline and saline waters contain considerable quantities of CO_2; but its effects, whatever they may be, are completely subdued by those of the mineral ingredients. In the acidulous waters, however, the carbon dioxid is the chief therapeutic agent, and for this reason it becomes necessary to discuss them as a separate class of mineral waters.

The physiological action of carbonated waters is comprised in a gentle stimulative effect upon the mucous membrane of the stomach, promoting peristalsis and thereby a more rapid evacuation of its contents. The pulse and respiration are said to be slightly accelerated, and a large quantity of urine is excreted. It seems, however, that this diuretic effect is not attributable to the carbonic acid gas, as is assumed by some authors, but rather to the large quantities of water which the patient is able to imbibe without distress, for the quantity of the gas absorbed into the blood through the walls of the stomach is certainly too small to produce systemic effects.

The acidulous waters have been chiefly recommended in gastric disorders, especially those of neurotic origin; and, owing to their agreeable taste, they form excellent table waters. They relieve nausea, increase the appetite, and aid digestion by stimulating the secretion of HCl. On account of their stimulating effect upon the peripheral cutaneous nervous system, they have also been employed as baths.

CHAPTER VI.

IMPORTANT MEDICINAL AGENTS IN GASTRIC THERAPY.

1. **Hydrochloric Acid.**—This acid should never be given except where the analysis of test-meals shows the absence of free HCl. For this purpose it is not sufficient to test with Congo paper or dimethylamidobenzol, but the amount of the HCl deficit—*i. e.*, the amount of decinormal HCl solution that must be added until the reaction of the chyme shows free HCl—must be determined, in order to be instructed how much behind the normal secretion the case may be.

HCl is given in the absence or diminution of the normal secretion mainly for three purposes: (1) To supplement gastric proteolysis; (2) to act as an antiseptic; (3) as a tonic and stomachic.

To these effects, that we have in mind in supplying HCl, may be added its influence as an important regulator of the gastric peristalsis; and that it brings the insoluble calcium and magnesium salts of the ingesta into solution; in fact, all of the objects and functions that are recognized as physiological to the HCl (see p. 49) may be at least partially accomplished by supplying it in sufficient quantity. But the question arises, Can it be supplied in sufficient quantity? The simple presence of free HCl does not contraindicate the administration of the acid. Positive reaction to Congo paper and phloroglucin-vanillin indicates, it is true, that HCl is secreted in excess of what is required to combine with the food. In healthy digestion it is always found that this excess amounts on the average to 30 c.c. of a decinormal solution of NaOH (in Baltimore); and it seems to be what is necessary or advantageous, not for digestive purposes (for even with a large excess of HCl it is not the rule for all the proteid matter to be digested in the stomach), but for destroying the exuberance of micro-organisms swallowed with the food. The frequently quoted cases without any gastric secretion whatever, who succeed in maintaining their nitrogen equilibrium, —and we have seen many such,—and the experiment with the dog (Kaiser and Czerny), whose weight was kept up although the largest

portion of the stomach was removed, constitute but a weak argument against the therapy of HCl. For although such patients manage to get along fairly well, it is only under the most careful diet and by taking very little exercise that they maintain their health. Permanent and perfect health with total absence of gastric secretion is rarely observed, except in those who are able to rest much and have their food prepared with great care. These facts must not be overlooked in the work of von Noorden ("Ueber die Ausnützungder Nahrung bei Magenkranken," *Zeitschrift f. klin. Med.*, 1890, Bd. XVII), which demonstrated that absolute and permanent deficiency of gastric juice may be accompanied by perfect health. This health is perfect under the conditions mentioned, but when they are taxed by work or the diet is not the usual one, suffering becomes manifest. If achylia gastrica could really exist without any subjective or objective disturbance, how is it that so many of these patients consult the stomach specialists and are reported by them in literature? When we must work for our living and can not have the benefits of the dietetic kitchen at all times, we must have a good gastric juice to partially, at least, disinfect and dissolve our food, and a person who secretes no gastric juice is or soon becomes a patient. In a recent article on achylia gastrica by F. Martius and O. Lubarsch (published by T. Deuticke, Leipzig, 1897), the authors arrive at the conclusion that neither simple achylia nor that dependent upon atrophy of the mucosa (anadenia) can bring about severe anemic or cachectic conditions unless motor insufficiency, atrophy of the intestinal mucosa or general diseases (tuberculosis, lues, infections, etc.) are added. Even if this is true, generally speaking it does not disprove the statement that absence of HCl in the gastric secretion compels the individual to lead the life of a patient. But over and beyond this, Flint (*loc. cit.*), Fenwick (*The Lancet*, 1877), Quinke (*Samml. klin. Vorträge*, No. 100, 1876), Nothnagel (*Deutsch. Arch. f. klin. Med.*, Bd. XXIV, 1879), Osler (*Amer. Jour. Med. Sciences*, April, 1897), Kinnikut (*Amer. Jour. Med. Sciences*, October, 1887), also Rosenheim and G. Meyer (both in article on "Achylia," by Martius and Lubarsch), have described cases of pernicious anemia in which atrophy of the gastric mucosa was, at the autopsy, found to be the only organic disease existing. It is conceivable that the intestine can not persistently digest an amount of proteid sufficient to maintain the nitrogen equilibrium during work; that it depends upon a certain

part of this proteolysis to be performed by the stomach; that the acid gastric chyme is necessary for the stimulation of the duodenal secretions. It is probable that digestion in the duodenum is not perfect without this acid chyme, which, as we know, certainly causes chemical changes in the bile.

So we take the ground that the supplementing of HCl is rational, even if we can not supply the deficit, because the amount necessary thereto could not expediently be administered. If we can not always add sufficient HCl to make the chyme distinctly acid, we can at least add enough to disinfect it and free it from a part, the surplus, of its germs, and perhaps produce some of the preliminary stages to peptone; for the acid albumins (syntonin), we believe, are of some further utility in duodenal digestion. In some cases, however, we are enabled to add enough to give the reaction of free HCl to the chyme.

According to Honigmann and von Noorden (*Zeitschr. f. klin. Medicin*, Bd. XIII), one part by weight of pure HCl is able to saturate 18 parts by weight of egg-albumen; 100 drops of dilute hydrochloric acid, containing 12.5 per cent. of the absolute HCl, will suffice to digest 15 gm., or 225 grs.—little less than four drams of pure egg-albumen. Riegel cites this statement (*loc. cit.*, p. 258), evidently to show how inefficacious 100 drops of a 12.5 per cent. solution of HCl are as a digestive. (The dilute hydrochloric acid of the U. S. Pharmacopeia is a ten per cent. solution.)

The conclusions of Honigmann and von Noorden, however, are, in our opinion, not calculated to inspire therapeutic skepticism; four drams of dried egg-albumen is a considerable quantity to be relieved of, and it can not fail to ease gastric digestion to give the acid, even if it can do no more work than this.

Riegel himself succeeded in causing a resumption of secretion of HCl in a patient who had not shown any for months, after he had taken 1.5 gm. of hydrochloric acid daily for fourteen days. He believes, however, that diet and lavage may have had much to do with the recovery.

Reichmann and Mintz (*Wiener klin. Wochenschr.*, 1892) report several cases in which free HCl could be again demonstrated after it had been missing for a prolonged time; the resumption of HCl secretion was attributed by them to a prolonged dosage with the same acid. As we shall see in the chapter on "Achylia," this disease may depend on a number of very different factors. Some-

times there is no evidence of pathological change in the mucosa, and naturally these may readily recover (neuroses), even without HCl treatment.

Professor Biedert claims to have used 120 drops of dilute HCl daily for a number of years, with much benefit (Biedert and Langermann, "Diätetik u. Kochbuch f. Magenkranke," 1895) to his achylia. Hänni introduced into the stomach 400 c.c. of a 2.5 per 1000 solution of HCl, containing also two gm. of pepsin, together with an Ewald test-breakfast. As early as fifteen minutes afterward, when some of the test-meal was withdrawn, the free HCl had completely disappeared and the digestive power of the sample was equal to zero (Hänni, *Zeitschr. f. klin. Med.*, Bd. XIX, Supplement, p. 307); and Boas cites this statement to show that the digestive value of HCl therapy is doubtful. Now, a patient who gets rid of 400 c.c. of liquid in fifteen minutes has hypermotility; so much could not possibly be absorbed in that short period. Nor could all of one gm. of absolute HCl which 400 c.c. of a 2.5 per 1000 solution contain enter into combination with the proteid of a single roll; for 100 gm. of wheat roll contain only seven gm. of nitrogenous or HCl-binding materials. We know, however, that one gm. of absolute HCl can digest 18 gm. of dried egg-albumen. Therefore the 400 c.c. had probably all been rapidly expelled into the duodenum before they could even be thoroughly triturated with the test-breakfast. Now, does this occur normally, and are we justified in drawing conclusions from such hyperkinetic cases regarding the value of HCl therapy?

As the amount of absolute HCl introduced in Hänni's experiments equaled one gm., and as so much could not enter into combination with the proteid of one roll, or 100 gm. of wheat bread, it stands to reason that if the motility had not been so exaggerated, some of the HCl would have been regained. Whenever Henne (*loc. cit.*, p. 306) succeeded in regaining some of the solution of HCl after it had remained in the stomach forty-five to sixty minutes, or even thirty minutes (see cases No. 3, Schmid, and No. 4, Hänni, p. 307, *loc. cit.*), the tests for HCl were positive and fibrin was well digested by the filtrate. The experiments of von Mehring, Moritz, and myself apparently agree in permitting the deduction that fifteen minutes is an abnormally rapid time for the expulsion of 400 c.c. of liquid (even if it were only water) into the duodenum, and whenever there is a fuller meal given than a simple test-

breakfast it does not occur, because solid and semi-solid matter can not be moved out so readily. Again, we must make allowance for a certain unavoidable nervous tension, and for the influence of suggestion, which takes hold of patients under experimentation, and which, from experience, we know has a decided influence on the rate of peristalsis.

A careful series of analyses, constituting a rational basis for HCl therapy, are those of Charles E. Simon ("The Modern Aspect of Indicanuria," *Amer. Jour. Med. Sciences*, Aug., 1895, p. 170). We submit a number of his conclusions:

"(1) The gastric juice possesses antiseptic and germicidal properties.

"(2) These properties are referable to the presence of free hydrochloric acid.

"(3) A subnormal amount of free hydrochloric acid will call forth an increased degree of intestinal putrefaction.

"(4) The conjugate sulphates form an index of the degree of intestinal putrefaction.

"(5) The increased intestinal putrefaction in cases of subacidity and anacidity of the gastric juice is largely referable to an increased formation of indol.

"(6) The elimination of indican in the urine may be regarded as an index to the amount of free hydrochloric acid present.

"(7) A normal acidity of the gastric juice is never associated with increased indicanuria.

"(8) Cases of ulcer of the stomach apparently form an exception to this rule, an increased indicanuria being usually associated with hyperchlorhydria.

"(9) In other cases of hyperchlorhydria a subnormal or normal amount of indican is eliminated."

We therefore recommend hydrochloric acid, believing in its efficacy in supplementing the digestive work of the stomach. Whenever it is indicated we usually give 20 drops of the diluted HCl (U. S. Pharm.) in two ounces of water every half-hour, beginning fifteen minutes *before* the meal; then 20 drops are taken during the eating, and 20 drops one half-hour after the meal. The medicine should always be taken through a glass tube, and the mouth rinsed with a weak solution of sodium carbonate afterward. As a remedy for improving the appetite, HCl is conceded, even by those skeptical of its digestive power, to be of

value. For this purpose it is best given in small doses diluted with water (10 to 20 drops in ℥iij H_2O), on an empty stomach, before meals. With regard to its disinfectant and anti-fermentative effect we entertain serious doubts, since it can not be given in sufficient quantity to be of much benefit in that direction when given with meals. Whenever there are decided fermentations in the stomach, lavage is the most effective means of combatting it, and for this purpose HCl in the form of a six per cent. solution may be used.

Hydrochloric acid is contraindicated when the normal gastric secretion is augmented. We have observed cases in which there was no free HCl to be detected by Congo-paper or phloroglucin-vanillin, but HCl given per os produced gastric distress and pain; so that there can be no doubt that cases of hyperesthesia toward HCl exist analagous to those described by Talma (*Zeitschr. f. klin. Med.*, Bd. VIII), which do not depend upon hyperchylia. One lady patient could detect whenever three drops of the diluted acid were given surreptitiously in the meals or medicine by the gastralgia caused thereby. This was a highly neuropathic case.

The amount of HCl consumed in the digestion of albumin has been very carefully studied by Fleischer. It takes 0.05 gm. of HCl to transform one gm. of egg-albumen into acid albumin. As human beings frequently take in 150 gm. of egg-albumen in twenty-four hours, it would require 7.5 gm. of pure HCl. (or 30 gm. of the 25 per cent. solution of the laboratories) to transform this amount into acid albumin. As the gastric juice contains HCl to the amount of two per mille, 3½ liters of gastric juice would be required to digest that amount of egg-albumen. Many children consume about one liter of milk daily; this would require 4.5 gm. of pure HCl or 18 gm. of a 25 per cent. solution of HCl. (100 gm. of cows' milk combined with 0.45 gm. of HCl). These amounts of HCl would bring the ingested albumen only to the stage of acid albumin or syntonin; but as hemialbuminose and peptone would require twice the amount of HCl, the quantity combined with must eventually be increased beyond the figures stated. A portion of the albuminous foods passes over into the intestine, however, and is there digested long before it reaches the stage of hemialbuminose; but when the transit of the food into the duodenum is obstructed, it is evident that enormous quantities of HCl must be secreted to digest all the albumin that is taken in. For a

purely physiological reason, it is not possible that the glandular layer can secrete the requisite amount. The absence of free HCl in these cases may be due to an invasion of the mucosa by the disease causing the obstruction. It is not impossible, however, that, even without this invasion, the mucosa has become exhausted, its secretory function being paralyzed.

The Alkalies.—Probably the earliest experiments upon the effect of alkalies on the gastric secretion are those by Claude Bernard, who found that in small doses they increased the secretion of, and in large doses they neutralized, the gastric juice in animals.

Leube (in von Ziemssen's "Handbuch," Bd. VII) stated, as a result of experiments on dogs with gastric fistulæ, that the carbonate of soda of the Carlsbad springs not only neutralized an excess of acid, but could cause a lasting increase in the HCl formation of a diseased mucosa.

Du Mesnil (*Deutsch. med. Wochenschr.*, 1892), and Linossier and Lemoine (Academie de Médecin de Paris, session of March, 1893) agree in stating that when sodium bicarbonate is given together with a test-breakfast, or shortly before it, it acts as an excitant to the mucosa and increases the percentage of HCl formed. Du Mesnil found in a case of hyperacidity that the amount of HCl was at once reduced. Indeed, the results of various experimenters differ according to the normal or abnormal state of the stomach with which they worked. It makes much difference, also, whether an alkali is given on an empty stomach, with very little or no secretion, when it may possibly act as an irritant to the mucosa and set up a secretory reaction, or whether it is given at the height of digestion and meets with free HCl; in the latter case it must of necessity combine with the acid, and can cause no further secretion.

It is unfortunate for the evolution of truth in this question—that is, whether or not small doses of alkali can stimulate secretion—that quite a number of experimenters (Ewald and Sandberg, Leube, Spitzer, etc.) worked with Carlsbad salts or water instead of with a chemically pure simple salt. The people of other countries do not share that intense interest in the Carlsbad and other springs with the physicians of Europe; or at least those of Germany and Austria.

There are not a few prominent representatives among the German clinicians who have expressed grave doubts whether the

cures and improvements reported are really due to the waters of Carlsbad, but that the credit must be given to the avoidance of bad home influences, the careful diet, the regular life, pure air, good sleep, and abstinence from alcohol (see p. 302). Personally, we consider it our duty to emphasize that the waters of the Congress and Hathorn Springs, of Saratoga, N. Y., and of the Bedford Springs, in Pennsylvania, have produced cures, and, where they were not possible, great alleviation of gastric diseases there treated. But even here it is impossible to ignore the good which the strict observance of the factors of hygiene and diet above mentioned may have worked.

The natural Carlsbad Sprudel salt has the following composition, according to Prof. E. Ludwig :

Sodium sulphate,	41.62 per cent.
Potassium sulphate,	3.31 "
Sodium bicarbonate,	36.11 "
Lithium carbonate,	0.2 "
Sodium chlorid,	18.19 "
Sodium borate,	0.03 "
Water,	0.44 "

The artificial Carlsbad salt, which, according to Boas, can fully replace the more expensive natural salt, contains the following salts, according to the German Pharmacopeia :

Sodium sulphate (dried),	44 parts.
Potassium sulphate,	2 "
Sodium chlorid,	18 "
Sodium bicarbonate,	36 "

In hyperchylia and hypersecretion (in gastric ulcer) it is given in doses of one to two dessertspoonfuls in ¼ of a liter of water, to reduce the excess of HCl and promote evacuation. In gastritis it is also recommended, and this has seemingly given the Carlsbad enthusiasts much difficulty, namely, to explain how the identical solution may produce reduction of the HCl, and in another case promote HCl formation.

We have studied eight cases who went to Carlsbad suffering from subacidity and from achylia ; we have not, in a single instance, observed a return of secretion where it was lost or an increase where it was deficiently formed.

Reichmann is one of the few who objected to applying the deductions found with Carlsbad water or salts, on account of their com-

plexity to the effects of pure, simple alkaline salts (*Therapeut. Monatshefte*, 1895). We have never shared the opinions of those who believe that small doses of alkali given on an empty stomach can produce a reactive secretion of HCl which may exceed the amount necessary to combine with the alkali given. We can understand that strong solutions of sodium and potassium sulphate, such as the Carlsbad water, may actually play the rôle of an irritant, to which the mucosa responds in the form of an increased secretion, just as the nasal mucosa would do if a crystal of salt were placed in the nasal passage. Indeed, N. Reichmann declares, after a series of careful analyses with Na_2CO_3, that the bicarbonate of sodium *does not act upon the secretory mechanism of the stomach, but only upon the juice already secreted, by neutralizing it and rendering the gastric contents alkaline* (Boas, *Archiv f. Verdauungskrankh.*, vol. 1, p. 44).

The actual therapeutic application of alkalies is limited to those dyspepsias associated with increased HCl formation, in simple neurasthenic hyperchylia in hypersecretion and in gastric ulcer. They are indispensable for lavage when it becomes necessary to neutralize acids and dissolve adherent mucus. The time to give alkalies in hyperacidity is from one-half to one hour after meals, when the HCl secretion is quantitatively at its height. The sensations of the patient are a very good guide, and the time can be learned by experience; the alkali should then be given a little previous to the time when the gastralgia, eructation, pyrosis, and distention set in. In hypersecretion there is a large amount of HCl present almost continuously in the empty stomach, in addition to hyperacidity after meals, so here we should give alkalies before meals in order to insure a certain time for action to the ptyalin; for this constant secretion a glass of Saratoga Vichy, or simply sodium bicarbonate ʒj, in plain cold water, before meals, is sufficient to permit amylolysis. In ulcer and chronic gastritis acida, alkalies find application also (refer to treatment of these diseases).

Determination of the Amounts of Alkalies Required.—Two groups of these bodies are in common use: (1) The alkaline earths, (2) the alkaline carbonates. Of the first group, magnesia usta or calcined magnesia, and the more expensive magnesium ammonium phosphate are the favorites; and of the second, the sodium carbonate and bicarbonate. Those alkalies which are capable of combining with the largest amount of HCl are preferable. It is expedient to avoid excess of sodium bicarbonate, because the liberation

of CO_2 in the neutralization may cause annoying distention of muscular walls already infirm.

Magnesia usta has the greatest binding power for HCl, and the reaction is expressed in the following equation:

$$MgO + 2HCl = MgCl_2 + 2H_2O.$$

Here 0.55 equivalents of MgO correspond to one equivalent of HCl.

The reaction with ammonio magnesium phosphate is the following:

$$Mg(NH_4)PO_4 + 3HCl = MgCl_2 + NH_4Cl + H_3PO_4.$$

Calculation here gives the result that 1.25 parts by weight of $Mg(NH_4)PO_4$ correspond to one part by weight of HCl.

The reaction with sodium bicarbonate is as follows:

$$NaHCO_3 + HCl = NaCl + H_2O + CO_2.$$

Calculation of the molecular weights shows that 2.3 equivalents of $NaHCO_3$ correspond to one of HCl.

According to Boas, the dose of sodium bicarbonate necessary to counteract a superacidity exceeding 2.5 pro 1000 is eight to ten gm., or four to six gm. of ammonio magnesium phosphate, or two to three gm. of magnesia usta. With an acidity of three pro 1000 HCl, the $NaHCO_3$ can be increased to 12 gm., the ammonio magnesium phosphate to 7.5 gm., and the magnesia usta to five gm. These calculations are made upon an amount of stomach contents equal to 400 c.c.; but as a part of the alkali is expelled into the duodenum, another part absorbed, and as the momentary quantity of HCl present can only be reckoned upon, the figures may be too low. With constipation and collection of gas in the intestines the preference is to be given to the magnesia salts. Germain Sée (*Semaine Médicale*, 1890, No. 12) recommends the following:

℞. Sod. bicarb.,
Cretæ præp.,
Magn. carbon., ... āā . . . 0.2 gm. M.
Take at once.

Boas' formula for continued excessive secretion is the following:

		Metric System.	
℞.	Magnesiæ ustæ, ...	15.0	gr. 231.5
	Bismuth. carbon.,		
	Natrii carbon., ... āā	5.0	gr. 77.2
	Extr. belladonnæ,		
	Extr. strychni, ... āā	0.1–0.2	gr. 1.7. M.

Sig.—One teaspoonful three times daily, half an hour after meals.

The amount of HCl secreted should be watched and the alkali discontinued if it becomes normal.

The Bitter Tonics and So-called Stomachic Remedies.— Experience has lent belief that the bitter tonics are agents which stimulate the appetite and the secretory and motor functions of the stomach. They are represented by preparations of condurango, quassia, colombo, gentian, angostura, absinthe, nux vomica and strychnin, creasote, guaiacol, orexin, lupulin, cetrarin, erythrocentaurin, rheum, resorcin, quinia, cinchona. Under certain conditions, HCl, sodium chlorid, and alcohol act as stomachics.

Some writers class sodium bicarbonate among these remedies, upon the supposition that small doses of this alkali may stimulate secretion of gastric juice; this therapy is, in our opinion, fallacious. As a general rule, these medicines are useful to improve the appetite, and as anorexia is mostly found in reduced or lost gastric secretion, the effect upon secretion is apparently the only one that can be attributed to them. What the bitter tonics really effect and how they act is an unsolved problem. There seems to be an absence of scientific exactness in many of the experiments, and a general diffusiveness regarding the special point of inquiry to be solved ("Fragestellung"). Thus, Penzoldt pretends that genuine stomachics must be able to improve all of the gastric functions (Penzoldt, on "Salzsaures Orexin," *Therap. Monatsh.*, 1890, No. 2). Loss of appetite may be present when the functional work is reduced, and then bitter tonics would be indicated; but it may just as well be present with normal or morbidly increased functions when stomachics would do harm. We have had many cases of anorexia with hyperacidity where the appetite returned after the use of bromid of strontium. In dilatations with fermentation the best stomachic is lavage. As every disturbed function or disease requires elucidation, so the anorexia based thereon demands its own adapted treatment. Bitter tonics and allied medication are, in general, stimulants to the mucosa, and although they have a large application, it is not rational to use them empirically. A sedative or an antiseptic may, under certain conditions, be a better medicine than the bitter tonic for anorexia. The most rational course to pursue is to ascertain the exact state of the gastric functions, and after the establishment of the diagnosis attempt to remove the cause of the anorexia, whether it is depressed motility, dilatation, or

achylia. For a fuller account of the physiological effects of these remedies the reader is referred to recent works on pharmacology and therapeutics (Hare, H. C. Wood, Remington, Sidney Ringer; Schmiedeberg's "Arzneimittellehre," Binz, "Pharmakologie"; the chapters on this subject in the text-books of Riegel, Ewald, Boas, Rosenheim, and Wegele).

Perhaps the most useful medicines of this class, in our experience, have been strychnin and condurango, which, according to the experiments of L. Wolff (*Zeitschr. f. klin. Med.*, Bd. XVI, S. 222), have no appreciable effect on the rate of secretion. Reichmann's very carefully conducted investigations (*Zeitschr. f. klin. Med.*, Bd. XIV, Heft 1 und 2) brought out the fact that some bitter tonics failed to cause any secretion of gastric juice when distilled water did. And whenever water failed to produce secretion the bitter remedies failed also. On normal digestive processes these agents have no effect; but when a juice was secreted that was acid, though not containing HCl, and if a gastric juice very weak in pepsin was secreted, then the bitter tonics, especially absinthe, were found to produce a stronger degree of acid and distinct reaction for HCl. Wherever there was atrophy of the glandular apparatus, all of these remedies failed to cause a secretion of HCl containing gastric juice. In brief, his conclusions are that the effect is very variable, sometimes less than that of water; but sometimes there is an increase of secretion after the bitter tonic has become absorbed and disappeared. They act best when given before meals, and when there is a gastric secretion still present but much reduced (hypochylia). In hypersecretion Reichmann found that the acidity was still further increased by bitter tonics. Riegel concludes that the bitter tonics should be given only in hypochylia or subacidity, and then one-half hour before meals. Our own formula for anorexia from hypochylia is the following:

		Metric System.		
R.	Strychnin sulphatis, ...	0.020	gr. ⅓	
	Acidi hydrochloric. dil., ...	14.787	f℥ ss	
	Extr. condurango, ...	45.361	f℥ jss	
	Elixir gentian, ... q. s. ad, . . .	177.442	f℥ vi.	M.

Sig.—f℥ ss. in ℥ ij aquæ, one half-hour before meals, through a glass tube.

Where there are evidences of anemia, with the hypochylia, the following acts satisfactorily:

		Metric System.		
℞.	Quinæ sulphatis,	1.193	gr. xviij	
	Strychnin sulphatis,	0.020	gr. ⅓	
	Ferri sulphatis,	0.775	gr. xij	
	Acidi arseniosi,	0.012+	gr. ⅕.	M.

SIG.—Fiant pil. No. xii. One pill, three times daily (must be prepared fresh and not coated).

Boas uses the following powder for anorexia:

		Metric System.	
℞.	Extr. strychn.,	0.03 to 0.05	gr. $\frac{7}{116}$–$\frac{9}{116}$
	Bismuth. carbon.,	0.5	gr. .43–.56
	M. f. pulv. Dent. tal. dos. xx.		gr. .56–$\frac{9}{116}$.

SIG.—One powder, three times daily.

Menche has warmly recommended resorcin sublimate, and it undeniably improves the appetite in cases of incipient gastric fermentation. It has also a slight sedative action. The following is Menche's formula:

		Metric System.	
℞.	Resorcin. resubl.,	2.0	gr. 30.5
	Acid. mur.,	1.0	gr. 15.4
	(Or if it be indicated in place of the HCl, one may order Natr. bicarb., 8.0.)		
	Aquæ destil.,	180.0	f℥vj
	Syr. simpl.,	20.0	℥iij.
	M. D. et ad vitr. nigr.		

SIG.—15 c.c. (℥ss) every two hours.

The following formulæ are recommended by Ewald for anorexia with fermentation:

		Metric System.		
℞.	Tinct. nucis vom.,	25.0	f℥vj	
	Resorcin. resublim.,	5.0	gr. 81	
	Tinct. amar.,	10.0	f℥iij.	M.
	Take 10 to 15 drops every two hours.			
℞.	Ext. condurango, fl.,	16.0	f℥ivss	
	Resorcin. resublim.,	4.0	℥i.	M.

SIG.—30 drops, four times daily.

Creasote is a remedy of doubtful efficacy in our experience, as it rarely benefits digestion except in tuberculous patients. Wegele says (*loc. cit.*, p. 53) that it will help if it is tolerated and causes no severe dyspeptic difficulties, but that is just what it will do in about

one-half of the cases. We have our doubts whether it can promote peristalsis, as is asserted by Klemperer (*Centralbl. f. klin. Med.*, 1891, No. 21), until enough is given to act as an irritant. Even when it is tolerated by the stomach, the repeated penetrating eructations are very annoying to nervous patients. Sommerbrodt recommended it to be taken in capsules. Bouchard advises the following formula:

	Metric System.	
℞. Creosot. puriss.,	13.5	
Tinct. gent.,	20.0	
Vin. Xerens,	800.0	
Spir. rectif.,	200.0	M.

SIG.—℥ ss four times daily.

Orexin (phenyldihydrochinazolin) has been strongly indorsed as a "genuine" stomachic by Penzoldt. In 273 cases he observed 144 successful restorations of appetite and secretion. Its special indications are gastric atony and beginning gastritis, and its action is attributed to its power of increasing the secretion of HCl (Penzoldt, "Weitere Mittheilungen über Orexin basicum," etc., *Therap. Monatshefte*, May, 1893.) The following formula is advised by Penzoldt for this useful drug:

℞. Orexin basic., ... ℥ ss. or 2 gm.

SIG.—Divide into six powders; inclose in small wafers. One to be taken in a cup of bouillon half an hour before meals, twice daily.

Digestive Ferments.—Artificial means of aiding digestion are certainly much abused, and if employed for long periods they frequently become, to a certain extent, injurious. Every organ is strengthened by activity and weakened by lack of exercise. The stomach will grow weaker and weaker the more artificial gastric juice is poured into it, and the finer and more subtle the nourishments are that are allotted to it (Einhorn). This agrees in the main with what we stated under "Dietetics," namely, that the diet should not be *leveled down* to the digestive capabilities of the stomach, but that digestion should be *leveled up* until it can deal efficiently with the amount of food required for the nitrogen equilibrium. In truth, the indiscriminate dosing with digestive ferments does more harm than good. The stomach is an organ which very rapidly adapts itself to cease performing the work that is done for it artificially. Then, again, there is such a thing as educating an apparently weak stomach up to digesting food which at first seems indigestible, and is taken with "fear and trembling." So we will

find that gastric training by graded diet may favor the development of what fragments of glandular elements may yet be slumbering in a diseased mucosa, but the irrational use of ferments may, by doing all the work itself, permit the gland-cells to go on to atrophy.

The artificial ferments have been recommended where there is a deficiency or absence of the natural secretion. They may be considered in two classes: (1) Those that have been isolated from the mammalian organism, viz., ptyalin, pepsin, pancreatin; and (2) those derived from the vegetable kingdom, viz., the various diastases, papain, bromilin. Some of the ferments of the human body have not yet been isolated; these are the milk-curdling ferments of the gastric and pancreatic juices, and the emulsifying and fat-splitting ferments. There are ferments in the succus entericus (invertin, etc., perhaps a curdling ferment) which we understand very little.

Ptyalin.—This ferment of the saliva is indicated in hyperacidity and hypersecretion, when the normal ptyalin may actually be destroyed. Boas has shown that with diminution of the acidity this ferment may, to a degree at least, resume its inversion of starches into dextrose. With very intense hyperacidity (0.04 pro 1000) the ferment appears to be so injured that it can not be restored to function, and a new supply may be necessary. Ptyalin is given in doses of 5 to 15 grs., with ℈j of sodium bicarbonate, immediately after meals. There can be no doubt of the greater amount of dextrose formed with the aid of ptyalin, and these patients are thereby enabled to eat more of carbohydrates.

Diastase.—Malt diastase, as manufactured in the form of liquid extract, or in dry form, as in Horlick's diastoid, is serviceable for the same purpose. Recently, Professor Leo, of Bonn (*Therap. Monatshefte*, Dec., 1896), reported to the Congress of German Naturalists and Physicians on taka diastase, an American product, which appears to have strong starch-inverting power, and to be able to act in an amount of acid equal to 0.1 per cent. HCl. We have assured ourselves that amylolysis is effectively carried out by this taka diastase, but the addition of an alkali is necessary, as with ptyalin, to render the effect prompt. Its tastelessness and moderate price are in its favor.

Ewald has found in a great many observations that absence or deficiency of ptyalin is an exceedingly rare thing; so that ptyalin is rarely required, because it is secreted in sufficient quantity,

but in some way is destroyed. The hygiene of the mouth should receive careful attention; a septic or acid mouth, with coated tongue and bad teeth, will offset any amount of ptyalin. To treat "amylaceous dyspepsia"—which is the objectionable name given to symptoms of hyperacidity and hypersecretion—by cutting off the carbohydrates, is irrational, because they can not be dispensed with, not on account of the starch only, but on account of the proteid which amylaceous foods contain. It will be found from the army rations of men under service of various nations that the carbohydrate portion of the foods is increased with harder work much more than the proteid and fat portion (see tables in Gilman Thompson's "Dietetics" and Munk and Uffelmann's "Ernährung des Menschen"). Therefore these foods should not be taken away, because they may not be perfectly digested; but the cause of the indigestion should be, if possible, removed. If possible, a large amount of natural saliva should be swallowed after meals; many times have we observed that, with the simple supply of additional saliva caused by chewing a piece of rubber, starch indigestion would not be demonstrated in the test-meal, although it had existed before. To Fothergill is attributed the saying that ferments are crutches; no doubt many an invalid would prefer walking on crutches than not at all. There are many crutch-walkers, however, who, by modern surgery, have been enabled to throw them away and walk by themselves unaided. Just so with the ferments; they may be used with success temporarily, but the best thing to do is to discover how the patient may digest without them.

Pepsin.—There is no lack of pepsin preparations in the market and their digestive powers as claimed, seemingly have no limit. Certain very popular compositions of pepsin should be emphatically condemned. For instance, all wines of pepsin are inefficient because very little of this ferment is taken up by alcohol. Recently, a preparation was brought to our laboratory containing hydrastis, rhubarb, pepsin, and pancreatin in one solution, showing a total disregard for the physiological fact that pepsin acts only in an acid and pancreatin in an alkaline medium. There is rarely any indication for the use of pepsin, for whenever a test-meal shows free HCl, pepsin must of necessity be present in sufficient amounts; and even when HCl is absent, pepsin or pepsinogen are, as a rule, still present. Assuming a case in which the last vestige of even pepsinogen secretion has been lost, the introduction of the ferment

might be of utility, but the enormous quantities of HCl necessary to bring about proper action of this pepsin could not be tolerated by any stomach (see chapter on the Therapy of HCl). And, again, in cases where pepsinogen is still formed, the addition of HCl simply, will suffice to convert it into the complete ferment. Pepsin is prescribed much too often.

Pancreatin.—Although there are many preparations of this ferment, and some of them very active, the substance spoils and loses its digestive power with age. As it is an easy matter to test its amylolytic and tryptic power in artificial digestion experiments, it is wise to do so in all cases where much dependence is placed in its action. The nature and value of the substance was scientifically explained by Sir William Roberts ("Digestion and Diet," p. 66). It can be obtained in a liquid form as well as in the form of a dry powder, from extraction of the pancreatic gland of animals. This ferment is completely destroyed in the gastric juice. This is why thinking practitioners should not use both pepsin and pancreatin together in the same solution, because the medium in which one must act is opposed to that of the other. In the majority of cases where pancreatin is given empirically, HCl is still secreted in the gastric cavity and the ferment is destroyed. Gilman Thompson (*loc. cit.*, p. 333) suggests that the pancreatin be inclosed in keratin capsules, keratin is unaffected by gastric juice, but readily dissolves, it is claimed, in alkaline media. Hence the pancreatin may pass through gastric digestion, and at its completion pass into the intestine where the coating is dissolved and the ferment acts upon the chyle. This idea is not supported by experiment nor by exact indications of the conditions for the employment of pancreatin. Keratin will not dissolve in the alkalinity of the duodenum except very slowly; pills coated therewith are found in the stools during normal digestion. There is no necessity for attempting to supply the ferment directly to the duodenum, since in the greater majority of cases, perhaps all, except where malignant neoplasm, cirrhosis, or abscess have destroyed the gland, there is plenty of pancreatic juice in that part of the bowel. In exceedingly rare cases pancreatic calculi and diseased states of adjacent parts may stenose the duct. The suggestion of Thompson was previously carried out by Unna. In all these attempts it is overlooked that the reaction of the normal duodenum is acid and will not permit the solution of keratin.

There is but one distinct indication for the use of pancreatin, and

that is permanent deficiency or complete absence of HCl and enzyme formation in the stomach. Experiment and experience have conclusively shown that when pancreatic digestion is in these cases started in the stomach, by giving the pancreatin with sodium bicarbonate, there is a more exhaustive utilization of the proteids and carbohydrates. We have frequently assured ourselves of this fact by analyzing the stools after weighed amounts of these food-substances had been ingested, at the same time making identical analyses with the same amounts of proteid and carbohydrate, but with pepsin hydrochloric acid as an artificial digestant; under the latter more food-substances passed through undigested than when pancreatin was used.

Pepsin and hydrochloric acid naturally suggest themselves in atrophic gastritis, but judging from our observations pancreatin is preferable. We have noticed cases in which there was a remarkable hypersensitiveness to hydrochloric acid even in doses of six drops of the dilute form, so that its use had to be dispensed with. The dose of pancreatin is from four to eight grs., together with the same amount of sodium bicarbonate in form of compressed tablets; of these, two to four are taken fifteen minutes after meals.

Papain, Papoid, Papayotin.—These ferment-containing substances are made from the milky juice of a tree belonging to the family of Papayaceæ, native in Central and South America.

Bouchut and Wurtz ("Sur la ferment digestiv du Carica Papaya," *Compt. Rend.*, 1879, tome LXXXIX) first prepared papain, and later Peckolt brought out papayotin. Papoid, an American preparation, according to Prof. R. H. Chittenden, is a vegetable ferment made up of vegetable globulin, albumoses, and peptone, with which are associated the ferments characteristic of the preparation. Papoid has the power of digesting to a greater or less extent all forms of proteid or albuminous matter, both coagulated and uncoagulated; its digestive power is exercised in a neutral, acid, as well as alkaline medium. Papoid is found in the stools, showing that it is not destroyed in the alimentary canal; the dose is from one to three grs. after each meal.

Finkler prefers papain to pepsin for aiding gastric digestion (*Therap. Gazette*, 1887, August 15), and G. Littmann has observed good results with it in acute and chronic gastritis, dilatations, carcinoma, and dyspepsia after chronic ulcer (Littmann, *Münch. med. Wochenschr.*, 1893, No. 29).

Papain seems to be a variable product and its digestive action not always the same (Rossbach and A. Eulenberg). It is an expensive preparation. Recently a highly concentrated extract of carica papaya has been brought into the market under the name of *Caroid*, which, according to Chittenden, has even a greater digestive power than papoid, and digests proteids, albumins, and starches in any medium. We append Chittenden's results with this energetic ferment, concerning the clinical application of which further observations are necessary:

With 0.05 per cent. hydrochloric acid.

	Undigested residue.	*Proteid digested.*
Caroid,	0.8024 gm.	20.2 per cent.
Papain, A.,	0.8959 "	10.9 "
Papain, B.,	0.8735 "	13.1 "

With 0.5 per cent sodium bicarbonate.

	Undigested residue.	*Proteid digested.*
Caroid,	0.4596 gm.	54.3 per cent.
Papain, A.,	0.5691 "	43.4 "
Papain, B.,	0.5927 "	41.0 "

If we examine these results critically it is plain that the digestive power of caroid on proteid matter is greater than that of the other two preparations. The difference in digestive strength is more apparent in these experiments with coagulated egg-albumen than with the other form of proteid matter, although quite marked with blood-fibrin.

2. *Starch-digesting Power.*—In starch-digesting power caroid is far superior to the other preparations, either papoid or papain. The following experiments will throw some light upon this point.

A starch paste was made from five gm. of dry arrowroot starch with 500 c.c. of water. Mixtures were then prepared as follows:

1. 0.5 gm. of caroid, + 90 c.c. water + 10 c.c. of starch paste.
2. 0.5 " " papoid, + " " + " " " "
3. 0.5 " " papain, A., + " " + " " " "
4. 0.5 " " papain, B., + " " + " " " "

These four mixtures were placed at 40° C., and tested from time to time with iodin solution. In five minutes No. 1 had reached the achromic point, while No. 2 did not give the achromic reaction until at the end of two hours. At the end of three hours, Nos. 3 and 4 still gave a bluish-violet reaction with iodin.

In another series of experiments, exactly similar to the above,

except that each mixture contained only 0.2 gm. of ferment, the caroid brought about a complete conversion of the starch into bodies noncolorable by iodin in eighteen minutes, while the others gave a blue reaction after two to three hours.

The presence of alkalies retards the diastatic or amylolytic action, but the caroid shows throughout very much greater amylolytic power than the other preparations.

The Ferments of the Pineapple.—The fruit contains very active proteolytic ferments, its juice being used in the production of the artificial predigested beef foods by a prominent American firm. These ferments are destroyed by boiling, and hence are no longer active in the preserved fruit. We have assured ourselves sufficiently of the proteolytic activity of raw, fresh pineapple juice to recommend it in achylia or subacidity, and to forbid its use in hyperacidity and hypersecretion, as well as in gastritis acida. It is allowed mainly because of its pleasant taste and because it stimulates desire for more food. The fiber must be removed from the mouth and not swallowed by gastric patients.

CHAPTER VII.

SURGICAL TREATMENT OF ORGANIC GASTRIC DISEASES.

In the pre-antiseptic time the stomach was regarded as a "Noli me tangere." Even in the beginning of this century gastric wounds were considered as directly fatal, and Larrey, the Surgeon-General of Napoleon, was one of the first to declare, "Plaies de l'estomac ne sont pas mortelles dans tous les cas" (*cf. Clinique Chirurg.*, tome IV, p. 10), which was, as is well known, confirmed by the notable observations of our countryman, Beaumont on Alexis St. Martin. Not only were surgeons timid about the almost unavoidable peritonitis, but there existed a universal belief that the solving and peptonizing action of the gastric juice prevented the wound from healing. The observation that undoubted gastric ulcers had healed, and that fistulæ produced by physiologists in animals healed spontaneously, and that gastrotomy and gastrostomy performed in pre-antiseptic years had not shown the corrosive effect of the gastric juice,—paved the way for experiments by Gussenbauer and von Winniwarter, proving that gastric wounds, when sutured, healed, as a rule, without interference from any digestive action of gastric juice.

The first proposition to treat organic gastric diseases by operation was made by Merrem, who originated the resection of the pylorus (pylorectomy), and, after performing it on dogs, suggested it for human beings (Dan'l C. Theodor Merrem, "Animadversiones quædam chirurg. experim.," etc., Giessæ, 1810).

Gussenbauer and von Winniwarter, later on, demonstrated that this operation was technically feasible, and that removal of the pylorus was not dangerous to life (*von Langenbeck's Archiv*, Bd. XIX, S. 347). They succeeded in showing that a certain per cent. of cases of pyloric carcinoma were indications for this operation. Czerny and Kaiser confirmed these opinions, and the latter managed to heal and keep alive a dog from whom he had almost entirely excised the stomach. As a surgical curiosity, Haberkant (*Arch. f. klin. Chirurg.*, Bd. LI, Heft III, S. 484) mentions a total extirpation of the stomach

by Dr. Connor, of Cincinnati, in a woman fifty years of age, who died before the esophagus could be united to the duodenum. According to Rydygier, a surgeon named Torelli, in 1878, executed the first gastric resection in a man, removing a piece 16 cm. long that had prolapsed from an abdominal stab wound (*Centralbl. f. Chirurg.*, 1879, S. 398). In the same year Billroth brought about healing of a gastric fistula by exposing the stomach and suturing it (*Wien. med. Wochenschr.*, 1881, S. 275). In January, 1881, Billroth executed the first successful resection of the pylorus for carcinoma.

The first total resection for ulcer was performed by Rydygier, and the first partial resection for ulcer was made by Czerny in 1882. Both were successful. Péan executed a pyloric resection in 1879, before Billroth, and so did Rydygier in 1880, but both were unsuccessful. In the first publication of Billroth's resection (*Wien. med. Wochenschr.*, 1881, No. 6), Wölfler defined the limits of the usefulness of total resection as existing in the transition of the carcinomatous tumors to the pancreas and duodenum. Cases in which the carcinomatous infiltration extended beyond the hepato-duodenal ligament should be excluded from resection. From these indications the plan to a second operation arose—"gastro-enterostomy," which is a type of entero-anastomosis (Maisoneuve), an artificial communication between the stomach and the jejunum, when the pyloric obstruction, for reasons given, can not be removed. In 1881 (September 28th), Wölfler performed this operation for the first time. But the very next case (performed by Billroth) was fatal, the patient dying with constant emesis of bile. The necropsy showed that the upward traction of the jejunal loop had caused what is termed a "spur," which returned the duodenal secretions (bile, etc.) into the stomach. The spur had divided the artificial gastro-intestinal lumen into two unequal parts, of which the larger belonged to the duodenal canal, the smaller to the jejunal loop leading away from the stomach.

As a necessary result of this the bile and pancreatic juice ran into the stomach, while nothing could pass out into the diminutive discharging outlet. In one of Lauenstein's cases ("Verhandl. d. Deutsch. Gesell. f. Chirurg.," Thirteenth Congress) the mesentery of the jejunal loop which had been drawn up to meet the stomach compressed the transverse colon. The adducent part of the loop was drawn across the colon like a tense ridge. Courvoisier, in 1883, invented another method calculated to avoid these difficulties.

Instead of inserting the jejunum to the ventral or anterior wall of the stomach, he made a slit in the mesentery of the transverse colon and inserted the loop into the posterior gastric wall. In order to secure the continued onward flow of the bile and pancreatic juice through the intestine, Courvoisier attached the adducent part of the intact loop to the stomach for a distance, then split the abducent part, and finally sewed the wound edges of the gastro-intestinal opening.

In 1885 von Hacker described a similar but much more improved method, which consists in the following: The colon and great mesentery are raised upward; the gaping edges of the slit in the mesocolon are attached to the posterior gastric wall; finally, the jejunal loop is attached to the stomach within this slit (von Hacker, "Verhandl. d. Deutsch. Gesell. f. Chirurg.," 1885, Fourteenth Congress).

A third method of gastro-enterostomy was suggested by Billroth and Brenner (*Deutsche Zeitschr. f. Chirurg.*, Bd. xxv, p. 502). In this method, openings are made both through the gastrocolic ligament and mesocolon, through which the jejunal loop was drawn up and sewed to the anterior gastric wall immediately above the greater curvature. Von Hacker has given these various operations very significant and explicit Latin designations ("Chir. Beitr. a. d. Erzherzogin Sophienspital in Wien," S. 42). These are his terms in English:

1. Gastro-enterostomy, anterior, antecolonic (Wölfler).
2. Gastro-enterostomy, posterior, retrocolonic (von Hacker).
3. Gastro-enterostomy, anterior, retrocolonic (Billroth-Brenner).

A number of other modifications must be passed over, since we are interested only in the clinical, not so much in the purely surgical, aspect of the subject.

VARIOUS FORMS OF OPERATIONS PRACTISED UPON THE STOMACH.

Gastrotomy is the operation of opening the stomach with the object of removing a foreign body; then sewing up the wound in the stomach, replacing the viscus, and sewing up the external abdominal wound. This operation must be looked upon as very successful, for, of 18 cases reported by Henry Morris (Ashhurst, "Encyclopedia of Surgery," vol. v, p. 589), 14 recovered.

Gastrostomy is designed to rescue a person from immediate

starvation when there is a stenosis in the esophagus, either from cicatricial contraction resulting from esophageal ulcer, syphilitic, tuberculous or malignant neoplasm, or corrosive toxic agents.

The same causes affecting the cardia—for instance, carcinoma of the cardia—may necessitate gastrostomy. Our experience is, that the sooner gastrostomy is performed in carcinoma of the cardia, the longer is the possibility of life. One should not wait until nothing but liquids will pass the stricture. It has been observed that the carcinoma will improve and show some tendency toward healing when food no longer passes over it and the dilatation above the stricture is kept clean and aseptic by esophageal lavage. Whenever possible, the dilatation should be washed out daily, even after gastrostomy has been performed. In cases where the esophageal stricture had become impassable, it has occasionally been noticed that after gastrostomy the stenosis again became permeable, and food could be swallowed for a while. Witzel has devised an oblique entrance of the fistula into the stomach, making use of the anatomical relations of the abdominal walls for that purpose. The canal is laid partially in the gastric and partially in the abdominal walls, being somewhat tortuous, and mostly closed to food trying to come outward (Witzel, "Z. Technik d. Magenfistelanlegung," *Centralblatt f. Chirurg.*, 1891, No. 32). Von Hacker's technic, as original with him, has been practised in a number of cases for dilating esophageal strictures with sounds introduced from the gastric side, when dilatation of the strictures from the mouth had failed. The unfavorable results of gastrostomy—Zesas reported only 19.5 per cent. of so-called recoveries in 131 operations (*Arch. f. klin. Chirurg.*, Bd. XXXII)—are largely due to postponing the operation until the general health is too low to assist in recovery.

Mikulicz has formulated his latest results in the following table (*Arch. f. klin. Chirurgie*, Bd. LI, p. 9, 1895):

GASTRECTOMY AND GASTROTOMY.

	Total.	Recovered.	Died.	Mortality Percentage.
For simple ulcer,	1	1	0	0.0
For ulcer and hemorrhage,	3	1	2	66.66
For ulcer with perforation,	1	0	1	100.0
Occlusion of pylorus with a gall-stone,	6	1	0	0.0
Total,	11	3	3	50.0

The results in *gastrostomy* for esophageal carcinoma are stated by Mikulicz (*loc cit.*) as follows: Of 28 patients that survived the operation longer than three weeks, 20 subsequently died of the fundamental disease. The shortest duration of life was three and one-half weeks, the longest twelve months, after the operation. The average duration of life after the operation was four and one-half to five months.

GASTROSTOMY.

	Total.	Recovered.	Died.	Mortality Percentage.
Toxic, corrosive stricture of esophagus, . .	9	9	0	0.0
Neurosis (cardiospasm), ...	1	1	0	0.0
Carcinoma of cardia or esophagus, ...	34	28	6	17.64
Total, ...	44	38	6	

Gastrorrhaphy is an operation for closure of wounds of the stomach.

Pylorectomy, or Resection of the Pylorus.*—In considering the value of this operation we must sharply distinguish between three types:

1. Typical, total, or circular pylorectomy.

2. Atypical pylorectomy, which consists of a combination of the former with a gastro-enterostomy.

3. Partial pylorectomy.

Typical or Total Pylorectomy.—Indications in 259 operations were the following: Carcinoma, 215 times; ulcer or cicatrix, 34 times; sarcoma, twice; angioma fibrosum, once; not stated, seven times. In judging of the benefit to be derived from these operations, we must distinguish sharply between (1) the immediate and (2) the remote results. Generally speaking, surgeons term a patient "recovered" when he succeeds in getting over the effects of the operation; this is the immediate result. The remote results are determined by the duration of life after the operation. The immediate results of the 259 cases above enumerated are the following: Of 34 cases of benign stenosis, 23 recovered; of 215 cases of car-

* For the statistics and historical information on the subject of the principal operations we are indebted to an article by Dr. Haberkant in the *Archiv f. klin. Chirurg.*, Bd. LI, p. 861, 1896; and to a report by Prof. J. Mikulicz, *Archiv f. klin. Chirurg.*, Bd. LI, p. 9, 1895.

cinoma, 98 recovered; both cases of sarcoma and the case of angioma fibrosum recovered. Haberkant (*loc. cit.*) found the mortality for ulcer to be 34.4 per cent., and for carcinoma, 56.7 per cent., in a total of 239 operations performed from 1879 to 1894. It is a very important question for the internist whether the mortality is becoming less as time progresses, which signifies an improvement in the technic and knowledge of the subject. Haberkant arranged 205 cases, operated on from 1881 to 1894, in two series of seven years each (from 1881 to 1888, and from 1888 to 1895). In the first series the total mortality was 62.8 per cent.; in the second series it was 45.1 per cent. For carcinoma a reduction of the rate of mortality from 65.4 per cent. to 42.8 per cent., and for benign pyloric stenosis a reduction from 42.8 per cent. to 27.7 per cent., is calculated. In 1882, of 13 cases of resected carcinomata, all died; in 1893, of eight cases, all recovered. There may be some objection to the absolute correctness of these figures, but they undoubtedly admit the belief that our methods of diagnosis and operative technic are improving. Some forms of gastric cancer are much more malignant and unfavorable to treatment than others. In 44 cases in which microscopical examinations were made, we found the following comparisons:

Nature of the Gastric Cancer.	Number of Operations.	Recovered.	Died.
Scirrhus,	16	10	6
Adenocarcinoma (epithelial carcinoma),	10	5	5
Medullary carcinoma,	9	1	8
Colloid carcinoma,	9	7	2
	44	23	21

According to this table, colloid carcinoma is the most favorable to operation, while the most unfavorable prognosis is to be formed of medullary sarcoma.

The remote results are best shown in the duration of life after the operation, which is expressed in the accompanying table (see end of this chapter), from which it is evident that the average expectation of life after pylorectomy for carcinoma is not very long. For of 26 so-called recoveries, or immediate good results, 17, or nearly two-thirds, died within one year after the operation. Furthermore, of 26 (different) cases, 12 died in from one and one-half to thirteen

months from return of the malignant trouble or metastases. One case of Billroth's lived five and a quarter years. One case of Kocher (*Centralbl. f. Chir.*, 1894, S. 221) lived five years and four months, and one case of Ratimmow's (*ibid.*, S. 1014) lived eight years. The boundary of the pathological tissue can not be determined accurately. As is the custom in most malignant neoplasms of other organs, the resection is made by cutting through the apparently or visibly healthy tissue one cm. from the limit of the diseased portion. Czerny, however, found, by careful microscopical examination of resected pieces, that the edges of the section, made through apparently healthy tissue, contained cancerous alveoli; he therefore advised that the cut be made, not one, but three, cm. from the limit of the carcinomatous tissue. Virchow holds that as long as a neoplasm is solitary, the hope for a successful operation must not be given up.

Pylorectomy is the only operation which can make a definite cure or a recovery of some duration possible; and although the prospects of complete cure are very few, we must hold fast to the encouragement which statistics furnish, namely, that more cases recover with improvement in the technic and diagnosis. Haberkant (*loc. cit.*, p. 26) takes too gloomy a view of the future of gastric operations when he asserts that we must expect no advance in the curability of gastric carcinoma, because, in his opinion, patients decide for the operation too late, even after the diagnosis is certain; and, secondly, because it will be impossible to diagnose gastric carcinoma at a time when a cure by extirpation would be possible. The early diagnosis of gastric carcinoma, he emphasizes, is in almost all cases impossible. The surgeon, as a rule, concludes to operate only when distinct stenotic symptoms are present, with emesis, dilatation, and palpable tumor. The only sign which Haberkant cites to be doubtful—that is, the absence of HCl in the gastric contents—is by no means the only one the clinician has to be guided by, as reference to the chapter on diagnosis of gastric carcinoma will show.

There can not be a moment of doubt about the feasability of operation when gastric dilatation is manifestly due to palpable neoplasm, even if it were not malignant. But we generally advise operation when dilatation is associated with cachexia, absence of HCl in the gastric contents, excess of lactic acid, and presence of the Oppler-Boas bacillus. Stenotic symptoms, accompanied with

these signs, are indications for operation, even in the absence of palpable tumor.

Exploratory laparotomy, which Haberkant states to be the *only reliable* means for making an early diagnosis of carcinoma, should be encouraged by the internist, not because carcinoma can be diagnosed with certainty thereby, for it really can not, as the stomach is the seat of many kinds of neoplasms, and even ulcer, with indurated edges, may simulate carcinoma; so that the finding of a new growth does not include a knowledge of its exact nature, and if a carcinoma of small size be at the posterior side of the lesser curvature, it may escape attention even at autopsies until the stomach is removed from the body. The article quoted is an excellent piece of work, and the pessimistic view on the future development of clinical diagnosis need not discourage the internist. For the progress which digestive physiology, pathology, and bacteriology have made in the last twenty years, and are still making, strengthens the belief that we will, in the near future, be able to make early diagnoses of gastric neoplasms. Whether they will be operable or not is another question, concerning which the internist and the surgeon must investigate together.

Even after the diagnosis is certain, much foresight is necessary in selecting cases for operation; the establishment of the indication must be done with exactness and care. That the mortality from cancer resections has sunk from 65.4 per cent., in the period from 1879 to 1887, to 42.8 per cent. in the period from 1888 to 1894; and of benign stenosis from 42.8 per cent. to 27.7 per cent. in the same period, shows that the importance of exact "Indicationsstellung" is being appreciated.

How many pyloric carcinomata are operable? In the records of the Vienna Pathological Institute, from 1817 to 1873, Gussenbauer and von Winiwarter found accounts of 542 pyloric cancers, of which 223 were entirely isolated, and 172 of these showed no adhesions; so 41.1 per cent were free from metastases, and 37.7 per cent. were, in addition to this, free from adhesions—the latter were suited for resection. In many of the instances where the necropsy showed adhesions, there must have been a time when they were not present, so that a big field for operative therapy is opened. Streit found, at the Bern Pathological Institute, that 25.9 per cent. (*Deutsche Zeitschr. f. Chir.*, Bd. XVI), and Kramer (König, "Lehrb. d. spec. Chir.," Bd. II), that 33.3 per cent., of pyloric cancers were operable. The best sta-

tistics state that from one-quarter to one-third of these neoplasms are operable. Adhesions increase the mortality; in 66 cases of pylorectomy in which records were kept concerning this point, the mortality was 72.7 per cent. with, and 27.2 per cent. without, adhesions. No immediate recoveries are on record where there were adhesions of the pylorus with the transverse colon, or with the colon and the pancreas together. Patients with tumors of the curvatures and fundus, although distinctly ascertained to be malignant, if they are causing no stenotic symptoms, so that the vicarious digestion of the intestines maintains the nitrogen equilibrium sufficiently, live longer, in our experience, if they are *not* operated. That is to say, the average duration of life after the date of exact diagnosis was longer in those cases of this type that were not operated than in those that were.

Can the secretory and motor function be restored after total extirpation of a malignant tumor?

Obalinski and Jaworski (*Wien. klin. Wochenschr.*, 1889, No. 5), Rosenheim (*Deutsche med. Wochenschr.*, 1892, No. 49), Känsche (*ibid.*), and Zawadski and Sohnan (*Deutsche med. Wochenschr.*, 1894, No. 8) assert that secretion is not restored by pylorectomy, but the last mentioned authors assert that the motility may again become good. In cases that are operated before a complete destruction of the gland-cells has taken place, the lost secretion of HCl has been observed to be restored. Rosenheim (*Berlin. klin. Wochenschr.*, 1895, No. 1) and Boas (*Deutsche med. Wochenschr.*, 1895, No. 5) have reported the only two cases of this kind, so that it must be an extremely rare occurrence.

Pyloric stenoses caused by simple benign adhesions can be removed by severing the constricting bands. These adhesions may cause pain and hematemesis without gastrectasia, as was shown in a case of Hahn's (*Deutsche med. Wochenschr.*, 1894, No. 43), where laparotomy revealed five adhesions binding the stomach to the colon. He ligated each one of the strong bands doubly and severed it, and from that moment the patient recovered perfectly. Median hernias of the linea alba have been known to cause intense gastric suffering, necessitating operation. Rosenheim has described such cases, which were, however, much benefited by lavage and diet, so that the motor insufficiency was much reduced (*Berlin. klin. Wochenschr.*, 1897, No. 11). Preperitoneal lipomata have been known to cause interference with the motility and necessitate surgical interference.

Adhesions may reunite after intersection and cause renewed trouble, as was shown in one case of Hahn's, in which the adhesions were divided again by W. Levy two years after the first operation; a few months after this Hadra executed a gastro-enterostomy, on Rosenheim's suggestion (*loc. cit.*), which gave no perfect relief, as the two previous operations had caused new adhesions. Referring again to malignant tumors of the lesser or greater curvatures where good motility is maintained, and that cases of this kind which are not operated live longer than those that are, we might add the case of a lady in whom Musser and Da Costa diagnosed a palpable tumor in February, 1896. Personally, we determined the location in July, 1896, when there was complete loss of all secretion and numerous Oppler-Boas bacilli were present in the gastric contents. A fragment of the neoplasm was obtained in September, 1896, during lavage, clinching the diagnosis. With daily lavage with HCl solution (4 : 1000), highly nutritious and concentrated diet, rest, and internal use of HCl, condurango, and strychnin, this patient has gained 12 pounds in six months, and is still (September, 1897) able to take walks of two miles a day, nineteen months after Musser first diagnosed the existence of a gastric tumor.

The number of authoritative advocates of pylorectomy for benign stenosis is growing less and less. Von Hacker recently again emphasized that gastro-enterostomy is not used enough for the treatment of cicatricial stenoses of the pylorus and duodenum, and that it has the value of a radical operation for many cases without sharing its dangers (von Hacker, "Magenoperationen," etc., etc., published by William Braumüller, Wien, 1895). Mintz considers pylorectomy unjustifiable for benign cicatricial stenoses (*Zeitschr. f. klin. Med.*, Bd. xxv). For mild stenotic cicatrices the pyloroplastic operation has proved sufficient. We shall refer to this operation in the following.

Atypical pylorectomy was executed first in 1885 by Billroth. It is a combination of resection of the pylorus with gastro-enterostomy recommended in cases where carcinomata, although operable, had so extensively involved the gastric walls that after resection it was impossible to suture the remainder of the stomach to the duodenum, or where traction upon the duodenum to meet the stomach would produce too much tension upon the stitches.

Von Eiselsberg executed the most extensive atypical resection of the pylorus in a very large but sharply limited carcinoma. His

incision began close to the cardia and descended perpendicularly downward, so that only a small portion of the left fundus remained (*von Langenbeck's Archiv*, Bd. XXXIX). Even this incision was not through healthy tissue, and the stitches tore through, terminating the case by perforation peritonitis. In 20 cases of atypical pylorectomy eight died,—a mortality of 40 per cent. The first case of immediate success by Billroth succumbed to a recurrence after four months. Krönlein performed this operation for traumatic cicatricial stenosis extending into the duodenum.

Partial Pylorectomy and Partial Resections of the Gastric Walls.—The indications are given by the round ulcer, both on the anterior and posterior walls, cicatrices that produce interference, or tumors of the neighborhood that have extended to the gastric wall without involving the entire circumference of the pylorus. Partial pylorectomy preserves the valvula pylori, which is itself rarely the seat of gastric ulcer. In a total pylorectomy the sphincter and valve are removed entirely and replaced by a gradually contracting scar. Haberkant records eight such partial operations—three by Billroth (reported by von Hacker, *loc. cit.*); three by Czerny (*Beitr. z. klin. Chir.*, Bd. IX, 1892); one by Spear (*Centralbl. f. klin. Chir.*, 1885), and one by Schuchardt (Twenty-third German Surgical Congress, 1894).

Billroth's and Spear's cases all died; the three cases of Czerny recovered. The indications in the cases of the first two surgeons were: cicatrices, four times in the anterior pyloric wall. The indications in the last three cases by Czerny were: ulcer, once; extension of sarcoma, twice. The case of stenosing ulcer, operated upon by the latter by this method, was still doing well, according to last reports, ten years after the operation.

The case of Schuchardt's is most instructive in bearing out our objection to Haberkant's assertion that exploratory laparotomy is the *only reliable* means for early diagnosis of carcinoma. Schuchardt's case had two open ulcers, only one of which was discovered when the stomach was opened, located at the lesser curvature and removed by excision of a piece as large as a 25-cent piece. Although no peritonitic symptoms appeared, death occurred under progressive cachexia in fourteen days. The necropsy showed a second, much larger, ulcer, which, in Haberkant's (*loc. cit.*, p. 514) own words, was not only inoperable, but could not even be palpated. If a very large ulcer can not be palpated when the stomach

is exposed, exploratory laparotomy is not infallible as a diagnostic method; it may desert us like our clinical methods, and is much more dangerous. The oldest *resections of the gastric wall* are by Billroth (*Wien. med. Wochenschr.*, 1881, p. 275) and Esmarch (quoted by Wölfler, *loc. cit.*), for the repair of fistulæ.

Rupp resected a subserous leiomyoma of the anterior wall near the cardia in this way (*von Langenbeck's Arch.*, Bd. XL, p. 756). The number of partial resections, including both those of the pylorus and of the anterior gastric wall, amount to 15 operations; eight were cured, seven were fatal, giving a mortality of 46.6 per cent.

Gastro-enterostomy.—In the beginning of this chapter we have described the various (three main) types of this operation that have been suggested. Which of these types of operation—Wölfler's, von Hacker's, or Billroth-Brenner's—is to be selected is a matter to be decided by the surgeon. But we would remark in parenthesis that the physiological rotation of the full stomach around its long axis, whereby the large curvature is turned anteriorly and the smaller posteriorly, which is asserted by Tiedemann, has been confirmed by no other experimenter. Betz and Lesshaft (*Virchow's Arch.*, 1882, vol. LXXXVII) have opposed the view. In many hundreds of experiments on the full stomachs of animals for the study of the peristalsis we have never observed it, and even if it were observed when the abdomen is opened, it could not then be considered physiological. The hypothetical gastric rotation has been adduced as an objection to Wölfler's anterior antecolonic operation, on the ground that the artificial lumen between stomach and intestine was compressed by bringing the loop between the abdominal wall and the stomach during this rotation. Von Hacker's method does avoid spur formation, and thereby retroflux of duodenal contents into the stomach, and also compression of the transverse colon by the inserted jejunal loop. In addition to this the entire intestinal canal remains unchanged in its natural anatomical relations. Von Hacker himself states that the mortality is not materially reduced by his method. The functional results are claimed by most German surgeons to be better with von Hacker's operation.

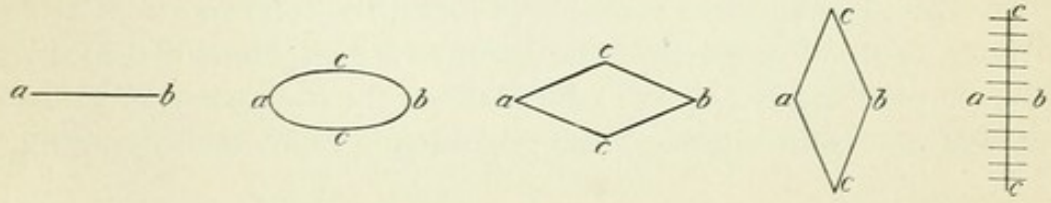
Results.—In 388 cases Haberkant found the total mortality to be 41.5 per cent. For gastro-enterostomy for carcinoma it was 43.5 per cent.; for ulcer, 25.5 per cent. One of the indications of gastro-enterostomy is the simple *atonic* dilatation. Four such operations

are reported, one of which (Selenkow) died and the remaining three (Renton, von Kleef, and Jeannel) recovered and the functional result was good. Concerning the remote results, it is self-evident that in carcinoma they can not be of long duration, as the growth is left intact; nevertheless the table at the end of this article shows ten cases in which the recovery lasted over a year. A singular gastro-enterostomy is that reported by Hahn (*Berlin. klin. Wochenschr.*, 1894, p. 1097). The operators were convinced that the neoplasm was a carcinoma; the patient lived seven years after the operation without complaints, the tumor always being palpable. This case will always remain a doubtful one.

Robert F. Weir has attempted to prove statistically that gastro-enterostomy keeps patients with pyloric carcinoma alive as long as pylorectomy, whereas the mortality is in the proportion of 12 per cent. for the former to 52 per cent. for pylorectomy.

Haberkant's statistics of a much larger number of cases shows a mortality of 54.4 per cent. for resection to 43.5 per cent. for gastro-enterostomy. But then many cases have formerly been resected that would in the present advanced state of knowledge not have been operated. Pylorectomy gives a better prospect for more lasting recovery than gastro-enterostomy. Of 47 cases recovered from pylorectomy, 22 lived longer than one year after the operation; but of 58 cases of gastro-enterostomy, only 12 lived longer than one year. With the exception of Hahn's doubtful case, there is *no* gastro-enterostomy in which the recovery lasted longer than two years; but of Haberkant's collected successful pylorectomies, 12 lived longer than two years.

Pyloroplastic Surgery (Pyloroplasty).—This operation was first devised by von Heinecke, in March, 1886. The first operation was a success. In February, 1887, Mikulicz rediscovered and applied the method independently of von Heinecke. The operation, which is applicable to some pyloric cicatrices, is carried out by slitting open the scars longitudinally in the line of the pyloric lumen and pulling the wound-edges apart by hooks inserted in the middle; then they are reunited by sutures transversely. Graphically the procedure is expressed thus:



Cases have been reported in which pyloroplastic surgery was attempted, and, failing, a pylorectomy had to be done (Löbker, "Verhandl. des XXI. Deutsch. Chir. Kongresses," 1, 60). Czerny reported a case in which resection had to be done because the scar was so rigid it could not be unfolded (*Beiträge z. klin. Chir.*, Bd. IX, 1892, p. 678). The cases that recover from a successful operation of this kind are, as a rule, cured permanently; no return of pyloric stenosis has been reported.

Up to 1894 (inclusive), 51 operations of this type have been compiled, 40 of which were successful and 11 fatal, making a mortality of 21.5 per cent. In 44 instances, where the indication was stated, there were seven peptic ulcers and 37 cicatricial stenoses; but of these 37 scars, 14 were produced by corrosive poisons.

Digital Divulsion of the Pylorus.—Loreta's operation consists of a simple gastrotomy and subsequent gradual expansion of the pylorus, by introducing first one and then two fingers; the dilating forceps has been used for the same purpose. The dangers of the procedure consist in (*a*) rupture or production of hemorrhages by lesions of the gastric wall, (*b*) frequent return of the stenosis.

According to Bull (*Centralbl. f. Chir.*, 1890, S. 149), Loreta himself has had the return occur in three cases. Haberkant (*loc. cit.*) has compiled 31 cases of Loreta operations, with 19 cures and 12 deaths—a mortality of 38.7 per cent. Three of the fatal operations were for carcinomata.

Novara had to execute a resection after divulsion had failed; the only justifiable indication is cicatricial stenosis. The operation has few advocates, and will have to give way to more exact and reliable operative methods.

For atonic forms of dilatation that resist all medical treatment, Heinrich Bircher (of Aarau, Switzerland) has devised a new operation, called *gastroplication*. This surgeon attempted to improve the motility of the stomach by a reduction of its size through making a fold or plait in the gastric wall. The greater curvature is raised to a much higher level by this operation. Bircher obtained good results in three cases, one of which, however, died three months after the operation. A certain amount of muscular tonicity must still be left in order to make this operation even a partial success (*American Jour. Med. Sciences*, 1892, vol. CIII, p. 333). The operation has not as yet been repeated in a sufficiently large number of cases to permit of a correct judgment of its real value.

The Fundamental Factors Influencing the Rate of Mortality in Gastric Operations.—These are partly under the control of the physician and partly not. Those over which we may exercise control are (I) defects in the technic, (II) selection of the kind of operation, (III) duration of the operation.

The factors that escape control are (I) age of the fundamental disease, (II) nature and extent of this disease, (III) age of the patient.

A. Factors Under the Control of the Surgeon.

I. *Faults in the technic*, as a rule, lead to peritonitis, of which one must distinguish two kinds: (*a*) The septic, produced by infection during the operation; and (*b*) perforation peritonitis, due to a technical defect in placing the sutures. Perforation peritonitis as a result of insufficiency of the sutures is much more common in pylorectomy than in gastro-enterostomy, because the lines of suture are much longer. However, peritonitis may be caused by errors in diet or by spontaneous perforations in other parts of the stomach, independently of the technic. In 165 fatal issues, with autopsies, the cause of death was peritonitis in one-fourth of the cases; only three fatal cases were due to spontaneous peritonitis.

II. *The selection of the proper operation* for any particular case is facilitated by an exact definition of the indications.

The indications for pylorectomy are: (1) the operable carcinoma or sarcoma, (2) the peptic stenosing ulcer or cicatrix, (3) perforation from pyloric ulcer.

The contra-indications are:

(1) (*a*) Firm adhesions, especially posteriorly on account of danger of injuring the hepatic artery and vein, (*b*) adhesions with the pancreas, (*c*) with the liver, (*d*) with the meso- and transverse colon.

(2) Infiltration of lymphatic glands (*a*) of the lesser omentum, (*b*) posterior surface of the stomach, (*c*) of the porta hepatis.

(3) Icterus from metastases or compression by the tumor.

(4) Great exhaustion of the patient.

Severe gastric hemorrhage can be treated in most cases by internal medication. Gastric ulcers that have given rise to repeated grave hemorrhages have been successfully excised by Czerny (*Archiv f. klin. Chir.*, 1884), Cordua (quoted in Debove and Rémond, "Traité de Mal. de l'Estomac"), and Mikulicz (*Deutsche med. Wochenschr.*, 1892).

Dunin asserts that ulcers in the pyloric region causing serious

hemorrhages would heal rapidly if the pyloric passage were put at rest by a gastro-enterostomy (Mintz, *loc. cit.*).

Küster cured persistent hematemesis from pyloric ulcer on the posterior wall by opening the anterior wall, producing scabbing incrustation with the thermocautery, and making a wide gastro-enterostomy. During the operation a cherry-stone was extracted from the depth of the ulcer. Mikulicz did a pyloroplastic operation for uncontrollable hemorrhage. The gastro-enterostomy, after cicatrizing the ulcers with the thermocautery, is generally a prophylactic measure to forestall a prospective pyloric stenosis. A pyloroplastic operation may accomplish the same object.

For perforation of gastric ulcer many operations have been executed. Pariser has recently reported 43 such operations with 33 deaths and ten recoveries. Only in four cases was the perforation in the pylorus (Pariser, *Deutsche med. Wochenschr.*, 1895). N. Senn suggested gastric distention with hydrogen, in order to rapidly find out the seat of the perforation.

Indications for gastro-enterostomy:

(1) Pyloric carcinoma with extensive adhesions and glandular metastases. Frequently it is not decided to do a gastro-enterostomy until the abdomen is opened; a resection has often been planned, but had to be replaced by a gastro-enterostomy. If the posterior wall alone is free from infiltration, von Hacker's method is indicated; in the reverse case, the methods of Wölfler or Billroth-Brenner.

(2) Stenosing ulcer (*a*) both when the pylorus is still isolated and free, and (*b*) when it is adherent with its surrounding. With this indication pylorectomy is unjustifiable, but partial resection and pyloroplastic surgery may yet compete with gastro-enterostomy. When, however, a cicatricial pyloric stenosis extends into the duodenum, nothing but a gastro-enterostomy should be done.

(3) Stenoses in the duodenum outside of the pylorus. Four operations, with three recoveries, have so far been executed for this indication.

(4) Stenoses by neoplasms of neighboring organs—the gall-bladder, periportal lymphatic glands, and pancreas. Novarro performed a gastro-enterostomy for pyloric stenosis caused by echinococcus cyst of the liver (*Deutsche med. Wochenschr.*, 1891, No. 4, S. 152). Stansfield did the same operation, with good result, for

tumor of the pancreas (*Brit. Med. Jour.*, 1890, pp. 294 and 1300), making use of Senn's bone-plates.

(5) Purely functional dilatation due to atony of the musculature without pyloric stricture. Four gastro-enterostomies for this indication are on record. Recently, Bircher has proposed gastroplication (resection of a fold of the stomach-wall) for this purpose.

III. *The Duration of Gastric Operations.*—It is evident that the sooner an operation is completed the less the danger of shock and sepsis. With the view of shortening the time of operation, Rydygier and Lauenstein advised the employment of continued sutures, which they claim abbreviate the time by one hour. The most celebrated time- and labor-saving devices in gastro-intestinal surgery are by our countrymen, Murphy and Senn. The advocates of Senn's bone-plates have claimed that the mortality under the older suture methods was from 42.8 per cent. to 47 per cent. (Herbert Page and von Hacker), and in 41 operations by the Senn method the mortality was only 24.5 per cent.

The decalcified bone-plates of Senn are not always digested. In one of the inventor's own cases they were vomited undigested forty hours after the operation. Haberkant asserts that the advantage of the saving of time is counterbalanced by less safety. For surgical opinions on the Murphy button and Senn plates we must refer to journals and text-books on abdominal surgery. But so much is clear: shortening of the time of operations by these contrivances is a great gain.

B. Factors that Escape Control.

I. *Age of the Underlying Disease.*—This can not be determined statistically, for both ulcer and carcinoma may remain latent for months, and it is impossible to ascertain the age of these conditions at the autopsy. We have observed a large carcinoma in a white woman who died at Bay View Hospital, occupying the posterior gastric wall, when during life there had been no gastric symptoms. Osler's case of very rapid course in gastric carcinoma—two weeks from the onset of severe dyspeptic symptoms—made it plain at the autopsy that the growth had been of considerable duration, but had for a long time not undermined the patient's health (*Univ. Medical Magazine*, January, 1895). The anamnesis given by patients regarding the period since when they have suffered from dyspepsia is frequently unreliable. The earlier the case is presented for operation the more favorable the prospects.

II. *Nature and Extent of the Fundamental Gastric Disease.*—Concerning this point the statistics show that the mortality in pylorectomy, as well as in gastro-enterostomy, is greater for carcinoma than for ulcer. Under the head of contraindications to these operations we have dwelt upon the dangerous influences of the extent of the disease.

III. *Effect of the Age of the Patient.*—In the cases as they are presented for operation there are so many other governing factors that the matter of age does not appear, from statistics, to exert much influence on the result of these operations, provided the other conditions previously mentioned are favorable. A difference becomes noticeable when the age is over sixty years. Among 176 resections, the percentage of mortality of those under fifty years was 50.4 per cent., and of those over fifty years, 52.9 per cent. With gastro-enterostomy the rate was 42.4 per cent. for those under fifty years, and 57.7 per cent. for those over fifty. These statistics, therefore, do not confirm a marked influence of age on the rate of mortality.

A critical consideration of these factors, in connection with other elements before mentioned, justifies the hope that diagnosis and gastric surgery have not reached their highest development as yet, and we may expect a further lowering of the rate of mortality.

An artificial communication between the stomach and intestines, as is performed in gastro-enterostomy, may become much smaller by cicatricial contraction. Kocher has reported two such observations. In one case Czerny made an opening three cm. in diameter; at the autopsy, five months later, it had contracted down to eight mm.

Heinsheimer has made careful analytical observations on the metabolism in two cases of gastro-enterostomy ("Mittheilungen a. d. Grenzgebieten d. Medizin u. Chirurg.," Bd. 1, S. 350). In this piece of work, which was done under von Noorden, Rachford's observation that fats require a very thorough and intense mixture with the secretions of the pancreas and liver for their digestion (*Centralbl. f. Phys.*, 1896, Heft 4) was confirmed. The further away the gastro-intestinal communication is laid from the duodenal orifices of these glands, the more defective the fat digestion and resorption. It is therefore suggested that in gastro-enterostomies a jejunal loop, as near as possible to the duodenum, be anastomosed with the stomach.

Gastro-anastomosis is an operation first performed by Wölfler for hour-glass stomach, by which one portion of the organ is anastomosed with the other at the greater curvature. Gastro-anastomosis remedies the separation of the organ into two distinct cavities separated by a narrow isthmus. Von Hacker reports and pictures cases of hour-glass stomachs complicated with cicatricial pyloric stenosis, for which he recommends a double operation—either a pylorectomy, or a pyloroplastic operation with gastro-anastomosis, or, best, a gastro-enterostomy and gastro-anastomosis (von Hacker, "Magenoperationen," etc., Wien, 1895).

For benign stenoses of the pylorus, pylorectomy is more and more deserted in favor of gastro-enterostomy, which gives the same functional results without the dangers (Ernst Siegel, "Mittheil. a. d. Grenzgebieten d. Medicin u. Chir.," Bd. 1, p. 347).

DURATION OF LIFE AFTER RESECTION IN 51 CASES OF CARCINOMA OF PYLORUS.—(*Haberkant.*)

Death.	Number of Cases.	Cause of Death.	Living at Date of this Report.	Number of Cases.	
After 1½ months,	1	Return of cancer.	After 2 months,	1	
" 2 "	3	One of metastasis in the liver.	" 3 "	1	
			" 3½ "	1	
" 2½ "	1	Acute lung disease, no return and no metastasis.	" 4 "	1	
			" 6 "	1	
" 4 "	2	Return.	" 7½ "	1	
" 5 "	2	One of lobular pneumonia, one of chronic pyemia.	" 8 "	1	
			" 9 "	1	
" 6 "	2	One of return, one of cicatricial stenosis.	" 1 year, .	2	
			" 1 year and 2 months,	1	
" 6½ "	1	Return.			
" 7 "	2		" 1½ years,	1	
" 8 "	1	Return.	" 2 "	2	
" 10 "	1	Return.	" 2 years and 2 months,	2	
" 11½ "	1	Rectal and pelvic carcinoma.	" 2½ years,	1	
" 1 year, . .	3	Two return.	" almost 3 years, .	1	
" 13 months,	2	Return.			
" 1¼ years,	1	Return.	" 3½ years,	1	
" 2½ "	1		" 5 years and 4 months,	1	Kocher.
" 3 "	1	Cicatricial stenosis.			
" 5¼ "	1	Not stated (Billroth).	Over 8 years, . .	1	Ratimmow.
Total, . . .	26		Total, . . .	21	

RECOVERIES AND DEATHS: PERCENTAGE OF MORTALITY IN 379 CASES OF RESECTION OF PYLORUS.—(*Haberkant.*)

Year of Operation.	Name of Operator.	Number Operated Upon.	Result.		Carcinoma.		Ulcer and Cicatricial Stenosis.		Sarcoma and Myoma.		No Indication Stated.		Remarks.
			Recovered.	Died.	Recovered.	Died.	Recovered.	Died.	Recovered	Died.	Recovered.	Died.	
1885	Rydygier, .	5	3	2	1	2	2	.	.	.	.	.	
1885	Gussenbauer,	6	2	4	.	4	.	.	.	.	2	.	
1889	Augerer, . .	6	1	5	.	.	.	.	.	.	1	5	
1890	Billroth, . .	41	19	22	12	16	6	6	1	.	.	.	Of these, thirty-six were total resections; 3 partial, 2 atypical pylorectomies.
									Sarcoma				
1890	Lauenstein,	12	4	8	.	.	.	.	.		4	8	
1890	Novarro, .	3	2	1	1	.	1	1	.	.	.	.	
1891	Tillmans, .	4	.	4	.	4	.	.	.	.	.	.	
1892	v. Heinecke,	14	5	9	4	9	1	.	.	.	.	.	
1892	Schönborn, .	5	2	3	2	3	.	.	.	.	.	.	
1893	Roux, . . .	5	3	2	.		.	.	.	.	3	2	One atypical, the rest typical, total resections.
1893	Doyen, . .	7	6	1	2	1	4	.	.	.	.	.	All atypical pylorectomies.
1893	Löbker, . .	13	7	6	5	5	2	1	.	.	.	.	
1893	Schede, . .	13	7	6	6	5	1	1		.	.	.	
1893	von Kleef, .	4	1	3	.	.	1	3	.	.	.	.	
1894	Kraske, . .	4	1	3	1	3	.	.	.	.	.	.	
1894	Czerny, . .	20	13	7	8	5	3	2	2	.	.	.	Three partial resections, the rest typical, total pylorectomies.
									Sarcoma.				
1894	Kocher, . .	9	7	2	7	2	.	.	.	.	.	.	In all 9 cases Kocher used his method with following gastro-duodenostomy.
	Krönlein, .	8	5	3	.	.	.	.	.	.	5	3	
1894	Kappeler, .	14	9	5	8	5	1	.	.	.	.	.	Total mortality, 35.7 per cent. For carcinoma alone, 38.1 per cent.
1895	Mikulicz, .	20	15	5	13	5	2	.	.	.	.	.	
	Other cases, exclusive of above, . .	166	79	87	60	76	8	6	2	.	9	5	Of these, 147 were total resections; 17 atypical, and 2 partial, pylorectomies.
									1 Angioma fibrosum. 1 Lymphosarcoma.				
	Total, . .	379	191	188	130	145	32	20	5	.	24	23	

RESULTS WITH GASTRO-ENTEROSTOMY FROM 1885 TO 1893.

Year of Publication.	Author.	Number of Operations.	Result.		Total Mortality. Per Cent.	Carcinoma.			Ulcer and Cicatricial Stenosis.		
			Recovered.	Died.		Recovered.	Died	Mortality. Per cent.	Recovered.	Died.	Mortality. Per cent.
1885	Kramer, ...	20	8	12	. .	5	11	68.7	3	1	. .
1886	Saltzmann, . . .	23	. .		. .	6	12	66.6	. .	.	. .
1887	Rockwitz, . . .	29	16	13	44.8	11	12	. .	5	1	. .
1890	Novarro, . . .	55	.	24	43.6	. .	. .	. .	. .	. .	. .
1890	Mehler, ...	. .	. .	. .	55.1	. .	. .	58.8	. .	. .	38.5
1891	Page, ...	36	. .	15	41.6	. .	. .	. .	. .	. .	. .
1892	Hadra, ...	76	. .	. .	. .	33	43	56	. .	. .	. .
1893	Zeller, ...	152	. .		. .	86	66	43.4	. .	. .	. .
		391	24	64		141	144		8	2	

RESULTS OF VARIOUS OPERATORS WITH GASTRO-ENTEROSTOMY.

(*Haberkant.*)

Year of Publication.	Operator.	Number of Cases Operated Upon.	Carcinoma.		Ulcer and Cicatricial Stenosis.		Dilatation of the Stomach.		Sarcoma.		No Indication Stated.		Remarks.
			Recovered.	Died.	Recovered.	Died.	Recovered.	Died.	Recovered.	Died.	Recovered.	Died.	
1887	Lücke, . .	8	5	1	2	.	.	.	.	.	.	.	
1890	Billroth, . .	28	14	14	.	.	.	.	.	.	.	.	
1890	Novarro, . .	10	5	3	2	.	.	.	.	.	.	.	
1891	Lauenstein, .	17	10	3	2	2	.	.	.	.	.	.	
1891	Hahn, . . .	11	.	.	.	.	.	.	.	.	5	6	
1891	Bowreman-Jesset, . .	5	.	.	.	.	.	.	.	.	3	2	
1891	Senn, . . .	13	.	.	.	.	.	.	.	.	4	9	Calculated according to Czerny.
1891	Remedi, . .	6	.	3	3	.	.	.	.	.	.	.	
1893	Roux, . . .	14	7	7	.	.	.	.	.	.	.	.	
1893	Doyen, . .	10	.	.	.	.	.	.	.	.	6	4	Two carcinomata, 8 cicatrices.
1893	von Kleef, .	19	.	.	.	.	1	.	.	.	13	5	
1893	Codivilla, .	6	2	.	4	.	.	.	.	.	.	.	Operated in one year by von Hacker's method.
1893	v. Heinecke,	6	4	2	.	.	.	.	.	.	.	.	
1893	Löbker, . .	7	4	3	.	.	.	.	.	.	.	.	All according to von Hacker's method.
1894	Czerny, .	23	12	7	2	1	.	.	.	1	.	.	
1894	Kraske, . .	10	7	3	.	.	.	.	.	.	.	.	
	Other cases, exclusive of above, . .	195	66	59	20	9	2	1	1	.	21	16	
	Total, . .	388	136	105	35	12	3	1	1	1	52	42	

DURATION OF LIFE AFTER GASTRO-ENTEROSTOMY.

Death.	Number of Cases.	Living and at the Time of Report in Good Health.	Number of Cases.
After 1 month,	8	After 2 months,	1
" 1½ months,	1	" 3 "	2
" 2 "	5	" 4 "	1
" 2½ "	2	" 5 "	2
" 3 "	4	" 6 "	2
" 3½ "	2	" 7 "	2
" 4 "	5	" 8½ "	1
" 6 "	3	" 9 "	2
" 7½ "	1	" 10 "	1
" 10 "	1	" 1 year and 11 months,	1
" 1 year,	3	" 2 years,	1 (Hahn).
" 1 year and 2 months,	1		
" 1½ years,	3		
" 1 year and 7 months,	1		
" 1 " " 8 "	1		
" 1 " " 10 "	1		
Total,	42	Total,	16

EFFECT OF AGE ON THE RESULT OF VARIOUS GASTRIC OPERATIONS.—(*Haberkant.*)

Age.	Total Typical Resection of Pylorus.			Gastro-enterostomy.			Atypical Resection of Pylorus.			Pyloroplastic Operation.		
	Number of Operations.	Recovered.	Died.	Number of Operations.	Recovered.	Died.	Number of Operations.	Recovered.	Died.	Number of Operations.	Recovered.	Died.
20 years.	2	2	. .	1	. .	1	. .	. .	. .	2	2	. .
22–30 "	9	6	3	21	14	7	3	1	2	5	2	3
31–40 "	44	21	23	47	26	21	2	2	. .	10	7	3
41–50 "	70	33	37	37	21	16	3	2	1	5	4	1
51–60 "	37	20	17	33	16	17	1	1		3	3	. .
61–70 "	13	4	9	11	2	9	. .	. .	. .	. .	. .	. .
Over 70 "	1	. .	1	1	1	. .	. .	. .	. .	1	. .	1
					(71)					(73)		

CHAPTER VIII.

INFLUENCE OF GASTRIC DISEASES UPON OTHER ORGANS AND ON METABOLISM.

Diseases of the stomach may, as is well known, affect general nutrition, the action of the heart, lungs, and the nervous system.

In all digestive diseases with apparent malnutrition the physician should ascertain the amount and the kind of food ingested, the state of the stool, and sleep. The cause of insufficient ingestion of food is anorexia in the majority of cases; in others the patients have a good appetite, but avoid food because it gives them pain, as in the case of gastric ulcer; others, again, will not eat because they vomit the food soon after ingestion. Loss of weight is of more serious significance in chronic than in acute stomach diseases. Instead of taking in 30 to 40 calories per kilogram of body weight, von Noorden found in chronic types, after careful observation, that they ingested only 21 calories of their own accord (v. Noorden, "Ueber Stoffwechsel d. Magenkranken," etc., *Berliner Klinik*, Heft 55). In cases in which the HCl secretion was so diminished that only a fraction of the proteids could be peptonized in the stomach and the largest portion passed into the intestines unchanged, von Noorden found that resorption of the main food-substances was sufficient. With a good gastric peristalsis, preventing delay and fermentation in the stomach, the intestine is capable of supplanting the deficient gastric digestion. In animals, total exclusion of the stomach from the digestive act need not injure general nutrition, provided the food is supplied in a proper form.

It is very probable that in certain forms of gastritis, in ectasias and carcinoma, poisonous substances are formed which are resorbed and injure the metabolism of the tissues. Friedenwald has recently found this to be the case in atony of the intestines and stomach (*Med. News*, Dec. 23, 1893).

Resorbable and combustible gases occur in gastrectasias with stagnating ingesta and have been described by many observers

(see Albu, "Die Autointoxicationen des Intestinaltractus," p. 19). Putrefaction of albuminous substances may occur in the stomach, as Boas has repeatedly observed. Some cases show the formation of sulphuretted hydrogen even with co-existent high acidity. Naturally, stagnation must be present to make albuminous decomposition possible. Müller has described a series of carcinoma cases in which more nitrogen was excreted than ingested in the food (*Zeitschr. f. klin. Med.*, Bd. XVI), which strengthens the conception of carcinomatous auto-intoxication, causing an increased albuminous breakdown in the tissues.

In all cases of subnormal nutrition all etiological factors must be sought out and an individualized, highly nutritious, concentrated, unirritating diet adapted to the patient after improving the appetite.

Influence of Gastric Diseases on the Heart.—It is natural to expect increased rapidity of the heart's action in all gastric diseases associated with fever, such as the various forms of acute gastritis, in perigastritis, and other complications (peritonitis). But tachycardia has frequently been observed by us associated with hyperacidity, gastrosuccorrhea, and pneumatosis. In one case of the latter disease the tachycardia was so persistent as to require special treatment by local ice bag, aconite, and bromid of strontium. In all of these cases fever was absent, and we have no experimental basis to explain the phenomenon.

Bradycardia is seen much more frequently, and the fact that it is aggravated and improves or disappears as the gastric trouble becomes worse or better, shows that it is not an accidental accompaniment, but is in some causal relation with the fundamental disease, *i. e.*, dilatation or ulcer. In animals, slowing of the pulse can be effected by distention or rough manipulation of the stomach. Stimulation of sensory nerves causes slowing of the heart beat, and this may partly be offered as an explanation of bradycardia in dilatation and ulcer, though it is far from satisfactory. It is difficult to prove that irregular heart's action or arrhythmia is dependent upon gastric diseases even when it actually is associated with the latter. Arrhythmia is so frequent that it may accidentally be present in an individual independently of any gastric disease.

Respiration.—It is undoubted that breathing is influenced by gastric troubles, although sufficient attention has not been given

to this matter. We have noticed in a number of cases that the respiratory expansion is lessened by gastric diseases impeding the excursions of the diaphragm. This we have produced experimentally in healthy individuals by inflating our stomach-shaped intragastric rubber bag within their stomach while they were under narcosis.

It appears, therefore, that undue or excessive gastric distention diminishes the amount of inspired air, independently of consciousness. In gastric fermentations toxic substances are produced, which, when injected into the circulation, caused dyspneic respiration of a paroxysmal character (Bouchard and Bouveret). It is probable, therefore, that gastric diseases may affect respiration either directly or mechanically, through interference with the descent of the diaphragm or by the absorption of toxins and action on the respiratory center. With the intimate, mutual correlation of the physiology of the circulatory and respiratory function, it is evident that a pathological disturbance of one will inevitably affect the other.

The Influence of Gastric Diseases on the Nervous System.—Nervous patients affected with a disease of the stomach frequently exhibit neuroses of sensation, hyperesthesia, intercostal neuralgias, and hemicrania. That there is some etiological connection between the stomach and these conditions is made very probable by the fact that very frequently they only occur after full meals (dinner), or, if they existed before, they are aggravated by copious eating, and become ameliorated or disappear as digestion is completed. The pains often return toward night and on going to bed, causing insomnia. Patients that are experienced in the use of the stomach-tube are able to arrest these pains at times by lavage. Occasionally the colon is the cause of the pain irradiation; this is especially to be looked for in membranous colitis or compression of the colon from tight lacing, or in the various forms of enteroptosis.

Gastric vertigo is a form of dizziness or partial unconsciousness without pain, but frequently with nausea and vomiting, occurring in gastric sufferers. Trousseau, who gave a classical description of this affection, argued that one of its peculiarities was that consciousness remained clear during the attack (Trousseau, "Clin. del' Hôtel Dieu," Paris, tome III, 1868).

We have observed transient loss of consciousness which at

times developed from typical stomach vertigo. This affection occurs in neuropathic patients with hyperacidity, particularly when the stomach is empty, and is associated more often with this gastric neurosis than others.

It is a more frequent complication of hyperchylia than is generally known, since many patients will not speak of their transient attacks unless especially questioned. Emesis often checks the attack, and Trousseau mentions that a cup of bouillon or a cake soaked in wine may check the vertigo.

Treatment of the fundamental gastric disease removes the vertigo, as a rule, but in explanation of the way in which the vertigo is caused, its nervous mechanism, etc., we have nothing but hypotheses.

Symptoms.—The symptoms of gastric vertigo are nausea, eructation, pyrosis, vomiting, sensitiveness in the epigastric region, pain in the stomach, and a feeling of pressure, fullness, and distention. In the majority of cases constipation exists, and the abdomen is frequently distended with gases. In some few cases a dilatation was found to be present. Vertigo has been observed to occur almost at any stage of digestion—before, during, and after meals. Sometimes the attacks of giddiness are announced by a sensation of great hunger or bulimia. Leube mentions that gastric vertigo may occur in some persons after the ingestion of certain foods. It may occur either at intervals of several days or several times in the same day. It is generally a chronic and permanent trouble, but in its lighter forms it has no serious influence on the condition of the patient. The treatment of gastric vertigo necessitates the treatment of the underlying condition of the stomach.

Leube has described an *intestinal vertigo* associated with intestinal diseases of various kinds, but generally not of a serious character. The most frequent causes are constipation and intestinal parasites, mainly lumbricoid and tapeworms. These attacks of gastric vertigo are as yet not satisfactorily explained. Leube has in some cases been able to produce the attack by pressure on the stomach or intestines (Leube, "Ueber den Magenschwindel"; Ziemssen's "Handb. d. spec. Path. u. Ther.," vol. II, p. 66). Mayer and Pribram claim to have observed excitation of the vasomotor center after irritation of the stomach, particularly of the serous coat (Mayer and Pribram, "Ueber reflect. Bezieh. d. Magens z. d. Innervationscentren f. d. Kreislaufsorg.," Sitzungsber. d. Wien. Akad.

d. Wiss., 1872). A second theory in explanation of stomach vertigo presumes that it is caused by cerebral anemia, or hyperemia, which is not described as a reflex act, but as a direct detrimental influence on the circulation of the brain. This hypothesis exaggerates the degrees of circulatory variation that can possibly occur in such light forms of digestive disturbance in which vertigo is observed. A third theory explains gastric vertigo on the basis of auto-intoxication. It is presumed that products of abnormal digestion, which collect when the motor function of the stomach and intestines is disturbed, are absorbed into the circulation, and act directly upon the brain. There is an abundance of experimental evidence, as well as clinical experience, which proves that such an effect of toxic chemical substances is possible. Such toxic irritation may be indirect, and is intermediated through the vasomotor center. Brieger has isolated a substance from dilated stomachs, which he has termed peptotoxin, which has an extremely poisonous effect when injected into the circulation of animals. In case of the presence of intestinal parasites, the toxic metabolic products of the helminthiasis are added to those of disturbed digestive function.

Tetany.—The term signifies characteristic convulsive attacks which occur in the course of gastric diseases, particularly with dilatation associated with hypersecretion. The term "tetany" was first used in 1852 by Corvisart. The spasms are prevailingly tonic contractions, alternating with less severe twitchings in the flexor muscles of the arms, calves, and generally also of the abdominal muscles. The facial, cervical, and maxillary muscles are occasionally attacked by the tetany, the eyes may be turned upward, and even emprosthotonos of short duration has been reported. The convulsions may be painful and consciousness may be clear or completely obscured. In one case of Kussmaul's the power of speech was lost; in another case the patient spoke disconnectedly and his pupils did not react to light. In a third case the symptoms referring to the cerebrum were absent, but a fourth case of Kussmaul's was of an epileptic form and character. Bouveret and Devic ("Rech. clin. et expérim. sur la tetanie d'origine gastrique," *Revue de Médic.*, 1892, 12, p. 48) have collected 23 cases of these tetanic attacks, and Albu ("Autointoxicationen des Intestinaltractus," Berlin, 1895) states that not more than 36 cases of this complication of gastric diseases have been reported. Kussmaul gave the first classical description of these attacks in his famous publica-

tion on the treatment of gastric dilatations by a new method by means of the stomach-pump (*Deutsches Archiv f. klin. Med.*, Bd. VI).

Clinically, it is not correct to designate all tonic muscular convulsions of gastric origin as tetany. In true gastric tetany there is an increased mechanical excitability of the muscles, and an increased mechanical and electrical irritability of the motor and sensory nerves, which precede the attack and may persist long after it. Cases have been reported by Fleiner and Kussmaul which strongly resembled typical tetanus. Cases are reported in which the clinical picture varied between tetany, tetanus, and epileptiform convulsions. In most of the cases Trousseau's phenomenon, *i. e.*, the production of spasms by pressure on the nerve trunks, was present. Among 27 cases that were collected by Riegel, 16 proved fatal. According to this, tetany is a very grave complication of gastric diseases. The gastric diseases with which tetany is associated are extensive dilatations, due mostly to stenosis of the pylorus or duodenum, by ulcer or cicatrix. In several cases the stenosis was due to a carcinoma that had developed from a cicatrix. Bouveret and Devic attribute great importance to hypersecretion for the production of tetany. This complication has, however, been observed in other dilatations in which there was no hypersecretion. Thus far three hypotheses have been put forward attempting to explain the origin of tetany: (1) That of Kussmaul, according to which it is caused by desiccation of the organism in consequence of copious loss of water. (2) The explanation according to which tetany is caused by a reflex irritation of the central and peripheral nervous systems, and that the irritation issued from the central branches of the gastro-intestinal tract. (3) That of auto-intoxication, which is the most probable.

Kussmaul's theory rested upon the apparent analogy between tetany and the cramps in the legs, occurring with Asiatic cholera, which are believed to be due to condensation and thickening of the blood, resulting from loss of water. We know, however, that these cramps occur also in cholera sicca. The loss of water, Kussmaul thought, was brought about by the exhaustive vomiting which usually precedes the attack of tetany, but cases have been reported in which tetany occurred without a preliminary attack of vomiting. In cholera nostras (the acute gastro-enteritis of children), where the loss of water is very great, tetany occurs very rarely, but it is a frequent accompaniment of chronic digestive diseases.

Blazicek described a case of gastric tetany in which the percentage of water in the blood was not reduced.

The second theory, that of the reflex origin, has been proposed by Germain Sée. The arguments of Bouveret and Devic, and of Ewald (*Berlin. klin. Wochenschr.*, 1894, No. 2), emphasize the fact that the reflex phenomena are based upon a preliminary chronic intoxication, which increases the irritability of the muscles and nerves. Tetany, according to these authors, is, therefore, not a reflex phenomenon, any more than are the convulsions of a strychninized frog, which result from the slightest cutaneous irritation.

The most modern authors (Gerhardt, Bouveret and Devic, Albu, Ewald, Heim, Loeb, Schlesinger, and Baginsky) favor an explanation of tetany on the basis of gastro-intestinal auto-intoxication. It is not a bacterial intoxication caused by metabolic products of pathogenic bacteria introduced with the food which these authors have reference to, but to poisons formed in the stagnating, fermenting contents of the dilated stomach. Kulneff has extracted toxic products from the gastric contents in carcinoma and dilatation, which, according to their chemical structure, were classed as diamins. These toxins were extracted by Brieger's method (extraction with alcohol and precipitation with mercuric chlorid). Bouveret and Devic extracted substances from the stomachs of three cases of tetany with hyperchlorhydria that produced spasms when injected into animals. Ewald and Jacobson have isolated alkaloidal bodies from the urine of tetany patients, and Albu isolated the double platinic and gold salt of an alkaloidal substance from the urine of a woman afflicted with tetany. This substance was absent from the urine when the patient was free from the attacks. Tetany occurs, in the majority of cases, only when abnormal fermentations and putrefactions occur in the stagnated contents of the stomach and intestines. This intoxication theory explains the nephritis which Loeb has observed in connection with tetany. The author has reported three cases of nephritis which undoubtedly owe their origin to auto-intoxication (Hemmeter, *Maryland Med. Jour.*, July 24 and 31, and Aug. 7, 1897).

Asthma Dyspepticum.—In 1876 Henoch described a clinical phenomenon in children, in which attacks very similar to asthma were associated with digestive disturbances (Henoch, *Berlin. klin. Wochenschr.*, 1876, No. 18), and in 1882 Silbermann described similar cases, also occurring in children (*Berlin. klin. Wochenschr.*, 1882,

No. 23). The attacks of asthma dyspepticum are characterized by a very abrupt, acute onset, after a very evident error in diet or after constipation or febrile gastritis. There is a pronounced dyspnea with cyanosis, very small, compressible, and hurried pulse, cold extremities, collapse, and, generally, no symptoms of severe gastric disturbance. The symptoms disappear as suddenly as they begin, after an emetic has taken effect or spontaneous vomiting has occurred. Strümpell ("Specielle Pathologie u. Therapie") doubts the existence of asthma dyspepticum, and Riegel also ("Die Erkrankungen des Magens," Wien, 1896, S. 192). The literature on this subject is very limited, and many of the cases reported do not impress us as strictly belonging to the clinical picture of asthma dyspepticum. O. Rosenbach (*Deutsche medicin. Wochenschr.*, 1879, No. 42) describes a number of cases, which, although he separates them from dyspeptic asthma, very much resemble this clinical picture. The patient complained of oppression, want of air, difficulty in breathing, and a sensation of fear. The scarcity of reports on this complication is explained by the fact that the physician very rarely has an opportunity for observing these cases during the attack; as a rule, they cease spontaneously within a few hours, and are frequently interrupted by the patients by mechanical manipulations to facilitate vomiting.

It is well known that conditions of more or less anxious oppression in breathing are observed occasionally in normal individuals, but more frequently in those afflicted with gastro-intestinal diseases. The attacks occur in connection with the larger meals, the patients having a feeling as if they could not breathe properly. The respiratory oppression and distress cease spontaneously during the course of digestion, or are relieved by eructation of gases. If these abnormal sensations are augmented, and when they occur at short intervals and after moderate ingestion of food, the condition becomes pathological. A fear of smothering, with cyanosis, cool extremities, greatly hurried pulse, and dyspnea, occurring in the sequence of gastro-intestinal disturbances, represent a clinical picture which we can designate as asthma dyspepticum. Oppler (*Allg. med. Centralztg.*, 1896, No. 71) and Lauterbach (*Wien. med. Presse*, 1894, No. 48) have each described one case of asthma dyspepticum as a sequence to gastric atony. The case of Oppler recovered under lavage, diet, massage, electricity, and the use of strychnin and belladonna. The cardinal symptom of the phenomenon is the paroxysmal dyspnea. It occurs most frequently among

women, and especially among the neurasthenic and hysterical. Potain (Association pour l'Avancement des Sciences, Montpellier, 1879) and Barié (*Revue de Médecin*, 1883, tome III, p. 1) have together reported 32 cases in France, a number of which gave indications that they were asthma dyspepticum. Boas (*Archiv f. Verdauungskrankheiten*, Bd. II, p. 444) gives a very interesting report of 11 cases,—ten males and one female.

Instead of going into details concerning the symptomatology, we will describe a case which has been observed by the author repeatedly during attacks.

The lady in question lived in the immediate neighborhood of the writer. Mrs. S., aged twenty-six, has suffered for years with symptoms of atony; mother living and healthy; father died with cancer of the stomach; she has been married four years, but has no children; heart and lungs normal. The dyspeptic symptoms are those of atony and nervous dyspepsia. There are no signs of enteroptosis; right kidney is firmly attached in its normal position; no history of uterine trouble; constipation. Results of analysis of test-meal: Total acidity, 90; free HCl, 50; combined HCl, 22; erythrodextrin present in excess; lactic acid absent. Examination of the urine for toxic products gave the following results when it was first examined; this was shortly after an attack, and was also followed by an attack on the next day: Preformed sulphates, 3.970 gm.; combined sulphates, 0.35 gm.,—ratio, 11.1; urea, 51.028 gm.; indigo blue, very strong reaction. On this date the patient was very melancholy, and suffered much from intestinal flatulence. The writer was called just as an attack was beginning, and found the patient on the sofa, with the servants rubbing her hands and feet, which had a bluish tint and were quite cold to the touch. She was gasping for breath; the pulse was 148. There was a very peculiar wheezing sound with each breath, and tenderness to pressure in the epigastric region; accentuation of the second cardiac sound. A stomach-tube was passed, and about 500 gm. of highly acid liquid drawn off, composed mostly of ice cream and strawberries. An enema was given containing warm claret and camphor, and hot bottles were placed to the feet. The patient broke out in a perspiration within thirty minutes of the enema, and had quite recovered two hours after the attack. This same patient has since that time, which was a year ago, been seen in two other attacks very similar to this one, both of them yielding to the same treatment.

There are no satisfactory explanations of asthma dyspepticum up to the present date. Potain (*loc. cit.*) believes in a reflex irritation from the gastro-intestinal tract, which causes contraction of the small pulmonary vessels. In the resistance to the pulmonary circulation which is thus brought about, the respiratory gaseous exchanges are interfered with, and Potain, as well as Barié, claim to have found dilatation of the right ventricle, with accentuation of the second pulmonary sound during the attack. A. Fränkel

(article on "Asthma" in Eulenburg's "Real-Encyclopädie," 3. Aufl.) considers asthma dyspepticum a reflex disturbance of cardiac asthma, caused especially by a weakness of the left ventricle, which then secondarily causes a passive congestion in the pulmonary circulation. This explanation concedes the trouble to be essentially cardiac asthma. There is a very intimate connection between disturbed digestion and cardiac action, which we have already dwelt upon, and the conception of Fränkel is not without foundation in those cases in which the heart's action is not perfectly sound. Boas has reported cases in which the attacks were brought on by a disturbed gastric digestion, with bronchitis and emphysema.

Abnormal gastro-intestinal meteorism may force up the diaphragm mechanically, and if there is any debility about the pulmonary capillaries, passive congestion can not fail to occur. With the evacuation or escape of the gas the attack will cease entirely. Senator (*Berlin. klin. Wochenschr.*, 1883, No. 22), G. Lewin, and Albu (*loc. cit.*) claim that dyspeptic asthma is caused by the absorption of toxic substances from the digestive tract. The theory of auto-intoxication has been criticized by Boas, since asthma dyspepticum is not met with in any gastro-intestinal diseases associated with extensive putrefaction and fermentation; whereas in those slight forms of gastric disease in which this asthma really does occur, there is very little formation of toxic products.

Prognosis is favorable. Boas, Lauterbach, and Oppler have reported cures. One of our cases has not had an attack for eighteen months.

Treatment is mainly a prophylactic and dietetic one. The stomach should be sparingly treated, the bowels kept open, and all food causing flatulence must be scrupulously avoided. The underlying neurasthenia and pulmonary or heart affections should receive therapeutic attention. In atony with hyperacidity, strychnin sulphate, $\frac{1}{30}$ of a grain, with ext. belladonna, $\frac{1}{10}$ of a grain three times daily, can be recommended. During the attack itself speedy evacuation of the stomach by the tube and of the bowel by warm-water irrigation are the most effective means of treatment.

The patients will usually not object to the tube in these attacks, because their suffering is so great that they are willing to undergo anything to be relieved; but where it can not be used on account of heart or lung trouble, emetics should not be used either, because

they are more depressing upon the heart than the use of the tube. When the heart is sound and emesis is absolutely indicated, we recommend the following:

℞. Pulvis ipecac, ... 1.5, or grs. xxiji
Antimon. et potass. tartrate, ... 0.05, or gr. $\frac{5}{6}$. M.
Sig.—Make two powders, to be taken one-half hour apart.

Prompt emesis may be effected by the use of apomorphin, hypodermically, in doses of $\frac{1}{10}$ of a grain.

The Influence of Nervous Diseases Upon the Stomach.—This subject will be considered in connection with the various nervous disorders of digestion. It is a well-known fact that emotional excitement may cause an alteration in the gastric secretions, and that intense nervous depression may produce gastric distress, fullness, pressure, eructation, nausea, constipation or diarrhea, meteorism, and tenesmus. Mental overexertion may lead to nervous dyspepsia. Anatomical alterations in the central nervous system may be accompanied by motor, secretory, and resorptive disturbances. In this connection we refer again to the gastric disturbances occurring with tabes, and to the fact that Koch and Ewald caused gastric hemorrhages by cutting the spinal cord (*Klinik d. Verdauungskrankheiten*, loc. cit.). Brown-Séquard and Schiff, as well as Ebstein (*Archiv f. exper. Pathol.*, Bd. 11, S. 183), produced gastric hemorrhage after experimental injuries to the anterior corpora quadrigemina. We have personally observed submucous hemorrhages and small areas of necrosis in the stomach after section of both vagi in cats, dogs, rabbits, and guinea-pigs.

Malaria.—It is a very well-known fact, and generally accepted by the physicians of the Southern and Eastern States, that malaria very often complicates gastric diseases, and may even be an underlying cause. It is very probable that a malarial state of the blood may be instrumental in causing gastric ulcer, which in this case has been asserted by London to be due to pigment emboli. At our clinic it is a standing rule to examine all persistent cases of stomach trouble for the presence of the malarial parasite in the blood. For the characteristics of this organism, and the methods of examination, we refer to the article by W. H. Welch and William S. Thayer, in the "Loomis-Thompson System of Medicine," and also to the able monographs of W. S. Thayer on this subject. In counties of the eastern shore of Virginia malarial gastralgia is frequent. Malaria does not, as a rule, affect the secretion or

motility, except in the various forms of pernicious malarial fever.* In gastric troubles showing any periodicity, or microscopic or clinical evidence of malaria, quinin should be promptly administered, and if not effective within twelve hours the hydrobromate of quinia should be injected hypodermically.

Anemia and Chlorosis.—The relation between pernicious anemia and atrophy of the stomach has been considered, and the claim of Austin Flint to the priority of this clinical association has been emphasized in the chapter on Achylia Gastrica. Anemia and chlorosis are influential etiological factors in the causation of gastric diseases, if they are primary conditions. This relation of the two states is very difficult to establish and probably very rare. Hayem ("Des Altérations du chimisme Stomacal dans la Chlorose," *Bulletin Méd.*, 1891, No. 87) asserts that the alterations in the stomach and intestines are the primary cause. Ewald and Rosenheim maintain that the digestive disturbances may be the results and not causes of the anemia. There are, undoubtedly, cases in which the anemia is the cause, and others in which it is the result. In some instances the treatment will throw light on this causative relation. If the secretory and motor functions of the stomach become normal with the cure of certain anemia, the gastric disturbance was the result of the state of the blood; but if the secretory and motor disturbances are marked, and perhaps of long standing, and examinations show only a slight deviation from the normal state of the blood, the digestive disturbance is the primary one. Often it is possible, when patients remain under observation for a long time, to observe the progressive anemia developing as a sequence to gastro-intestinal atrophy.

The effect of **syphilis** on gastric digestion has been considered in a separate chapter.

Pulmonary Diseases.—The most prominent among these is pulmonary tuberculosis. W. Fenwick found gastritis to be present in nearly all the cases of pulmonary tuberculosis, chronic bronchitis, emphysema, and acute pneumonia. He asserts that in diseases of the brain no gastric involvement was observed by him (*Virchow's Archiv*, 1889, Bd. CXVIII, S. 187); he found gastritis in

* A very reliable and accurate Southern colleague informed us of a case of periodical hematemesis, which he had observed near Savannah, occurring every third day, which was cured by quinin. A form of the algid pernicious malarial fever is called by some Southern doctors "gastric malarial fever."

11 cases out of 15 of phthisis. Marfan ("Troubles et Lesions Gastriques dans la Phthisie Poulmonaire," Paris, 1887) found but five cases in 61 of tuberculosis in which the gastric symptoms preceded the pulmonary. It is very difficult to decide, when a dyspeptic is at the same time affected with pulmonary tuberculosis, which trouble is primary. As a rule, diseases limited to the stomach can not so weaken the general state of health as to predispose to pulmonary tuberculosis. Rapid exhaustion from localized gastric diseases occurs only in carcinoma, which is in itself rapidly fatal before lung trouble is developed to any great extent; but when the gastric disease is associated with intestinal disturbances, so that the digestion is very much interfered with, general nutrition may be so impoverished that tuberculosis can be more readily acquired. Hutchinson ("The Morbid State of the Stomach and Duodenum," London, 1878) publishes an analysis of a large number of cases, and states that the digestive disturbances precede the tubercular infection in about one-third of the cases. It is in these cases of suspected pulmonary disease, associated with digestive troubles, that the ability of a good auscultator will tell. Gastro-enterologists should not fail to perfect themselves in the technic of auscultation and percussion. Whenever sputum can be obtained it should be examined for tubercle bacilli. The state of the gastric secretion and the motor function in tuberculosis have been studied by Edinger (*loc. cit.*), Rosenthal (*loc. cit.*), Shetty (*loc. cit.*), O. Brieger (*loc. cit.*). Immermann (*loc. cit.*), Hildebrandt (*loc. cit.*), and Einhorn (*loc. cit.*), The state of the secretory and motor functions in pulmonary phthisis varies, in our experience, with the stage of the pulmonary disease. In the incipient stages of phthisis, secretion and motility may be normal for a long time; they will become more and more deranged as the pulmonary trouble progresses, so that in the final stages of pulmonary caseation, breakdown, and formation of cavities, all gastric function may be extinguished. Brieger (*loc. cit.*) states, that in the initial stages the cases of normal and disturbed secretion are about equally divided. In moderately severe cases secretion was normal only in one-third, or 33 per cent.; in the remainder, secretion was variable, but generally depressed. In 6.6 per cent. there was no secretion whatever. In advanced cases of phthisis secretion was normal only in 16 per cent. of the cases. It was more or less defective in the rest of the cases, and in 9.6 per cent. there was complete arrest of secretion. Immermann (*loc. cit.*) found the gastric

peristalsis normal in 53 out of 54 tests, whereas Klemperer (*loc. cit.*) claims to have found marked inhibition of the peristalsis by his method. The amount of gastric secretion and the state of the peristalsis are not satisfactory exponents of the digestive powers of phthisical patients. The only correct way to find out whether such patients have digestive power sufficient to maintain the nitrogen equilibrium is by quantitative experiments on *metabolism*. By giving weighed amounts of certain foods, and determining the quantity that is digested and the quantity that is excreted undigested, together with careful determination of the amount of nitrogen in the urine, we have been able to discover that tuberculous patients, with absolute achylia gastrica, may, with care as to diet, still be able to retain their nitrogen equilibrium, provided the gastric peristalsis was preserved. In future, the exact state of the pulmonary disease, its duration and extent, together with a statement of the condition of all the remaining organs, would be desirable, if the correlation existing between gastric and pulmonary troubles is to be put upon a basis of approximate exactness. Although the treatment of the tuberculosis is the main object, it will be impossible to maintain nitrogen equilibrium with a defective digestive apparatus; it is, therefore, essential that the functions of the stomach should be improved as far as possible. In this way a system of forced alimentation, such as has been very successfully employed by Debove (*loc. cit.*), Dettweiler, Liebermeister, Leyden, Rühle, and Peiper, may become possible. In each individual case the diet and the medicine should be ordered according to the state of the gastric functions found from test-meals. We have had three patients affected with pulmonary tuberculosis at the Maryland General Hospital during the winter of 1896 and 1897, who gained considerably in weight by treatment of the existing gastritis. One patient with pulmonary tuberculosis and a tubercular rectal fistula gained 14 pounds in two months under daily lavage and administration of HCl and strychnin, together with nutritious diet. The tuberculous fistula was treated by Dr. Samuel T. Earle, and healed up completely before the patient left the hospital.

Diseases of the Heart.—We have already spoken of the effect of gastric disturbances in producing tachycardia, bradycardia, and arrhythmia. The diseases of the stomach which are caused by valvular affections of the heart, are brought about by the venous stasis and passive congestion. Under the head of chronic gastritis

we have spoken of the efficacy of digitalis when valvular disease is in clear etiological association with the gastric affection. Concerning the state of the secretion in heart diseases, there is no agreement in the observations thus far reported. In 20 patients with heart disease, Adler and Stern (*Berl. klin. Wochenschr.*, 1889, No. 49) found free HCl always present in 16, variable in two, and always absent in two cases. Hüfler states that in ten cases of mostly valvular lesions, suppression of the secretion of HCl and absence of albumin digestion was found nine times, and hyperacidity in a single case. Most of his patients are stated to have been in the stage of perfect compensation. These observations of Hüfler are not intelligible in the light of the pathological physiology of cardiac diseases. For perfect compensation means that the arterial and venous pressure in all the organs is normal; under this state we can not conceive of any passive congestion in the stomach. In our experience, gastric secretion was normal in eight cases of mitral regurgitation, two cases of aortic regurgitation, and two cases of mitral insufficiency. As soon as compensation becomes defective, the gastric symptoms make their appearance, and secretion is found altered.

Diseases of the Liver.—The close anatomical, physiological, and pathological relationship between the liver and the stomach explains the sympathetic manner in which diseases of one organ frequently reflect upon the other; excepting in the diseases of the biliary passages and gall-bladder, it is impossible to say which organ is primarily affected. During the passage of gall-stones gastric secretion is suppressed; this suppression is due to a reflex influence caused by the intense pain. We have tested the vomited matter which was brought up during attacks of biliary colic. In three cases it was neutral or alkaline; in one case it showed presence of combined HCl—no free HCl. The alkalinity of this vomit was not due to the presence of bile, or pancreatic juice, because they were found to be absent. Cases of cirrhosis of the liver, and even of cancer of the liver, may run a latent course for a long time, the symptoms being those of chronic gastritis.

Gout and Rheumatism.—Burney Yeo claims that dyspepsia is a frequent and prominent manifestation of gout (*Brit. Med. Jour.*, Jan. 7 and 14, 1888). This specific gouty disorder of the stomach is claimed to exist in states of uric acid diathesis by a number of contributors to British medical journals, but Sir William Roberts, Sir Dyce Duckworth, and Haig mention nothing about it. Ewald

states that he has not met with a single case of true gout with coincident gastric disturbances, but that he has seen numerous such examples in chronic articular rheumatism, in which the dyspepsia was so marked that the pains in the joints were comparatively insignificant. Anomalies of secretion in gout have been repeatedly observed by us. The most frequent secretory trouble is hyperacidity.

Diabetes Mellitus.—Although there is no constancy in the character of the secondary gastric symptoms accompanying diabetes, there are few cases of this disease in which the stomach is not involved. Diabetes affects the stomach in two ways principally, either by arresting its functions through auto-intoxication or by production of gastritis. The presence of great thirst, polyuria, polyphagia, ocular disturbances, pruritus, emaciation, usually means co-existent gastric involvement. Rosenstein and Gans have examined the gastric functions in diabetes (Rosenstein, *Berl. klin. Wochenschr.*, 1890, No. 13). Their results show that the disturbances, although present, show no constancy in type. The polyphagia and polydipsia of diabetes have been known to cause gastrectasia.

Diseases of the Kidney.—The stomach is always more or less affected in renal diseases, and the symptoms of disturbed gastric digestion very often appear long before albumin is present in the urine. In the *Maryland Medical Journal*, July 24 and 31, and Aug. 7, 1897, we have reported three cases of nephritis which were undoubtedly due to chronic auto-intoxication from the gastro-intestinal tract. In this connection we wish to emphasize the gastric diseases which are caused by pre-existing affections of the kidneys. Naturally, it is unavoidable that a certain amount of auto-intoxication will accompany the association of renal with gastric disease, no matter which is the primary affection. Albu (*loc. cit.*) and Biernacki (*Berl. klin. Wochenschr.*, 1891, No. 25 and No. 26) emphasize the influence of retained metabolic products in producing gastric disturbances. These retained products of metabolism injure the stomach in two ways: (1) By acting as toxins through the vascular channels directly upon the parenchyma of the gastric walls, and (2) by irritation of the surface of the stomach, since they are very frequently excreted in this manner. Fenwick (*loc. cit.*) states that the gastric mucosa is capable of secreting urea like the intestinal mucosa, and that the excretion of this product causes an acute catarrh of the gastric

glands. A variety of gastric diseases has been found to exist in connection with chronic Bright's disease. We have seen that acute and chronic gastritis, fatty degeneration of the glandular epithelium, and, according to Ewald, amyloid degeneration may occur. Edema of the gastric walls is a very rare complication. The effects of floating kidney in producing stenosis of the duodenum have been considered in the chapter on Enteroptosis. Allan A. Jones ("Gastric Conditions in Renal Disease," *New York Med. Jour.*, Jan. 19, 1895) has frequently found suppression of gastric secretion in patients with kidney diseases. Einhorn reports a case of achylia gastrica due to renal calculus, which had existed for a long time. After removal of the stone by operation, the gastric symptoms at once disappeared. According to Biernacki, the secretory function is arrested in renal affections.

Skin Diseases.—There can be no doubt that digestive troubles have an influence in the production of eczema, urticaria, erythema, the various forms of acne, and pemphigus; but there is no evidence, vice versâ, that skin troubles have any effect upon the gastric function, excepting extensive cutaneous burns. When the skin has been destroyed over large areas, duodenal and sometimes gastric ulcer were observed to develop. The homeopaths assume a great many digestive troubles to be caused by so-called "systemic" itch, and Pedioux (*L'Union Méd.*, 1866, p. 235) considered dyspepsia an expression of a herpetic state of the system. These inferences are too absurd to be considered seriously.

LITERATURE

ON THE CORRELATION OF DISEASES OF THE STOMACH TO THOSE OF OTHER ORGANS.

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CHAPTER IX.

THE BLOOD AND URINE IN STOMACH DISEASES.*

In general we may say that, while we are unable to make, in any given case, the diagnosis of stomach disease from an examination of the blood alone, it will, in many instances, render great assistance in connection with other symptoms.

The presence of an oligocythemia is found, in cases of long-continued stomach disturbances, serious enough in character to interfere with the nutrition of the body. For example, in chronic gastritis there is always a moderate degree of oligocythemia, the decrease in the number of red corpuscles running nearly parallel with the disturbance of nutrition.

In the severe forms of atrophic gastritis the decrease is sometimes enormous; so much so, that many of these cases are considered as cases of primary pernicious anemia, the true cause not being discovered until the postmortem examination is made.

In cancer of the stomach the oligocythemia is usually marked, in cases of well-developed cachexia the number of red corpuscles often being found to be between one and two million, in some instances falling below one million.

In ulcer of the stomach quite variable conditions may be found. In cases of chronic ulcer of the stomach with slight hemorrhages, the blood changes may be those of a simple secondary anemia, or the blood may approach the normal in its proportions. In acute or subacute ulcer of the stomach the blood may show a normal number of corpuscles, unless there has been a recent hemorrhage of considerable severity. In case of hemorrhage, the decrease in the number of red corpuscles is in proportion to the amount of blood lost.

In simple or benign dilatation of the stomach the anemia is proportional to the disturbance of nutrition which it produces, other things being equal.

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* The articles on "The Condition of the Blood and Urine in Stomach Diseases" and on "The Stomach Gases" were written by Dr. E. L. Whitney.

Leukocytosis.—Considerable may be learned from a study of the occurrence and degree of leukocytosis in stomach diseases.

Under normal conditions a moderate degree of leukocytosis (10,000 to 15,000) develops after meals, depending upon the absorption of proteid materials from the gastro-intestinal tract. This does not take place in the majority of cases of cancer of the stomach, but does occur in ulcer of the stomach—a fact of considerable diagnostic importance.

There is usually present in cancer of the stomach, as in malignant disease in other situations, a constant increase in the number of white corpuscles, varying from 10,000 to 50,000, the normal number being taken as about 7000 leukocytes.

In the acute inflammatory diseases of the stomach, such as any of the forms of acute gastritis, there is present leukocytosis of varying intensity. This is an important fact to remember in making a diagnosis between acute gastritis and typhoid fever in its early stages,—acute gastritis being accompanied by a moderate leukocytosis, typhoid fever showing a normal or decreased number of leukocytes.

After severe hemorrhage from a gastric ulcer, gastric cancer, or from varices in the esophagus or stomach, as from any loss of blood, the so-called "posthemorrhagic leukocytosis" occurs—a fact which should be taken into account in forming any conclusions from leukocytosis in the course of gastric diseases. This leukocytosis, as a rule, disappears in about three or four days, and can thus be excluded by frequently repeating the examination.

Red Corpuscles.—In cancer of the stomach in its later stages, the red corpuscles frequently show the changes in form known as poikilocytosis, in an exquisite manner. In cases of severe anemia of any kind, poikilocytosis may occur, but in pernicious anemia and cancer this change is most pronounced.

Hemoglobin.—In the various anemic states depending upon diseases of the stomach, the hemoglobin is decreased.

In ulcer of the stomach it is a common observation to find a normal or only slightly decreased number of red corpuscles with a considerable decrease in the amount of hemoglobin, the so-called "chlorotic blood." After hemorrhages, especially when severe, the number of red corpuscles may in a short time decrease considerably.

In cancer of the stomach in its early stages the blood may pre-

sent a similar picture; but in the later stages the number of corpuscles is decreased extremely, the hemoglobin not being diminished proportionally.

In other diseases of the stomach the alterations are usually those of secondary anemia, the red corpuscles and hemoglobin being reduced in a corresponding ratio.

Stained specimens of blood from patients suffering with the cancerous cachexia in a severe form will usually show the presence of a considerable number of normal-sized nucleated red corpuscles, as well as megaloblasts, the latter being much in the minority, a point which may be of importance in the diagnosis of severe cancerous cachexia from primary pernicious anemia.

After severe hemorrhages from gastric ulcer, fairly numerous normoblasts may be found, in addition to a decrease in the number of red corpuscles, and in the amount of hemoglobin. These changes may be of importance in the diagnosis of true gastric hemorrhage from the attempts at deception made by malingerers and hysterical patients.

In chronic atrophic gastritis the blood may show the exact picture of a primary pernicious anemia, viz., marked oligocythemia, increase in color index, decrease in specific gravity, presence of nucleated red corpuscles, normoblasts, microblasts, and megaloblasts, with a decrease in the number of white corpuscles. In by far the larger number of cases, however, the blood changes are simply those of a severe secondary anemia.

Alkalinity of the Blood.—The researches of Loewy concerning the alkalinity of the blood in health and disease, by the method which he has devised, have shown the sources of error in the methods formerly in use, and rendered a revision of our opinions necessary. The method is one which will probably supersede all others for the clinical laboratory, on account of simplicity of execution and accuracy. At present there are not enough observations recorded to permit us to speak of its application in diagnosis. In the observations made with reference to digestion, it has been shown that a rich secretion of HCl by the stomach increases the alkalinity of the blood, and vice versâ. Whether this fact can be of utility in the study of gastric diseases must remain at present undecided.

In addition to the examination of the blood for the preceding constituents, it may be of importance in cases of continued fever

with marked gastric symptoms, in which the diagnosis lies between some severe inflammatory disease of the stomach and typhoid fever, to make a trial of the Widal test for typhoid.

This test is of no value in the early days of the disease, the reaction seldom appearing before the seventh day, and rarely only on the fifth or sixth day.

The results of our knowledge of the blood changes in the various stomach diseases may be summed up as follows:

Acute Gastritis.—Usually a slight degree of leukocytosis, increasing with the intensity of the inflammation.

Chronic Gastritis.—A decrease in the number of red corpuscles and hemoglobin, the leukocytes showing normal numbers, as a rule.

Chronic Atrophic Gastritis.—The blood may show the same changes as in the simple chronic gastritis, or may show the blood changes of pernicious anemia (poikilocytosis), marked decrease of red corpuscles, a marked decrease in hemoglobin, the decrease being less in proportion than that of the red corpuscles, a decrease in the number of leukocytes, and the presence of a large number of nucleated red corpuscles, normoblasts, megaloblasts, and microblasts.

Gastric Ulcer.—In the old chronic forms of ulceration the blood usually shows the changes of a secondary anemia, as in chronic gastritis.

In ulcers of recent origin the blood may show no variations from the normal, or it may show the characteristic changes of chlorosis, viz., nearly a normal number of red corpuscles with a considerable decrease in the percentage of hemoglobin.

After hemorrhages, the changes are those common to loss of blood from any part of the body—a decrease in the red corpuscles and hemoglobin, an increase of the leukocytes for a few days, and the presence of normoblasts in the blood.

In ulcer, digestion leukocytosis occurs, a point of some value in the differential diagnosis between ulcer and cancer.

Cancer of the Stomach.—In the early stages the changes may be simply those of secondary anemia. In the later stages, when the cachexia becomes apparent, the blood changes are rather characteristic. There is a marked decrease in the number of red corpuscles and in the amount of hemoglobin, the former being often between one and two million, the latter from 20 to 30 per cent. There are

often a number of nucleated red corpuscles, both normoblasts and megaloblasts. The red corpuscles may show variations in size, averaging smaller than normal, often with an exquisite poikilocytosis. A leukocytosis is usually present, varying greatly in its intensity. There is, with rare exceptions, no digestion leukocytosis.

In dilatation of the stomach from benign causes, the changes are simply those of secondary anemia, the alterations being proportional to the disturbances of nutrition.

THE GASES OF THE STOMACH.

Under normal conditions the stomach contains a mixture of gases, derived in part from air swallowed with the food, in part from chemical and fermentative processes in the stomach, and possibly from a small amount of CO_2 eliminated from the blood flowing through the gastric mucosa. The contents of a normal stomach, removed at the height of digestion, and placed in a fermentation tube at the body temperature, exhibit for several days only slight gas formation, this occurring only when the free HCl has been nearly or completely neutralized by the food products. After this, fermentation and putrefaction proceed as usual in fluids rich in proteid and carbohydrate material.

Under pathological conditions, such as marked dilatations with stenosis, especially when due to malignant disease, the case is altered. The food, which usually contains a variable number of bacteria, is not properly sterilized in the stomach on account of the partial deficiency or absence of HCl, and it remains for a long time in the stomach; in addition, it is not subjected to a normal peristalsis. The requisite conditions for an abundant bacterial growth are thus present, viz., an animal fluid (containing both carbohydrates and animal proteids), heat, and moisture.

Various gases have been found in the stomach in such conditions, among which may be named acetylene, hydrogen, carbon dioxid, nitrogen, oxygen, marsh gas (CH_4), and sulphuretted hydrogen.

The question of the special variety of gas is not of as much importance as that of the formation of any gas. Accurate gas analyses have, up to the present time, yielded little of diagnostic value and from their difficulty will seldom be attempted by the general practitioner.

The presence of combustible gases in dilated stomachs has been

first demonstrated by G. Hoppe-Seyler ("Verhandl. d. Congr. f. innere Medicin, 1892," S. 392) and F. Kuhn (*Zeitschr. f. klin. Med.*, Bd. XXI, S. 572). These investigators demonstrated that hydrogen, marsh gas, etc., could be formed notwithstanding the presence of a considerable amount of free HCl. The influence of various antiseptic agents on the process of gas formation in the stomach has been carefully investigated by F. Kuhn, whose results constitute an important practical contribution to the therapy of gastrectasia.

To test for the presence and amount of gas formation, the freshly drawn stomach contents are well mixed and broken up into a finely divided state, poured into a fermentation tube (that devised by Einhorn for the estimation of sugar in urine, or the ureometer of Doremus may be used), and the tube (loosely stoppered with cotton) placed in a warm oven at the temperature of the body. If none of these is at hand, a fair substitute may be improvised by filling a large test-tube with the stomach contents and inverting over a small beaker partially filled with the same material, allowing the lower end of the test-tube to dip into the stomach contents in the beaker, to retain the fluid in the tube by atmospheric pressure.

If evolution of gas take place within a few hours the presumption is that we have to deal with a case of stenosis of the pylorus; and if, at the same time, we find a marked formation of lactic acid and an absence of HCl, we may assume that it is a case of malignant stenosis of the pylorus with a high degree of dilatation.

In non-malignant stenosis we find, as a rule, that the gas formation goes on rather less rapidly and is not associated with an excess of lactic acid as in malignant stenosis.

In dilatation unaccompanied by stenosis, and even in the presence of small quantities of free HCl, we find that gas is formed; often, however, only after the tube has been allowed to stand for several days.

The gas may be submitted to various tests to determine its character.

A few c.c. of a strong solution of caustic soda are placed in the lower part of the fermentation tube and allowed to stand for some time. If the gas is composed partly or wholly of CO_2, it will be absorbed by the alkali, and its volume percentage may be read off directly by the decrease in volume of the gas.

A small amount of the gas is allowed to bubble out and a piece

of filter-paper, previously dipped in a solution of lead acetate, is held in the gas as it escapes. If any sulphuretted hydrogen is present, the paper will turn black, due to the formation of lead sulphid.

A portion of the gas may be tested for inflammability by allowing it to flow out as before and attempting ignition by holding a lighted match to it as it escapes. If it take fire or give a slight explosion, the probabilities are that hydrogen, marsh gas, or acetylene are present.

URINARY CHANGES IN STOMACH DISEASES.

While many interesting and valuable observations upon the urinary alterations in stomach diseases have been made, it must be stated that, up to the present, little of diagnostic importance has been determined. It is not without interest, however, to take a short review of the topics so far as it concerns the subject of this book.

The Amount.—So long as appetite, digestion, and absorption from the stomach and intestinal tract are little interfered with, there are only trifling alterations in the quantity of urine.

The quantity of urine sinks in cases of vomiting; as, for example, in acute gastritis and gastric ulcer, the decrease in the amount of urine being in an almost exact ratio to the amount of fluid lost by vomiting.

It is, however, in marked cases of gastric dilatation and pyloric stenosis that the greatest decrease in the amount of urine is noticed. In well-marked cases of dilatation the amount of urine often sinks to from 300 to 500 c.c. Under these conditions a continued low quantity is an unfavorable prognostic sign, while an increase indicates an improvement in the motor function.

The specific gravity has little diagnostic significance, but, in general, has about the same value as the amount of urine. In cases of dilatation, in which the element of inanition is beginning to be a factor in the clinical picture, we find that the solids of the urine, as indicated by the specific gravity, fall; while in cases in which the nutrition is well preserved, the total solids of the urine approximate the normal value.

The Reaction.—Bruce Jones, in 1819, first explained the well-known relation existing between the secretion of gastric juice and the reaction of the urine. After a meal the acidity of the urine decreases, often becoming neutral, and, occasionally, alkaline in

from three to five hours. Subsequently the acidity of the urine increases, reaching about the average a short time after the food has been propelled from the stomach into the intestines. The range of variation is greater following a full meal than a light repast, so that a greater fall in the acidity is found after dinner than after either of the other meals.

This phenomenon Bruce Jones explained upon the ground of a greater alkalinity of the blood, as a consequence of abstraction from it of acids or acid-forming substances. The presence of an increased alkalinity of the blood leads to increased excretion of alkalies in the urine and a diminished acidity. An additional factor may be the presence of alkalies or of alkaline carbonates in the food, which neutralize the acidity of the gastric juice, and assist also, after absorption, in increasing the alkalinity of the blood.

From these physiological facts the variations of the reaction in disease are easily understood. In the vomiting of ulcer, if profuse, a large amount of acid is withdrawn from the system ; and, in some cases, the urine may exhibit for some time a neutral, or even alkaline, reaction. In cases of hyperacidity of the gastric juice, in which a larger quantity of acid (than normal) is withdrawn from the blood, the curve of urinary acidity undergoes greater variation than under normal conditions. In cases in which there is a considerable diminution, or a total absence of the acid secretion of the stomach, this variation in the reaction of the urine does not occur, or if any variation does take place it is less marked than under normal conditions. Hence it will be seen that in cases of atrophic gastritis, severe chronic gastritis, and in carcinoma of the stomach, with an absence of HCl, little or no variation in the acidity of the urine occurs,—a fact that may be of some importance in the differential diagnosis between carcinoma and ulcer of the stomach, especially where the occurrence of hematemesis or bloody stools contraindicates the use of the stomach-tube.

The Chlorids.—The amount of chlorin depends primarily upon the amount of food, and, with it, on the amount of chlorids absorbed from the stomach. The amount of chlorids present in the urine is also in an inverse ratio to the amount of HCl secreted by the stomach.

In ulcer of the stomach, associated with considerable vomiting of a large amount of highly acid gastric juice, a considerable decrease in the amount of urinary chlorids will be found, as also in other cases of vomiting in which a large amount of HCl is lost.

In hyperacidity without vomiting, the amount of urinary chlorids decreases as secretion goes on, diminishing in well-marked cases to a very low percentage, increasing again as absorption from the stomach and intestine goes on, finally reaching the highest part of the chlorid curve five or six hours (sometimes later) after the ingestion of the meal.

In cases of anacidity this curve of urinary chlorids does not occur, no fall of chlorids occurring after the meal, a slight increase taking place as the absorption of the food goes on.

In severe cases of gastrectasia with pyloric stenosis, the amount of chlorids in the urine sinks very considerably, the decrease being in proportion to the severity of the affection. An increase in the amount of chlorids without a corresponding change in the diet may be taken as a symptom of improvement, in this respect being even of more prognostic importance than the increase in the amount of urine which usually occurs at the same time.

Taken in connection with the total nitrogen of the urine, the amount of chlorids may be of assistance in determining the question of a benign or malignant stenosis of the pylorus.

If one finds, for example, in the urine a small amount of chlorids and a proportionally small amount of nitrogen, it speaks for a simple inanition, a benign stenosis; but if, on the other hand, one finds a small amount of chlorids and a relatively large amount of nitrogen, it speaks for the presence of a malignant stenosis.

The Phosphates.—The investigation of the excretion of phosphates in stomach diseases has yielded little in the line of diagnosis as yet. The deposit of basic phosphates in the freshly voided urine passed after meals is quite frequently seen in cases of hyperacidity, though the same change may also occur in perfectly healthy individuals. The deposit of the basic earthy phosphates in these cases is due to the alkaline tide spoken of in the paragraph upon the reaction of the urine.

In hyperacidity, according to Robin, the excretion of phosphoric acid is considerably increased.

According to F. Müller, the excretion of phosphates is increased in cancer of the stomach, though not in all cases.

Of more diagnostic importance, however, is the relation existing between the excretion of nitrogen and phosphoric acid. Under normal conditions the proportion is about as follows:

$$N : P_2O_5 :: 100 : 17 \text{ to } 20.$$

In malignant disease in general, the proportion of P_2O_5 rises, in one case of gastric carcinoma, recorded by Chas. E. Simon, the relation being 100 : 34.

The Sulphates.—Of the two forms in which sulphuric acid occurs in the urine, the preformed sulphates have only a passing interest as being a measure of the proteid metabolism going on in the system; while the ethereal sulphates have a more direct bearing, as they seem to be formed chiefly by putrefactive changes within the intestine. As a normal secretion of HCl and a normal motility seem to be the chief checks upon intestinal putrefaction, it can be readily seen that in cases of sub- or anacidity, especially when associated with an impaired motility, the putrefactive changes in the intestine are increased and lead to an increased formation and excretion of the ethereal or combined sulphates.

In expressing an opinion as to the condition of the stomach from the amount and proportion of the ethereal sulphates, care must be taken to exclude any intestinal or peritoneal troubles, which would, by themselves, have a tendency to increase the amount of putrefaction in the intestine.

Under normal conditions the amount of the total sulphuric acid is from two to three gm., increasing under a meat diet, decreasing under a vegetable diet; the amount of ethereal sulphuric acid being from two to three decigrams.

The average normal ratio between the preformed and ethereal sulphuric acid is as ten is to one, the proportion being stated, as a rule, as follows:

A : B :: 10 : 1

In cases of nervous anacidity with periods of normal secretion, it may assist in forming an opinion as to the severity of the case to find a normal ratio of the sulphates. In a case of anacidity due to some organic trouble, the amount of ethereal sulphates will, as a rule, be increased, and the ratio, instead of being one to ten, may be increased to a marked degree, in some cases reaching nearly equal amounts.

Indoxyl-sulphate of potassium has, for the most part, the same significance as an excessive amount of ethereal sulphates or an increase in their ratio.

This chromogen of the urine is a product of the putrefactive bacteria of the intestinal canal. As the result of their action upon the

proteid bodies of the intestinal canal, indol is produced from which, by successive oxidations, indigo-blue is formed. This, by combination with the elements of sulphuric acid and potassium, forms the substance,—indoxyl-sulphate of potassium, or indican.

Owing to the fact that certain species of bacteria may be present which do not form indol, the absence or presence of this body in normal amounts in the urine does not prove the absence of putrefactive changes in the intestinal canal.

Recent observations of Charles E. Simon (*loc. cit.*) show that indican is present in excess in the urine, in cases of marked subacidity, anacidity, gastric carcinoma, stenosis of the pylorus from any cause, and also in cases of gastric ulcer with hyperacidity.

Careful study of a series of cases of gastric diseases with relation to the urine seems to show that those cases of gastric disease with a marked increase in the amount of indican are those which are associated with a lack of motility.

It must be said, however, that the ratio of the ethereal to the preformed sulphates is a better index to amount of decomposition in the intestinal canal.

Indigo-red, also called urrhodin, has essentially the same significance as indigo-blue or indican.

Urea and Nitrogen.—The amount of nitrogen eliminated in the urine may come direct from the food ingested or from the nitrogenous metabolism of the body.

In general it may be said that those diseases which are attended with impaired digestion of proteid foods are attended with a decrease in the amount of nitrogen eliminated in the urine. In the cachexia which results from cancer of the stomach, the elimination of nitrogen is greater than is the elimination in simple inanition of the same degree of severity.

There is at present no explanation of this fact unless it be due to some product of the tumor itself, which is eliminated by the urine, or to the increased metabolism of the body resulting from some product of the neoplasm. This theory as to the production of some poisonous substance by the tumor itself is supported by the fact that even small carcinomata, situated in a part of the body where they in no way influence the bodily functions, give rise occasionally to an increase in the elimination of nitrogen by the urine.

The proportions of the various nitrogenous bodies in the urine, according to the few published observations, depart considerably

from the normal in carcinoma. In normal urine the nitrogenous matter is divided about as follows: Urea, 96 per cent.; uric acid, 1.8 cent.; ammonia, 1.2 per cent.; and extractive matters, 0.6 per cent. to 0.8 per cent.

In cases of carcinoma examined by Töpfer the proportions were as follows: Urea as low as 80 per cent.; uric acid, one to five per cent.; ammonia, 0.2 per cent. to 13 per cent., and extractive matters 13 per cent. to 23 per cent. Von Noorden also found the ammonia increased (10.2 per cent. to 13.9 per cent.).

It is thus seen that in these cases there is a relative decrease in the amount of urea with or without a rise in the amount of uric acid, and a considerable increase in the relative amounts of ammonia and extractives.

Acetone and Diacetic Acid.—Acetone and diacetic acid occur in the urine in various disturbances of the digestive tract, affecting both the stomach and intestines, and, according to Lorenz, their occurrence is not infrequent.

Acetone occurs frequently in the digestive disturbances of children, and is by some authors considered as the cause of the convulsions which so often accompany these derangements.

Acetone is found in the urine in increased amounts in cases of inanition and in cachectic conditions, and to this fact may be assigned the occurrence of acetone in increased amount in cases of cancer and of dilatation of the stomach.

In general it may be said that the increase in amount of acetone and the presence of diacetic acid simply indicate an increase in albuminous disintegration.

Albumin.—Albumin is not of infrequent occurrence in cases of gastric disturbances. It may be the result of any severe stomach disease.

Von Noorden found albumin with relative frequency after the onset of severe gastric pains, such as occur at intervals in ulcer of the stomach and also after profuse hematemesis. In cases of cancer of the stomach, especially in the later stages, albumin is found, either regularly or temporarily, in a large proportion of cases.

Peptonuria.—The question as to the occurrence of peptone in the urine—as the word peptone is now employed—is not definitely settled.

Under certain conditions the urine fails to react to the ordinary tests for albumin, such as Heller's nitric acid test, Purdy's acetic

acid and potassium ferrocyanid test, and the boiling test; while it certainly contains some form of proteid which reacts to the biuret test.

The question whether this is a secondary albumose, pure peptone, or a mixture of the two, is of minor importance from a clinical standpoint. Recent investigations show that, in the course of absorption, peptones are acted upon by the epithelium of the gastro-intestinal tract and reconverted into the coagulable proteids.

This theory renders the occurrence of peptonuria in the course of gastric ulcer, carcinoma of the stomach, erosions of the mucosa, and ulceration of the intestine easy of comprehension. The occurrence of these bodies in such diseases is general in this class of cases, though many observers have failed to demonstrate them in all cases.

Ferments.—Under normal conditions the urine contains a variable quantity of pepsin and rennin ferment. The maximum excretion of pepsin is found from four to six hours after meals. Pepsin is often found to be decreased or entirely absent in the urine in cases of cancer of the stomach, and would probably be found, in the course of extended observations, to vary in an exact ratio to the amount of pepsin formed by the stomach.

Rennin is found to undergo the same variations as pepsin, and its variations have the same clinical significance.

Ehrlich's Diazo Reaction.—The presence or absence of this color reaction of the urine was formerly thought to be of great diagnostic importance for the recognition and differentiation of typhoid fever. In the two diseases of the stomach (acute and phlegmonous gastritis) with which, in its earlier stages, typhoid fever might be confounded, it would be of great value were its accuracy undoubted. Quite an extended experience with this test, used on all patients entering the hospital for several months, showed conclusively that it could not be relied upon as a differential test, several patients giving a typical reaction whose history before, during, and after the examination excluded typhoid absolutely. On the other hand, even in typhoid fever the reaction frequently is absent, so that any absolute deductions as to the disease from this reaction are apt to be misleading.

PART THIRD.

THE GASTRIC CLINIC.

CHAPTER I.

ACUTE GASTRITIS.

Simple Acute Gastritis.—Phlegmonous or Purulent Gastritis.—Suppurative Inflammation of the Gastric Mucosa.—Abscess of the Stomach.—Infectious Gastritis.—Gastritis Mycotica or Parasitaria.—Gastritis Diphtherica and Crouposa.—Toxic Gastritis.—Gastritis Venenata.

Gastritis is a collective or generic term which comprehends all inflammatory processes of the stomach proper, including the so-called catarrh of the superficial layer of columnar epithelium, the inflammation of the glandular parenchyma and interstitial connective tissue, the purulent infiltration of the submucosa and muscularis, and also the penetrating excoriations of corrosive poisons.

It is natural that these manifold morbid conditions should present considerable variations in etiology as well as in the intensity of the symptoms. It is almost impossible to draw a sharp limit separating the simple superficial catarrhs from the deeper, penetrating inflammations. Penzoldt suggests the line between mucosa and submucosa.

We may designate as simple gastritis that inflammation of the gastric mucosa which involves not only the superficial columnar epithelium, but, as a rule, the glandular parenchyma. This condition may occur in an acute and in a chronic form, and under each classification—the acute and the chronic gastritis—one may arrange two subdivisions: (1) the primary and (2) the secondary gastritis.

We therefore have (1) the acute, simple, primary gastritis, which occurs as the original disease; and (2) the acute secondary gastritis, known as the gastritis sympathica acuta, which occurs not as the original disease, but as a frequent accompaniment of numerous acute febrile disorders. All the exanthematous infectious diseases—

measles, scarlatina, variola, typhus and typhoid fevers, puerperal fever, pyemia, dysentery, croup, and diphtheria—are known to effect pathological changes in the gastric mucosa directly, or to influence it detrimentally by reflex nervous action (Hoppe-Seyler, "Allgemeine Biologie," 1877, p. 242).

There is a very plausible desire evident in some recent works on the subject to avoid the name *stomach or gastric catarrh*, because the word *catarrh* has reference to a superficial inflammation, but in gastritis we are dealing also with parenchymatous inflammation. Penzoldt uses the expression "simple gastritis" for an inflammation reaching no deeper than the submucosa (*gastritis simplex*); for the penetrating results of suppurative or purulent inflammation he uses the term "grave gastritis" (*gastritis gravis*). He does not favor the terms "toxic" and "infective gastritis," for to a certain extent all forms of this disease are toxic and infective, and in his book, "Specielle Therapie innerer Krankheiten," vol. IV, p. 320, he discusses only (1) simple and grave acute gastritis, (2) chronic gastritis, and (3) purulent or suppurative gastritis. Fleischer ("Specielle Therap. u. Pathol. d. Magen- u. Darmkr.," S. 793) describes (1) simple acute, (2) secondary or sympathetic acute, (3) phlegmonous or purulent, (4) toxic, (5) diphtheritic, croupous, mycotic, parasitic, (6) chronic gastritis. Excepting those forms mentioned by Fleischer under group 5, Boas describes all of these in separate chapters.

Ewald mentions and describes all of these forms, and subdivides the suppurative inflammation (the *gastritis phlegmonosa purulenta*) into an idiopathic primary and a metastatic secondary form. Sidney Martin's treatise on "The Organic and Functional Diseases of the Stomach" deals with the symptomatology, pathology, and treatment of acute and chronic gastritis in one chapter (chap. VIII), and then goes on to speak of toxic and infective gastritis in the next chapter (chap. IX). Albert Mathieu, of Paris, briefly mentions acute and chronic gastritis, and the varying amount of mucus and acid accompanying these diseases; none of the other forms are referred to. (New York edition.)

Rosenheim ("Pathol. u. Therap. d. Krankh. des Verdauungsapparates," p. 99) describes gastritis as acute, simple, phlegmonous, toxic, diphtheritic, and chronic. Einhorn approaches the simple classification of Penzoldt, and divides acute gastritis into (1) simple, (2) phlegmonous, and (3) toxic, and then proceeds to the consideration of chronic gastritis.

Alois Pick describes (1) acute, (2) infectious, (3) phlegmonous, (4) toxic, (5) parasitic, and (6) chronic gastritis ("Vorlesungen über Magen- u. Darmkrankheiten," S. 73); and Fleiner ("Lehrbuch d. Krankheiten d. Verdauungsorgane") gives an account of (1) gastritis catarrhalis acuta, for which he also uses the name gastricismus; (2) gastritis toxica; (3) interstitial suppurative gastritis, stomach abscess and stomach phlegmone, or gastritis phlegmonosa or interstitialis, or submucosa purulenta, or also linitis suppurativa; (4) mycotic gastric inflammations; (5) chronic gastritis.

Osler, in his new "Principles and Practice of Medicine," pp. 348 and 359, considers (1) acute simple, (2) phlegmonous or acute suppurative, (3) toxic, (4) diphtheritic or membranous, (5) mycotic or parasitic, (6) chronic gastritis. Under the latter he gives a special paragraph to the chronic forms with extreme connective-tissue proliferation and increase in thickness of the submucosa and muscularis, under the name of sclerotic gastritis.

These references are sufficient to demonstrate the discrepancy existing in later works concerning the separate and distinct recognition of the various forms of this disease, and that a more uniform classification would be desirable.

In accordance with Penzoldt, this treatise will describe only (1) simple acute gastritis, (2) simple chronic gastritis, and, separately, (3) the forms in which the element of pus formation is a factor—the suppurative gastric inflammations; and in a supplement the forms due to toxic or corrosive agents, and the remaining very rare varieties, may be appropriately described.

Nature and Concept.—One should be very careful not to diagnose every temporary, transient gastric disturbance as acute gastritis, nor a prolonged loss of appetite, with eructations, coated tongue, and no other demonstrable signs and symptoms, as chronic gastritis; as Penzoldt correctly says, this is, in the majority of such cases, neither justifiable nor conducive to the scientific development of diagnosis. We can agree with him in the opinion that it is inconceivable that all the functional and anatomical changes which one is accustomed to find in acute inflammations in other tissues, should really be present in every brief digestive disturbance after dietetic errors, alcoholic abuse, etc.

We could not designate a brief irritation of the nose, with sneezing, secretion of mucus, and hoarseness, lasting several hours, as nasal catarrh. By catarrh of the air passages we understand a

more lasting affection, with a somewhat typical course, and more permanent changes, of both a structural and functional nature, in the mucosa. Indeed, Sidney Martin very appropriately considers these functional, lighter forms of gastric disturbance in separate chapters, and classifies them under (1) gastric irritation, and (2) gastric insufficiency. Functional disorders, then, are irregularities of gastric motility, absorption, and secretion, and also of the innervation and vascular supply, in which organic diseases of the stomach —ulcer, gastritis, neoplasm, etc.—are absent. It can not with certainty be stated that all histological changes are absent in functional disorders; at least, not in functional disorders of secretion. We have become convinced of certain changes in the acid and ferment cells that are apparently quite constant.

Ever since Beaumont's pioneer observations it has been known that every severe inflammatory irritation of the gastric mucosa produces an alteration in the gastric secretion, the quantity and effectiveness of which is much reduced; it is known, furthermore, that the impairment of one function of the stomach, as a rule, rapidly involves that of another. The inner lining of the stomach can not, in the true anatomical meaning of the word, be called a mucous membrane, because it is devoid of one of the essential attributes of a mucous membrane—the mucous glands. The mucus of a normal stomach is surprisingly small in amount, and owes its origin not to glands but to mucoid degeneration of the superficial columnar epithelial cells.

As this cylindrical epithelium continues down into the alveoli of the peptic tubules without any distinct border line, all irritants striking the former must of necessity affect the parietal or border cells as well as the chief cells of the gland-duct. It is characteristic of the pathology of gastric digestion that impairment of one important function, or rather of one of the many physiological processes of which the digestive act is composed, soon creates sympathetic disturbance in the remaining functions, so that the clinical picture of an acute or chronic gastritis is that of a combination of disturbances.

It is not established, nor very essential, which function suffers first, but probably in most cases a derangement of secretion starts the morbid series, and the remaining functions follow in the affection. For example: If by ingestion of food which is already in a state of fermentation an acute gastritis has been induced, the reduc-

tion in the amount of hydrochloric acid produces a hindrance, not only in the normal chemistry of the stomach, but resorption and motility are also very soon retarded. This pronounced subacidity has in its consequence an imperfect digestion of the proteids, so that very small amounts of acid albumin and hemialbumose are detectable in the vomit, and peptone is found in traces only. A further step, then, is that these undigested proteids continue to remain in the stomach longer than with normal proteolysis. This means a much more prolonged burdening of the gastric walls; the stomach does not gain sufficient rest in which to prepare itself for the demands of the following meal; the distention by the weight of the food lasts longer.

On the other hand, much more of the carbohydrates will in this subacidity be converted into soluble starch, maltose, and dextrose, than with a normal secretion of hydrochloric acid. With the progressing stagnation and putrefaction of proteids, these products of starch inversion mean more ready food for bacteria, which are constantly introduced with the saliva, and, finding in the moisture and suitable temperature of the gastric contents congenial conditions for their development, the danger of progressive decomposition is very great. The toxic products of this carbohydrate and proteid decomposition are irritants to the mucosa, and increase the already existing inflammation.

When this inflammation has reached a certain stage, an inflammatory edema of the muscular layers sets in, effectually destroying the motility, and, simultaneously (as in most all serous and mucous inflammations), an alkaline transudate exudes into the mucosa, neutralizing the last vestige of hydrochloric acid that may yet be secreted. Lactic, butyric, and acetic acid are evolved from the fermenting carbohydrates, and, further on, H_2CO_3 and H.

When these gases begin to expand, and the already impaired motility can not expel them by eructation, the stomach is still further distended. The normal hydrochloric acid not only acts as an antiseptic and antifermentative, but, as we know, undoubtedly brings about energetic peristalsis, which effects a thorough mixing of the ingesta, and, frequently, repeated contact and friction with those portions of the secretory membrane whose glands produce the hydrochloric acid and ferments. This mixing and triturating peristalsis is at the same time a most essential stimulus to absorp-

tion, and eventually effects the timely expulsion of the chyme into the duodenum.

With impaired motility, therefore, the food masses remain too long in one and the same place. An intimate contact of the ingesta with the membrane, as is produced by healthy peristalsis, is essential for normal stimulation to continued secretion; hence, the secretion of the oxyntic and ferment cells, already damaged by inflammatory infiltration, soon ceases entirely. Resorption is not only impaired by absence of intimate contact with ingesta, but by the fact that the epithelial surface is, in the various forms of gastritis, covered with a tough glassy mucus, epithelial detritus, and sometimes pus. In addition to this, one must not overlook the element of the effects of the inflammatory changes on the rate, tonicity, quality, and quantity of the circulation on all of the gastric functions.

The damaging effects of inflammation might be partly made up again by a healthy peristalsis, but, as this is not present, resorption and secretion are inhibited. The suspension of the resorption must be looked upon as an act of self-protection, as there are nothing but poisons to absorb in these conditions. There is, fortunately, no excessive formation of peptone, as this is prevented by the subacidity. We say "fortunately," because it would simply be food for bacteria. So it is evident that, in an acute gastritis, there are numerous concurrent deleterious elements and changes which are essentially similar to those of most light and severe gastric inflammations.

The clinical picture is a very manifold one, as in the individual cases one may observe first one and then another function that is most seriously damaged. It is natural to observe exceptions from the rule: thus, in prolonged anacidity we may find cases in which the motility is unimpaired, which of course favors intestinal digestion by timely evacuation of the chyme, so that even the symptoms of dyspepsia may be lacking.

Etiology.—In the majority of cases, acute simple gastritis is caused by errors in diet. Irritation may be caused by quantity as well as by quality of the food. Decomposed articles of liquid or solid nature will set up inflammation through the bacteria they contain. These germs must not be thought to invade the mucosa proper in all cases; they exert effects by their action. Ewald (*loc. cit.*) says

he has never found bacteria in the gastric tissues in these cases. Spoiled or decomposed meat, fish, or vegetables, cheese, wine, cider or beer that has not completed its fermentation, infected milk, and impure pond water have been known to produce severe acute gastritis.

Excessive indulgence in perfectly healthy food can provoke the trouble, not only by the mechanical distention and irritation which are caused thereby, but by the inability of the motor power to move the ingesta about and to expel them into the duodenum, and also by the deficiency in the secretion of gastric juice, which may be able to digest a normal but not an excessive amount of food. The amount that can be digested under normal conditions without causing acute gastritis will naturally vary considerably in different individuals.

Chemical Causes.—Among these may be mentioned quinin salts in large doses; all metallic salts, particularly those of copper, antimony, arsenic, lead, gold, mercury, and silver; acids, and alkalies, unless properly diluted.

We have observed an acute gastritis follow the use of two gm. of sodium salicylate three times daily, and feel convinced that iodid of potassium, if not given properly mixed with food (right after meals), may lead up to gastritis. The various drugs used internally for gonorrhea—cubebs, copaiba, and the oil of sandalwood—may, in susceptible individuals, bring about, after long use, a condition of the gastric mucosa in which acute gastritis is readily set up.

Psychic Causes.—It is said that grief, sorrow, terror, anger, and even excessive joy (?), have been observed to produce gastritis. Sexual excesses, particularly in neurasthenics, are on record as causes.

Thermic Causes.—Large quantities of very cold or very hot liquids, particularly the former, can produce the disease, when taken in rapidly when the body is in an overheated state.

Mechanical Causes.—It is possible that pieces of fish bone, egg shells, or oyster shells, or fruit seeds, if accidentally ingested, may, by mechanically scratching and bruising the mucosa, cause a gastritis. We had occasion to observe a singular case of this disease in a professional base-ball player, caused by a blow from a base-ball pitched with great speed. The bruise extended from the xyphoid cartilage to the left hyprochondriac region. The player was knocked senseless, and, after partial recovery, vomited a meal, which he had taken two

hours before, mixed with blood and much mucus; later on he vomited some milk that was given him, and on being tested this vomit was alkaline.

The pain was so severe that morphin had to be injected hypodermically, and food was kept out of his stomach altogether for three days, during which period he was fed by Boas' nutrient enemata. The attack lasted two weeks, and the patient made a perfect recovery.

Predisposition.—Manassein has shown that fever produced experimentally in dogs which he had made anemic by depriving them of much blood, caused considerable suppression of the secretion of hydrochloric acid. Kussmaul, Uffelmann, Leube and von den Velden have confirmed this subacidity in cases of fever in the human being. It is not surprising, therefore, if we find acute gastritis developing in convalescents from severe diseases; also in tuberculous, cancerous, and syphilitic patients. Functional gastric disturbances predispose to acute gastritis as well as pre-existing or concomitant diseases of the heart, lungs, liver, and kidneys. Ewald believes in hereditary predisposition to gastritis, as some families show numerous cases of the trouble in spite of the best care they take of their stomachs.

Idiosyncrasy.—It is a very perplexing fact that some persons in good health acquire acute gastritis after eating certain articles of food. While the author was resident physician of Bay View Asylum, Baltimore, he had a colleague, a perfectly robust, vigorous man, who was not at all neurasthenic, who developed the disease every time he ate oysters. He could not be induced to eat them after he had established the causal relation, but convinced us by consenting to an experiment.

Influence of Sex and Age.—Acute gastritis occurs more frequently in men than in women; of 36 cases observed by the author, of which records were taken, ten occurred in females and 26 in males. Females are more frequently attacked during menstruation and puerperium. Old persons and very young, feeble children are more likely to be attacked than those in middle age. In nursing infants a very slight change in the milk may be enough to cause it. According to Booker, of Baltimore, acute gastritis in infants is accompanied by prolongation of the time that the milk is retained in the stomach, at times over five hours. The gastric contents occasionally show epithelial and pus cells.

Rotch ("Pediatrics," p. 854) holds that the acute form is more common in infants, and that the chronic form, while it does occur in them, is more frequent in children toward puberty. The frequent attacks of gastritis occurring during the hot summer months are undoubtedly largely due to the consumption of unripe fruit. Bouveret, however ("Traite des Maladies de l' Estomac," p. 384, Paris, 1893), attributes them to the abusive consumption of water. According to Pick, the disease has been observed to develop after taking cold.

The effect of fever on the secretions of the stomach is not always evident. Edinger found the secretion of hydrochloric acid normal in five cases of fever; having examined hectic, recurrent, intermittent, and typhoid fever patients (L. Edinger, "Zur Physiol. u. Path. d. Magens," *Deutsch. Archiv f. klin. Medizin*, Bd. XXIX, S. 555). G. Klemperer ("Dyspepsie d. Phthisiker," *Berlin. klin. Wochenschr.*, 1889) and Schetty ("Untersuch. ü. Magenfunction bei Phthisis," *Deutsch. Archiv f. klin. Med.*, Bd. XLIV, S. 516) confirm the finding of Edinger. Ewald (*loc. cit.*, p. 301) found almost normal digestive power in a case of facial erysipelas. From these studies it is plain that not in all cases of secondary acute gastritis can we attribute the stomach affection to the functional disturbances which the primary disease produces: for, in the first place, these may be entirely absent; secondly, the frequency of the gastritis is not at all dependent upon the height or intensity of the fever; thirdly, the secondary sympathetic gastritis may set in concomitant with the fever or even before it, ushering in the main infectious symptoms as a prodromal affection.

The secondary sympathetic gastritis is therefore more likely to be originated by localization of the specific, organized disease-producers of the fundamental disturbance—in the mucosa of the stomach, or even by the toxic metabolic products of these microbes. In addition to the infectious diseases already mentioned, this sympathetic form may be a consequence of diseases of the heart, lungs, kidneys, and liver, causing venous, passive congestion of the gastric mucosa (Stauungskatarrh). In cardiac and nephritic diseases the passive gastric congestion may be relieved by appropriate medication directed to the fundamental disorder; *i. e.*, the use of digitalis, strychnin, and diuretics.

Pathological Histology.—According to Orth ("Specielle patho-

log. Anatomie," Bd. 1, S. 702), our knowledge concerning the pathological histology of the exudative inflammation of the stomach is very limited; in the first place, because uncomplicated, simple acute gastritis rarely ends in death, and, secondly, because post-mortem changes and autodigestion exert a most disturbing and disfiguring effect, particularly in these superficial diseases. In a case which M. Laboulbéne observed—twenty-four hours after death by rupture of an aneurysm—there existed hyperemia of the mucosa, localized ecchymoses, swelling of the mucous alveoli, and augmentation of the mucus.

Delafield and Prudden give essentially these same changes (" Text-book on Pathology "), also Ziegler (" Lehrbuch d. allg. und spec. pathol. Anat.," Jena, 1890), which may be summarized as follows: The surface of the mucosa is covered by a tough, glassy, cloudy, or reddish mucus. The mucosa itself is injected, swollen, and characterized by a hyperemia, which is limited generally to the pyloric region, and rarely extends to the entire mucosa. Red spots, either well circumscribed or diffuse, are very evident, and ecchymoses are scattered throughout the mucous membrane. Larger suggillations occur also, but are rare.

The histological changes are, by most German authors, said to be out of proportion to the degree and intensity of the symptoms (Fleiner, *loc. cit.*, p. 233): That is to say, they expect a greater extension and degree of inflammation to correspond to the severity of the symptoms, and are surprised not to find it. Fischl asserts this particularly of the gastro-enteritis of children (Fleiner, *loc. cit.*, p. 233). However, the exact and very instructive investigations of William D. Booker ("Johns Hopkins Hospital Reports," vol. VI, pp. 159–258, plates xvi to xxi) show quite the contrary. Booker's researches demonstrate destruction of the superficial epithelium in parts and infiltration of the mucosa with polynuclear leukocytes. Many oxyntic cells are without nuclei, and show only loose, granular protoplasm remaining. Epithelial cells and fragments of glands are collected in heaps on the surface, but not to so marked an extent as in the intestine (Booker, *loc. cit.*, p. 251). In a few cases of acute gastritis associated with enteritis he found the entire gastric mucosa destroyed. Bacteriological cultures were made in 23 cases; in 19 the colonies were very numerous, in two moderately numerous, and in two there were no colonies of bac-

teria, but many of oïdium albicans. Tabulated, his results appear as follows:

	Predominant.	Numerous.	Few.	Absent.	Pure Culture.
	Cases.	Cases.	Cases.	Cases.	Cases.
Oïdium albicans, ...	3	3	1	14	0
Bacillus coli communis, . . .	5	8	4	6	0
Bacillus lactis aerogenes, . .	7	2	5	7	2
Proteus vulgaris, ...	3	0	2	18	0
Streptococci, ...	0	4	3	16	0

Booker, like A. Czerny and P. Moser, concludes that the gastro-enteritis of children is a general infectious disease, with auto-intoxication, in which other organs of the body participate, either as a result of an invasion of the body by bacteria, as is often the case with the lungs, or from the effects of poisons absorbed from the gastro-intestinal canal. This infantile digestive affection is undoubtedly a more severe and acute disease than any gastritis that occurs in adults, but its study certainly aids our knowledge of the allied pathological states of adults. There are a number of inflammations, occurring in adults as well as children, that are followed or preceded by digestive disorders, the etiology of which is much cleared up by the work of the authors above mentioned. We refer to the obscure attacks of parotitis, tonsillitis, and pharyngeal abscess sometimes following well-defined gastric ulcer, and to the disorders of the heart and nervous system concomitant or succeeding gastro-intestinal lesions.

These secondary attacks at times may be auto-intoxications; then, again, they show the unmistakable signs of direct infection secondary to digestive trouble, for in the superficial epithelium is to be found the chief protection of the mucosa against the invasion of bacteria. When the epithelium is well preserved, bacteria are not found in the mucosa beneath, whereas they may be seen entering it where the epithelium has been lost or injured (Booker, *loc. cit.*). The first step in the pathological process is probably an injury to the epithelium from abnormal or excessive fermentation in the stomach, or from toxic products of bacteria and the many other conditions that have already been described. (See Plate VI.) To prevent the effects of autodigestion and postmortem digestion on the gastric mucosa, Ewald suggested washing out the stomach immediately after death and filling it with alcohol. This may in

future save a large number of futile investigations. Formerly one depended largely on the studies of gastritis experimentally produced in animals for recognition of the pathological changes. Thus, Ebstein produced gastritis by injecting absolute alcohol into the stomachs of dogs.

Ewald and Ebstein describe a granular, cloudy swelling in the superficial epithelium. While there is no differentiation possible between the parietal or oxyntic and the central, chief, or ferment cells, both varieties are either swollen or contracted, granular, cloudy, and with very indistinct nuclei. Between the different epithelia and in the interglandular connective tissue there are considerable masses of round cells. In these, as well as in the emigrated leukocytes and the cylindrical, superficial cells, numerous karyokinectic figures are very evident, and were claimed by Sachs (*loc. cit.*) to be characteristic of acute gastritis, but this is denied by Ewald.

Beaumont (*loc. cit.*) gives some strikingly correct descriptions of the conditions observed in the stomach of his patient, Alexis St. Martin, when it was acutely inflamed in consequence of overfeeding or of abuse of alcoholic beverages. He states that the mucosa was mostly very hyperemic, even when no digestion was going on, swollen, and covered with a thick layer of tough mucus. After ingestion the food was not digested, but remained in the stomach from four to six hours. The secretion, which was much diminished, was only rarely faintly acid, mostly it was found alkaline or neutral. After a few days the mucus became still thicker, but the hyperemia grew less. This and the following account of Beaumont on the state of the mucosa in gastritis—" its surface was marked with numerous white spots and vesicles like coagulated lymph, between which were very dark red spots "—is considered by Fleiner (p. 232, *loc. cit.*) and Fleischer (p. 802, *loc. cit.*) as unintelligible in the light of our present knowledge. These remarks of the American pioneer of gastric pathology, considered in that very light, impress us as a surprisingly acute and exact description of the mucosa in certain types of gastritis, and inspire the latter-day student with respect for the powers of observation in the man. Fisch (" Fleiner's Lehrbuch," p. 233), after what he considers very detailed and careful investigations, differentiates three forms of gastritis in children: (1) An interstital gastritis, which he supposes to start from the connective tissue; (2) a parenchymatous inflammation having its seat in the

glandular tubes; and (3) a combined parenchymatous interstitial inflammation. The interstitial affection may be interglandular or submucous.

Symptomatology and Course.—Immediately after gross insults to the gastric physiology, characteristic signs and symptoms appear. These are fullness in the epigastrium, which is distended and painful on pressure. Eructation, which at first may bring relief, later on increases so as to be a great annoyance. Thirst, anorexia, and even disgust for food, may accompany this. The tongue is often thickly covered with a tenacious white fur, retaining the impressions of the teeth, and colored by food or drugs; the breath is offensive. The secretion of saliva is augmented, the pulse small and rapid. There may be painful contractions of the esophageal musculature, spasmodic yawning, and herpes labiales. A burning pain in the epigastrium, which may radiate to the hypochondriac regions, arises under the sternum (pyrosis) toward the throat, causing burning all the way, and sometimes raising sour or bitter stomach contents. As water and other liquids diminish the gastric burning, the patients usually show great thirst. The appetite, however, is absent, or there is a perverse craving for piquant, acid, or salty foods, while the habitual diet is detested. Taste is much disturbed. The nervous symptoms are general malaise, indisposition to mental or bodily work, prostration, cerebral pressure, and frontal headache. Palpitation of the heart, giddiness, and a feeling of fear, with profuse sweating, are sometimes present. Nervous and less resistant patients (children) may have delirium. Fleiner declares that general convulsions or loss of consciousness are not rare in his experience.

All these symptoms may arise directly from the stomach or reflexly from the central nervous system, which in these cases suffers intensely at times through the absorption of toxins from the stomach. If the nausea increases to emesis, there will be at first vomiting of food that has been eaten many hours before. This vomited material is mostly badly digested, and imbedded in mucus. After emesis the symptoms may ameliorate, and the nausea cease; very frequently, however, the vomiting continues when no more food is in the stomach. Then saliva, mucus, bile, and even blood, may be forced up under great retching and suffering. Intestinal parasites have in this way been forced into the stomach and vomited up. Skoda first directed attention to cases in which vomiting was

much impeded (at times prevented) by spasm of the sphincters, particularly at the cardia.

If the last meals contained an abundance of carbohydrates or fats, the vomited material will, on testing, show an abundance of lactic, butyric, and fatty acids; it will also contain acetic acid from the alcohol which was either the cause of all the difficulty or which, in nine cases out of ten, has been administered by laymen. But the most characteristic chemical condition is the entire absence of free hydrochloric acid in the vomited matter, which is the cause of the perverse fermentations and decomposition in the gastric contents.

The occurrence of sulphuretted hydrogen in the contents of the stomach and in the urine, which has been reported by Senator, indicates a degree of albuminoid decomposition which is extremely rare.

State of the Urine.—The quantity is, as a rule, diminished; in febrile cases the specific gravity is high, and, when constipation is present, it contains an excess of indican.

Fever.—While about one-half of the cases transpire without rise of temperature, in the other half fever is present, appearing suddenly, and reaching at times 105° F. (40° C.). This form may, in the beginning, occasion some difficulty in the diagnosis, because of its strong resemblance to developing enteric (typhoid) fever. Under these circumstances Widal's method of ascertaining typhoid fever, by the effect of the serum of such patients on the typhoid bacillus, is worth trying for diagnostic differentiation (*Le Bulletin Médical*, 1896, Nos. 59, 61, 64, 78, 83; 1897, No. 4). Also C. Fränkel (*Deutsch. med. Wochenschr.*, 1897, No. 3) and Wyatt Johnson (*N. Y. Med. Jour.*, Oct., 1896, and *Med. News*, Jan., 1897). Some German writers still speak of *gastric fever* as an infectious disease peculiar to itself. (See F. Schmidt, "Dissertation," Berlin, 1885; "Z. Frage d. Existenz d. gastrisch. Fiebers, als einer eigenartigen Krankheit.")

Though it is difficult to furnish proof of a direct infection in these febrile forms at present, it is not at all impossible that such a gastritis may exist. Future bacteriological studies in this disease may throw much light on this point. The fever of acute gastritis is usually preceded by repeated chilly sensations or by a typical shaking chill.

Duration.—If the rules of hygiene are regarded and the patient observes a careful diet, the disturbances will disappear entirely in

from three to four days; there are, of course, much shorter attacks. The stomach remains very sensitive to errors of diet, etc., for a varying time. A number of neglected cases, or those occurring in very weakened individuals, may, by a gradual transition, turn to the subacute or chronic form.

Diagnosis.—In cases that are not accompanied by any fever there should be no difficulty in determining the nature of the disease, especially as the direct cause is, in most instances, apparent. The febrile form may be confounded with beginning enteric fever; during the first three days of the attack it may be impossible to differentiate the two, Widal's method giving a negative result if instituted before the second week of typhoid fever. The existence of fever blisters (herpes labialis), which, according to Leo (*loc. cit.*, p. 66), speaks against typhoid, is, in our experience, an unreliable sign; the results of the blood examinations are contradictory, and in the urine no diagnostic feature is known. The diazo-reaction of Ehrlich, even when performed in the originator's latest method (*Charite Annalen*, 1886, Bd. II), has, in our experience, been of no diagnostic value. In this respect we can confirm the opinions of von Jaksch and Eichhorst ("Klinische Untersuchungsmethoden," p. 777). Most infectious diseases (see above) are in the beginning accompanied by an acute gastritis; in most of them, particularly the exanthemata, a differentiation is not difficult. It is good advice that von Leube ("Specielle Diagnose," I Theil, Leipzig) gives when he says: "In all cases with high fever think of other sources and causes before settling upon gastritis." There are two conditions which, as far as can be judged at present, are reliable factors in the early diagnosis between acute gastritis and enteric fever. (The early diagnosis is the only one we are here discussing; the element of time is very important here, as simple gastritis is only of three days' duration.) The differentiating factors are the manner and rise of the fever and the state of the spleen.

In enteric fever we mostly meet with a gradual rise of temperature and a gradual fall when the fever subsides. In gastritis the temperature rises abruptly, the remissions are slighter, and the fall is more sudden. (See Osler, "Principles and Practice of Medicine," p. 349.)

Therefore frequent, regular, thermometrical studies are not to be omitted. The second diagnostic sign of value is the presence or absence of splenic tumor; its presence points to enteric fever.

Unfortunately, the splenic enlargement is not invariably present in enteric fever at the outset.*

Prognosis.—Speaking generally, the prognosis of simple acute gastritis, except in very old patients and in young children, is favorable.

Treatment.—1. Prophylactic. 2. Dietetic. 3. Medicinal.

Prophylactic treatment will especially be applicable to cases that are known to have enfeebled digestive organs or in which attacks of digestive disease have repeatedly occurred. Attention must be directed to avoidance of injurious influences that may affect the stomach directly from external causes and those that affect it from internal causes.

(*a*) The external causes are, of course, the manifold varieties of trauma that are possible in modern life; not only those that can occur accidentally, but those that occur gradually by pressure upon the abdomen from without, such as is requisite in the execution of certain trades, the manipulation and handling of machines, and even the continuous pressure of tables.

A very important matter in this respect is clothing, particularly that of the female sex. Female clothing of to-day, as far as the maintenance of healthy digestive organs is concerned, is not at all conformable to this object. The much-condemned corset is not even the worst part of the female outfit, for a properly constructed and correctly applied corset need not necessarily effect damage; but for the greater number of ladies wearing them it would be more hygienic to discard it altogether, and preserve form and insure support to the breast and graceful carriage in the style of the ancient Greeks, *i. e.*, by broad, soft bandages applied immediately to the skin, over the underwear, or even externally (Julia Marlowe style). A more harmful thing than the corset is the tying of the skirts and dresses around the waist.

The most judicious female clothing, conformable to the object of relieving the abdominal organs of pressure, would be represented by garments made in one piece, of which the upper part supports

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* Dr. Edward L. Whitney and myself have observed that the Widal test for typhoid fever failed when instituted during the first week. Apparently, a certain time is required before the serum acquires the characteristic effect on the typhoid bacilli. This, of course, —if confirmed by further observations,—would render it useless in the differential diagnosis between typhoid fever and acute gastritis.

the lower from the shoulders. (Kleinwächter, *D. med. Zeit.*, 1894, S. 82; also Meinert, *Volkmann's klin. Vorträge*, 115, 116.)

The abdomen should always be kept warm, not by special bandages, but by garments that are made of wool, fitting quite comfortably to the skin, and closed below. All digestive sufferers should take special care against cooling or sudden chilling of the surface.

(*b*) The internal causes or injurious influence must chiefly be avoided in the food. Exclusive of corrosive and irritant poisons that may be swallowed accidentally, the food articles may contain adulterations in the form of organic or inorganic additions that are incompatible with sound digestion, or the food may be decayed, fermenting, or decomposed. Among the adulterations might be mentioned that of—

Milk, with water, sodium carbonate and bicarbonate, borax, and salicylic acid; or it may contain bacteria (tubercle and typhoid bacilli).

Cheese, adulterated with decomposable gelatins, and may contain lead and tin from the packing, and also mineral impurities.

Sausages may contain flour, fuchsin (for coloring), organic poisons, bacteria, ptomaïnes. (Botulismus: poisoning by sausage.)

Butter may be adulterated by mineral substances, gypsum, lime, coloring matters, lead chromate, cresol, dinitronaphthol, and the caustic alkalies.

Vegetable Food.—Flour has been found adulterated by sand, gypsum, and alum, and also mixed with the fungi of rye or wheat; ergot poisoning by rye flour has been observed in Russia. Some confectioners use dye-stuffs of various kinds, all of which are dangerous. Coffee is sometimes adulterated with copper or lead salts to give it the desired color. Wine, beer, and whisky are subject to numerous adulterations to effect cheaper manufacture, to preserve or color, or to give any desired taste. In beer, picric acid, colchicum, and strychnin have been found as substitutes for hops; impure grape sugar for malt; alkalies to prevent souring, and salicylic acid to preserve it or check fermentation.

Furthermore, the prophylaxis must be directed to the (1) quality, (2) quantity of the food, (3) the proper preparation of the food by chewing and insalivation, and proper conduct after eating.

These subjects are best studied in the section on dietetics (p. 177 to 284).*

Dietetic Treatment.—Acute inflammation of any structure is best treated by rest, and the stomach forms no exception. Hence, total abstinence from food and great reduction of the quantity of fluid imbibed is often curative after an interval of thirty-six hours. So, for the first two days as little food as possible should be allowed. To accomplish this very simple and logical object is, in private practice, a most difficult thing. There is an incorrigible custom among relatives of stuffing the patient with all manner of articles, which it is hard to combat. At the bottom of all this probably lies the popular superstition that a human being can not exist twelve hours without food. A total abstinence from food is borne very well, and leads most rapidly to recovery. For the intolerable thirst, cracked ice should be given, a wineglassful in two hours. If there are signs of collapse, champagne or brandy can be added with safety, even if alcohol was the cause of the trouble.

After the twenty-four hours of total abstinence, the first food to be given is milk, or beef bouillon, with soft rice or an egg beaten up in it. A good stimulating food, when there are signs of prostration, consists of one raw egg beaten up with ½ of a pint of Hocheimer, or a full pint if desired, and sweetened to taste, with a slight flavor of lemon added. The wine may have to be diluted if the gastric mucosa is very sensitive. Of the above, a small wineglassful (two ounces) may be given every two hours (quite warm, if preferred). On the third day a few soda crackers or cakes may be allowed. On the fourth day a gradual return to more reconstructive food is advisable, such as calf's brain, free from all stringy and membranous parts, boiled first in bouillon, then rapidly broiled; sweetbread or thymus gland broiled; breast meat of broiled squab, pigeon, or chicken. Finally, on the sixth day after the attack, finely scraped broiled beef, potato purée, stewed apples, rice,

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*Gilman Thompson, "Dietetics."

Wegele, "Dietetische und medicamentöse Behandlung der Magenund Darmkrankheiten."

Penzoldt, "Handbuch der speciellen Therapie innerer Krankheiten," volume IV.

Biedert u. Langermann, "Diätetik u. Kochbuch," Stuttgart, 1895.

J. Burney Yeo, "Food in Health and Disease," Philadelphia, 1897.

Sir William Roberts, "Digestion," etc.

tapioca, very soft omelette. A plan that is generally successful is to follow out the Penzoldt diet order given on p. 219.

Medicinal Treatment.—Acute gastritis must be treated without drugs wherever it is at all possible. If the dietetic rules of total abstinence from all food for twenty-four hours and cautious return to light diet are carried out, two-thirds of the cases will recover without medicines. Not a few patients, even children, will do this instinctively, and not permit any cramming with food until the stomach has become rested and a natural desire therefor returns. The most important indication of treatment is usually done by the injured organ itself, *i. e.*, evacuation.

If emesis does not occur easily at the outset, both Ewald and Boas recommend the following emetic:

℞. Pulvis ipecacuanhæ, ... 1.5 grs. xxiij
Antimonii et potassi. tartratis, ... 0.05 gr. ⅘. M.
Sig.—Fiat chart No. 1. To be taken at once or in divided doses.

In children, Ewald favors a teaspoonful of the syrup of ipecac. We have so far been able to accomplish all that was necessary without emetics, and are loath to advise their use. Where emesis must be brought about it is more expedient and reliable to use ¼ of a gr. of apomorphin hypodermically. Another drug which gives satisfaction to both patient and physician in these attacks, particularly when there is constipation, is calomel. Sometimes, when persistent nausea follows thorough emesis, it may even act as a gastric sedative. Ewald advises six grs., repeated in an hour. While this dose seems large, it is by no means too large, and will produce a cholagogue and sterilizing effect that may terminate the gastritis then and there. Formerly we used tablet triturates of ½ of a gr. of calomel every hour until purgation; they are more pleasant to administer. The larger dose recommended by Ewald produces more of an antiseptic action, since a portion of it is converted into mercuric chlorid.

Calomel can not be given at the beginning of the gastritis very well; the second day is best suited for its administration. Although we mention these drugs, it is not with a view to routine treatment, but to aid in meeting special indications. When pain in the stomach is attended by chilliness, we advise hot poultices over the entire abdomen, turpentine stupes, or spongiopilin dipped in hot water and 10 to 20 drops of tincture of opium sprinkled over before it is applied to the epigastrium. But when there is gastric pressure that seems to embarrass respiration, associated with explosive eructa-

tion, cold hydropathic applications are more effective than hot ones. When there is fever these applications should be made with ice-water or the ice-bag. Intense pain is met with hypodermic injections of morphin ¼ of a gr., and atropin sulphate $\frac{1}{150}$ of a gr. The following suppositories of Boas may be applied.

℞. Codein phosphoric, ... 0.05 grs. v
Ext. belladonnæ, ... 0.03 grs. ij.
F. c. butyr. cacao suppositor No. X.

SIG.—One every hour until relieved. Where the pain must be relieved, and the hypodermic injection is not permitted and medication per os not retained, they are very useful.

By the mouth, codein is best given in the following manner:

℞. Codein phosph., ... 0.4 gr. vj
Aqua menth. pip., ... 40.0 f℥iss. M.

SIG.—One teaspoonful every three hours.

If symptoms of hyperacidity, keeping up the annoying pyrosis and thirst, are predominant, it may be impossible to avoid alkalies. They are expediently prescribed in the succeeding formula:

℞. Magnesia, calcined,
Sodium bicarbonate, ... āā . . . . 10.0 ℥ijss
Menthol, ... 2.0 grs. xxx.
Mix thoroughly.

SIG.—One-half teaspoonful pro re nata followed by ℥iii water.

It is not rational to give purgatives, because they irritate the inflamed mucosa; calomel is the only drug of this nature that can safely be given, but not before the fermenting stomach contents have been removed by emesis or lavage. To effect purgation before the stomach is emptied exposes the intestine to infection from the septic mass forced through it. Persistent vomiting may call for especial treatment; here, morphin hypodermically, mustard plasters to the epigastrium, and small pieces of ice will be sufficient. A singular case of very exhausting and persistent vomiting was in our practice relieved by bismuth salicylatis gr. x, cocain hydrochlorate, gr. ½; menthol, gr. ii; aquæ camphor, f℥ss. M. Every two hours until relieved. Vomiting of this character is bound to bring on collapse. It is, fortunately, a rare complication, but must be met energetically if it develops. In concluding the medicinal treatment, we desire to refer to a successful therapeutic measure which does not properly belong under this heading, because it is not medicinal but mechanical.

This consists of evacuating the stomach with the tube, and immediately thereupon disinfecting it by washing it out with a solution of the following composition :

℞. Thymol, ... 0.5 gr. viij
Acid. boracic., ... 16. ℥ss
Warm water, ... 500. Oj (one pint). M
Sig.—Lavage fluid.

The water during lavage must be used quite warm and the antiseptic not used until the plain water runs out clear. Catch up the outflowing antiseptic fluid and ascertain that it approximates one pint ; a few ounces retained will not do harm. Vomiting, as a rule, ceases entirely after this. Six hours later wash out the colon by large enemata of ten per cent. solution of boracic acid, no matter whether the patient has diarrhea or constipation. If diarrhea exist, it is absolutely rational to effect the removal of the putrefying colonic contents by large enemata (given in the knee-chest posture), and if constipation exist, the stagnation of feces certainly aggravates the symptoms by increasing flatulence and abdominal pressure. If there is any therapeutic measure in addition to abstinence from food that merits confidence, it is this mechanical cleansing of stomach and colon. Rare cases of high temperature may need special therapeutic measures for the fever. Here, also, drugs must be avoided and the temperature reduced by sponging with cold water or the cold bath.

In case the appetite fails after the attack, or there is protracted weakness with timidity and aversion to food, the following tonic may become useful :

℞. Strychnin. sulphatis, ... 0.021 gr. ⅓
Acidi hydrochlorici dilut., ... 12. f℥iij
Elixir gentianæ q s. ad mis., ... 192. f℥vj. M.
Sig.—One tablespoonful diluted with ℥ij H_2O three-quarters of an hour before meals, through a glass tube.

Forms of acute gastritis associated with copious vomiting of bile are frequently seen in our latitude and termed "bilious attacks." There is no liver disease associated with the attacks, and they are generally brought on by errors in diet and mental strain.

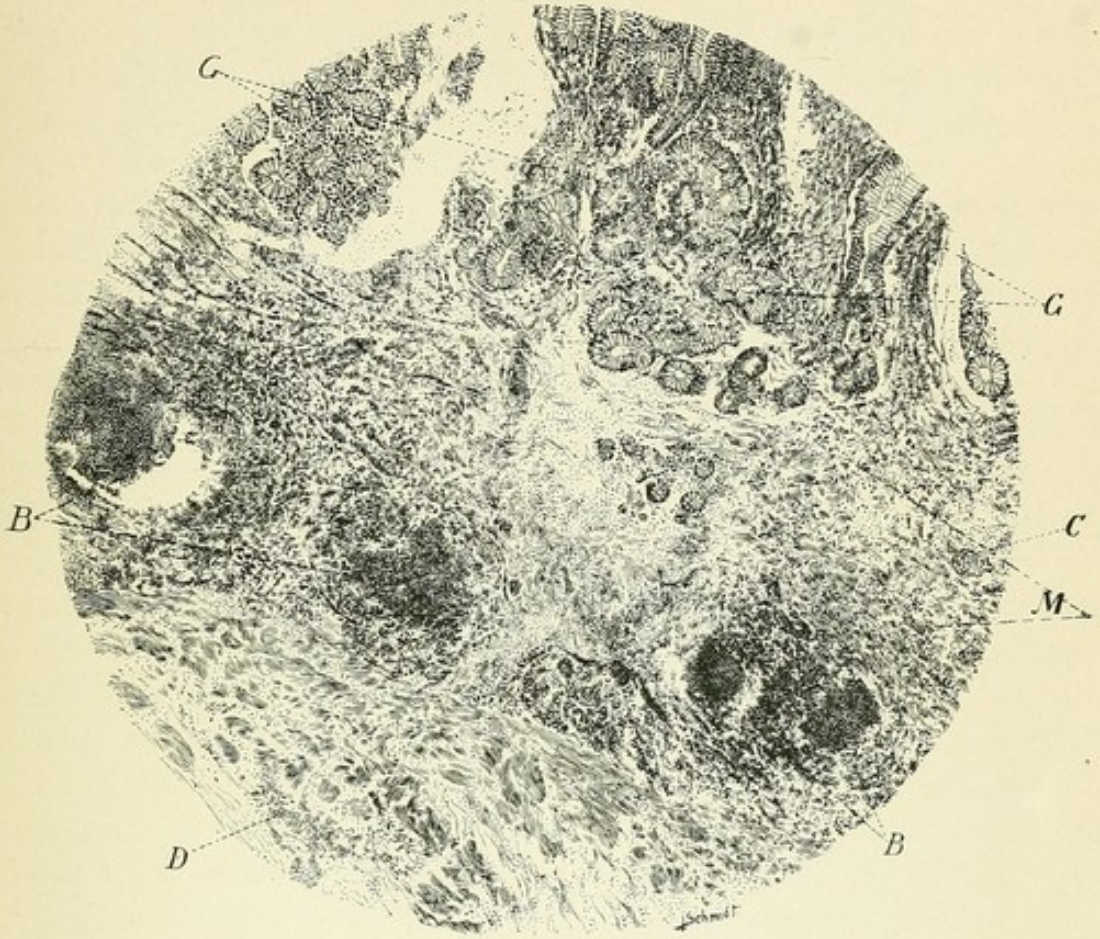
PHLEGMONOUS OR PURULENT GASTRITIS—SUPPURATIVE INFLAMMATION OF THE STOMACH—GASTRIC ABSCESS.

This is a very acute, fatal, and, fortunately, very rare affection of the gastric walls, apparently set up by an invasion of pyogenic

cocci. It is a purulent inflammation invariably originating in the submucous connective tissue, and from here extending to the mucosa. Ziegler ("Lehrbuch d. allgem. u. spec. path. Anat.," 1887, Bd. II, S. 516) describes large numbers of streptococci occurring partly free in the tissues and partly in the protoplasm of the cells. In case the serosa is invaded, the disease, as a rule, produces a general fatal peritonitis by perforation, unless an infection of the peritoneum is prevented by an agglutination with adjacent organs. We have seen but one case of this sort; it was not diagnosed, but our associate, Dr. Delano Ames, discovered it at the autopsy. It had followed ulcus carcinomatosum of the pylorus. The submucosa and muscularis mucosæ were pushed apart by numerous miliary abscesses. (See Hemmeter and Ames, *N. Y. Med. Record*, Sept., 1897, "A Case of Phlegmonous Gastritis," etc.) We submit an excellent drawing showing four small abscesses forcing apart the fibers of the muscularis mucosæ. As far as one is able to judge from the literature on this subject, the disease is inevitably fatal, running most always an acute, rarely a subacute, course. Ewald (*loc. cit.*, p. 303) has seen only one case, and that at the clinic of his teacher, Frerichs. It occurs much oftener in men than in women; of 41 cases, 33 were men and eight women. In a report by Glax ("Die Magenentzündung," *Deutsche med. Zeit.*, 1884, No. 3) it is stated that but 51 cases had been observed up to that time.* Most authors who have had experience with the disease, distinguish, first, an idiopathic primary purulent gastritis, the etiology of which is obscure; and, secondly, a secondary, metastatic, phlegmonous or purulent gastritis, which is an accompaniment or a sequence of other infections, such as pyemia, puerperal fever, anthrax, typhus, or variola. Anatomically, one may distinguish a diffuse and a circumscribed purulent inflammation of the submucosa; the latter is spoken of as stomach abscess.

Etiology.—The direct cause of the rarer idiopathic phlegmonous gastritis is unknown. The predisposing causes may be the same as stated under the etiology of simple gastritis. The direct causes, judging from anatomical specimens, are undoubtedly bacterial invasions of the submucosa, principally by pyogenic cocci that find portals of entry through lesions in the superficial epithe-

*We have collected the entire literature on the subject of phlegmonous gastritis, which is appended.



PHLEGMONOUS GASTRITIS IN THE SEQUENCE OF ULCUS CARCINOMATOSUM. SECTION SHOWING THE LOWER PART OF THE MUCOUS COAT WITH THE ENDS OF SOME OF THE GASTRIC GLANDS, THE MUSCULARIS MUCOSÆ, AND A SMALL PORTION OF THE UPPER PART OF THE SUBMUCOSA.
—(*Original observation from the Author's Clinic.*)

Objective, one-sixth. Eyepiece, one inch. Stained with hematoxylon and orange G. Magnification about 320 diameters.

In all sections the small round-cell infiltration is well marked, the cells chiefly filling up the muscularis mucosæ and invading the lower portion of the mucous coat. In the muscularis mucosæ these cells are aggregated into a number of small, circular, dense masses (*B*), *miliary abscesses*, *between* which they are but little less numerous. The fibers of the muscularis mucosæ have been widely separated by these cells. Few cancer cells are to be seen in this portion of the tissue, but in one place (*C*) they are found plugging completely a small vessel. In the upper part of the submucosa a few of the cancer cells can also be seen (*D*).

lium of the stomach, such as occur in most gastric diseases, especially in so-called exfoliation in carcinomata and old ulcers, or after trauma caused by fish bones, seeds, foreign bodies, etc. Ziegler's (*loc. cit.*) studies have already been mentioned. The secondary, metastatic phlegmonous gastritis, which seems most frequent, is that following puerperal fever, and owes its origin to localization in the stomach of the specific organisms producing the fundamental disease. Whatever they may be, it is self-evident that only an enfeebled organ is liable to such an inflammation, since pyogenic cocci can not resist the action of the free HCl of the gastric juice.

Pathological Anatomy.—The diffuse inflammation rarely invades all parts of the stomach with the same intensity, even if the whole organ is involved. The pyloric portion is generally invaded more than the others; toward the cardia the inflammatory process is less and less marked, whilst the esophagus is rarely attacked. The submucous layer is most extensively altered; on cross-section it is swollen, showing an edematous, purulent, or, at times, a bloody infiltration. From here the inflammation spreads along the interglandular tissue between the glandular tubules, effecting fine or larger perforations in the mucosa, which may assume a sieve-like appearance. Pus wells up through these cribriform perforations as out of a swollen sponge. It may occur that the mucosa is lifted from the submucosa by accumulations of pus. Rokitansky has described a case in which the mucosa was only strikingly anemic, otherwise unaltered. Macleod (*Lancet*, 1887, vol. II, p. 1166) describes a gastric abscess in which mucosa was said to be unaltered.

Toward the deeper portions of the engorged layers, the process spreads along the bundles of muscular fibers in the muscularis, which undergo fatty degeneration, and show infiltration with pus cells and proliferation of nuclei. The serous or peritoneal layer may also be lifted from the subserous or muscular layers, and perforation, as a rule, rapidly follows inflammation of this layer. Circumscribed abscesses, which must be differentiated from the diffuse inflammation, are usually small, varying from the size of a hazelnut to that of a goose egg (Leube, *loc. cit.*). On cutting into the swollen elevated areas of mucous membrane, the abscess is found in the submucosa, but may extend through the muscularis to the serosa.

Symptomatology.—The symptoms are very much like those of a very intense acute simple gastritis; the pain of gastric phlegmon

is not materially increased by change of position or pressure. There is very rarely any vomiting of pus in diffuse purulent gastritis. Gastric abscess may be attended by copious vomiting of pus, after which a tumor that may have been palpable before may become much smaller, or disappear entirely; this phenomenon might be significant for the diagnosis of gastric abscess if it were not for the fact that pus tumors of the neighboring organs sometimes break through into the stomach and cause the same symptoms. The fever reaches 104°–105° F., the patient being aware from the outset that he is very seriously ill. The sensorium is much disturbed by great restlessness, headache, insomnia, delirium. To the symptoms of acute gastritis those of a sudden peritonitis may be added at any time.

Diagnosis.—The important conditions for diagnosis are the pain, vomiting, meteorism, fever, diarrheas, and general phenomena. The pain is localized in the epigastrium and said to have been absent in some cases. The emesis is always present, and consists of bile, mucus, and food debris; in diffuse purulent gastritis, pus has not been noticed in the vomit, which strongly resembles so-called peritoneal vomiting.

The fever is very high, and the temperature curve is said to resemble those of pyemic fevers, with marked remissions and exacerbations. Tympanitis and diarrhea are more frequent than constipation. Other symptoms are: rapid compressible pulse, cold peripheral parts, hurried respiration, thirst, and a much-coated tongue. The course of gastric abscess is not characteristic, and Leube states ("Spec. Diagnose d. inneren Krankheiten," S. 237) that the diagnosis is a matter of chance. The attack may resemble a circumscribed peritonitis or one of the various perigastric inflammations or abscesses; according to Ewald (*loc. cit.*) it may so mimic abscess of the spleen or left lobe of the liver, that a differential diagnosis is absolutely impossible. Deininger (*Deutsches Archiv f. klin. Med.*, Bd. XXIII, S. 268) held that high fever, constant and intense gastralgic pain that is not increased on movement, and increased resistance in the epigastrium, should be sufficiently characteristic to justify a diagnosis. These symptoms, however, occur also in above conditions referred to by Ewald. Chvostek (*Wiener Klinik*, 1881, and *Wiener med. Presse*, 1877, Nos. 22–29), however, seems to have made the diagnosis in one of his cases. The case reported by the author ("A Case of Phlegmonous Gastritis," etc.,

by Hemmeter and Ames, *New York Medical Record* (*loc. cit.*), was not diagnosed. The condition of diffuse suppurative gastric inflammation had followed an ulcus carcinomatosum, which had been recognized and successfully treated as a simple ulcer by Dr. E. L. Whitney fourteen months before death occured. Where there is probability of diffuse or circumscribed phlegmonous gastritis, the exploratory puncture with an aspirating needle or the exploratory incision is, in our opinion, justifiable. In Penzoldt u. Stintzing's new "Specielle Therapie innerer Krankheiten," Volume IV, p. 446, von Heinecke gives suggestions for the operative treatment of phlegmonous gastritis.

Prognosis is almost always unfavorable, especially in the diffuse form. After the circumscribed form and evacuation of the abscess, several clinicians have reported recoveries (Deininger, *loc. cit.*, Glax, *loc. cit.*, Kirchmann, *loc. cit.*, also Buckler, "Idiopathic Phlegmon. Gastritis," *Bayerisch. Aerztliches Intelligenzblatt*, 1880, No. 37), but it is impossible to confirm whether they were really gastric abscesses. Dittrich has found cicatrices in the submucosa pointing to the possibility of healing.

Treatment.—If a diagnosis could be made, it seems to me that these cases, the diffuse as well as the circumscribed forms, had best be treated surgically. Under the existing difficulty, the treatment can be only symptomatic and limited to relieving pain by hypodermic injections of morphin, applications of ice, ice bag to the stomach, crushed ice by the mouth. To counteract collapse, wine enemata and hypodermic injections of strychnin are recommended. Medicines by the mouth are worse than useless.

INFECTIOUS GASTRITIS.

(*Gastritis infectiosa, diphtheritica, crouposa, mycotica, parasitaria.*)

As Penzoldt correctly remarks (*loc. cit.*), every gastritis is to a certain extent infectious; for this reason a number of authors reject the conception of infectious gastritis as a separate and distinct disease. Lebert (*loc. cit.*) and Oser (article on Gastric Diseases in "Eulenburg's Realencyclopädie," second edition, volume XII, p. 410) believe that there is a characteristic infectious gastritis peculiar to itself. Boas ("Spec. Therapie d. Magenkrankh.," p. 6) is of the opinion that there is a form of acute gastro-enteritis, well characterized clinically, which differs from simple gastritis by the

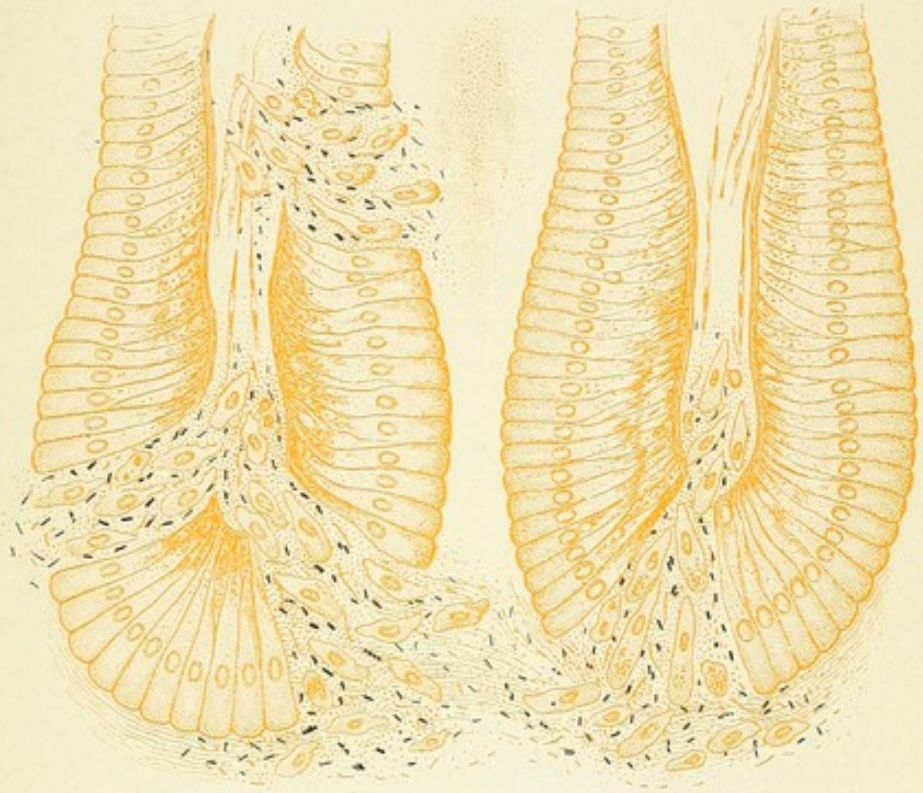
gravity of the symptoms, and particularly the course of the fever, so that it merits separate consideration. Ewald, on the other hand, holds that there is no sufficient specificity of inflammatory processes affecting the stomach for establishing a separate class of infectious gastritis. Fleiner, Penzoldt, and Einhorn give no separate consideration to infectious gastritis. Those that establish a separate category for this affection, class under this head all gastric invasions by infectious germs, so that all forms, as remarked before, are to a certain extent infectious.

The symptoms are said to be very similar to acute simple gastritis, and therefore require no further description. The course is more protracted, as it may last, according to Boas, three to ten days. According to Lebert (*loc. cit.*), some cases may have fever for two to three weeks. In this case the Widal method (*loc. cit.*) should be made use of to decide the nature of the infection. Gaffky (*Deutsch. med. Wochenschr.*, 1892, No. 14) gives an account of severe gastro-enteritis in three persons who drank the unboiled milk of a cow affected with hemorrhagic enteritis. Gaffky believes that the infecting germ was a particularly virulent type of the bacillus coli communis. A number of similar mass epidemics are on record (Husemann, *Deutsch. med. Wochenschr.*, 1889, S. 960) that tend to strengthen the conception of a special infectious gastritis. There seems no necessity as yet for a separate classification of this kind; the subject is still too hypothetical to be ranked as equal in importance with other well-characterized forms of gastritis. The diagnosis, prognosis, and treatment are said by Boas to be the same as for acute simple gastritis.

Diphtheric gastritis is a rare affection, occurring not only as a sequence to laryngeal and pharyngeal diphtheria, but also as an accompaniment to pyemia, septicemia, puerperal fever, scarlatina, variola, endocarditis, ulcerosa, typhus, etc. The disease is, as a rule, not discovered until the autopsy is made, and for that reason has more of a pathological than clinical interest.

Mycotic Gastritis.—When the vitality of the mucosa and the secretion of hydrochloric acid have been reduced, suppressed, or destroyed, certain pathogenic fungi are known to invade the mucosa, producing ulcerations and necrosis.

Most of these mycotic gastritic inflammations can not be recognized during life as such. Among those that have been described



Bacterial Invasion of Gastric Epithelium. From a Case of Diphtheric Gastritis.—(*Hemmeter.*)

are the *anthrax gastritis*, produced by spores or bacilli of anthrax lodging in the mucosa or submucosa and giving rise to inflammation, ulcertion, and necrosis.

Sidney Martin observed a case of anthrax of the anterior wall of the stomach at Guy's Hospital ; the primary infection was in the left cheek, where a malignant pustule developed (*Journal of Pathology and Bacteriology*, vol. 1).

Gastritis caused by the *favus fungus* (Achorion Schönleinii) has been reported by Kundrat (" Ueber Gastro-enteritis Favosa," *Wien. med. Blätter*, 1884, No. 49). The case was that of a drunkard whose gastric mucosa was predisposed by alcoholic chronic gastritis ; he had favus universalis, and in the stomach and intestines the fungi had caused diphtheric inflammations, with fibrous exudations, ulceration, and sloughing ; death was caused, it appears, by a terminal colitis.

The *thrush fungus* (German, Soor ; Latin, Oïdium albicans) has been reported as setting up a mycotic gastritis; in some cases in which the stomach alone appeared infected, throat and esophagus were intact.

The *yeast fungus* (torulæ or saccharomyces cerevisiæ), sarcinæ, the common molds (penecillium glaucum and mucor), and various schizomycetes occur in the gastric contents and set up irritation of the mucosa ; not by direct invasion, it appears, but by the toxic products of the fermentation which they cause.

Sarcinæ, according to Hühne, do not bring about any fermentation.

Miller's interesting investigations concerning the bacterial flora of the mouth have been referred to on page 64. In the first volume of his excellent text-book (" Specielle pathol. Anatomie," Bd. 1, p. 704), Johannes Orth describes an interesting bacterial invasion near an old chronic gastric ulcer which had largely become healed. At several places there were grayish, bran-like incrustations, partly adherent, which anatomically had to be designated as diphtheric. In the scabs or crust, and in the deeper parts of the mucosa, and partly lodged distinctly in lymph vessels, were numerous bacilli that had some morphological resemblance to those of anthrax; this supposition could, however, not be confirmed by cultures. The case was complicated by the fact that a fatal hemorrhage had occurred from a very small arteriole at a place where only a very tiny defect in the mucosa was observable. In the immediate neigh-

borhood of this defect the bacilli were found also, but not in sufficient numbers to attribute to their destructive agency the tearing of the arteriole, which was not aneurysmatic.

Orth then refers to the bacillus gastricus, or polysporous brevis of Klebs ("Ueber Infectiöse Magenaffectionen," *Allgemein. Wien. med. Zeit.*, 1881, Nos. 29 and 30), which this pathologist claims to have found free in the lumen of the glands as well as between the cells of the glands and the tunica propria; there was also an interglandular, small, round-cell infiltration.

Böttcher (*Dorpater med. Zeitschr.*, 1875, p. 184) also defended the view that gastric ulcers are in part due to mycotic and bacterial invasions. Unfortunately, Klebs' and Böttcher's statements have not been confirmed by later investigators.

Animal parasites are also on record for producing gastritis. C. Gerhardt ("Magenkatarrh durch lebende Dipterenlarven," *Jenaer med. Zeitschr.*, Bd. III, S. 522) gave an account of acute gastritis set up by larvæ (maggots) of diptera, a class of insects of which the common fly, the flea, etc., are examples. The eggs of these larvæ were said to have been swallowed with raspberries. Meschede ("Ein Fall von Erkrank. durch im Magen weilende, lebende Maden," *Virchow's Archiv*, Bd. XXXVI, S. 300) reports gastritis caused by maggots eaten with cheese. Senator reported gastritis set up by living maggots of the common fly which occurred in the mouth and stomach (*Berlin. klin. Wochenschr.*, 1890, No. 7); the same observation was made by Hildebrandt (*Berlin. klin. Wochenschr.*, 1890, No. 19). Fermaud observed a case of gastritis and gastralgia caused by an earthworm (*Journal de Med. Practique de Paris*, 1836, tome VII, p. 57). It is known also that ascarides and tapeworms may reach the stomach in rare cases and give rise to severe inflammations, which may subside at once as soon as the offending parasite is vomited.

Toxic Gastritis (*Gastritis Venenata*).—This form of acute gastric inflammation is caused by poisons or corrosive chemical bodies. The poisons that have been taken, either by mistake or with suicidal intentions, are mercuric chlorid or corrosive sublimate, phosphorus, arsenic, chloroform, creosote, potassium chlorate, oxalic acid, nitrobenzol, carbolic acid, the concentrated inorganic acids, sulphuric, hydrochloric, and nitric acids; the caustic alkaline hydroxids in strong solution, and, furthermore, alcohol in all its forms, and some substances used as medicines (see etiology of

acute gastritis), particularly croton oil, antimonium and potassium tartrate (tartar emetic); also ammonia.

The *pathology* will necessarily vary considerably, as it is not only dependent upon the kind, but upon the quantity and concentration of the poison, and also upon the circumstance whether the poison is taken on a full or an empty stomach, as food and drink dilute the drugs. There may be only a slight superficial inflammation, or a very penetrating corrosive effect involving the entire gastric wall and even leading to perforation. Different drugs produce different effects upon the mucosa. Phosphorus, arsenic, antimony, and alcohol produce, in excessively large toxic doses, a milky, yellowish-white, or opaque appearance. The epithelia of the alveoli of the tubular glands are partly in a state of mucoid degeneration, partly finely granulated, cloudy, and showing fatty degeneration; the same is the case with the secreting cells. The tissue between the cells is crowded with a small round-cell infiltration. In this condition auto-digestion by the gastric juice may cause peptic ulcers, *i. e.*, when the poisons are not taken sufficiently strong to effect ulceration or to destroy secretion.

Dilute acids and alkalies induce the pathological picture of a simple acute gastritis; while, in concentrated form, the same agents cause a deeply penetrating necrosis, formation of crusts and intense reactive inflammation with serous infiltration, suppuration, and blood extravasation. The scabs or crusts show different colors with different corrosives. Under the effect of sulphuric acid they are black; of nitric acid, yellow; of alkalies, brown; of copper salts, green or blue; of silver salts, black. Dislodgment of these crusts leads to fatal bleeding, tearing of the serosa, or perforation, with peritonitis. Oxalic acid is said to produce a jelly-like, semitransparent swelling. Ammonia causes a pustular inflammation.

Symptoms.—After taking the poison there is generally an indescribably severe pain, intolerable burning, and vomiting which increases the pain and at times causes fainting. The vomit, as a rule, contains blood or bloody mucus; the thirst is great. There is most frequently diarrhea containing blood. Severe general symptoms follow; small, very fast pulse; jactitation; delirium. In case much of the poison has reached the general circulation, hematogenous icterus, petechiæ, albuminuria, and hematuria may follow. Death follows in a few hours or a few days from collapse; or later by perforation peritonitis. Even if the patients are tided over the first period

of acute gastric symptoms, they may die later from hemorrhage when the scabs and crusts are sequestrated, or by the consequences, *i. e.*, stenosis of the esophagus, cardia, pylorus, or atrophy of the mucosa.

The *diagnosis*, after learning the history of the case, will not be difficult. One should not fail to make a thorough examination of the mouth and throat, where the corrosive effect, if any, will be evident. An analysis of the vomit will probably inform of the nature of the poison.

The *prognosis* of severe toxic gastritis is necessarily fatal; if not by the direct poisoning or first destructive effect of the drug, certainly by the severe secondary effects.

The *treatment* will vary with the nature of the poison. In recent poisoning with strong acids, magnesia usta (calcined) should be given as soon as possible. If no drug-store is near, chalk, or even powdered lime, which can be scraped from the wall, should be given. Whenever possible, the stomach-tube should be used at once for all poisoning of recent date.

Boas, Fleischer, and Pick advise that the tube should not be used in severe acid or caustic alkali poisoning, because of the danger of perforating the stomach. As most such cases will probably die of perforation anyway, we certainly should use the tube and let the patient take his chances. About 250 c.c. of water, with sodium bicarbonate in case acids were taken, or vinegar in case alkalies were taken, will doubtlessly dilute and combine with the destructive agent present. Lemon juice will also answer for the alkaline caustics. In all other poisonings the stomach-tube, or, if convenient, the pump, should be used as soon as possible, and the stomach washed out thoroughly. For other poisons the approved antidotes should be given (freshly prepared hydrated oxid of iron for arsenic, etc.), that will be found in various text-books on toxicology and therapeutics (H. A. Hare's system; H. C. Wood; Lauder Brunton; Binz, Schmiedeberg; Penzoldt and Stintzing's system, vol. VI). After carbolic acid ingestion wash out the stomach, and then pour in 250 c.c. olive oil. In all corrosive poisoning cases the pouring in of olive oil or molten vaselin, after neutralization and washing out, will, if possible, diminish the corrosive effect. Where not too much acid or alkali has been taken, the subnitrate of bismuth or subgallate of bismuth, one dram three times a day, if possible, swallowed with oil, will favor rapid cicatrization and inhibit

bacterial infection of the necrosed, charred areas. A suspension of bismuth subnitrate, three drams to one pint mucilage and water, has proved advantageous in a case of carbolic acid poisoning in our practice; it was used in form of lavage.

If the pain is severe, morphin must be promptly given, hypodermically, in ¼ to ½ gr. doses, repeated until relief comes. It is our duty to give relief to the pain at any risk, even if chloroform anesthesia is required; for after the suffering ceases our efforts to save the patient can be more easily executed. Nutrition must be carried on by rectal enemata only. By the mouth, ice is about all that is permissible; it will tend to diminish the pain, fever, and inflammation. We make such an explicit statement of treatment because we had experience with two cases where the autopsy showed that recovery might have been possible (as not much sulphuric acid had reached the stomach) if the treatment had been more heroic, *i. e.*, if the tube had been used for timely removal of the poison.

CHAPTER II.

CHRONIC GASTRITIS.

Little over a decad ago it was customary to designate all stomach diseases that were not acute, and that could not be diagnosed as dilatation, ulcer, or carcinoma, as "chronic gastric catarrh." We agree with Ewald and Penzoldt, in the objections to the word "catarrh," and have given the reasons under the chapter on Simple Acute Gastritis. Even at the present day there is no absolute uniformity in the conception and limitations of the term "chronic gastritis."

With the aid of improved methods of diagnosis, particularly such methods as permit of an exact study of the various gastric functions, the so-called gastric neuroses have been recognized as separate and distinct diseases; formerly they were believed to be symptoms of chronic gastritis. This chronic inflammation of the mucosa affects all the important functions, although one or the other of these is generally most involved. There are observed many variations in kind and intensity of disturbed function, from a trivial reduction of secretion of gastric juice or interference with motility, to complete suppression of glandular activity and pronounced insufficiency of peristalsis. There are two pathological processes inseparable from every chronic gastritis; these are: degeneration and desquamation of the glandular cells, and infiltration of connective tissue. Bearing in mind these conditions, we may distinguish two main types of chronic gastritis: first, the hypertrophic; and, secondly, the atrophic. The hypertrophic form consists of proliferation of the connective tissue, leading to change of form and folding or warty elevations of the mucosa ("état mammeloné," or polyposis). The result of this process is: first, either complete destruction, or, secondly, cystic degeneration of the glands. A grayish-brown, or in places a dark brown, color is peculiar to this swollen and proliferated mucosa, which is covered with an adherent, gray coating of mucus.

The atrophic form consists of contraction of the connective

tissue, loss of epithelium, and more or less complete destruction of the glands; in rare instances, superficial ulcerations. The mucous membrane is much thinned out, very smooth, and of a grayish-white or pale slate-gray color. If this process attacks the muscularis and submucosa, it may cause atrophy of the muscle fibrils, with or without thickening of the entire gastric wall due to new formation of connective tissue. Then, again, we may meet with a genuine hypertrophy of the muscularis, particularly at the pyloric portion, or in the pylorus itself. The lumen of the stomach in these forms may show a normal capacity; or it may be much diminished in size by connective-tissue thickening of the gastric walls and subsequent contraction. This process is known as "gastric cirrhosis" (Brinton). By French writers it is termed "hypertrophic sclerosis of the stomach" (Hanot and Gombauldt, *Archiv. de Physiol.*, IX, p. 412; also Dubey, *Gazette Hebdomin*, 1883, p. 198), and it may reduce the normal capacity to 160 c.c. (Leube, Penzoldt). Or, again, the capacity is much increased by a dilatation in consequence of chronic gastritis and hypertrophic pyloric stenosis. So the anatomical picture may present: (*a*) atrophy of the mucosa with wasting of the peptic glands and of the muscularis; thinning of the entire gastric wall, and, very frequently, dilatation; or, on the other hand: (*b*) inflammatory hyperplasia of the layers of the stomach, with excessive connective-tissue proliferation (cirrhosis ventriculi); hypertrophic pyloric stenosis; atrophy of the glandular layer and sometimes of the muscularis. This form may lead to marked reduction of the lumen; or, more frequently, if a stenosis exist, to a dilatation. Both forms produce grave disturbances of motility, secretion, and absorption.

The cause of the elevated, warty, or polypoid projections of the glandular layer is to be sought in the fact that, in certain forms of the disease, the mucous layer grows much more rapidly than the submucous layer, bringing about a rough, wrinkled, mammillated surface that has been described as "gastritis polyposa," and by some French writers is termed "état mammeloné" (see Orth, "Specielle pathol. Anat.," Bd. I, p. 709). A number of Germans describe a variety of special forms of chronic gastritis under the names of "saurer Katarrh" (sour, or acid, gastritis), "Schleimkatarrh" (slimy, or mucous, gastritis), also termed "gastritis atrophicans," and a simple chronic gastritis, or "einfacher Katarrh." All of these terms are unfortunately chosen and unscientific because they are

artificial. The so-called "saurer Katarrh" is not a gastritis at all (Ewald), but a neurosis of secretion; a hyperacidity, the result of secondary irritation of the mucosa.

Etiology.—Chronic gastritis is a wide-spread disease, occurring in all stations of life, but most frequently among the poorer classes, where the quality of the food may be so inferior as to keep the stomach in a state of constant irritation. All the numerous injurious influences which arise from a defective and inappropriate diet have been referred to under the head of the pathogenesis of acute gastritis. It may evolve from the acute or subacute form, where the mucosa has been damaged by the altered circulation and its resistance to disease lessened. It may arise from all processes that lead to venous congestion of the stomach, *i. e.*, affections of the organs of the portal system, especially of the liver and spleen; it may also be caused by diseases of the heart. There are certain conditions which may bring about a chronic gastritis by effecting alterations in the composition and structure of the blood; among these are: anemia, chlorosis, scrofula, secondary anemias following typhus and typhoid fevers, the exanthemata, pregnancy, tuberculosis, diabetes, gout, and nephritis. Irritating substances brought continuously in contact with the mucosa, either from without or within (*i. e.*, from the blood), are believed to cause the disease. Ewald states that it may result from direct local irritation of alterations in the mucosa itself, such as cicatrices and neoplasms. Our experience is that in the vicinity of such structural changes pre-existing in the mucosa there is indeed a gastritis observed, but it partakes mostly of an acute or subacute type. Among the most pronounced causes of the frequency of chronic gastritis are: defective *chewing* and insalivation, hurried eating and swallowing of large pieces of food, putrefaction of the mouth from carious teeth, or the manifold forms of stomatitis and gingivitis, and, in this country, excessively hasty eating, with the abuse of ice-water at meals and of tobacco and alcoholic liquors between meals. The majority of American people residing in cities live under commercial and social customs pernicious to the digestive organs. Foremost among these conditions are the high mental pressure evoked by the demands of business, the constant worry and nervous tension caused by force of competition, the anxiety to get rich rapidly by straining all mental and physical powers; all these things bring about a hasty, nervous manner of taking food. Chewing is a process which most

business men execute in a perfunctory manner only, allowing no time for insalivation. If it were possible they would gulp the food down dry; as it will not go down that way, it is washed down with ice-water. Tobacco juice is responsible for much of this disease; also condiments used habitually (pepper, ginger, mustard, horse-radish), and the habitual use of drugs (arsenic, silver salts, iodids).

Chronic gastritis is most frequent among habitual consumers of alcoholic liquors. From what was said, under acute gastritis, of the experimental production by Ebstein of this disease with alcohol, the frequency of the chronic form among the devotees to Bacchus and Gambrinus is very intelligible. As Ewald correctly remarks, the disease may be classified among those in which the patient's indiscretions play a very important rôle. But, as most persons treat their stomachs badly, and neither eat with proper mastication, nor are able to resist culinary temptations, gastritis is one of the "best nourished" and most prevalent diseases in the world. "Indigestion is the remorse of a guilty stomach," says Ewald; and F. Albin Hoffmann ("Vorlesungen über allgemeine Therapie," Leipzig, 1885) expresses a sentiment that deserves to be an apothegm: "*Jeder Mensch hat den Magen den er zu haben verdient*" ("Every one has the stomach which he deserves"). It is not intended here to do injustice to a large number of sufferers from weak stomachs, who take the greatest possible care to avoid dyspepsia, and, nevertheless, are liable to acute or chronic gastritis. The etiology explains why the male sex is much more frequently affected than the female.

The Pathological Anatomy.—The changes are, as in the acute form, most pronounced in the pyloric region, and from here extend to the fundus. The alterations of structure occurring in the course of chronic gastritis present varying pictures, according to the duration of the disease. In the later stages the variations are considerable, since, at this period, the connective-tissue changes may at one time incline to inflammatory hyperplasia; at another, may show an atrophic character; again, either the mucosa or submucosa only, or, in other instances, the deeper layers, may be involved, with alternating intensity and extent. The inflammatory process is not at all limited to mucoid degeneration and desquamation of the surface epithelium, but pre-eminently affects the glandular elements and interstitial tissue, whence it attacks the deeper layers of the gastric wall. In early stages there is a general, diffuse redness, due

to hyperemia; in later stages this color exhibits a peculiar pigmentation, which first assumes a bluish or brownish shade, and finally gets to be of a dirty red-brown, or slate-gray, or both. This pigmentation is generally limited to the pyloric region, but, in spots, it may be spread over other sections of the inner surface of the stomach. The color is caused by blood pigments which have become stored up in the cells and interstitial tissue; also by blood-corpuscles that have left the vascular channels and undergone pigment metamorphosis during the long-standing chronic hyperplasia. This pigmentation must not be confounded with post-mortem discoloration.

Inflammatory Hyperplasia.—In this form the gastric mucosa may either preserve the velvety appearance peculiar to the normal inner surface of the contracted stomach, or it may be covered with irregular warty projections, and exhibit immense development of the pyloric "plicæ villosæ." This is due generally to inflammatory infiltration of the interglandular and subglandular connective tissue, but particularly to the same process occurring in the connective-tissue ridges ("Leisten") existing between the vestibular entrances to the gland-ducts ("Vorräume" of the Germans), or peptic-duct alveoli, as we prefer to call them. If these hypertrophic-hyperplastic processes are confined to circumscribed areas, they may assume exaggerated degrees, forming polypoid proliferations, which, as a rule, are attached by broad bases; in consequence, however, of connective-tissue contraction, they may also occur pedunculated. In this way papillomatous excrescences may be developed which project into the lumen of the stomach (Orth, "Gastritis Polyposa," *loc. cit.*, p. 710). When the submucosa is attacked with inflammatory infiltration and new formation of connective tissue, the loose tissue is first transformed into one much richer in cells, subsequently, into a tougher, more inelastic layer, resulting naturally in a much reduced movability of the mucosa upon its substratum. When this chronic process results in cicatricial contraction in the hyperplastic submucous tissue, it may lead either to partial, localized change of form, or to a more or less general, uniform contraction ("Schrumpfung," "cirrhosis ventriculi"; "linitis plastica," Brinton). In the pyloric portion this process may lead to stenosis. Frequently, the muscularis also is hypertrophied, as a consequence of the chronic inflammation transmitted through the submucosa. This muscular hypertrophy is most pronounced at the pylorus. The

localization at the pylorus of the maximal intensity of the inflammatory process in the mucosa, submucosa, and the muscularis, makes the origin of a pyloric stenosis in consequence of chronic gastritis intelligible. This kind of stenosis is usually spoken of as benign, in contradistinction to the malignant stenosis of carcinoma.

Much diversity of opinion exists concerning the origin of the *état mammeloné* ("mammelon" means the nipple of the mammary gland). Frerichs held that it was due to accumulations of fat in the mucosa and inflammatory hyperplasia of its contained lymph-follicles. Rindfleisch maintained that a greater growth of the mucosa than of the submucosa was the cause. Ziegler explained the mucosa polyps by proliferation of the submucosa. Ebstein assumed an inflammatory hyperplasia of the tissue between the glands. Jones assigned as a cause an excessive contraction of single bundles of the muscularis mucosa. Undoubtedly, this gastritis polyposa, with its mammelonated appearance, may be formed by a great diversity of processes.

Inflammatory Atrophy.—The progressive plastic character of the inflammation just depicted may lead to retrograde metamorphosis before it has progressed very far; in some cases it may not develop at all, but the disposition to break down and atrophy may start early in the disease. These atrophic changes are most marked in the glandular elements, and may be limited to these. Sometimes the inflammations of the mucosa and gland-cells have, from the outset, a degenerative tendency, and no hypertrophy or hyperplasia enters into the anatomical picture. The surface columnar epithelium and the cylindrical epithelium of the vestibular alveoli fall prey to a mucoid degeneration and desquamation. The epithelial cells of the peptic glands undergo fatty degeneration. During this atrophy, the mucosa changes to a thin, smooth, pigmented, or slate-gray membrane. This atrophy may be limited to the mucosa, while, at the same time, hypertrophic changes go on unhindered in the submucosa and muscularis; again, the atrophy may extend to the latter layers, and bring about a wasting of all gastric strata. This last condition was formerly designated "tabes of the stomach" (the "phthisis ventriculi" of Rokitansky). Under these irreparable atrophic states, anomalies in the gastric volume may develop, but dilatation is here more frequent than contraction.

Atrophy of the stomach may occur without preceding chronic gastritis. It then appears as a simple degenerative process, and

follows severe anemic, cachectic states, and also grave infectious diseases and poisonings.

When confronted with cases of gastric atrophy, with absence of hydrochloric acid, the ferments, and enzymes, and co-existent anemia, it is sometimes very difficult to decide as to the primary causative disease. In these cases it is well to bear in mind that anemias, even those of a grave pernicious character, may be a consequence of, or rather secondary to, atrophy of the gastric mucosa which has extended to the intestinal mucosa. Our countryman, Austin Flint, was the first to call attention to the relation between anemia and atrophy of the gastric glands. In 1860 (Austin Flint, *American Medical Times*, 1860) he expressed the opinion that some cases of obscure and profound anemia are dependent upon degeneration and atrophy of the glands of the stomach. (Further contributions of Flint to this subject are to be found in the *New York Medical Journal*, March, 1871, and in his "Principles and Practice of Medicine," p. 477, Philadelphia, 1881.) Since Flint's publications, cases have been reported by Fenwick (*The Lancet*, 1877, July 7th, *et seq.*); Quinke (*Volkmann's Samml. klin. Vorträge*, No. 100, case *b*); Brabazon (*The British Medical Journal*, 1878, July 27th); Nothnagel (*Deutsch. Archiv f. klin. Med.*, Bd. XXIV, p. 353); Bartels (*Berlin. klin. Wochenschr.*, 1888, No. 3); Scheperlen (*Nordisch. Medic. Arkiv*, 1879, Bd. XI, No. 3); Osler ("Atrophy of the Stomach, with the Clinical Features of Progressive Pernicious Anemia," *American Journal Med. Sciences*, 1886, No. 4). Rosenheim reported two similar cases which appeared to be pernicious anemia (*Berlin. klin. Wochenschr.*, 1888, Nos. 51, 52).

Inasmuch as these cases of atrophy of the gastric mucosa are accompanied by marked changes in the blood, signs of breakdown in the red blood-corpuscles, increase in the white corpuscles and formation of macrocytes and microcytes, the question may arise whether pernicious anemia is really an independent disease or the result of gastric atrophy. Atrophy of the mucosa,—not secondary to well-known stomach or general diseases, but occurring as a primary disease,—has been claimed to exist by Fenwick (*loc. cit.*). Professor William H. Welch (Pepper's "Amer. System of Medicine," vol. XI, p. 616), however, was, at the time of that publication, of opinion that the existence of atrophy of the stomach as a primary, independent disease had not been established, the histol-

ogical examination of many of the cases reported as such having been defective. Professor Welch has since modified his views on this subject. From the statements of some writers the impression might be gained that the hypertrophic-hyperplastic form of chronic gastritis was, from its fully developed stage, changed into the atrophic form. This would mean the total disappearance of the papillary, polypoid proliferations of the "état mammeloné," because the mucosa of the atrophic form is very smooth. According to Orth (*loc. cit.*, p. 710), this is very improbable, for he is of the opinion that the atrophic form is developed uniformly by transformation of cellular interstitial tissue into contracting cicatricial tissue, bringing about thinning of the mucosa and degeneration of the glandular elements without the intervening features of hyperplasia above referred to.

Ulcerative processes are said to occur (Ziegler, *loc. cit.*) when, in the course of the disease, intense (hemorrhagic) inflammation produces necrosis of the epithelium and submucosa, and its subsequent "sequestration." In this way the so-called catarrhal gastric ulcers and hemorrhagic erosions are formed, which may be associated with hemorrhage. Cruveilhier ("Anatomie Pathologique du Corps Humaine") records a follicular gastritis, in which ulcers were said to originate in the follicular glandular apparatus.

The catarrhal gastric ulcers of chronic gastritis are mostly small, round, or irregularly indentate. They are supposed to heal and form flat pigmented cicatrices. Förster asserts that they may lead to perforation. Orth (*loc. cit.*), whose statements merit confidence because of his scientific conservatism, is of the opinion that ulcerative processes in the course of chronic gastritis are very rare. The minute anatomy of the process is that of a parenchymatous and interstitial inflammation. The glandular cells are partly destroyed, partly granular, and partly shriveled up; differentiation between the principal (Hauptzellen) and the parietal cells (Belegzellen) is impossible. In many places, especially in the pyloric region, the ducts have lost their regular order of lying alongside of one another, and show atypical manifold ramification like glove fingers. Isolated glands become separated at the fundus and appear as cysts at the border of the submucosa; these are either empty, with a smooth lining membrane, or are filled with the remains of glistening hyaline cuboidal epithelium. There is an abundant small-celled infiltration which is especially marked near

the surface of the mucous membrane; the cells lie between the glands, and, in places, push their ducts far apart. In the hyperplastic form we see processes of connective tissue which proceed upward between the glands from the submucosa, like the branches of a tree. The free surface of the glandular layer is covered with a

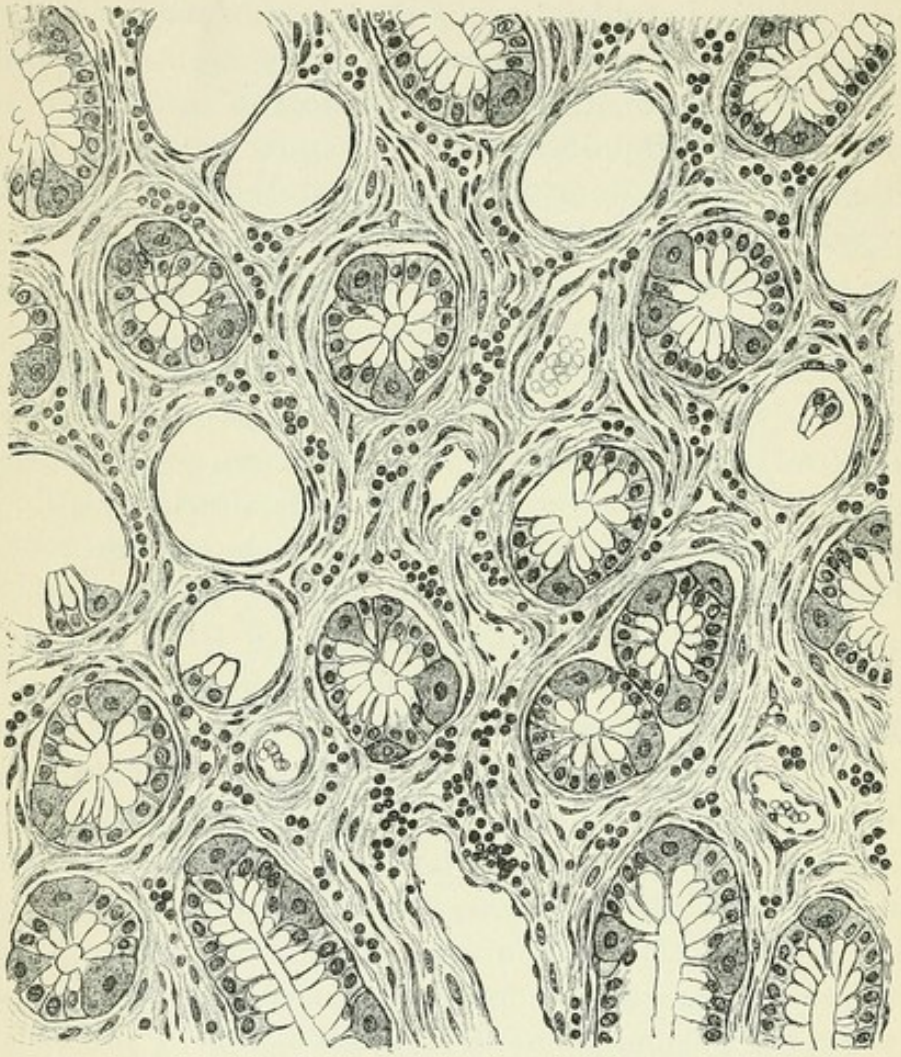


FIG. 34.—ATROPHY AND VACUOLIZATION OF GLANDULAR ELEMENTS—MUCOID DEGENERATION OF PEPTIC CELLS—INCREASE OF INTERSTITIAL CONNECTIVE TISSUE—SMALL ROUND-CELLED INFILTRATION.

In some places the glandular elements have disappeared, leaving empty, circular spaces. From a case of chronic (alcoholic) gastritis (found in the wash water).

film of mucus inclosing many leukocytes and nuclei (Ewald). The superficial layer of the epithelium of the mucosa is loosened, and can be separated in adherent shreds, which may sometimes be found in the wash water after lavage of the stomach. In sections, one can readily see the mouths of the glandular ducts and the surrounding epithelium. The epithelial cells of the vestibular

alveoli (Vorraum) are, for the greater part, filled with a pale mucous mass, which projects sharply against the lumen without any inclosing membrane, as described by Kupffer in the normal stomach. Ewald has been able to study this and the following conditions, in specimens which were obtained immediately after death, or from living persons after resection of the pylorus. In the conditions (to be described presently) of gastritis mucosa or mucipara, this mucoid degeneration may be observed to extend to the base of the glands, so that in place of the ordinary chief and oxyntic cells, we find only cells in the most varied stages of mucoid degeneration (see Fig. 34). This condition is especially marked in the pyloric region. Some isolated cells may be found which are still intact, the mucus filling only a small part of them, while the rest of the cell is occupied by granular protoplasm and a large nucleus. In others, the mucus occupies the greater part of the cells, and crowds the protoplasm and the flattened nucleus against its base. In still others, the cell membrane has ruptured, and the mucus has escaped into the lumen of the duct of the gland, where it has been precipitated in streaks by the alcohol. This gives rise to very delicate figures which resemble a row of horseshoes with their openings toward the lumen of the gland. That this is really mucus, and not the isolated formation of vacuoles as described by Stöhr and Sachs, is easily proved by the reaction with acetic acid, and the grayish color with hematoxylin. Ewald emphasizes the fact that these features are found only where the mucous membrane has been placed in alcohol while still warm; in old tissues he has never met them. Thus there is a mucoid degeneration of the protoplasm of the cells, which extends deep down into the fundus of the gland.

Symptomatology.—As a general rule, the onset of gastritis can not be determined with certainty, because, first, it develops very gradually and insidiously, either as a continuation of acute gastritis and of other diseases, or as an independent disease; and, secondly, the initial symptoms, not being very pronounced, are generally ignored. Only the sudden aggravation caused by dietetic errors, and other injurious influences, lead to the conclusion that a serious disease is present. The clinical picture varies considerably, although the signs of a disturbed digestion, as indicated by dyspepsia, absence of appetite, eructation, nausea, vomiting, pressure and fullness in the gastric region, repeat themselves in various

cases; first one symptom and then another will manifest its presence or be entirely absent. Perhaps the most constant of the early symptoms is absence of appetite.

Absence of Appetite (*Anorexia*).—Even in less serious attacks this symptom, as a rule, exists, and may eventuate in disgust for the customary diet. After prolonged fasting the patient feels that the stomach is empty, but there is present no desire to eat and no hunger. There is, however, a strong craving for "piquante," salty, or acid food. It seems as if an instinctive knowledge existed that the production of gastric juice is depressed and that the mucosa requires a stronger incentive to secretion. Sometimes a slight appetite is, at rare intervals, developed, which, however, a very few mouthfuls of food suffice to satisfy completely. Incidentally, the desire for food will increase if the patients force themselves to eat; now and then bulimia—an intense hunger—may develop at extraordinary times, *e. g.*, during the night; but this is more frequent in the neurosis of hypersecretion, which was formerly classed as a gastritis. Thirst and salivation are increased.

Taste.—We have rarely observed a case of chronic gastritis in which there was not present one or more of the following complications: Pharyngitis, posterior nasal catarrh, laryngitis, or a form of stomatitis; the last occurring most frequently. This condition of the mouth perverts taste, rendering it pasty, sometimes distinctly unpleasant, acid, bitter, or metallic. The breath is frequently offensive, caused by caries of the teeth and by decomposition on and in the lingual epithelium, and eliciting the remark that "food has no taste." Almost all foods then taste alike. Occasionally, the breath will first become offensive at the height of indigestion, one or two hours after meals, and especially so after ill-smelling eructations; this should lead to the suspicion that there is gastric decomposition.

Nausea is an early symptom, generally preceding emesis; it may exist by itself for many hours without emesis, and may even occur on an empty stomach. When it occurs after eating, it subsides upon vomiting the food. The ingestion of food may diminish or increase the nausea, which is not always a direct effect of ingesta or fermenting contents on the stomach itself. We have observed it when no food has been taken by the stomach for ten days, where daily lavage has been carried out and nutrition conducted by rectal feeding. This form of nausea may be an effect of intestinal auto-

intoxication of a severe type, as these chronic cases of gastritis are occasionally subject to "ptomaïn storms."

Eructation is in all cases present at some time. The gases brought up are air, carbon dioxid; in some rare instances, inflammable gases, such as hydrogen and marsh-gas, CH_4, have been eructated (Ewald, Rupstein). The gases may be tasteless and odorless, or may have an offensive after-taste of rancid or bitter character, particularly when small portions of ingesta arise with the belching. Sometimes the contents of the stomach are very rich in organic acids, this being most likely when the motility and the secretion of normal HCl are suppressed. A very high total acidity, showing no free nor combined HCl at all, will then be composed entirely of lactic, butyric, and acetic acids. This is a very rare occurrence in chronic gastritis in our experience, and, as a rule, associated with some disturbance of motility. When this acid mass is forced up into the esophagus during the eructations, a very annoying heartburn, or pyrosis, ensues, which seems localized at various parts of the gullet or cardia, and may last for hours.

Vomiting, though not so frequent as in acute gastritis, nevertheless occurs quite often. In the chronic gastritis of drinkers it is often a regular event each morning, and is then known as the "morning vomit," or *vomitus matutinus* (water-brash), which Frerichs attributed to the swallowing during the night of saliva and the secretions from the pharyngeal catarrh. The morning vomit is usually alkaline, inverts starch to sugar, and gives the red rhodankalium KCNS reaction with chlorid of iron. A tough, glassy, morning vomit occurs in some patients who are not drinkers; after severe retching, the mucus may be found tinged with blood. We have under observation at present a female patient with chronic gastritis, who vomits this glassy mucus almost the moment she raises her head from the pillow in the morning. Vomiting which occurs after meals brings out food in a more or less partially digested state, according to the duration of its retention in the stomach and the condition of the secretions. The eructated ingesta are imbedded in tough mucus, and may be in a state of fermentation. Bile may form part of the admixture. If the gastritis is due to secondary passive hyperemia ("Stauungskatarrh") accompanying hepatic cirrhosis, the vomit may contain blood from the rupture of minute varicosities on the mucosa. The

ejected food contains organic acids (particularly after carbohydrates have been ingested), but no free acids. We have been struck with the frequency of the occurrence of excessive amounts of acetic acid when the gastritis has been set up by long-standing abuse of alcohol. Yeast cells, sarcinæ, and a large variety of bacteria may be present. With incipient and not very grave cases the ferments, pepsin and rennin, are yet to be detected; but, in later stages, they are evident only after adding HCl slightly in excess of the deficit; this really shows that the pro-enzymes, not the perfect ferments, are present. Finally, pepsinogen and rennet zymogen are absent; and, in very advanced forms, even the mucus will cease to be secreted. This last symptom is an indication of the complete atrophy of the mucosa.

The tongue is very frequently coated with a grayish-white deposit, most marked on the back and root of the organ. The impressions of the teeth are retained by it. At the edges and apex the tongue presents a deeper red color, with swollen papillæ. The coating may disappear toward evening, to reappear in the morning. Henoch (*Klinik der Unterleibskrankheiten*, Berlin, 1863, p. 382) holds that the appearance of the tongue is really not always a mirror of the stomach, but that its condition is to be regarded simply as an index of the existing state of the oral mucous membrane. Certainly the tongue is the more frequent organ of the two to first become diseased, as it is nearer to the outer world and its infections than the stomach. Therefore, it might be supposed that catarrhal states of the tongue, mouth, and throat may occur more frequently as independent diseases, not secondary to antecedent diseases involving the stomach. Schech ("Krankheiten d. Mundhöhle"), in addition to malformations and inherited or acquired defective forms of the mouth, describes 16 distinct diseases of the human mouth, not including neoplasms, tumors, and results of nervous diseases. Seifert (Penzoldt and Stintzing's "Handbuch der spez. Therapie, Bd. IV) describes 23 mouth diseases. In the primary form, all these arise in the mouth, and occur as secondary forms in acute inflammatory conditions of the digestive tract, particularly after infectious diseases. We have paid particular attention to the state of the tongue, esophagus, and stomach at autopsies, and also during a large number of analyses of stomach contents, and must admit that the condition of the tongue is one of the most variable signs in gastric symptomatology. The cases of manifest disease of

the stomach where a primary disease of the mouth is out of the question, are extremely rare. A critical review of the etiology of gastric diseases can not fail to evince the fact that the prominent causes can, and most often do, affect alike the mouth and stomach. The gastric disorders in which the tongue is most frequently unaffected are those associated with little gastric sepsis, *i. e.*, ulcer hyperacidity, neurasthenia gastrica; whereas, in diseases associated with much gastric fermentation or histological changes in the mucosa that may extend to the mouth, or involve it through circulatory or nervous channels, the tongue is most often affected. These diseases are gastritis, carcinoma, and dilatation.

In reviewing the statements of most authors on the condition of the tongue, one can not fail to notice a lack of clearness and precision, which doubtless indicates that the relation between remote and local causes is not well understood concerning this matter. A systematic bacteriological and histological study of coated tongues is very much needed in association with gastric diseases. The attempt to establish a definite, characteristic condition of the tongue for every gastric disease has thus far failed. The extension of stomatitis and glossitis to the stomach by the deglutition of infective material is very intelligible. But the various forms of gastric diseases may also extend upward, either by eructations or direct cellular continuity. Then, again, the oral and gastric cavities are in intimate correlation, and may mutually affect each other through the vascular and complex nervous channels. Fleischer (*loc. cit.*, p. 820) holds that the importance of the coating of the tongue as a sign of gastritis has been much overrated, and that the tongue may be clean notwithstanding very evident chronic gastritis and may be coated when this disease is absent. Nevertheless, he considers the frequent coincidence of coated tongue and gastritis remarkable, but attributes it to a concomitant stomatitis.

General Nutrition.—Chronic gastritis of long standing, left untreated, will inevitably affect the general nutrition. As von Noorden repeatedly emphasizes, "most dyspeptics do not eat enough," and in consequence of this, emaciation ensues to such a degree that even physicians suspect a grave underlying disease (tuberculosis or carcinoma) where there is only a chronic gastritis. The absence of appetite is most frequently caused by suppression of secretion of HCl.

Feeling of pressure and fullness in the epigastric region is, in many cases, complained of, and may be evident on awakening or develop after ingestion of food. The epigastric region in these cases is very likely to be arched forward and outward, and very sensitive to pressure, even the weight of the clothes being annoying. If no dilatation exists, the lower border of the stomach is found in its natural place. It must not be forgotten that a stomach may be dilated considerably and yet the lower border be found in normal position, for the organ may be enlarged upward or laterally, displacing the diaphragm. Prof. J. Schreiber, of Königsberg, has repeatedly called attention to the fact that the horizontal umbilical line is a misleading landmark by which to judge a dilatation, and that the upper border should, in all cases of suspected dilatation, be determined (*Archiv f. Verdauungskrankh.*, Bd. II, Heft 4). It may be possible to ascertain by palpation whether the gastric walls are thickened or not. If a dilatation be present there exists, generally, a stenosis of the pylorus; very rarely it is due to so-called atony. The feeling of pressure may increase to a constant dull pain, which should, if it becomes intense, lead to suspicion of carcinoma or ulcer. Some patients with chronic gastritis suffer during digestion from an active peristaltic unrest in the stomach and intestines, their attention being directed to it by abdominal rumbling and gurgling (borborygmus).

Conditions of Gastric Contents; Secretion.—The results of microscopical and chemical analysis after test-meals, or of lavage water early in the morning before any food has been taken, will vary according to the particular kind of chronic gastritis and the present state of the disease. Boas recognizes, with regard to these points, four varieties, viz.: (1) Acida; (2) Anacida; (3) Mucosa; (4) Atrophicans.

Gastritis Acida. State of the Secretions.—Prior to the results of recent investigations, it had been uniformly maintained that absence or great diminution of HCl was a constant symptom of chronic gastritis. Boas argues that there is a form of typical inflammation of the stomach—termed by him "Gastritis Acida"—in which there is present a normal amount of acid, or even superacidity (Boas, "Ueber Gastritis Acida," Mittheil. d. Naturforscher-Versamml. in Wien, 1894). Even the mucus from the fasting stomach may turn Congo-paper blue. In gastritis subacida, or anacida, the free HCl is reduced or entirely absent, but combined HCl still may be present.

Gastritis Anacida.—In this subdivision free HCl is diminished or entirely absent, but combined HCl is still present. Egg-albumen discs are but slowly digested, or not at all, in the filtrate, even after addition of HCl. The difference between this and the atrophic form is but one of degree, as in the latter all secretion is lost completely.

Gastritis Mucosa, or *Mucipara.*—As was pointed out (page 121) before, when rhinitis, laryngitis, pharyngitis, and bronchitis can be eliminated, large quantities of mucus in the gastric contents, as a rule, speak for chronic gastritis mucosa. The cases not forming much mucus are rare. The mucus formation can be best estimated by washing out the fasting stomach. There should be no difficulty in differentiating gastric mucus from that derived from the respiratory passages. The former is generally thin, clear, glassy, stringy, and flowing; the latter, thick, opaque, yellowish-gray, and lumpy. In the washing from the fasting organ, one frequently finds the organic, structural form-elements of the mucosa, minutely described in the last chapter (and on p. 129). If these bits of mucosa are found at repeated washings, showing these elements either in conglomeration or singly, there can be no doubt of the existence of glandular chronic gastritis. Frequently the morning contents of the fasting organ show numerous leukocytes. The contents should be drawn by expression; if possible, without using water. In gastritis chronica mucipara the contents may show a normal amount of HCl, or may be either neutral or alkaline.

Gastritis Atrophicans.—In this variety both free and combined HCl are absent, and the tests for enzymes and pro-enzymes are negative. Milk taken or poured into the stomach is returned mostly in unchanged condition. Martius and Lüttke (*loc. cit.*), von Noorden, and others, maintain that absolute disappearance of pepsin and rennin is never seen. From large clinical experience we are prepared to state that the end stages of atrophic gastritis give no evidence of ferments in gastric contents by any of the known tests. Nor would it be rational to suppose that in hypertrophic gastritis, in which the stomach is converted into a hyperplastic, dense, hard mass of muscle and connective tissue, with no histological remnant of a glandular layer, there should be any possibility of the formation of enzymes. In atrophic gastritis, more than in the other forms, there are very characteristic, lancinating pains.

The digestion of albumin discs or fibrin in the thermostat is much retarded, or may be wanting entirely, denoting the suppression of the secretion of pepsin.

Disappearance of rennin and its zymogen goes on simultaneously with that of pepsin. In cases with loss of rennin the zymogen of this ferment must be tested for. Among other observers, Bouveret ("La pepsine et le ferment lab.," *Gaz. Méd. de Paris*, 1893, No. 22) declares that the absence of this zymogen is an important criterion of the degree to which the destructive process has advanced. For the same purpose, Jaworski suggests the introduction of decinormal solutions of hydrochloric acid into the stomach, to awaken any slumbering remnants of pro-enzyme formation and convert them into perfect enzymes. In no case that shows the presence of rennin zymogen need hope of complete or partial restitution be resigned.

Age.—This is pre-eminently a disease affecting adults, for the young are not so liable to abuse their stomachs, or so subject to the manifold factors composing the etiology; besides, their reconstructive and compensatory powers are greater. The majority of cases are over forty years of age, but Litten (*Zeitschr. f. klin. Med.*, Bd. XIV, S. 573) has reported a case of eighteen years, and Einhorn one of twenty-one years. The case of Westphalen (*St. Petersburger med. Wochenschr.*, 1890, Nos. 37 and 38) was, however, verified by autopsy; it occurred in a young man twenty-eight years old. We have had under our personal observation since 1888 a young printer, at that time in his twentieth year, whose case showed absence of enzymes and HCl, with much mucus. Numerous leukocytes were evident in the contents (mucus) before food had been taken. Although we have frequently since analyzed his stomach contents, no hydrochloric acid or pro-enzymes have ever been detected. But on several occasions there appeared fragments of gastric mucosa, showing glandular atrophy and chronic inflammation.

The condition of the bowels most frequently exhibits constipation. Absence of the antiseptic action of hydrochloric acid favors intestinal fermentation, flatulence, and meteorism. When there is much decomposition of ingesta, intestinal irritation will eventually set in, accompanied by diarrhea.

The urine is rich in urates and phosphates and often gives a strong indican reaction. The total acidity of the urine is reduced.

The general health is variable; the body weight may either be reduced or remain constant for years: this last indicates that the intestinal digestion is good. Many changes of the general condition, from good to bad and vice versâ, may occur, but as the chronic inflammation progresses there are marked symptoms of general discomfort and indisposition to bodily or mental exertion. The least exertion rapidly tires, bringing on pains in the limbs, and despite this exhaustion there may be insomnia. This leads to a depression of spirit which may control the entire mentality, and brings on hypochondriasis and melancholia. This leads us to refer to the *psychic* and *nervous* symptoms, of which there may be many, beginning with timidity and worry at every new symptom, precordial fear, oppression, and cardiac palpitation accompanied by occasional attacks of dyspnea. The so-called stomach vertigo, first described by Trousseau, we have never observed in chronic gastritis, nor the agarophobia (*i. e.*, terror in crossing wide and empty localities alone) which Fleischer (*loc. cit.*) says occurs as a psychic accompaniment. From practical observation on many cases, we are disposed to believe that these psychic and nervous phenomena have been exaggerated, as they occur only in very protracted cases, and then even inconstantly.

Disturbances of Motility.—A great number of cases of chronic gastritis have been examined at the Maryland General Hospital with regard to the peristalsis; and, in the large majority, this has been found normal, or slightly exaggerated. We use the method described on pages 76–80. Boas declares that he has never observed a dilatation arise from a chronic gastritis (" Diagnostik u. Therap. d. Magenkrankh.," 2d edition, p. 21). It is evident that stenosis of the pylorus can occur which is not caused by cicatricial contraction nor by neoplasm, but by hyperplasia of the muscular sphincter of the pyloric region. If a chronic gastritis lasts long enough, it is a fair presumption that it may result in a gastrectasia, due either to atrophy of the muscularis from connective-tissue invasion, or to the muscular hyperplasia, producing a stenosis. Boas has also conclusively shown that lactic acid is not, as a rule, formed in glandular gastritis.

Complications.—The most frequent is the extension of the inflammation to the intestines. The frequent association of chronic duodenitis with the disease explains the occurrence of catarrhal icterus, which is an extension of the intestinal inflammation to the

gall-ducts. The results of chronic gastritis are, in protracted cases (particularly where the intestines have been involved), marked disturbances of nutrition and anemia, which, as we have had occasion to observe, may assume very serious forms.

The **duration** may vary from several months to years, particularly if the patients have not the means nor the will-power to follow dietetic régime.

Atypical forms of chronic gastritis are by no means rare occurrences, and sometimes make a clear diagnosis very difficult. In the foregoing description of the disease, the lack of very characteristic and peculiar symptoms is evident. In addition to this, the symptoms of loss of appetite, pressure, fullness, eructation, vomiting of mucus, may be absent in atypical forms, and it has been observed that chronic gastritis may run its course in a latent, undetected manner. Again, it may exist under the manifestations of a nervous dyspepsia, or there may be such prominent intestinal symptoms as to disguise the gastritis.

Diagnosis.—The determination of chronic gastritis is one of the most uncertain things in the domain of digestive clinical pathology. It requires careful study not only to distinguish chronic gastritis from other diseases, but also to distinguish the simple, mucous, atrophic, and chronic gastritis acida (of Boas) from each other. As a rule, the primary and secondary forms can be distinguished without much difficulty. Generally speaking, the diagnosis of chronic gastritis can only be satisfactorily established after the possibility of the existence of other affections of the stomach has been excluded. This disease may strikingly resemble the clinical pictures of the gastric neuroses, of ulcer, and even carcinoma. As dilatation is a very rare complication, it is not a confusing factor in diagnosis. One should not make the diagnosis definite at the first examination, but reserve opinion until the patient has been studied during three or four visits. It has, in some cases, taken a much longer time than that to obtain satisfactory evidence of the disease. The best evidence is afforded by repeated microscopical and chemical examination of the wash-water and test-meals.

It will be necessary to dwell upon the differential diagnosis between chronic gastritis and the neuroses, ulcer, and carcinoma: The neuroses may present all the symptoms of a chronic gastritis, particularly the absence of HCl; but, after patient and repeated test-meal analysis, it will be found that the neuroses will some day

show a normal and even excessive amount of HCl. The course to pursue is to wait for this evidence. The presence of much mucus, epithelial cells, and leukocytes in the wash-water from the jejune stomach indicates chronic gastritis. Demonstration of enzymes and pro-enzymes is very valuable, as a normal amount of pepsin and rennin (when HCl is absent), speaks for neurosis and against gastritis. In the absence of HCl, Jaworski's method of pouring in decinormal HCl should be used to stimulate the formation of enzymes.

In the incipient stage of chronic gastritis, the enzymes may be present, even in normal amount; but they disappear gradually as the disease progresses. By the time the physician is consulted, the enzymes are very much diminished or entirely absent; and, according to Boas, this is an indication of an inflammation of the mucosa, not a neurosis.

The differential diagnosis between *idiopathic chronic gastritis* and *ulcer* is decided by the symptom of pain, which is always present in ulcer, and usually absent in chronic gastritis. The ulcer-pain is localized, well circumscribed, very intense, and occurs at definite times after partaking of food. Hematemesis, of course, points to ulcer. The vomit of ulcer shows hyperacidity, which is, as a rule, absent in gastritis. In atrophic gastritis there may be lancinating pains, but they are not so frequent or constant as in ulcer.

From carcinoma the differentiation is very difficult when no palpable tumor can be detected. This is intelligible when one reflects that carcinoma is generally complicated with chronic gastritis. If a pyloric carcinoma be present, the most noteworthy symptoms are: stenosis, motor insufficiency, and stagnation of food with large amounts of lactic acid. A carcinoma seems to strike a stomach suddenly with very severe symptoms and general disturbances—pain, emaciation, and vomiting; whereas, chronic gastritis is characterized by slow increase of the gravity of symptoms, with alternating improvements and aggravations. It is an important fact that the motility is not disturbed in chronic gastritis, and, therefore, the jejune stomach rarely contains anything but mucus, isolated cells, and leukocytes. But in carcinoma the peristalsis is seriously impeded from the onset, and therefore there must be stagnation, retention, and acid-fermentation. These products of retained ingesta occur even where there is no stenosis of the pylorus, as a result of carcinomatous invasion of the muscularis. Gas-

trectasia is an exceedingly rare result of gastritis, and can only occur from hyperplastic thickening of the pylorus, a thing seldom reported in the literature of this subject. As stated before, the presence of marked amounts of lactic acid is not observed in gastritis, but in carcinoma its occurrence is very frequent. Organic acids are rare in the test-meals of gastritis, whereas in carcinoma there is, as a rule, an excess of lactic and fatty acids early in the disease (Boas, *loc. cit.*). For further differentiation see article on Carcinoma.

Synopsis of diagnostic points in various types of chronic gastritis:

	Contents of Fasting Stomach.	Acidity.	Ferments.
(1) Simple Chronic Gastritis. Subacid or anacid.	Limited amount of watery mucus; leukocytes; epithelial cells; round cells.	Variable free HCl rarely present, but if present lessened in amount; combined HCl present.	Pepsin and rennin present in small amount; propeptone formed in the stomach.
(2) Chronic Mucus Gastritis.	Much mucus; epithelial fragments.	At the beginning there may be a normal amount of free HCl, later on the amount is low; HCl absent; HCl deficit.	Pepsin absent; rennin absent; both pro-enzymes present; experimental digestion occurs on supplying the HCl deficit.
(3) Chronic Atrophic Gastritis. Lancinating pains present in this form.	Empty; no mucus.	HCl absent; HCl deficit; no combined HCl.	No enzymes; no pro-enzymes; no curdling of milk on adding HCl.
(4) Acid Gastritis.	Much mucus; giving HCl reaction.	Normal amount HCl, or hyperacidity.	Ferments increased.

Prognosis.—Chronic gastritis is a tedious, but not a very serious affection, as many cases recover under suitable treatment. The prognosis must vary with the stage of the disease as presented, and the intelligence and will-power of the patient. Patients who will study to avoid further detrimental influences, and who have the determination to carry out the dietetic and hygienic management, will recover. With incorrigible eaters or drinkers, who retransgress against their stomachs on the slightest improvement, permanent recovery is doubtful. After the establishment of partial or complete atrophy of the glandular mucosa, perfect recovery is impossible; but, as it is well known that a good state of general

health may be maintained with complete suppression of the gastric juice, provided the intestines still function normally, atrophy of the mucosa need not necessarily imperil vitality. On the other hand, there are numerous well-authenticated observations (see literature) that demonstrate severe disturbances of nutrition, particularly pernicious anemia, as a consequence of gastric atrophy. Fenwick (*loc. cit.*) suggests that this pernicious anemia may be due to autointoxication from the stomach. Other authors, again, hold that the anemia may be the primary factor, and bring about the atrophy of the mucosa. This entire question still partakes of a speculative nature, since exact and logical experiments and deductions are wanting.

Treatment. *Prophylactic Treatment.*—The prevention of the development of the disease implies avoidance of the causes given under the head of etiology of acute and chronic gastritis. Special attention should be directed to the avoidance of continued abuse of alcohol. Every acute gastritis, be it an independent, idiopathic affection, or secondary to other diseases, must be carefully treated in order to prevent its transition into the chronic form. Explicit directions regarding diet and mode of life must be given to all sufferers from liver, lung, heart, and kidney diseases; also to diabetics, in order that they may be saved from secondary gastritis, for disturbances of appetite and impairment of digestive powers must inevitably render the fundamental disease more serious.

The chief predisposing factors in chronic gastritis are passive congestion and accumulation of injurious metabolic products in the heart muscle, with renal insufficiency. In cases of threatened passive engorgement, digitalis should be used early. One need not fear the appetite-disturbing effect of the medicine, as this is usually transient, and we can confirm Leube's statement that an improvement of the appetite and of nutrition in general is observed after treatment by digitalis, we usually combine it with large doses of strychnin. The passive engorgement of the mucosa is more harmful than the drug. If it is noticed in several attacks that the gastric symptoms improve on administration of digitalis, it is expedient to give the remedy at the outset of the slightest disturbance of appetite, since our experience has taught us that this will unfailingly become aggravated by delay in the use of the heart tonic. If the stomach rebels against the remedy, the infusion should be given by enema into a rectum previously

cleaned by warm normal salt irrigation, or digitalin injected hypodermically.

Lavage.—When it is no longer possible to remove the causes that lead to a chronic gastritis, we may yet be able to remove those that maintain or aggravate the malady. These causes are: the accumulation of mucus, and the mechanical as well as chemical irritation of the stagnating contents, particularly when atony and hypertrophic stenosis exist. To accomplish this, emetics are impracticable, because they rarely effect a thorough cleansing, and may increase the inflammation by the convulsive contractions they excite and by their direct irritation. If an emetic is absolutely not to be avoided, apomorphin hypodermically is the safest. Purgatives are even more deleterious, for several reasons: first, they also increase gastric irritation; secondly, they can not be used habitually; and, thirdly, they hurry decomposing masses into the intestines, thereby precipitating an involvement of this tract and the dangers of intestinal putrefaction and auto-intoxication. Lavage is the only correct procedure in chronic gastritis wherever increase of mucus, absence of HCl, decomposition, and a protracted stomach digestion are evident. The mucus often adheres very tightly to the gastric walls, since it only appears, as a rule, toward the close of the washing. Its evacuation is facilitated by allowing the water to run in under high pressure, and directing the patient to change his position—*i. e.*, lying on his back, rising or turning on his side—during the lavage; or by employment of gastric massage. The solution of the mucus is effectually accomplished by adding one tablespoonful of salt and two tablespoonfuls of sodium bicarbonate or biborate to a liter of warm water. To disinfect the stomach after the removal of mucus and fermenting ingesta, the following remedies are approved aids: Salicylic acid, 1 : 1000; thymol, 0.5 : 1000; boracic acid, 10 : 1000; chloroform water, 5 to 10 : 1000; shake the chloroform with the water, and after settling pour off the water, using only the latter; hydrochloric acid, 8 : 1000; resorcin resublimate, 10 : 1000; benzol, 5 : 1000. The solution must be prepared immediately before the washing.

The frequency of the lavage depends upon the state of the stomach. There may be cases that do not require it oftener than once in two or three days; others require it twice in twenty-four hours; usually, once a day is sufficient. The time of the washing should be so selected that the exhausted stomach may enjoy the

longest possible rest. For this purpose six o'clock in the evening is most suitable, as it is then about six hours after the main meal of the day, and only very light diet is taken after the lavage and before bedtime. In other cases this hour may be inconvenient, and an early hour before breakfast must be chosen. Washing out the stomach is advisable only when there is much formation of mucus and where there may be stagnation of food. In atrophic or chronic gastritis without much mucus, frequent lavage is not necessary. In these cases the stomach-tube is recommended, not to remove fermenting ingesta or mucus, but to treat the mucosa directly, to stimulate its sluggish secretion by irrigating with decinormal solution of HCl; if enzymes are still to be detected, common salt solutions are useful for this purpose (about one tablespoonful to the quart). Solutions of NaCl must not exceed the strength of one per cent., as solutions of four per cent. NaCl check the HCl secretion and are available in the treatment of hyperacidity (see "Achylia Gastrica").

Dietetic Treatment.—In each case it is advisable to give the patient a written diet-list, based upon a chemical study of the individual's digestive power. Sometimes it will be impossible to give a diet at first that will at the same time suit the patient's palate and digestive power. The most digestible food will at times disagree with chronic gastritics, and food which would seem *a priori* very indigestible, agrees well. A good plan is to inquire minutely into each patient's accustomed diet and ascertain what food especially disagrees. At the beginning, the diet should be of a light kind; one that makes but slight demands upon the working capacity of the stomach. As the motility is good in this trouble, the diet, as far as possible, should be liquid or semi-liquid, and four to six small meals per day are preferable to three large ones. Nutritious soups, such as beef bouillon enriched by the addition of butter, eggs, beef-meal or jelly, somatose, or peptone, are generally well borne, but as a rule do not suffice to maintain strength and body weight. It is well to insist on slow eating, thorough chewing, and insalivation as important. The teeth should be looked after, and, if necessary, repaired, or artificial ones provided. Should there be a normal amount of HCl and pepsin secreted, then a diet rich in proteid will be advisable. Suitable articles of diet are all meats, fish, and eggs, properly prepared; which means that the roasts or broiled meats, even after they are on the table, must be very finely divided

on the plate, before placing in the mouth. Light vegetables are permissible in form of purées, viz.: potatoes mashed in milk, peas or beans driven through a sieve, spinach, etc. When the HCl, although present, is considerably reduced, the diet will be similar; but spices and well-salted food are more adapted. If the secretion is completely suppressed, it is not expedient to greatly restrict the diet, as these patients are more liable to suffer from inanition. The greatest care is to be employed in the preparation of the food, which must be presented in an appetizing and finely divided form. All meats and fish must be prepared in the steam broiler, and, if needed, they should be previously minced and then re-formed into any desirable shape, held by a supporting substance such as experienced cooks are familiar with, generally consisting of bread crumbs, eggs, salt, and butter. Milk, if it agrees well, is a valuable adjunct to the diet, and even if not well digested, or if there is an aversion to it, should be surreptitiously added to soups, chocolate, rice, sago, gelatins, and farinaceous foods. When it is thus mixed with other foods it is generally very well digested, and adds to their nourishing quality. Von Mehring has recommended a chocolate (Kraftchocolade) which contains 20 per cent. of fat; it is very palatable and usually causes no digestive distress. When there is emaciation we give small amounts of alcohol, upon the authority of Chittenden's experiments, that alcohol up to three per cent. favors the formation of peptone and amylolysis. If we are sure, from test-meals, that there is no gastric fermentation (and according to our experience there rarely is in chronic gastritis), we recommend the genuine Oporto, Malaga, or imported Hungarian Tokay wines (J. Palugyay & Sons, Pressburg). When it is evident that the gastritis was caused by bacchanalian excess, it is, naturally, a good plan to exclude alcohol as far as possible. Indeed, when the emaciation is not marked, or where, after a trial, the collective symptoms appear to become worse, alcoholics are best avoided. There are, however, cases of distinct alcoholic gastritis in which, after well-observed test-meals, the proteolysis is carried on better when wine is taken.* The wines we have recommended have not only a stimulating, but, on account of their large percentage of

*The "rationale" of the administration of alcohol is governed by its effects on the gastric digestion as observed in test-meal analysis—if it impedes digestion it must be forbidden.

grape sugar, a nutritive value. This grape sugar will, however, increase the lactic acid formation if it be already present. In this last case a standard champagne—Mumm's Extra Dry, "Roederer," Piper-Heidsick—is preferable. Beer and claret are, according to our experience, not well borne, and frequently augment gastric distress. In cases of marked anorexia a palatable dilution of brandy or whisky, taken half an hour before meal times, very often produces an appetite. The so-called "Angostura Cocktail" is sometimes useful to sufferers from anorexia. The physiological reasons for the administration of alcohol are explained in the chapter on The Dietetics of Alcoholic Beverages (also in the *Dietetic and Hygienic Gazette*, May, 1896, p. 289; and R. H. Chittenden, *Amer. Jour. Med. Sciences*, Jan. to April, 1896, "Influences of Alcohol on the Chemical Processes of Digestion").

Constipation in chronic gastritis should always be treated dietetically, never by medicines per os. A glass of cold water, or, preferably, of Bedford Magnesia Spring water, before breakfast, is a simple thing, and yet, if persisted in, very often gives an evacuation. The breakfast should contain honey, milk-sugar or levulose, some plum, fig, or prune preserves, and Graham bread. Twice daily, a glass of buttermilk or kefyr may be administered, if agreeable to the patient. Where the constipation resists this diet at the beginning, a trial for the first week should be made with large colon irrigations, with one liter of normal salt solution, introduced in the knee-elbow position. Fleiner's enemata of 250 c.c. (½ pint) of pure olive oil are more lasting in their effects, one enema sometimes keeping the bowels regular for a week. When *diarrhea* is present large irrigations of warm water, by removing fermenting and putrefactive colon contents, frequently cure it without other medication. But strict dieting for a few days is always advisable in exhaustive diarrheas, as it shortens the attack. In diarrheas, as well as in constipation, the state of the gastric secretion must be regarded, and HCl or alkalies must be supplied, as the case may be. Excess of HCl secretion may provoke diarrhea by causing carbohydrate indigestion. A diet of Pasteurized milk and some stimulant, as brandy, and, perhaps, albumen water for forty-eight hours, to the exclusion of everything else, is most effective. Soup made of bouillon and thickened with wheat-flour toasted brown in hot butter, is quite binding. "Eichelcacao," a palatable German preparation of chocolate, can be recommended for its constipating effect, as it contains much tannin.

For special full diet lists and further dietetic directions concerning this disease, as well as other recipes, we refer to the section especially devoted to this subject,—the comprehensive chapter on Dietetics (pp. 223–226).

As Gilman Thompson points out, there are some persons in whom the digestion of salt and smoked meats seems to be more easily accomplished than that of prepared fresh meat. Niemeyer offers the explanation that these preparations are less likely to decompose in the stomach. We agree with Boas (*loc. cit.*, p. 21) in the observation that there is very little fermentation and formation of organic acids in chronic gastritis. After carefully observing this point, we maintain that it is not necessary to withhold the saccharine and farinaceous foods, as Gilman Thompson suggests (*loc. cit.*, p. 508). On the contrary, they should be liberally supplied, as amylolysis progresses rapidly in stomachs that secrete no HCl, and test-meals do not, as a rule, show the excess of organic acids asserted by von Leube, Ewald, and Rosenheim.

Balneological.—There is much truth in what Prof. Ira Remsen said when he opined that the effect of the use of natural or mineral spring waters was not attributable to the chemical constituents or salts of these waters, but more to the favorable mode of life, the better diet, the greater introduction of plain water into organisms which previously received very little of it, and, lastly, to important psychic influences. To these may be added the perfect rest, comfort, and auxiliary methods of treatment employed at the springs. Thousands of Americans visit the German, Austrian, and French spas annually, when they might have almost the same waters—and sometimes much better ones—in their own country.* Observations are very numerous on the treatment of chronic gastritis by mineral waters, but are rather inexact and based upon imperfect histories, as many cases are called chronic gastritis which, in fact, do not deserve the name. One can not well judge of the effect of the waters alone, as they are always combined with dieting. The systematic drinking of alkaline waters must not be estimated to be worth more than that of a poor substitute for lavage. As the object is to promote the solution and evacuation of mucus, all waters will be equally serviceable, even ordinary spring or hydrant water. The salts can be

* In extended travels through our Eastern States, we have seen over 20 mineral springs discharging very healthy—sometimes carbonated—waters, that are not at all known except to people living in the immediate vicinity.

added to imitate the real composition of the famous springs, and are made by several wholesale manufacturers of effervescent salts for this purpose, so that the poorer patients may have the effect of mineral springs at home.

(For the effects and contraindications of mineral waters the reader is referred to the section on Mineral Springs. The composition of artificial Carlsbad salts is given on p. 323.)

In the use of alkaline chlorids it is expected to stimulate the secretion of HCl. Hot spring waters must be cooled and the cold water warmed; and, in dilatation and atony, the patient had best abandon the use of water in this manner entirely.

Baths.—As in chronic gastritis the general metabolic processes are much depressed, a cold sponge bath taken before breakfast will gradually make the dyspeptic more resistant by its stimulation of cellular oxidation and its hardening effect. Warm baths we advise, for purposes of cleanliness only, once or twice a week.

Gymnastics.—All patients with chronic gastritis should be encouraged to take moderate exercise : walking, bicycle riding, horseback riding; also rowing and swimming. A pair of four-pound dumb-bells for men and two-pounders for women should be used three times daily, each time for five minutes, with three minutes of rest intervening between the five minutes of exercise. This will make fifteen minutes of training, and should be done before dressing, in the under-garments, immediately after the cold sponge bath. Great care should be bestowed upon the tonicity of the abdominal muscles. Loss of the unconsciously and continuously acting tonus of these muscles is a most potent factor in the etiology of dilatation, gastroptosis, and floating kidney. There are, of course, other causes; but, even if the attachments of an organ are loosened, it can not wander far from its normal location with a vigorous, unrelenting, external abdominal wall. Therefore, all patients subject to digestive diseases, except ulcer and carcinoma, should train their abdominal muscles and keep them active. Sandow's directions for accomplishing this, as described in his book, are excellent.

Electricity.—The faradic and galvanic currents are useful in the treatment of chronic gastritis. The former may be used as general external faradism, which is one variety of a general massage. One pole, in shape of a broad, flat electrode, is moved slowly up and down over the spinal column, while the other is moved over both

arms and limbs, and particularly over the abdominal muscles. With one pole over the spine, and the other over the epigastrium, the current appears to go directly through the stomach, and yet there is no evidence that the organ does contract. In this case it is doubtful whether any current reaches the stomach at all. The good effects observed after this method must be attributable to the abdominal massage and the psychic influence. For the intragastric application of both the faradic and galvanic currents, the practical intragastric electrode of Einhorn is possibly the most convenient. The secretion of gastric juice can not be influenced by either the faradic or galvanic current, nor can the motility be enhanced (J. C. Hemmeter, *New York Med. Journal*, June 22, 1895, p. 769). As the currents usually employed for this purpose are too weak to effect a contraction of the muscularis, Meltzer (*New York Med. Journal*, June 15, 1895) holds that percutaneous and direct faradization of the stomach and intestines can not produce any contraction of these parts. Max Einhorn (*Archiv f. Verdauungskrankheiten*, Bd. II, S. 454), in his recent contributions to the subject, is of entirely opposite opinion. As mentioned in the theoretical part of this work, we have been able to demonstrate that the human stomach can be made to contract under faradic stimulation by introducing the intragastric bag (which terminates in an electrode near the pylorus) and connecting it with the kymograph. Some of the intragastric bags ended in two brass electric end-knobs, one on the cardia and one on the pylorus. However, the currents required to cause gastric contraction were so strong as to become hazardous (see part I, p. 60). As Ziemssen (" Electrizität i. d. Medizin," 1887, p. 445) has emphasized, it is not necessary to effect gastric contraction in order that electricity should prove beneficial. In fact, it would appear that a neuro-metabolic or -trophic effect of electricity is becoming more and more understood, so that the faradic and galvanic current should be employed, both externally over the spine and epigastrium, and internally with the intragastric electrode; not because of any undeniable evidence that it can influence secretion, motility or absorption, but because of the general uniformity of opinion among experienced clinicians, that chronic gastritis is undoubtedly benefited by electrical treatment. Even Goldschmidt (*loc. cit.*), whose results regarding the effect of the faradic and galvanic currents on secretion and motility are entirely negative, admits that these are useful agents in the treatment, even bene-

fiting stomach diseases depending upon organic changes. It is evident that while experimental evidence of the manner in which electricity acts on the stomach is very necessary, the clinical approval of its therapeutic utility is more important.

Medicinal Treatment.—Two chemicals seem to have maintained their reputation as being able to benefit the disease; these are argentic nitrate, either in form of gastric spray (1 : 1000), or lavage (1 : 2000), or in form of solution, 0.3 to 120 of peppermint water; of this, one tablespoonful three times daily, on an empty stomach. The second drug is bismuth subnitrate, recommended by Penzoldt (*loc. cit.*), Fleiner (*loc. cit.*), and Pick (*loc. cit.*) in large doses, 4 to 6 gm., in wafers. With both remedies we have had experience, and prefer the latter in the following form, together with bismuth subgallate, which certainly diminishes the amount of mucus formed in alcoholic gastritis:

℞. Bismuth subnitratis, ... 48 gm. ℥ xij
Bismuth subgallatis, ... 16 gm. ℥ iv. M.
Fiant chart. No. xxiv.
SIG.—One powder in a wafer four times daily.

Unfortunately, this treatment is constipating, and must, therefore, be combined with a diet promoting evacuation and the use of Saratoga Congress water. Argentic nitrate is best employed in form of the intragastric spray, or in the lavage. These drugs are permissible, particularly when diet and massage can not be properly carried out. They were originally suggested for the treatment of ulcer; their efficacy in some cases of chronic gastritis is, however, undoubted.

The Hygiene of the Mouth.—The frequent association of stomatitis, gingivitis, and glossitis with this disease make it all-important that the mouth should be in a healthy condition. Dental defects and their repairs have already been referred to; but, in addition, the mouth should be disinfected after each meal. After removing the food débris by toothpick and brush, one of the following antiseptic lotions should be used both on the brush, applied to the teeth and the root of the tongue, and as a mouth wash:

℞. Acid. thymol, ... 0.25 gr. iv
Acid. benzoic., ... 3.0 gr. xlv
Tr. eucalypt., ... 15.0 f ℥ iijss
Alcohol, ... 100.0 f ℥ iijss
Ol. menth. pip., ... 0.75 ♏ xij. M.
SIG.—Pour sufficient into ½ of a glass of water until turbidity results.
If much decomposition be present 0.8 hydrarg. bichlorid corrosive can be added.

℞. Spirit. lavandul.,
Spirit. myrciæ, ... āā . . . . . 50.0 f℥ xijss
Tinct. myrrh., ... 5.0 f℥j
Saccharin, ... 1.0 gr. xv
Menthol, ... 1.0 gr. xv. M.

SIG.—One-half to one teaspoonful to a glass of water.

Treatment of the Symptoms.—This is of subsidiary importance to systematic treatment by diet, hygiene, and lavage; but, in cases that have progressed too far, or in secondary forms that are incurable (albuminuria, diabetes, etc.), a special therapy for symptoms may be indispensable.

The *treatment of loss of appetite* which we advocate is the following: Lavage with chlorid of sodium, ℥ss to the quart or a decinormal solution of HCl; fluid extract of condurango in doses of ℥j; tincture or elixir of gentian. Lavage with quassia, colombo, or calisaya solutions will often produce appetite. Orexin, a new stimulant to the appetite and HCl secretion (first recommended by Penzoldt), is best given in the following form:

℞. Orexin basic, ... 0.2, grs. iijss.

SIG.—Make one wafer. Take one wafer in a cup of bouillon half an hour before meals, t. i. d.

Our favorite tonic for anorexia in chronic gastritis contains strychnin and hydrochloric acid in the following proportion for adults:

℞. Strych sulphatis, ...02 gr. ⅓
Acid. hydrochlorici dilut., ... 19.4 f℥ v
Elixir gentianæ, ... q. s. . . . . 180. f℥ vj. M.

SIG.—f℥ ss in ℥ ij aquæ after meals, through a glass tube.

Fluid extract condurango f℥ xij may, if desired, be added.

Some old gastritics can not tolerate so much hydrochloric acid. Then the dose must be reduced to five drops, t. i. d. Always precede the administration of HCl by a test-meal analysis, so as to find out the degree of HCl deficiency.

Pyrosis and eructation are best treated with magnesia and sodium bicarbonate, according to the principles laid down in the management of hyperacidity.

Pain.—If diet and lavage do not relieve this, it is best to subject the patient to a rest cure of eight days, with hot external fomentations to epigastrium. The galvanic current has been a very reliable means of easing pain in this affection. Opiates and other narcotics must be avoided; but, in the very rare cases where this is impossible, they should be given by the rectum or (morphin) hypodermically.

The same treatment applies also to vomiting, which is, as a rule, relieved by diet, lavage, small pieces of ice, or champagne; very rarely does it become so distressing a symptom as to require a hypodermic injection of morphin.

Deficiency of gastric juice and ferments may be supplanted by the use of HCl internally, as per formula stated above. If HCl is no longer tolerated, it is well to convert the entire gastric chemistry into an alkaline proteolysis by pancreatin and bicarbonate of sodium. In long-standing cases the mucosa acquires a strange hypersensitiveness to all acids, which points the way to this plan of treatment. Reliable pancreatin and sodium bicarbonate, of each five grs., are recommended by Boas, Witte, Simon, Schering, and Penzoldt. In the private sanitarium, we have, by a study of test-meals, found Reichmann's preparation of fresh ox pancreas an effective digestant. It is made by finely mincing one ox pancreas and extracting it with 15 per cent. alcohol or brandy for forty-eight hours, and straining. The dose is a wineglassful after meals. We very rarely found it necessary to give pepsin; for if HCl is still secreted, pepsin will be found also, and if HCl be absent, although the ferments may be wanting, it is expedient to give only the acid, as proteolysis is sufficiently effective in the intestine, and the effect of the HCl is to improve the appetite and prevent intestinal fermentations. It would require enormous quantities of pepsin to do sufficient proteolytic work, which is more readily accomplished by pancreatin. In addition to this, HCl is cheap and pepsin expensive, so that it is impossible to have patients continue its use for a long time.

Motor insufficiency is, fortunately, a very rare occurrence in the disease, but, if present, may be met with intragastric use of lavage, electricity, hydrotherapy, massage, and strychnin. This will be more fully treated in the section on this defect.

Diarrhea and constipation must be treated by diet to the exclusion of drugs; but large colon enemata are valuable and permissible adjuncts to their management.

Psychic depression may, according to the most prominent underlying cause, require one, or other, or several of the methods of treatment mentioned. But regular bowel movements, electricity, a daily tepid or cold sponge bath, moderate exercise, massage, surf baths, and climatic changes are the most reliable means to be employed. Some of these cases will not recover until brought

to a properly managed institution for nervous and digestive sufferers.

Advanced Chemical and Mechanical Defects.—When the glandular elements have been completely destroyed as a result of hypertrophic or atrophic metamorphosis of the mucosa and degenerative processes in the muscularis, and also of cirrhotic contraction of the stomach, secretion, absorption, and motility no longer exist. The gravest defect now is the loss of motility. For, in the total absence of all gastric digestion, no food except a small fraction of the carbohydrates (ptyalin) enters into solution. The ingesta are not reduced sufficiently in size because there is no churning peristalsis and no secretion; they are not squeezed out into the duodenum, because the propelling peristalsis is missing. Now, although there is no stenosis of the pylorus from cicatricial contraction or neoplasm, nor even a pyloric hyperplasia, under these conditions there is what we may term a "relative pyloric stenosis"; that is, the pylorus is relatively too small and peristalsis too defective for the passage of the insufficiently macerated ingesta. This combination of things may occur in the last stages of chronic gastritis accompanied by the clinical aspects of progressive anemia, due to inevitable malnutrition, and may simulate carcinoma.

The intestine, although it may be healthy, can not supplant the absent digestion of the stomach by its vicarious action, since it gets no chance to do so, the gastric contents fermenting, and eventually being expelled by emesis, rather than propelled into the duodenum. This state should be treated exactly as if there were a real pyloric stenosis, namely, by operation,—either by gastro-enterostomy or by dilatation of the pylorus; or, if an excessive atonic gastrectasia with immense enlargement of the stomach and normal pylorus be present, by gastroplication (Bircher). So far, it appears that gastro-enterostomy has been done but once under these conditions for typical gastric atrophy (Westphalen, *Petersburger med. Wochenschr.*, 1890, 37, 38) occurring in a tuberculous patient. As the expelling force of the peristalsis is much reduced, gastro-enterostomy will probably be preferable to dilatation of the pylorus. The indications for surgical operations upon the stomach have been separately considered (pp. 336–358). They are, at the present time, more clearly and precisely defined for cases of *pyloric* stenosis from cicatrices or neoplasms than for cases of motor insufficiency due to gastric atrophy. We have seen one case of well-defined motor insuf-

ficiency, with a history of two years of gastric pain, hematemesis with tar-colored stools, even hyperacidity of the vomit, where the operation revealed a normal pylorus, and no ulcer cicatrix could be detected. The gastrectasia was considerable, but the stenosis was evident only when the stomach was filled, *i. e.*, by kinking off the pyloric exit at its hepatic attachment. This kinking off at the duodenal juncture by stomachs that are dilated occuring only when distended with food, but apparently normal in size when empty, has been first described in an interesting report by Broadbent (*Brit. Med. Jour.*, vol. II, 1893, p. 1193). We have advocated the operative removal of the stenosis in the following cases: Dilatation with motor insufficiency due to the various conditions that may arise from the atrophic and hyperplastic forms of chronic gastritis; in either an absolute (hypertrophic) or a relative stenosis of the pylorus with complete atrophy of the glandular layer and loss of secretion, provided the intestine can be ascertained to function normally. In extreme dilatations with no absolute stenosis gastroplication, as first carried out in this country by Weir (*N. Y. Med. Jour.*, LVI, 29), is an available procedure after medical treatment has failed. Gastroplication does not strike at or remove the cause of the dilatation. Weir (*loc. cit.*) designates the operation of reducing the size of a dilated stomach by making a fold or plait in it as "gastrorrhaphy." This is the same operation that was originally designed by Bircher (p. 349) and termed by him "gastroplication." Weir has used the term "gastrorrhaphy" in a new sense—similarly as the term "perineorrhaphy" is employed to designate an operative reduction in the size of the perineum. Originally the meaning of "gastrorrhaphy" was an operation for closure of the wounds of the stomach (p. 340).

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In addition to the text-books of—

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Bamberger,
Birch-Hirschfeld,
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Brinton,
Cohnheim,
Cruveilhier,
Dujardin-Béaumetz,
Eichhorst,
Ewald,
Fleischer,
Förster,
Henoch,
Jürgensen,
Kunze,
Lebert,
Leo,
Leube,
Liebermeister,
Hayem,
Niemeyer,
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CHAPTER III.

ULCER OF THE STOMACH.

Ulcus Ventriculi, Pepticum, Rotundum, Perforans, Rodens, Corrosivum, e Digestione.

Ulcer of the stomach is a loss of substance of the gastric mucosa, characterized by very little tendency toward healing, but rather by destructive progression both in a longitudinal direction, *i. e.*, in a line with the surface, and toward the depth of the mucosa. It may occur in two forms, (1) the acute and (2) the chronic. The acute form extends so rapidly from the mucosa toward the peritoneum, with such small lateral involvement, that Rokitansky's original comparison, "as if the ulcer were cut out with a punch," has become the classical expression of the text-books. In the chronic form the destructive process is not so rapid; it extends more laterally, producing a terraced or shelving appearance of the edges and sides, so that it may be funnel-shaped. Perforation into an artery, vein, or into the peritoneal cavity occurs in both forms. The chronic form has a tendency to healing, but in so doing causes cicatricial contractions and deformity. The acute form may terminate in healing, but owing to its limited lateral extent, the small cicatrix rarely causes deformity. It is very probable that the acute ulcers have a different etiology (corrosives, toxic action, trauma by sharp, hard materials in the food, in conjunction with other factors to be considered) from the chronic eroding type, to which the following description appertains more especially.

Self-digestion of the Stomach (**Gastromalacia**).—If an animal be killed while in full digestion, the stomach may undergo self-digestion after death if the body is kept warm. In human beings who died suddenly while the gastric digestion was at its height, it was found at the autopsy that not only the stomach had been digested, but also the spleen, and that this process had extended through the diaphragm into the lungs. The question naturally arises, What protects the stomach from autodigestion from its own secretions under normal conditions? This is an inquiry

that concerns the fundamental properties of living matter, for it includes the nondigestion of the intestinal tract by the alkaline pancreatic juice and succus entericus, the same property as observed in the digestive tracts of invertebrates and even in the unicellular organisms, the amebæ and plasmodia of mycetozoa. For instance, Metschnikoff, C. Le Dantec, Greenwood, Saunders, and the author have shown that a secretion is formed in the digestive vacuoles of these unicellular organisms which digests foreign proteid material, but not the living substance of the cell itself (see "On the Rôle of Acid in the Digestion of Certain Rhizopods," by J. C. Hemmeter, Philos. D., etc., in *American Naturalist*, August, 1896, p. 619).

The following explanations have been offered for the protection of the human stomach from its own secretion:

1. By Hunter: That the principle of life in living things protected them from digestion. Resorting to a hypothetical principle in explanation of the properties of living matter, can not be seriously considered in the science of biology.

Moreover, Bernard succeeded in demonstrating that the hind leg of a frog, introduced into a dog's stomach through a fistula, undergoes digestion. This will also happen if the leg be placed in a vessel containing gastric juice at the proper temperature.

2. Bernard explained the exemption of the normal stomach from autodigestion by assuming a protective power in the living epithelium, which he thought prevented the absorption of gastric juice.

3. Stricker believed that the mucus formed on the surface of the stomach acts as a protective covering.

4. Pavy ("Guy's Hospital Reports," vol. XIV, 1868) held that the alkaline blood circulating through the gastric walls saved them from digestion, since it neutralized the acid as fast as it was absorbed. None of these explanations is sufficient. Bernard's suggestion simply shifts the problem by assuming an immunity of the living epithelial cells without attempting to explain why these are not digested. The coating of mucus which Stricker believed to be a protection is the contrary; for in gastric diseases in which there is excessive formation of mucus, autodigestion is more frequently observed than in normal stomachs, provided HCl is secreted.

Pavy's theory that the alkaline reaction of the gastric circulation prohibits self-digestion, is untenable, because under these condi-

tions one could not explain why the pancreas does not digest itself, and is also disproved by Samelson, who produced a neutral reaction of the blood by gradual introduction of acid, and then poured dilute HCl into the animals' stomachs ; but even then no autodigestion was observed. On the whole, the question of the exemption of the stomach from self-digestion remains unanswered. When Hunter, over one hundred years ago (1786), referred the immunity to a specific property of the living cells, "the vital principle," he gave as good an explanation as any given up to date. The expression "vital principle" may sound mysterious in the light of modern physiological knowledge, but it undoubtedly implied that gastric immunity from self-digestion was due to physical and chemical forces possessed by the protoplasm of living cells and which are not as yet understood. In the latter term we use more accurate expressions, but still leave the whole question as unexplained as Hunter.

Elsässer agreed that gastromalacia was always a cadaverous process, and was supported in this view by Virchow, Foster, Oppolzer, Bamberger, and others, so that his opinion became the prevailing one. A contrary view was held by Rokitansky, who represents the belief that there is a gastromalacia that occurs *intra vitam* particularly in the end stages of grave diseases of the brain and its membranes (basal and tuberculous meningitis) and in other severe exhausting affections. The occurrence of intravital autodigestion was proved in a case reported both by W. Mayer and Leube from Ziemssen's clinic, and also by numerous animal experiments.

Results of animal experiments in producing secondary injury and consequent self-digestion of the stomach are the following: Schiff, by intersection of the thalami and cerebral peduncles, produced hemorrhagic infiltrations, partial softenings, erosions, and even ulcer formation in the gastric mucosa, and interpreted his results as consequences of neuroparalytic hyperemia caused by injury to the central vasomotor nerve-tracks of the stomach. Ebstein and Brown-Séquard obtained identical effects after circumscribed destruction of the anterior corpora quadrigemina. Panum injected an emulsion of tiny wax globules into the femoral arteries of dogs and effected small gastric hemorrhagic infarcts and ulcers. Cohnheim injected suspensions of plumbic chromate into the stomach circulation, by which he succeeded in blocking only the branches of the mucosa and submucosa, while the circulation of the muscularis remained free. At the autopsy he discovered large

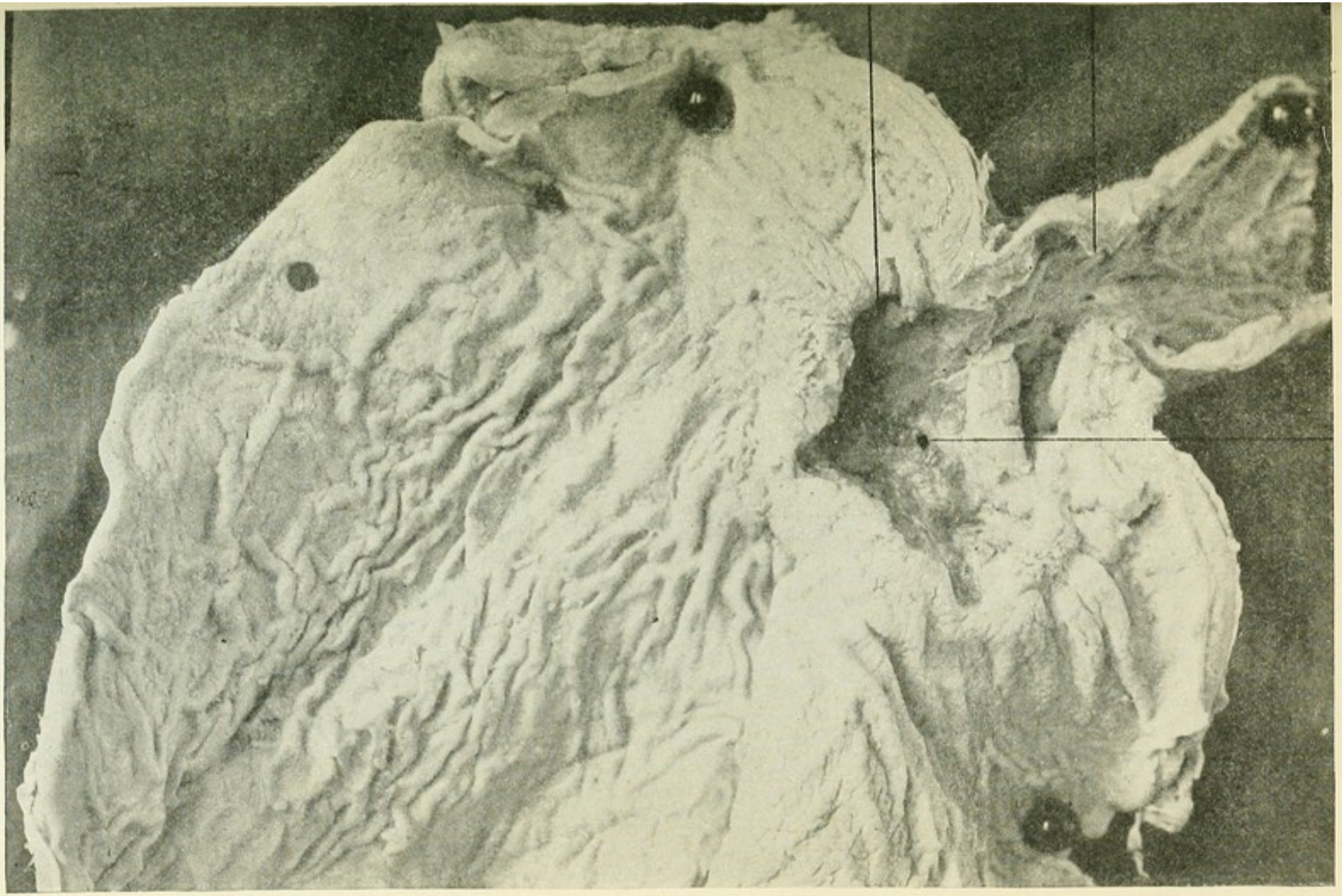

PLATE VII.

CARCINOMATOUS ULCER (ULCUS CARCINOMATOSUM) OF THE PYLORIC ANTRUM.

Reproduction of a photograph of the open stomach showing the nearly circular ulcer, with comparatively smooth edges near the pyloric opening. The swollen, raised condition of the ulcer walls is well shown, as is also the normal, wrinkled condition of the gastric mucosa. The point where perforation occurred is seen as a dark spot to the right and a little above the center of the ulcer. The stenosed condition of the pylorus is not shown, as the pyloric opening and the duodenum have been laid widely open. It can be seen, however, that the ulcer does not involve the duodenum. At one place a small cube of tissue has been removed. (*From the Author's Clinic at the Maryland General Hospital.*)

ulcers with abruptly descending edges and clean bases. Koch and Ewald brought about gastric hemorrhagic infarcts by intersection of the spinal cord (Schiff's method), and after this introduced strong solutions of hydrochloric acid, five per 1000, into the stomach, thereby producing penetrating ulcerations. After severe traumatism,—for example, bruising the epigastric region with a hammer,—and after thermic irritation, as by introducing very hot gruel, Ritter and Decker produced ulcus ventriculi. Silbermann brought on gastric ulcers that healed with difficulty, by causing hemoglobinemia with substances that dissolved the blood-corpuscles.

His results are significant, as explaining the pathogenesis of gastric ulcer after extended skin burns and malaria. This is to a certain extent explained by the investigations of Klebs and Welti, who have shown that broken-down red corpuscles, blood pigment and thrombi of blood plaques, or undeveloped elements may occlude the gastric vessels and cause ulcer; and London explains the gastric ulcers in malaria by the occurrence of pigment emboli. Tolma produced gastromalacia and gastric ulcers by ligating the esophagus of dogs above the cardia and the duodenum below the pylorus (Tolma, "Untersuch. über Ulcus ventric," etc., *Zeitschr. f. klin. Med.*, Bd. XVII, S. 10). This experiment constitutes too violent an interference with normal gastric physiology to permit of any correct deductions.

It is impossible to differentiate the effects of violent trauma, interference with the venous, arterial, and lymph supply, intragastric stagnation, fermentation, and sepsis, that the experiment of Tolma brings about.

Views Concerning Causative Circulatory Disturbances.—Virchow called attention to the frequency of gastric ulcers in anemia and chlorosis, explaining it by the diseases of the vessel walls, fatty degenerations, aneurysmal and varicose dilatations, and their consequences, viz.: thrombosis and embolism. Cohnheim conceded the casual relations of these states, but disputed the frequency of their occurrence, (1) because the abundant anastomoses between the gastric vessels facilitate a compensatory collateral circulation; (2) because the diseases of the vessel walls referred to are rare in young, but frequent in old persons, which would indicate that in these, gastric ulcer should be found frequently, whereas in later life it is very rare. Klebs has a theory attributing gastric

ulcer to local ischemia, supposed to be caused by spastic arterial contractions. Rindfleisch's opinion is that venous stasis in the gastric walls may lead to ulcer, since occlusion of the exit of the blood may occur easily on account of the compressibility and the few anastomoses in the gastric veins; this, he thinks, may cause hemorrhage, erosions, and ulcer. Cohnheim opposes this view also, because gastric ulcer is a rare thing in the passive congestion due to hepatic cirrhosis. One must not overlook the fact, however, that in this state the secretion of HCl is much reduced. Axel Key assumes that long and persisting contractions of the musculature may cause local ischemias or disturbances in the venous outflow. Böttcher and Letulle attribute the causation of ulcer to bacteria, which they could demonstrate in colonies in the floor and in the surroundings of ulcer.

Most of the observers mentioned make the statement that gastric ulcers produced experimentally in animals heal rapidly. The mucosa is replaced almost completely; a new formation of peptic glands has been observed by Grifinni, Hauser, and Vassali. At autopsies, cicatrices are often found in the human stomach, where no symptoms referable to ulcer were evident during life. It is known, also, that pieces have been torn loose by the suction of the lower end of the stomach-tube, and yet this loss of substance healed without forming an ulcer; so that repair may follow injury to the human stomach, and it is very evident that some other causatory factors besides injury are necessary to bring about an ulcer. A pathological composition of the blood has been thought to be one of these factors, particularly as gastric ulcer is very frequently found in anemia and chlorosis. This view is supported by the experiments of Quinke and Daettwyler, who produced a high degree of anemia by gradual withdrawal of blood from dogs. Thereafter, they produced gastric injuries by mechanical, chemical, and thermic irritants, and discovered that ulcers were formed that healed with much difficulty. Clinical experience confirms these observations, that an impaired state of the blood may greatly protract healing. On the other hand, there are numerous records of severe and recurrent attacks of gastric ulcer in persons whose blood was found perfectly normal.

Hyperacidity (one of the etiological factors).—In the second volume of Reynolds' "System of Medicine," page 930, W. Fox expresses the opinion that the cause of chronicity of ulcer may be

"an excessive acidity or secretion of the gastric juice, particularly when the stomach was empty." But to Riegel belongs the credit of having placed this condition of hyperacidity with gastric ulcer upon a scientific basis. His results were confirmed by von den Velden, Jaworski, Korcynski, Ewald, and Boas.

According to our experience, hyperacidity is present in 88 per cent. of undoubted gastric ulcers. Riegel at first asserted that it was a constant accompaniment (F. Riegel, " Beiträge zur Diagnostik d. Magenkrankheiten," *Zeitschr. f. klin. Med.*, Bd. XII, S. 434). But Gerhardt, Rosenheim, Ritter, von Mehring, Cahn, and Hirsch published cases of gastric ulcer with normal and even subnormal acidities.

The methods of Cahn and v. Mehring we have thoroughly tested, and assured ourselves that it gives values that are too low for the free HCl, which may explain, in part, some of the results and discrepancies of Rosenheim and the originators of the method. We do not deny that there are undoubted cases of gastric ulcer in which there is a subnormal amount of HCl, but they are exceedingly rare. It is well known that ulcer may be associated with gastritis, which may have reached the stage of glandular atrophy and become the cause of the sub- or anacidity. Whenever the glandular layer is intact it is reasonable to expect hyperacidity, because the presence of an ulcer is a never-ceasing irritation. Riegel asserts that in consequence of and by means of this hyperacidity an ulcer may develop from an erosion or injury of the mucosa, which, though seemingly unimportant in itself, is retarded in its healing by autodigestion. Of course, it is intelligible (as Ewald, Ritter, and Hirsch point out) that the hyperacidity may be the result just as well as the primary cause of ulcer. It is probable that the hyperacidity may change normal clonic contractions of the gastric muscularis to intense tonic spasms that last an unnaturally long time and produce local ischemia and impediments to the exit of venous blood. This, if it occurs, must inevitably be followed by circulatory disturbances and changes in nutrition of the mucosa, and eventually by hemorrhages of a circumscribed character, out of which the autodigestive process may form erosions and ulcers.

Etiology.—The deductions from the preceding summary of experiments and observations are, above all, the establishing of four principal factors in the etiology of ulcer:

I. An impaired vitality or resistance of portions of the mucosa.

II. Hyperacidity or supersecretion.

III. An altered state of the blood.

IV. Local bacterial infection.

There are a number of well-authenticated cases on record, proving that direct trauma may cause gastric ulcer (vide Einhorn, *loc. cit.*, p. 191; also others reported by C. Hoffmann, Leube, and Eichhorst).

According to Sidney Martin (*loc. cit.*, p. 410), there are three common causes of the death of the tissue which precedes ulceration:

(1) *Mechanical and Chemical Causes.*—Ingested fish-bones, egg and oyster shells, seeds, etc. Corrosive poisons lead to ulceration by directly destroying the tissue; and an injury to the mucous membrane, which is subsequently exposed to the continued action of an irritant, will also lead to an ulcer.

Direct injury and wounding of the gastric mucosa occur very frequently, and, as a rule, heal very rapidly. There are a number of cases on record of persons swallowing glass, nails, and knives, which passed through the entire intestine without causing injury. Marcet ("Med. Chirur. Transactions," vol. XII, p. 72) narrates the case of an American sailor swallowing some 30 pieces of knife-blades, which were found in his stomach, together with a number of handles. Two blades were in the colon and rectum, placed transversely, and had perforated the intestinal wall without causing peritonitis. No recent or old ulcers were found in the stomach.

The following report from the German Hospital, of Kansas City, goes to show that this class of human ostriches has not died out. The main fact that is proved by such cases is that something else is needed in addition to direct injury to the stomach in order to produce an ulcer.

GERMAN HOSPITAL, KANSAS CITY, MO., June 14th.—Harry Whallen, the "human ostrich," who was operated upon at the German Hospital last Saturday, and from whose stomach the surgeon took an assortment of cutlery and hardware, died at two o'clock this morning, the result of the operation.

Whallen got into trouble by swallowing a big Barlow knife, in Pilot Grove, Mo., the other day. When he was operated upon at the German Hospital these articles were removed from his stomach:

Two jack-knives, one three inches long and the other four inches; five knife-blades, from one to three inches long; 32 wire nails, eightpenny or larger; 34 sixpenny nails, 26 shingle nails, 16 carpet tacks and small wire nails, one barbed wire staple, one horseshoe nail, three screws, three ounces of glass, and several bits of crockery.

Whallen was offering bets that at least 100 nails and three knives would be found in his stomach, and if the referee had decided that five single blades are

equivalent to one knife, he would have won his bets. He was a professional showman. He began swallowing glass and nails when he was ten years old, and says he has eaten a lamp chimney nearly every day during the seventeen years he has been at it, but the Barlow knife, which he swallowed last week, was too much, even for his long-suffering stomach.

When the surgeons operated upon him, the stock of hardware inventoried was found imbedded in a solid mass in his stomach and partially encysted. After it was removed the stomach was thoroughly washed out and sewed up.

(2) *Interference with the Vitality of the Tissue.*—The vitality of a particular part of the mucous membrane may be diminished by local and chronic disease or by interference with the circulation over a certain area. This latter usually occurs by means of thrombosis or embolism. Thrombosis takes place in connection with disease of the vessels and in association with inferior quality of the blood and a slowing of the local circulation; embolism may be infective or noninfective, and is usually capillary.

(3) *Bacterial Infection.*—The infective processes of the digestive mucosa with which we are most familiar, are the ulceration processes of typhoid fever, certain dysenteries, and tuberculosis. In the gastric ulcer, however, there is another kind of bacterial infection, which is not accompanied with the signs of active inflammation, and is termed by some authors "bacterial necrosis."

The process is characterized by the invasion of bacteria, usually in the lower depths of the mucous membrane, by their growth and subsequent necrosis of the tissue. Although the secretion of HCl is germicidal to many bacteria, it must be remembered that the spores are not destroyed by it, and that the invasion may take place during the periods of rest of the glands in the intervals of digestion when no, or very little, HCl is secreted. There is room for the suggestion that the primary necrosis is due to bacteria and the ensuing ulceration caused by the action of the gastric juice. The bacteria can exist in the cells around and beneath the floor of the ulcer, and notwithstanding a very high degree of hyperacidity.

In a number of cases which we examined by the most approved cellular and bacterial stains, the bacteria were present throughout the layers, even in the peritoneum, while the floor of the ulcer was in the muscularis. It is conceivable that they pave the way for autodigestion by causing necrosis of the tissues in which they are imbedded. No bacterium was so far obtained in pure culture, but the one most frequently observed was a bacillus

very much resembling that of anthrax and in two cases of ulcus carcinomatosum the Oppler-Boas bacillus.

Thermic causes are the ingestion of very hot food and drink, taken when the organ is empty.

An interesting etiological relation exists between cutaneous burns and gastric or duodenal ulcers.

The last two factors, hot food and large cutaneous burns, explain the rather frequent occurrence of gastric ulcer which is said to occur among cooks, who are in the habit of tasting foods that are still on the fire, and also liable to frequent burns.

Constitutional causes are generally brought about by such diseases as effect alterations and degeneration either in the composition of the blood or in the vessels. These are chlorosis, anemia, syphilis, tuberculosis, arteriosclerosis, fatty, amyloid, and aneurysmal degenerations of arteries, thrombi, emboli, trichinosis, and malaria.

Effect of pressure exerted upon the stomach by the costal margins is claimed to induce anemia and atrophy of the mucosa, especially in the region of the smaller curvature. Habershon and Rasmussen have advanced this view, in explanation of the frequency of gastric ulcer in those whose occupations necessitate continual pressure on the stomach.

Influence of Age.—In order to determine from postmortem records the age at which gastric ulcer most frequently occurs, all cases in which only cicatrices are found should be excluded, because a cicatrix gives no evidence as to the age at which the ulcer existed. The best statistics on this subject are contained in Welch's article on Gastric Ulcer in Pepper's "System of Medicine," volume II, page 483. The statistics of Brinton, which are still cited in the last editions of Boas, Fleischer, Sidney Martin, Fleiner, Debove and Rémond, and others, include all cicatrices found at autopsies as open ulcers. The following table is given by Welch, representing the age in 607 cases of open ulcer, collected from hospital statistics:

Age, .	1–10	10–20	20–30	30–40	40–50	50–60	60–70	70–80	80–90	90–100	Over 100
No. of Cases,	1	32	119	107	114	108	84	35	6	0	1
Totals,	33		226		222		119		7		

From this table it is apparent that the largest number of cases is found between twenty and thirty. Three-fourths of the cases occur between twenty and sixty.

In 41,688 cases, constituting the clinical material in Zürich and Breslau between the years 1853 and 1873, 252 cases of gastric ulcer were diagnosed during life by Lebert; nearly seven-tenths were between twenty and forty years of age,—a preponderance sufficiently great to be of diagnostic value in the differentiation, as we shall see later from carcinoma, for in this disease the largest number of cases is found between fifty and sixty years. Goodhart has described a case of gastric ulcer in a child thirty hours old.

Influences of Sex.—Females are more frequently affected than males; the following are the figures given by various authors:

	Males.	*Females.*
Welch, ...	40 per cent.	60 per cent. in 1699 cases of gastric ulcer found at autopsy.
Brinton's ratio, . . .	1 male to every 2 females.	
Anderson, ...	3 males and 32 females in 35 cases.	
Habershon, ...	74 males and 127 females in 201 cases.	

The nursing period, puerperium, and menstruation are also claimed to increase the susceptibility to ulcer.

Geographical Distribution.—There seems to be an unequal geographical distribution of the disease, which seems to be more common in northern than in southern countries. It is less common in this country than in England and Germany, according to Da Costa, Keating, and Welch (*loc. cit.*, p. 485). The last-mentioned author found only six cases of gastric ulcer in 800 autopsies made by him in New York. In 444,564 deaths in New York City, from 1868 to 1882, ulcer of the stomach was assigned as the cause in only 410 cases. To these statistics little importance can be attached, because they are compiled from reports of practitioners of varying diagnostic skill, and concern a disease that presents many difficulties of recognition.

The Frequency of Gastric Ulcer.—We quote the following from Professor Welch's article (*loc. cit.*):

In 32,052 autopsies made in Prague, Berlin, Dresden, Erlangen, and Kiel, there were found 1522 cases of open ulcer or of cicatrix in the stomach. If all the scars be reckoned as healed ulcers, according to these statistics gastric ulcer, either cicatrized or open, is found in about five per cent. of persons dying from all causes.

It is important to note the relative frequency of open ulcers as compared with that of cicatrices. In 11,888 bodies examined in Prague, there were found 164, or 1.4 per cent., with open ulcers, and 373, or 3.1 per cent., with cicatrices. Here scars were found about two and one-fourth times as frequently as open ulcers. The observations of Grünfeld in Copenhagen show that when especial attention is given to searching for cicatrices in the stomach, they are found much more frequently than the figures here given would indicate. It would be a moderate estimate to place the ratio of cicatrices to open ulcers at three to one.

The statistics concerning the average frequency of open ulcers are much more exact and trustworthy than those relating to cicatrices. It may be considered reasonably certain that, at least in Europe, open gastric ulcers are found on the average in from one to two per cent. of persons dying from all causes.

It is manifestly impossible to form an accurate estimate of the frequency of gastric ulcer from the number of cases diagnosed as such during life, because the diagnosis is in many cases uncertain. Hence the importance of autopsy statistics.

Von Jaksch (cited by Bamberger, "Handbuch d. speciel. Path. u. Therap.," von Virchow, VI, 1. Abth., 280) states that 113 ulcers or cicatrices were found in 2330 autopsies, *i. e.*, 4.8 per cent. Orth gives five per cent.

Berthold's statistics from the "Charité," Berlin, from 1868 to 1882, give 294 cases,—2.7 per cent. ("Statist. Beiträge z. Kennt. d. chronischen Magengeschwürs," Sections-Protocoll d. Path. Inst., Berlin).

Nolte, München, 1876 to 1883, gives 3500 autopsies, with 43 ulcers, or 1.23 per cent. ("Häufigkeit d. Magengeschwürs in München," Dissert., 1883).

Berthold (cited from Ewald, "Diseases of the Stomach," p. 233) gives the percentage of ulcer of the stomach for Berlin as 2.7 per cent.; Nolte, for Munich, as 1.23; Gries, for Kiel, as 8.3; Stark, for Copenhagen, as 13 per cent. Von Sohlern ("Der Einfluss der Ernährung auf die Entstehung des Magengeschwürs," *Berlin. klin. Wochenschr.*, 1889, No. 14) has lately called attention to the fact that the Roen Mountains and the Bavarian Alps (Germany) and the greater part of Russia are nearly exempted from gastric ulcer. The diet upon which the inhabitants of these countries subsist consists largely of amylaceous and vegetable substances containing

a large percentage of potassium salts. The blood of persons living largely or exclusively on a vegetarian diet (Japanese) is very rich in potassium phosphate. According to von Sohlern, the exemption from gastric ulcer observed among these peoples is due to the large amount of potassium introduced in their food. We are not aware that von Sohlern has supported his theory by quantitative blood analyses.

Symptomatology.—The most characteristic subjective signs of gastric ulcer are localized pain, vomiting, hematemesis, disturbances of secretion, the presence of blood in the stools, and the state of the appetite. Frequently all these symptoms occur together; at other times only one or the other single symptom becomes prominent. There are cases of gastric ulcer that run a latent course, without any characteristic symptoms whatever.

We will begin with a consideration of the excessive secretion of HCl. This is a result of the irritation of the gastric nerves, either by the inflammation caused by the ulcer itself, or by irritation of exposed nerve-fibers, caused by the contents of the stomach. Although the secretory nerves of the stomach have as yet not been demonstrated as definitely as the secretory nerves of the salivary and lachrymal glands, we are justified in assuming their existence on pathological and clinical grounds. Just as the eye will overflow with tears until an offending foreign body has been removed, and just as the saliva will be secreted when the mucous membrane of the mouth is stimulated by food, or when there are ulcers or inflammations present in the buccal cavity, so in a similar manner the gastric mucosa will respond to irritation of its nerve-fibers by an augmented secretion. This irritation may be caused by the ulcer itself, exposing and corroding the terminal nerve-fibers.

The entire gastric nerve apparatus is placed in a state of increased excitability through the presence of an ulcer, and when food reaches the stomach the mucosa is stimulated with an intensity that is much greater than in the normal stomach. The percentage of HCl present varies from three to five per 1000; this strong gastric juice rapidly dissolves albuminous constituents of the food, while the carbohydrates remain undigested. Organic acids are absent. It may happen, in rare cases, that the peristalsis of the stomach is inhibited, causing retention of the food; in such cases the irritation of the nerves is kept up as long as food is present in the stomach, constituting continued hypersecretion. We have to distinguish in

these cases between two kinds of excess of gastric juice: (1) the *digestive hyperacidity*, which occurs when the motility is good, only during the normal presence of ingesta in the stomach; (2) the *continued hypersecretion*, which occurs as soon as the motility is impaired, when food is present at all times in the organ. With a continued hypersecretion the glandular cells gradually become exhausted; they eventually secrete a juice which is much poorer in HCl and pepsin than the normal product, just as the exhausted salivary gland-cells secrete a saliva which is very poor in ptyalin. The exhaustion of the gastric gland-cells may explain the observation referred to, where hyperacidity was absent in cases of undoubted gastric ulcer. The fact was, the glands had become so exhausted that it was impossible for them to form their characteristic product.

The pain of gastric ulcer is caused by irritation of the sensory nerves in the base of the corroded area. It occurs with great intensity immediately after the ingestion of food, and, as a rule, increases with the augmentation of acid during digestion. The pain during the digestive act is most probably caused also by the peristaltic movements, drawing upon and compressing the ulcer. External pressure will produce sharp pain in the locality of the ulcer. The pain is of a burning, stinging character, and in some cases it causes a spastic contraction of the sphincter of the pylorus reflexly—a reflex pylorospasm, which in itself may be very painful. Some patients complain frequently of a sore spot in the epigastrium. In cases of gastric ulcer associated with pylorospasm the pain radiates from the epigastrium toward the right and left, reaching the spinal column. Traube called attention to well-defined irradiations of the pain into the domain of other nerves outside of the stomach. Attacks of angina pectoris, intercostal neuralgias, and neuralgias in the left brachial plexus have been described by Brinton; sympathetic neuralgias in the arms and legs have been referred to by M. Müller.

The intercostal nerves of the left side may be in a more sensitive condition earlier than those of the right side; this may reveal itself by a hyperesthesia of the skin and soft parts in the lower left parts of the thorax, upper portions of the abdomen, and in the lumbar region. The slightest touch, the pressure of the clothes and bed-covers may be unpleasant to such patients.

Female patients can not wear a corset. Very frequently the pain

has a penetrating, lancinating character, shooting from the epigastrium straight through to the spinal column. The influence of the ingestion of food on the pain is very evident, although there are painful sensations when the stomach is empty; these sensations partake more of the nature of soreness and hunger. This may be momentarily relieved by the taking of food, only to become more severe by the stimulation and the hyperacid secretion that are set up by it. Liquid food may pass through the stomach without causing much annoyance, whereas solid food is always distressing. Very cold or very hot food invariably causes this gastralgia. The pain usually occurs within a half hour after ingestion. Should it not occur until an hour and a half to two hours after meals, this would justify the suspicion of an ulcer below the pylorus in the duodenum, whereas if the pain occurs at once, during the act of deglutition, an ulcer in the lower part of the esophagus should be suspected. Lying on the left side increases the pain (Leube), whereas absolute quiet and resting on the back relieves it.

Pyrosis.—There is in most cases a very annoying burning feeling in the left hypochondrium and epigastrium, frequently rising to the throat. Some patients locate it posterior to the sternum, or even between the shoulder-blades; this so-called "heart-burn" is caused by irritation of the stomach and esophagus by excessively acid gastric contents. If the burning is very marked in the esophagus we may presume that abnormal peristalsis of the stomach and insufficiency of the cardia are co-operative in bringing about the pyrosis.

The Condition of the Appetite.—In our experience the appetite is either normal or increased in the majority of cases. The instances where the appetite is positively lost are very rare. Before accepting a state of anorexia it is necessary to distinguish whether food is refused because the patients have no feeling of hunger, or whether they will not eat because they dread the pain caused thereby. Thirst is usually increased, and the tongue is clean.

Vomiting.—The irritation and the hyperacidity set up by the presence of the ulcer, cause increased peristalsis and antiperistalsis. The peristaltic unrest is accompanied by a feeling of boring undulation in the epigastrium. It may involve the intestine, causing gurgling, rumbling noises. The rapid evacuation of the stomach, caused by the intensified peristalsis, is rather favorable to

recovery, because it brings on a speedy return of the contracted state which favors approximation of the edges of the ulcer and healing. When the pylorus is tightly closed by spasmodic contraction, the food masses remain much longer in the stomach, and the mucosa is excessively irritated by the intensely acid contents. The stomach is then distended by the constant afflux of gastric juice and saliva; also by the aspiration of air, which occurs frequently in these conditions. The distention causes a drawing apart of the edges of the ulcer, pain, antiperistaltic movements, and eventually vomiting. Pylorospasm is a very grave accompaniment, since it gives rise to gastric hemorrhages and new erosions by the development of the conditions just described. The vomited matter generally shows a good digestion of proteids and imperfect digestion of carbohydrates.

Hematemesis.—This is probably the most characteristic sign of ulcer. It only occurs in about half the cases. Jaworski and Korczynski (*loc. cit.*) assert that the acidity is very much increased immediately before and after the hematemesis. This, of course, would explain the digestion of the blood and the conversion of oxyhemoglobin into hematin hydrochlorate. The amount of the vomited blood does not give a correct impression of the degree of the hemorrhage, because considerable quantities of the blood escaping into the stomach reach the intestine and are passed out in form of tarry stools. The intestinal evacuations may contain blood several days after the hematemesis.

The production of gastric hemorrhage is favored by bodily movement, but it may occur during rest, even during sleep. When very small quantities of blood escape into the stomach, they mix with the contents, are partially digested, and eventually come up in the form of coffee-ground material. When larger vessels are corroded by the ulcer we have copious hemorrhages of dark-red, pure blood. A profuse hemorrhage, therefore, as a rule, points to a deep ulcer. Gastric hemorrhages are accompanied by the systemic phenomena of internal hemorrhages in any other part of the body, such as sinking of arterial pressure, marked pallor, sensations of warmth and pain in the stomach, cardiac oppression, nausea, cold sweat, fainting, and collapse. Death has been known to occur in the state of collapse before any blood was vomited, the autopsy showing enormous quantities of liquid and coagulated blood. In one case a solid blood-clot filled the entire

stomach. Bodily exertions, the external application of force of any kind to the region of the stomach, and straining at stool have repeatedly been reported as the direct causes of gastric hemorrhage. In patients with persistent pain in the stomach, and dark-colored stools, the latter should be examined for blood-coloring matters by testing for the hemin crystals. Iron, bismuth, tannic acid, tea, claret, and huckleberries may produce a black color of the stool.

The state of the bowels is mostly that of persistent constipation; sometimes the evacuations are normal; this is generally the case when much water has been ingested to quench the intense thirst. The small quantity of the evacuations is explained by the fact that very little food is ingested, and this is so thoroughly dissolved in its proteid constituents by the very active gastric juice that little work remains for the intestine. Often the pylorospasm, the cicatricial contraction of the pylorus, and the frequent vomiting are agents in producing constipation, because they prevent the transit of the food into the duodenum. Colitis and membranous dysentery may co-exist with gastric ulcer in rare instances.

The urine is very much diminished in quantity, and is frequently highly acid when no emesis has occurred; but when there has been much vomiting, or when the stomach has been washed out frequently, the urine may become alkaline. Maly and Quincke have observed that the excretion of the alkaline constituents of the blood goes hand-in-hand with the increased acid secretion of the stomach; at the same time the total chlorids of the urine are very much reduced. The results of Chas. E. Simon (*loc. cit.*) indicate that exact analyses of the urine respecting its alkalinity, the subnormal amount of chlorids, and the excess of indican, etc., may eventually instruct us concerning the secretory processes in the stomach, where it is impossible to gain the gastric contents for examination. (For detailed information see chapter on The Urine in Gastric Diseases.)

Diagnostic Pain Points.—Of these there are two that are of importance: (*a*) the epigastric; (*b*) the dorsal. The epigastric pain point is in the median line, or slightly to the left, very rarely to the right of it. It can not be correctly called a point, because the pain is more or less diffuse, usually spreading over an area as large as the palm of the hand. The exact limitation of the epigastric painful area varies with the location of the stomach. In most

cases it is close to the xyphoid cartilage, but it may be several centimeters below that in cases of descent of the stomach, gastroptosis, dilatation, etc.

The epigastric pain, which is very sharply circumscribed and intense, may be associated with a sensation of throbbing and pulsation. The *dorsal* pain region, which was first described by Cruveilhier, is also sharply circumscribed. It was found in about one-third of our cases at a level with the tenth to the twelfth thoracic vertebra. Its lateral extent amounts to from two to three cm. and its vertical extent from two to five cm. In very rare instances there is a painful zone corresponding to the fourth or fifth thoracic vertebra. Usually the dorsal pain point is only present on the left side, and Boas mentions a case in which the dorsal pain was present and no epigastric pain complained of. The only gastric sensation this patient had was a feeling of pressure after the ingestion of food. There was no vomiting nor blood in the stools. Pressure on the epigastric region did not cause pain, but there was an intensely painful spot at the level of the twelfth dorsal vertebra. The patient later on suffered from severe hematemesis.

Diagnosis.—Wherever a chronic morbid process can be determined upon with accuracy and the characteristic pain points are

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DESCRIPTION OF PLATE VIII.—ULCUS CARCINOMATOSUM OF THE PYLORUS.

FIG. 1.—A SECTION THROUGH THE WALL OF THE STOMACH, SHOWING THE EDGE AND A PORTION OF THE BASE OF THE ULCER.

Objective, two-thirds; eyepiece, two inches. Stained with hematoxylin and eosin. The drawing is built up from a series of microscopic fields. × about 15 diameters.

d. Mucous membrane. m. Muscularis mucosæ. s. Submucosa. a. Base of the ulcer. mm. Muscle-coat of stomach. mc. Groups of cancer cells between the bundles of muscle-fibers. dc. Groups of cancer cells in the edge of the ulcer in the mucous membrane. sc. Groups of cancer cells in the submucosæ. a. Necrotic membrane lining the base of the ulcer.

FIG. 2.—A SMALL NODULE FROM THE SEROUS COAT OF THE STOMACH OVER THE BASE OF THE ULCER.

Objective, two-thirds; eyepiece, two inches. Stained with hematoxylin and eosin. × about 15 diameters.

This figure gives a good idea of one of the nodules in the serosa. It is composed entirely of a collection of groups and masses of packed cancer cells, so closely packed that the outlines of the individual cells can not be made out. Except for these nodular thickenings, the serosa was not altered. pc. Cancer masses in peritoneal coat.

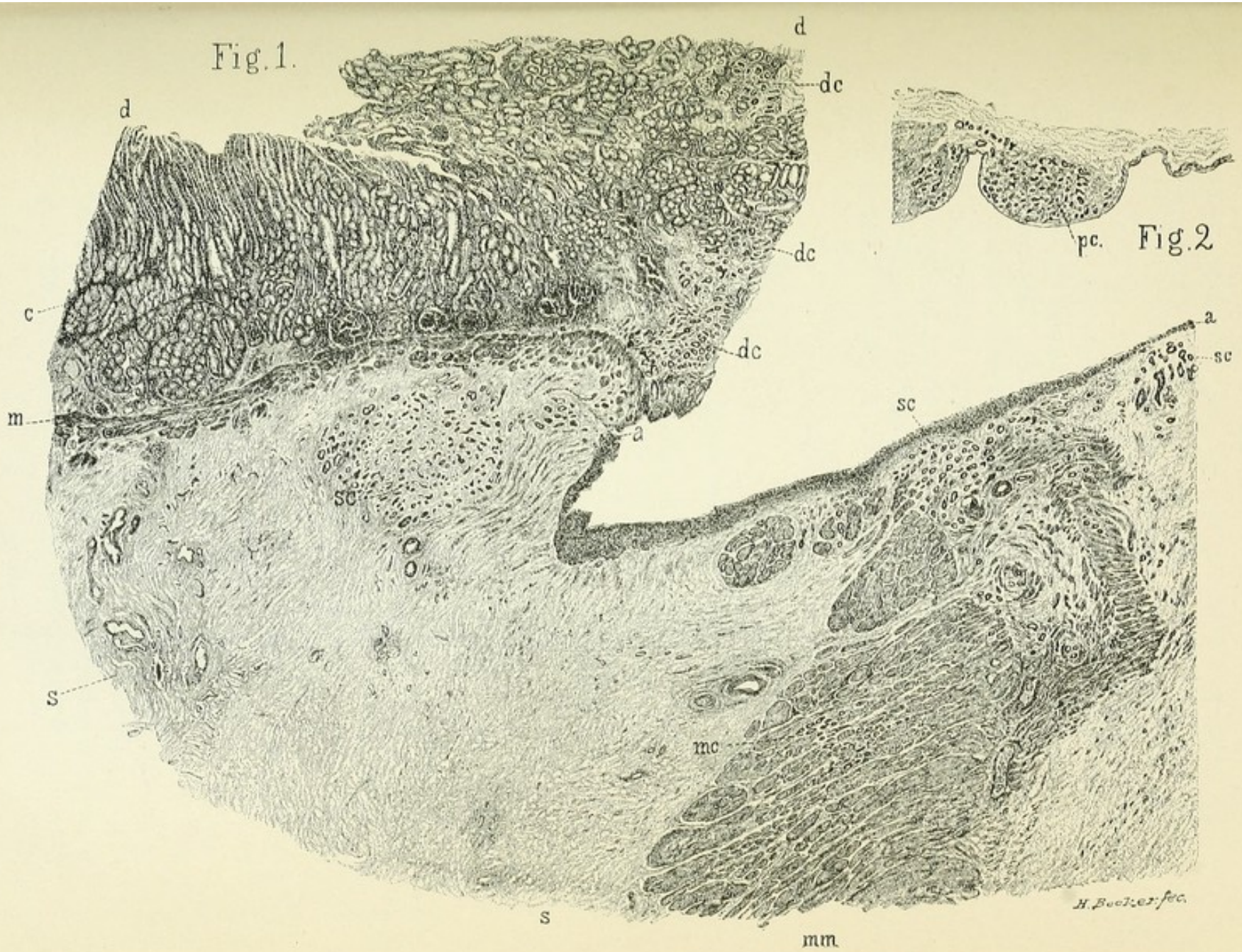

Fig. 2

PLATE VIII.

present at the same time, the diagnosis, according to Boas, should be certain (*loc. cit.*, p. 41). He attributes less importance to analysis of the gastric contents. There are, however, atypical forms which present some difficulty in diagnosis. Thus there are cases rarely observed in which the patients never complain of pain, nor has the food any distressing effect upon the stomach. In other cases, although pain is present, it is not aggravated by taking food. In some well-diagnosticated cases, food of all kinds was well borne. In all of these well-authenticated forms the diagnosis was assured by characteristic unmistakable symptoms, such as hematemesis and bloody stools, coming on afterward. Concerning hematemesis, it should be said that the differentiation of pulmonary from gastric hemorrhage may become necessary. The differentiation may be facilitated by a study of the subjoined scheme:

HEMORRHAGES FROM THE

Lung.	Stomach.
1. Blood is bright red, foamy.	1. Blood is dark brown, partly coagulated, frequently mixed with food, sometimes acid.
2. Physical signs point to a pulmonary or cardiac affection—the stomach may be affected secondarily.	2. Physical examination evinces a gastric or hepatic affection, or stasis in portal circulation.
3. Pulmonary hemorrhages followed by rusty-colored sputa for days (generally), but there is no blood in the stools.	3. Gastric hemorrhages are frequently associated with tar-colored stools.
4. Physical signs of pulmonary or cardiac disease—moist râles.	4. Physical examination of heart and lungs usually negative.

The diagnosis becomes complete if the characteristic pain points are present, with prompt aggravation of pain soon after taking food, vomiting showing hyperacidity, hematemesis, and a history of chronic trouble.

The blood coming from the stomach does not necessarily originate from an ulcer. One may, in rare instances, be called upon to exclude carcinoma, portal vein stasis producing passive congestion, gastric varicosities, toxic corrosions, traumatisms, scurvy, acute yellow atrophy of the liver, and yellow fever. The hemorrhages of carcinoma are small in quantity compared to those of ulcer, and in cancer the blood is more frequently decomposed and of a coffee- or chocolate-brown color, and there are rarely any bloody stools.

Charcot has reported hematemesis in hysteria (crises gastriques), but Debove suggests (*loc. cit.*) that organic and functional nervous diseases may be coincident with ulcer. In sudden gastric hemorrhages the previous history will, as a rule, enable one to distinguish between the above-mentioned possibilities. In hemorrhage from passive congestion due to stasis of the portal vein, the epigastric pain is very slight or entirely absent.

Cholelithiasis may be confounded with ulcer when there has been no blood in the vomit or stools, nor any grit, sand, or stones in the evacuations. The following signs and symptoms are then of value. The pain in hepatic colic is not in connection with the taking in of food; it draws from the median line to the right. The dorsal pain point of ulcer is located at the level of the twelfth thoracic vertebra, to the left and very close to the body of the twelfth vertebra. But the dorsal pain point of cholelithiasis is located to the right, about two to three fingers' breadths from the twelfth dorsal or first lumbar vertebra. In ulcer there is rarely any pain on the right side; even if there is, it is much less intense, and in cholelithiasis there is rarely any pain to the left of the spinal column.

In cholelithiasis the right lobe of the liver and the gall-bladder is enlarged after an attack; and during the intervals between the attacks all kinds of foods can be eaten with impunity. In cholelithiasis the amount of HCl in the gastric contents is normal or subnormal, or the contents may not show any free HCl; in ulcer there is hyperacidity. Icterus, when repeatedly observed, following attacks of pain, strengthens the diagnosis of cholelithiasis, but it must be emphasized that with duodenal ulcer icterus is occasionally observed. In private practice we have observed two cases in which cholelithiasis and gastric ulcer occurred contemporaneously.

Diagnosis of the Complications and Consequences of Gastric Ulcer.—These are: (1) The perforation peritonitis. (2) Cicatricial stenosis of the pylorus. (3) The transition of ulcer into carcinoma, or ulcus carcinomatosum. (4) Hour-glass stomach from cicatricial contractions. (5) Subphrenic abscess. (6) Progressive pernicious anemia.

The diagnostic signs of perforative peritonitis are: (*a*) great rigidity of the abdominal muscles, flat abdomen; (*b*) disappearance or diminution of liver dullness; this sign may be absent, how-

ever, if only liquid gastric contents and no air escape into the peritoneum; (*c*) vomiting.* According to Rosenheim (*Zeitschr. f. klin. Med.*, Bd. XVII, S. 116) about five to six per cent. of gastric ulcers develop carcinomata at their margins, and these carcinomata are said to be associated with a pronounced hyperacidity.

The so-called *hour-glass stomach* may be produced by one or more cicatrices in the neighborhood of the antrum pylori. Cicatrices of the duodenum may cause a dilatation beyond the pylorus, by which the latter will itself constitute the narrowing or isthmus of what very much resembles an hour-glass stomach (Reiche, "Jahrb. d. Hamburger Staatskrankenanstalt," 1890, p. 180).

Subphrenic Abscess (Pyopneumothorax subphrenicus).—In 1880, Leyden first described a combination of diseases which followed perforative peritonitis or escape of pus from the intestines into the peritoneum. A purulent exudate forms in the lower parts of the right or left thoracic cavity under symptoms of inflammation, but no coughing or expectoration is connected therewith. The posterior and lower thoracic regions give dullness on percussion, absence of vesicular murmur, and fremitus. Metallic sounds can be made out when one percusses and auscults simultaneously. The succussion sound is distinct. The lung is distinctly intact above these parts. The respiratory murmur is vesicular and the fremitus is maintained down to the fourth or fifth rib; from here on the respiratory murmur suddenly ceases. The dullness that corresponds to the exudate, changes with various positions of the body. The signs of equally distributed pressure in the pleura are wanting. The movements of the corresponding half of the thorax are not co-ordinated, the intercostal spaces are almost obliterated, and the heart is slightly pushed to the other side.

If the exudate is on the right side, the liver projects far into the abdomen, and can be felt at or below the umbilicus. The exudate may perforate into the respiratory passages and cause sudden and abundant expectoration of foamy pus containing hepatic cells. In

* The diagnosis of perforation has been attempted, when other signs failed, by puncturing through the abdominal walls with a sterilized hypodermic needle, when the gaseous, bacterial, and cellular evidences of perforation can be aspirated. (Test for H_2S by lead-acetate paper; when the abdomen is very tympanitic, this sign is almost pathognomonic.) The puncture is made when the patient is in the dorsal position. The escaped gases will rise upward between the intestines and the peritoneal wall. There is danger of puncturing the intestines in this method.

1894 Maydl collected 179 cases of subphrenic accumulations of pus. In 20 per cent. of these cases perforating ulcers of the stomach or duodenum were found to be the causes. ("Subphrenic Abscess," Meltzer in the *N. Y. Med. Jour.*, June 24, 1893. Progressive pernicious anemia as a concomitant phenomenon of ulcer can be recognized by the reduction of the number of red corpuscles and the appearance of the poikilocytes, microcytes, and macrocytes. (See chapter on The Blood in Gastric Diseases, p. 376 *et seq.*)

Treatment of Gastric Ulcer.—*Prophylactic.*—If gastralgias are frequent in a person afflicted with hyperacidity, the diet must be very mild and unirritating; two weeks of a milk diet will be the safest. Sudden deviations in the temperature of the food must be avoided, daily evacuations must be effected by suitable diet, and, if need be, Carlsbad salts, and the hyperacidity remedied. The dietetic and medicinal treatment will vary according to the presence or absence of hematemesis.

Treatment of Hematemesis and the Period Immediately Following It.—During the stages of blood vomiting the patient must remain absolutely quiet in bed and not even arise for urination or defecation. Positively nothing should be permitted by the mouth, not even ice. If the patient is well nourished no alimentation by the rectum is advisable, because this necessarily disturbs the rest and compels the stomach to move because of the changes in position required. If the patient is weak and anemic, a nourishing enema may be imperatively indicated every four hours. The enema most favored is that of Boas, consisting of 250 gm. of milk, the yolks of two eggs, a teaspoonful of salt, one ounce of good claret and one tablespoonful of aleuronat flour. Previously to giving an enema for nutritive purposes, the rectum and colon must be cleaned by a high irrigation with one liter of warm water. The above ingredients are thoroughly mixed by means of an egg-beater, warmed to about 99° F., and permitted to run in under gentle pressure, care being taken that the tube is introduced as far up into the sigmoid flexure as possible.

When the hematemesis is copious and persistent, a hypodermic injection of ergotol, 20 to 30 minims, should be given at once. With this preparation of ergot we have had extensive experimental and clinical experience (see *Med. News* for Jan. 31, Feb. 7, Mar. 7 and 14, 1891, "An Experimental and Clinical Study of Ergot," by J. C. Hemmeter). At the same time an ice-bag is placed over the epi-

gastrium, and if the pain is severe an injection of ¼ of a gr. of morphin should not be delayed, as by the ease and quiet it brings about this drug acts as an adjuvant to the hemostatic. For three days following hematemesis this treatment should not be changed, and no food allowed by mouth. The treatment from the fourth to the seventh day after, consists of absolute rest in bed, a wet pack covered with oiled silk and bandage being applied to the epigastrium. And now one may resume feeding by the mouth, but in form of liquids only,—half milk, half lime-water, or milk with a small addition of coffee or tea, never more than lukewarm; also beef-tea, to which lactose, meat powder, or somatose have been added, and egg-albumen water. Chocolate, yolks of eggs, and all alcoholic beverages must be forbidden in this stage.

In the second week after the hemorrhage a typical cure for ulcer, according to principles laid down by Wilson Fox ("Diseases of the Stomach," 1872, p. 146), v. Leube ("Ziemssen's Handbuch," Bd. VII, 2, p. 120), and v. Ziemssen (*Volkmann's Sammlung klin. Vorträge*, No. 75), should be instituted. These systematic treatments are in the main rest-cures combined with the daily use of a glass of Carlsbad Mühlbrunnen water, liquid or semiliquid diet, and hot applications to the epigastrium. Every morning the patient takes a glass of (40° R.) warm Mühlbrunnen in which five to ten gm. of natural or artificial Carlsbad salts have been dissolved. Spongiopilin cut into any requisite shape and dipped into hot water, is applied externally to the epigastrium, and renewed every three hours night or day. The diet consists mainly of milk and whipped eggs; if there is great weakness, the above enemata, containing perhaps two ounces of claret, should be given, and if the pulse is feeble, hypodermic injections of digitalin $\frac{1}{30}$ of a gr., and strychnia $\frac{1}{30}$ of a gr. In one case of profuse hematemesis we gave an intravenous injection of 500 c.c. of sterilized normal salt solution. The pulse had left the wrist, and was barely perceptible at the carotid; the effect was prompt, and the opinion of the assisting colleagues was that life was saved thereby,—the case recovering later on under the nitrate of silver treatment.

In the third week, when the pain in the epigastrium and general cardialgia have ceased, the patient may be permitted to rest on the sofa, and the Carlsbad water is continued. We might remark here that the Saratoga Carlsbad and the Hathorn spring waters act quite as well as the imported. In fact, the only objects of the Carlsbad

water in the cures of Leube and Ziemssen are the neutralization of the hyperacidity and the promotion of intestinal evacuation. One must not gain the impression that Carlsbad waters or salts have any direct or specific curative effect. Ewald (*loc. cit.*, p. 275) declares that many a patient who went to Carlsbad might have recovered more rapidly if he had taken the rest-cure at home. To neutralize the hyperacidity and prevent autodigestion we usually give the following:

℞. Magnesiæ ustæ,
Sodii carbonatis,
Potassii carbonatis, ... āā . . . . 5.0 ℥j + grs. xv
Sacchar. lactis, ... 25.0 ℥vj + grs. xx.
SIG.—Half a teaspoonful dry on the tongue every three hours.

In the third week one may permit dipped cakes, toast or Zwieback; broiled sweetbread or calf's brain, dumplings made of finely divided meat, broiled pike, bluefish, trout, oysters, in very small quantities. In the fourth week purées made of potatoes, peas, or beans rubbed through a sieve, stewed apples, pears, and plums. Saratoga Vichy may be allowed; all vegetables that can be prepared in purée (gruel) form, such as spinach, carrots, peas, etc., etc. For many years the patient must avoid raw fruits, all sour, acid, or spiced food and drink, ice cream and all cold and hot beverages. If there has been no hematemesis the treatment had best be carried out along these lines also. On pages 232, 233, and 235 detailed diet lists for cases of gastric ulcer are given. In rebellious cases of recurrent gastralgias, vomiting, and hyperacidity, McCall Anderson (*Brit. Med. Jour.*, 1890, May 10) and H. B. Donkin (*The Lancet*, Sept. 27, 1890) recommend a total abstinence cure of two to three weeks, during which the patients are fed exclusively by rectal enemata (three to four in the day); hot applications to the epigastrium are also used. After ten days of rectal feeding they cautiously and slowly return to feeding by the mouth (milk, bouillon, egg-albumen). We have tried this in a number of cases in which relapses had occurred after the rest-cure, and can speak in favor of the method. Gerhardt and Boas speak very favorably of nitrate of silver in light cases of gastric ulcer. The latter begins with: ℞. Argenti nitratis 0.25 to 120 of peppermint water; one tablespoonful three times a day on an empty stomach. Then the dose is increased to 0.3 to 120 of water, of which two bottles are taken, and finally 0.4 to 120 of water, of which also two

bottles are advised. This is combined with a sparing diet and as much rest as possible.

Fleiner and Kussmaul recommend bismuth subnitrate in all irritative conditions of the gastric mucosa—old ulcers, erosions, excoriating carcinomata. Fleiner employs it in the following manner: 10 to 20 gm. (150 to 300 grs.) of bismuth subnitrate are stirred in 200 c.c. of warm water; after the stomach has been thoroughly cleansed by lavage, this suspension is poured into the stomach and allowed to remain three minutes; then the clear water is siphoned out, the bismuth remaining behind and forming a coating to the injured places in the stomach. It is a modified direct or local treatment. We usually employ three drams of bismuth subnitrate and one dram of bismuth subgallate in a pint of warm water, having previously thoroughly cleansed the stomach with solutions of sodium bicarbonate (℥ss to a pint), the state of the ulcer permitting.

In chronic cases in which Fleiner's treatment can be employed it relieves pain promptly, reduces the hyperacidity, and promotes healing; it is worth trying in cases of long standing. Direct or local treatment of this kind is permissible when there have been no hemorrhages or tarry stools for one month. During this time the ordinary cures by diet, rest, Carlsbad Mühlbrunnen, etc., must have been employed. There must be no sensitiveness to pressure on the epigastrium. Chronic ulcers that have resisted dietetic and medicinal treatment, have been successfully treated by this method by Mathes (*loc. cit.*), O. Fischer (*loc. cit.*), and Stintzing (*loc. cit.*). The anemia following ulcer may require iron, arsenic, strychnin. Iron preparations must contain no acid.

Surgical treatment becomes necessary when, after a trial of the aforesaid methods, the ulcer or ulcers prove very obstinate and not amenable to medical treatment, or because hemorrhages may become so abundant and frequent as to endanger life, or, lastly, because of perforation. Nelson C. Dobson (*Bristol Medical and Surg. Jour.*, 1883) first advocated surgical interference for perforating gastric ulcer. In this country, Robert F. Weir, of New York, has contributed the most important work to this domain of surgery. His last important paper (Robt. F. Weir and E. M. Foote, "The Surgical Treatment of Round Ulcer of the Stomach and Its Sequelæ," etc., *Medical News*, April 25 and May 2, 1896) contains an account of 72 cases of laparotomy for acute perforation of gastric ulcer.

Gastric ulcers have been excised entirely, the sequelæ thereof have been removed by the severing of peritonitic adhesions, and hour-glass stomach much improved by gastro-anastomosis (see von Hacker, "Ueber Magenoperationen bei Carcinom u. b. narbigen Stenosen," published by Wilh. Braumüller, Wien and Leipzig, 1895).

For further details concerning the operations on the stomach for recent ulcers and for cicatrices, we refer to the sections on Surgery of the Stomach.

Treatment of Exhaustive Gastric Hemorrhage by Transfusion and Intravenous Injection of Normal Salt Solution.—Michel transfused successfully in a case of extreme anemia following gastrorrhagia (*Berl. klin. Wochenschr.*, 1870, No. 49). In a case of profuse and repeated hematemesis, which followed washing out the stomach, Michaelis infused into the veins 350 c.c. of solution of common salt. Reaction gradually followed, and the patient recovered. This case, which was one of probable ulcer, illustrates the advantages of infusing a small quantity (*ibid.*, June 23, 1884). The sudden infusion of quantities of liquid exceeding 500 c.c. will cause such an abrupt rise in arterial pressure that the injured blood-vessels in the gastric mucosa may reopen, causing renewed profuse hemorrhages. The dangers are illustrated by a case reported by von Hacker, who infused 1500 c.c. of salt solution into a patient in a state of extreme collapse resulting from hemorrhage from gastric ulcer. The patient rallied, but he died three hours after the infusion from renewed hemorrhage (*Wiener med. Wochenschr.*, 1883, No. 37). In Légroux's case of gastric ulcer, renewed hemorrhage and death followed the transfusion of only 80 gm. of blood (*Arch. Gen. de Méd.*, Nov., 1880). In a case quoted by Roussel, Leroy transfused 130 gm. of blood into a girl twenty years old, who lay at the point of death from repeated hemorrhages from a gastric ulcer. In the following night renewed hemorrhage and death occurred (*Gaz. des Hôp.*, Sept. 22, 1883). According to the experiments of Schwartz and Ott, the transfusion, or, rather, infusion, of physiological salt solution is as useful as that of blood, and it is simpler and unattended with some of the dangers of blood transfusion. The formula is chlorid of sodium, 6 parts; distilled water, 1000. Our personal experience is confirmatory of the observations of these last-mentioned experimenters.

Fleiner (*loc. cit.*) favors the excision of simple gastric ulcer, when

external (social) conditions render a suitable diet and treatment, impossible. We can not advocate this heroic treatment for simple uncomplicated ulcer, feeling convinced that the various treatments with which we are now acquainted are eminently successful. But if a laparotomy has been undertaken and the stomach has been opened for other indications (suspicion of peritonitis, perigastritis, carcinoma, perforation), and an uncomplicated ulcer is discovered, the excision of the latter is undoubtedly justifiable, and has been successfully carried out by Cordua, Känsche, Maurer (at Czerny's Clinic), and Mintz (*loc. cit.*). In the latter case the gastric functions were entirely recovered. Extreme and persistent gastric pain has been the indication for gastro-enterostomy in a case of Cahn's (*loc. cit.*).

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CHAPTER IV.

MALIGNANT TUMORS OF THE STOMACH.

(A) CARCINOMATA.

Pathology.—Orth distinguishes four forms:

1. The cylindrical cell, or adenocarcinoma.
2. The soft glandular, or medullary carcinoma.
3. The hard glandular carcinoma, or scirrhus.
4. The mucous, or colloid carcinoma.

In the section on the Surgical Operations on the Stomach, we have spoken of the relative frequency with which these various types of carcinoma attack this organ. It is an error for clinicians to speak of gastric cancer as if this were the only type of malignant neoplasm that can attack the stomach. Inasmuch as these various types show different rates of mortality after operation, and as they can occasionally be distinguished clinically by small bits of the new growth, which come up in the wash-water or are found caught in the eyes of the stomach-tube, it is essential that a brief pathological description of them should be given. The types which we have mentioned are not sharply distinguished from each other, but many gradations and transitions exist between them. The structure of a gastric malignant neoplasm is by no means a matter of indifference, both for the clinical history and the prospective surgical treatment. The scirrhus exhibits the most protracted course; the medullary (soft glandular) a disposition to disintegration and formation of metastases; while the colloid has a tendency to extend to the peritoneum, and rarely forms metastases.

The *cylindrical* cell, or *adenocarcinoma*, presents a soft, distinct prominence, or tumor, upon the surface of which smaller fungoid elevations develop, being attached to the fundamental tumor by broad or narrow bases, which give the surface a papillary appearance. In this case the tumor regularly has a red color, because each individual fungosity contains a small loop of blood-vessels. The little vessels in the outer, as well as those in the inner, sections of the neoplasm frequently show an irregular spindle-shaped or

spherical dilatation, so that this form of carcinoma has been called by Orth, *telangiectatic*. This condition of the blood-vessels may explain why, in this type of cancer, smaller or larger extravasations

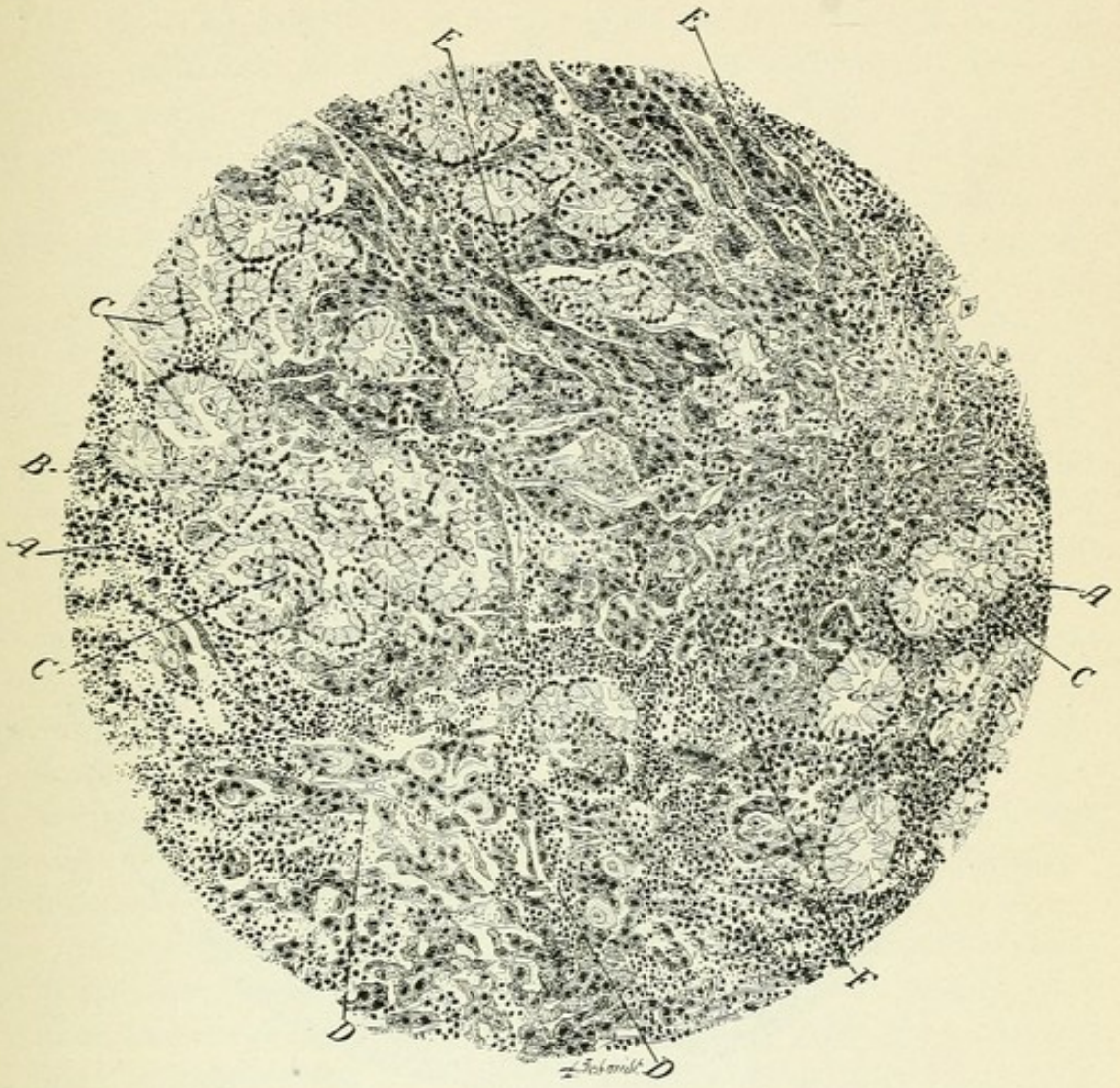

FIG. 35.—CANCEROUS INVASION OF THE GLANDULAR LAYER. A PORTION OF THE MUCOUS COAT.

Objective, one-sixth. Eyepiece, one inch. × about 320 diameters. Stained with hematoxylin and orange G.

This cut shows very well the small, round-cell infiltration between the cross-sections of the gastric tubules, with here and there the cells very much crowded, *A*, *A*.

The exfoliation of the cells lining some of the glandular acini is also shown in places, *B*.

At one or two places the proliferation of the epithelial cells that line the glands, with breaking of these glandular structures and the escape of some of the cells into the surrounding tissue, is seen, *C*, *C*, *C*.

The entire obliteration of some of the glandular structures by masses of cancer cells, *D*, *D*, which in many places are strung out for some distance, *E*, *E*, and in a few others have taken on the pseudo-glandular arrangement, *F*, is also well shown.

of blood are found on the surface, as well as in the parenchyma; and also accounts for the frequent effusion of blood into the cavity of the stomach. On section, the so-called "carcinoma juice" ap-

pears abundantly on the surface, and in this "juice" typical cylindrical cells are generally exclusively found in sections examined microscopically. Such sections present varying pictures, according to whether they are taken from the surface or from deeper

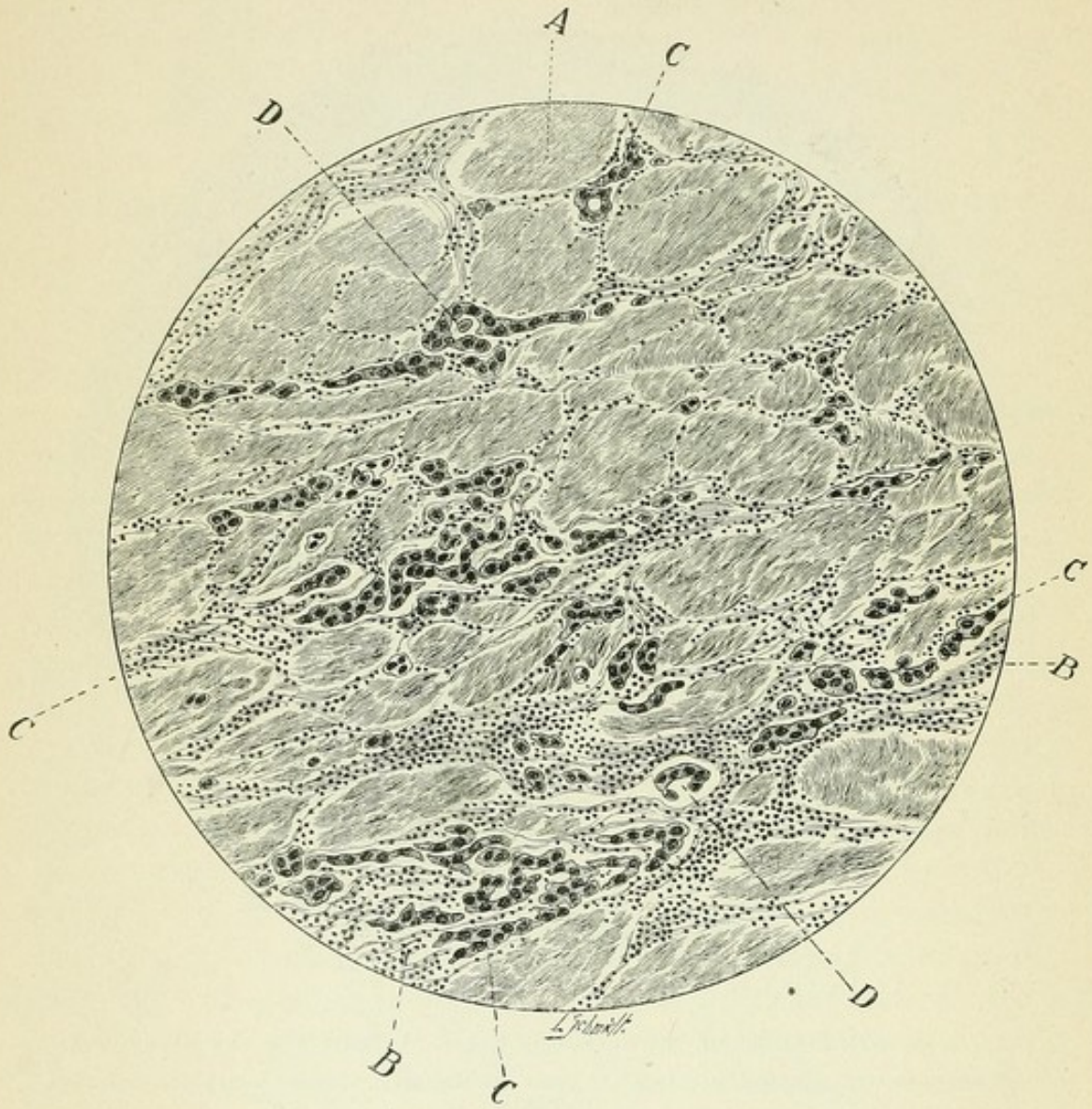

FIG. 36.—CANCEROUS INFILTRATION OF THE MUSCULARIS. SECTION OF A PORTION OF THE MUSCULAR COAT OF THE STOMACH.

Objective, one-sixth. Eyepiece, one inch. Stain, hematoxylin and orange G. × about 320 diameters.

Cross-section of bundles of muscle-fibers from the muscular coat are shown, *A*, between which there are a large number of small round cells, *B*, *B*, in places, and here and there large clumps of cancer cells, *C*, *C*, *C*, *C*, a few of which show the attempt at pseudoglandular formation, *D*, *D*, in some instances arranging themselves in complete circles, while in others only a portion of an acinus is formed.

regions of the neoplasm. On the surface the aspect closely resembles that of a papillary fibroma, but in the deeper regions a glandular structure becomes very distinct, for here cylindrical

cells may be seen lining tubular, hollow spaces in a regular manner, these tubular ducts being separated by connective tissue, which generally shows small-celled infiltration; nor are these glandular hollow spaces always regular in distribution or in size. The order of the lining cell is, in so far, a typical one, as the whole cavity is filled with cells of which only the outer ones are cylindrical and arranged in regular order, while the rest show very irregular relations, both in form and position. The cylindrical cell carcinoma is most frequently found in the pyloric region, its favorite place being close to the valve, and generally sharply limited toward the duodenum. It is probable that the neoplasm originates here from the pyloric glands, while at other locations of the stomach the surface epithelium and the cylindrical cells of the gland vestibules form the bases of origin. The papillary forms of these cancers particularly have a tendency to grow toward the surface, for they may last a long time; *i. e.*, the cancer mass may assume a considerable size before the infiltration will invade the outer layers of the stomach wall. The development of secondary carcinomata may also take a long time, so that with very large malignant neoplasms perhaps only one or a few lymph glands will be found secondarily involved. Finally, ulcerations of the surface may prolong development; or, if a loss of substance does occur, it may be compensated for by proliferation of the tumor tissue; eventually, however, with the co-operation of necrosis, a larger destruction occurs. The ulceration is usually surrounded by a projecting fungus-like wall.

Occasionally the ensuing necrosis—which presumably arises from disturbances in the circulation—may become so extensive that the entire tumor, with the exception of very few remnants, may be sloughed off, leaving behind an ulcerating base. The breakdown and destruction of the tumor mass frequently progresses in a gangrenous manner, and then we may find not only formations of cavities within the tumor, but the entire stomach walls may also be perforated, while large cancerous proliferations are still left close to the perforation.

The second main type, the soft *glandular*, or *medullary*, carcinoma, likewise forms knotty projections on the inner surface of the stomach, but it is very rare that these are observed intact; on the contrary, this type of cancer usually appears at the necropsy as a cancerous ulceration. Its form is quite characteristic. It presents a navel-like, deepened center and an external surrounding

wall which is formed by the mass of the tumor; it is either broad or narrow, high or low; at times it exhibits a uniform appearance; again, it is irregularly ragged in outline. In the bowl-shaped central depression the tumor mass is found breaking down in

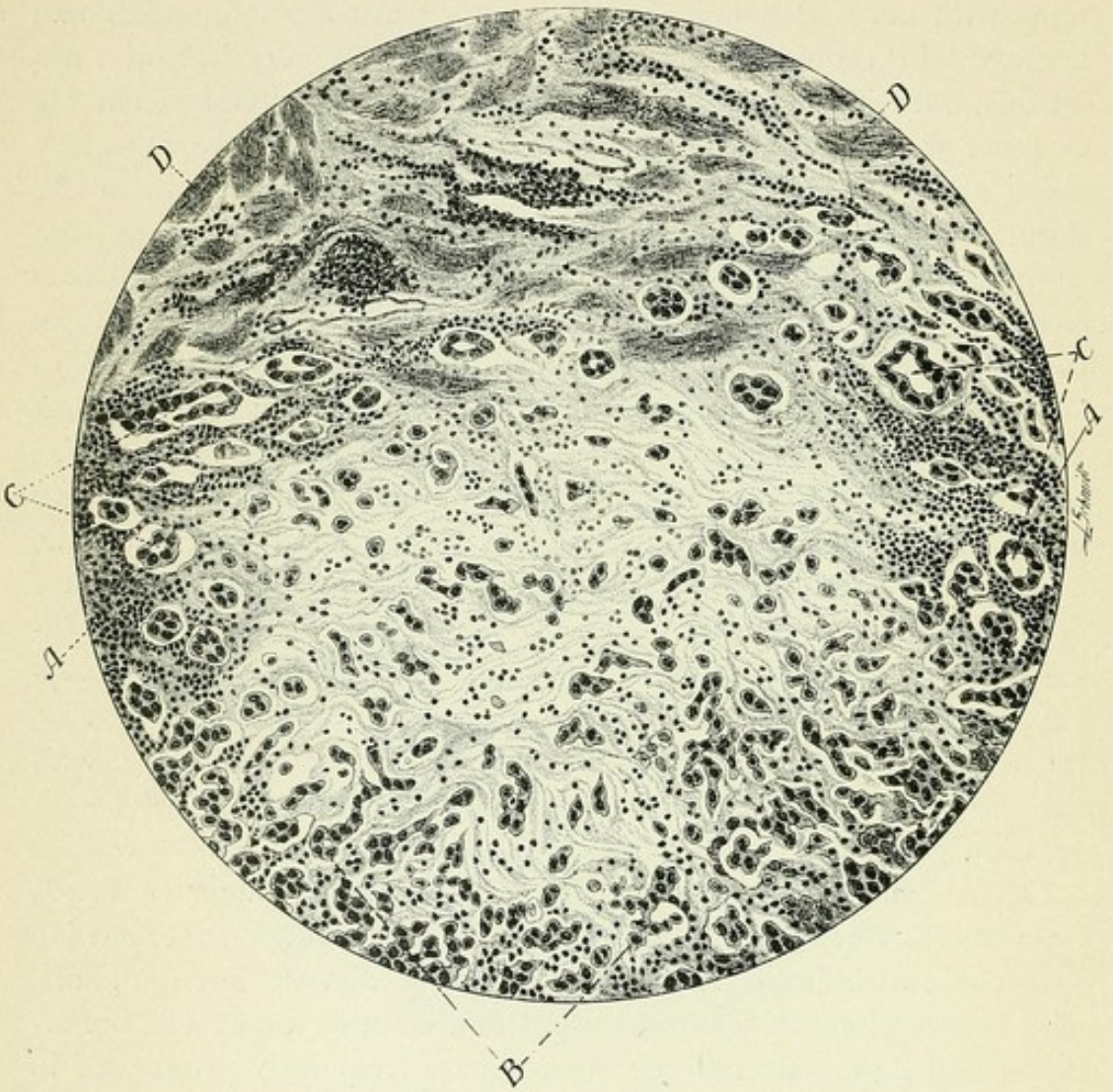

FIG. 37.—A PORTION OF AN AREA IN THE SUBMUCOSA, LARGELY COMPOSED OF GROUPS OF CANCER CELLS.

Objective, one-sixth. Eyepiece, one inch. Stained with hematoxylin and orange G. × about 320 diameters.

The fibrous tissue of the mucosa is infiltrated with many small round cells, which in some places are very numerous, *A*, *A*. The most prominent change appears in the numerous clumps of cancer cells, most all of which lie in open spaces in the tissue, *B*. These clumps are like those seen in other coats of the stomach, but the attempt at glandular formation is more marked here than in any other locality, *C*, *C*. In the upper part of the cut is seen the lower portion of the muscularis mucosæ, *D*, *D*, infiltrated with many small round cells, and containing a few of the masses of cancer cells.

fragments, or, occasionally, this depression may be found smooth, since the more resistant muscularis may have been exposed and is, presumably, destroyed much more slowly than the other layers by

the action of the gastric juice. In this tumor also the destruction may go on to complete perforation of the gastric wall. The masses of the tumor which surround the ulceration denote more or less extensive retrogressive metamorphoses, accompanied by hemorrhages, and, not rarely, an ichorous deterioration of the tumor mass. On microscopical examination of these masses, it is noticed that the

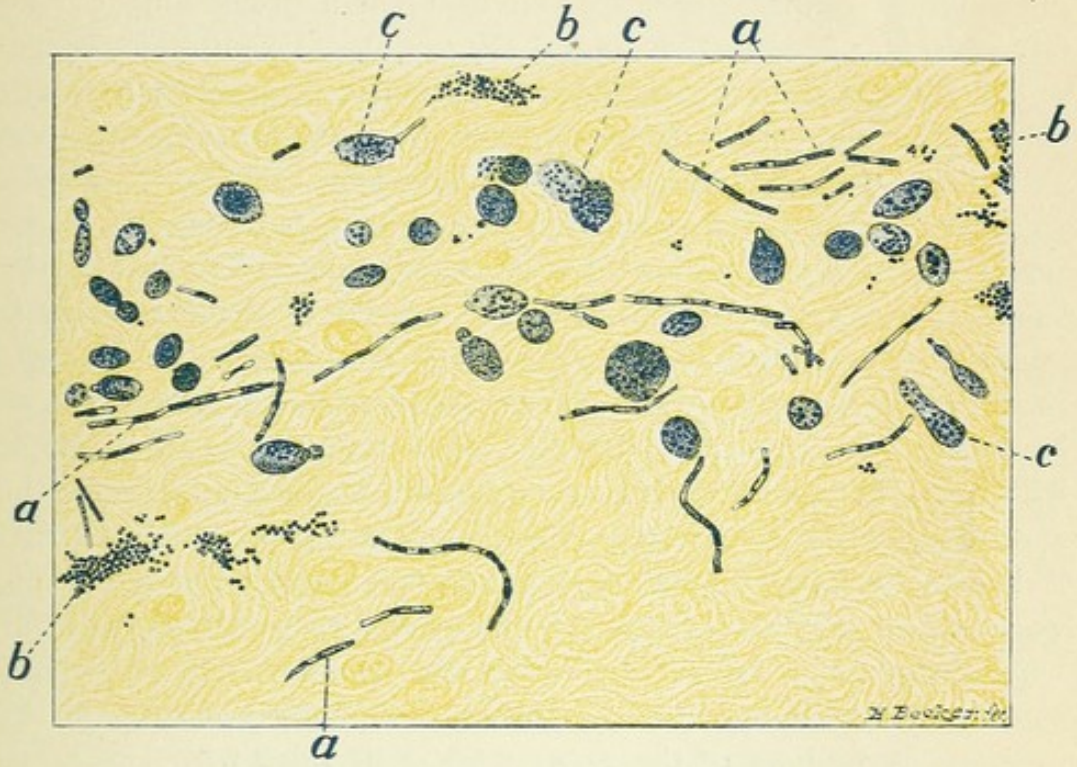

FIG. 38.—SECTION OF TISSUE NEAR THE BASE OF A CARCINOMATOUS ULCER, SHOWING MICRO-ORGANISMS.

Objective, one-twelfth. Eyepiece, one inch. Stained with methyl-violet, anilin-oil solution by Gram's method. × 1060 diameters.

a, a, a. The Oppler-Boas bacillus, singly and in chains. The peculiar base-ball bat shape is shown in some cases, while in others it is seen that one end of the rod is narrow and the other broad, the change in size being sudden. Some of the rods stain solidly, while in others there are clear spaces.

b, b, b. A micrococcus which occurs singly and in clumps, but never in chains. These, as well as the Oppler-Boas bacillus and the next organism to be described, were found both in the necrotic tissue over the base of the ulcer and in the healthy tissue below the same.

c, c, c. A peculiar yeast-like organism, that is probably some protozoan. It is much smaller than a yeast cell. Budding forms are seen, and the granular protoplasm in some, and the few large deeply staining dots in others, are well represented.

cancer cells are, as a rule, quite small and irregularly shaped, similar to oxyntic cells, but that their numbers far exceed the stroma, which in many places consists chiefly of very thin, delicate partitions. Larger supporting partitions of the stroma exist, of course, in addition to these, and in this latter type "small-celled infiltration" is regularly present. Microscopical examination evinces the

fact that the soft glandular cancer rapidly invades the exterior gastric layers, for small nodules (tumor knots) appear at the serosa at an early stage, which nodules distinctly show, and correspond to, the course of the lymphatic vessels. These nodules have arisen by a direct advance of the cancer into and through the muscular layer, in which, microscopically, a distinct thickening is observed, this thickening being dependent upon proliferation of the muscular substance itself, as well as upon a broadening of the intermuscular connective septa (Fig. 36). Examining microscopically a section through the muscularis, it at once becomes evident that the cancer masses, in their invasions between the muscular fibers, follow the septa which conduct the lymphatic vessels. In older cases, small foci are found in the muscle-bundles themselves, where they have forced apart the muscle cells to assume the shape of spindle-like spaces. As the growth of the carcinoma is much more restricted in the denser and closer netted muscularis than in the subserosa, the cancer masses outside of the muscular layer are generally considerably more voluminous than those within it. The (soft) medullary carcinoma may extend toward the surface as well as toward the interior, and it will then be seen that it habitually follows preformed passages—namely, the lymphatic vessels. In very rare cases this neoplasm may extend over the whole stomach, except, possibly, the fundus; and in such a case Orth has found that the entire lymphatic network of the mucosa, as well as of the submucosa, was filled with cancerous masses. The greater tendency of the medullary carcinoma for local dissemination corresponds to its relations toward the general organism. With this neoplasm particularly, one finds extensive lymphatic gland carcinomata, not only in the epigastric, celiac, portal, and retroperitoneal, but also very frequently in the left supraclavicular lymph-glands; one may find metastases in the lymph- as well as in the blood-channels, and, besides, a dissemination of cancerous nodules in the abdominal cavity.

Concerning the seat of medullary cancers, it may be said that they are not limited to any particular part of the stomach, for they may be found at the cardia, the anterior and posterior walls, and the lesser curvature as well as in the pyloric region, for which they have an unmistakable affinity. Frequently, cancers of the cardia extend to the esophagus, while the duodenum remains intact.

The *scirrhus* (meaning hard glandular) is distinguished from the

two preceding types of carcinoma mainly by its hardness. It produces no large tumor nodules, but rather simple thickenings of the entire wall. The surface of the mucosa shows, as a rule, a flat ulceration, which has either a smooth or an actually cicatricial basis, or else a papillomatous, irregular, and corroded appearance. The edges of the ulcerations are generally entirely flat, without a trace of the wall-like elevation, and for that reason the transition into the surrounding mucosa is very gradual. On cutting through a scirrhous gastric wall considerable resistance is met with, so that the tissue actually grates on cutting. Microscopical section reveals a thickening of all layers, particularly of the muscularis, by a grayish-white, striated, cicatricial connective tissue. Typical cancerous proliferation is not apparent on the mucosa nor in the remaining layers of the gastric wall, so that it may be doubtful whether one is dealing with a cancer or with a simple chronic ulcer. To decide this question one must observe the relation of other parts, and, particularly, search for secondary cancer formation.

As a matter of fact, one occasionally sees small, flat tumor nodules on the serosa directly over the neoplasm ; but as extensive adhesions of the pylorus with neighboring organs (liver, intestine, omentum) are invariably present with this form of cancer, implicating the peritoneum, such nodules are difficult to recognize, even if they are present. A more reliable sign is the condition of the lymphatic glands, which usually show cancer formation ; and, besides this, the liver and other organs, which are otherwise rarely invaded (for instance, the spinal column), may be found to contain it. Such cases easily give rise to deception, because these secondary tumors may show a medullary structure and attain considerable size, in which case the seemingly unimportant scirrhous ulceration in the stomach may be overlooked. The surest indication of the character of these changes is obtained from microscopical examination, though in a section-preparation hardly anything else but fibrous connective tissue is seen, particularly in the very much thickened muscularis, which is permeated with broad, grayish-white stripes. But when a larger number of preparations are carefully examined the histological peculiarities of carcinoma, the connective-tissue stroma, and carcinoma bodies are discovered.

The last-named are diminutive and consist of small-celled rows. The stroma is massive, and composed of tough, rigidly fibered connective tissue. The longitudinal direction of the small-cell rows is

parallel to the course of the fibers of the stroma. It is noteworthy that the secondary cancer nodules of scirrhus are richer in cells, and therefore more closely resemble the medullary carcinoma; and also that, alongside entirely fibrous places in the gastric wall, here and there at the edges of the ulcerations, places can be found where the cancer cells are not yet so scarce in proportion to the stroma, and where the latter does not as yet possess the characteristic callous consistency. It may, from this, be concluded that the scirrhus is, in fact, an atrophic cancer (*cancer atrophicans*); *i. e.*, that the callous formation represents nothing more than a later stage, or result, of the initial cancerous process. The question has arisen, whether complete healing may not be produced by a total callous metamorphosis of the neoplasm; but, up to the present time, no convincing observations confirmatory of this question have been made. It is reasonable to assume that a very localized callous cicatricial healing may be brought about in certain places, while in other portions (the very youngest parts of the neoplasm) very slow but gradual cancerous progress is made. The callous stroma of the scirrhus has a tendency to contract such formations.

This fact is of great significance, when the microscopical relation of the scirrhus is considered, for it explains the stenosis which it causes at the pylorus,—its almost exclusive locality. This constriction is further increased by the very much thickened and callous-like alteration of the gastric wall, which becomes unresistant and inelastic, resembling a hard, rigid ring or stiff tube. It is self-evident that a pylorus changed in this manner is no longer capable of closing off the stomach toward the duodenum (incontinence of the pylorus). The extent of the scirrhus from the pylorus toward the cardia may be variable, rare cases occurring in which the entire gastric wall is in a state of scirrhous degeneration. The entire organ is then, as a rule, considerably contracted, and at the same time the walls are very much thickened. On the inner surface little mucous membrane remains in these cases. We have seen elsewhere that a similar condition may be brought about by chronic inflammation (cirrhosis of the stomach). The differential diagnosis is very difficult to establish from the local conditions, but the majority of stomach contractions are to be attributed to scirrhus (Orth, *loc. cit.*).

The *colloid* carcinoma in typical cases has a very characteristic appearance. It does not produce circumscribed tumor masses so

much as diffuse thickenings of the entire wall similar to scirrhus. In this growth the stroma is not a bright fibrillar tissue, but a gelatinous, translucent, colorless or light-brown material. These masses are recognizable on the inner surface, which, as a rule, presents an extended flat ulceration. Where the tumor tissue lies exposed there appears a distinct, alveolar, grayish framework, which incloses the colloid granules, in dimension the size of a pinhead or a millet seed. The whole mass has a slimy, mucoid feeling, but it is not nearly as soft as is genuine mucus. Microscopically a similar picture obtains; for here, also, the connective-tissue alveolar framework, containing a transparent mucocolloid mass in its meshes, is prominent. This mass may be entirely devoid of cellular elements, but generally a number of cells and cell fragments are detected, in which it can be distinctly recognized that these cells themselves furnish the colloid material of the alveoli, for one frequently sees many cells in a swollen state, either with hyaline granules or in a condition of disintegration. In other places cells may be found in better preservation, while the colloid matter is not so pronounced, so that, in this form of carcinoma, just as in scirrhus, there are transitions to the medullary type. Here, likewise, the youngest portions of the growth are those most rich in cellularel ements, and production of colloid material is a phenomenon which occurs in the course of further development of the tumor.

"Colloid tissue" several centimeters thick may be found throughout the entire gastric wall, and here again the lymph-vessels offer the channels in which the cancerous masses take their course, and in which they ramify both interiorly and superficially. Occasionally, larger colloid tumor nodules may appear on the serosa; and, in fact, the colloid carcinoma not rarely invades the peritoneum and produces an extensive carcinosis—as a result of which the omentum is transformed into a short, thick, and board-like band. Affections of the lymphatic glands, liver, lungs, and other organs, are by no means absent. The colloid carcinomata also have their favorite location in the pyloric region, whence they may extend to the duodenum, and also to the liver, by direct continuity. The extension to the liver generally occurs after the formation of a previous adhesion. A transition to the esophagus from the cardia has likewise been observed. Although the colloid carcinoma produces no large prominent tumors it may extend far over the gastric

surface, and frequently takes in the entire wall, reducing the size of the stomach somewhat but not to such a degree as scirrhus. The wall is hard and immovable, the inner surface ulcerated, the outer coarsely granular from small and large cancerous nodules of the peritoneal covering.

Although the ulcerations of the colloid carcinoma may have considerable superficial extent, still a perforation rarely results, although the ulcerations may, at places, reach even to the peritoneum. The colloid tissue is not subject to rapid disintegration, hence new tumor masses may be formed in front of the basis of ulceration.

Structural Effects of Malignant Gastric Neoplasms.—The development of gastric carcinoma is accompanied by adhesions of the serosa with the pancreas, liver, the transverse colon, the anterior abdominal wall, and the omentum; and, at the same time, there occurs a callous hyperplasia of all connective tissue in the immediate neighborhood. The result is that the stomach, particularly the part most frequently affected (namely, the pylorus), becomes fixed, while in other rare cases such adhesions may not be formed, and the stomach is dislocated downward by the tumor masses, in which case the pylorus may extend as far as the symphysis pubes. Frequently the cancerous stomach exhibits changes of size and form. We may have diminutions in size accompanying the total degenerations of stenosing cardiac carcinomata, or, what is more common, dilatation accompanied by marked muscular hypertrophy. The dilatations originate from the obstruction of the passage through the pylorus, a pyloric stenosis existing. This may be caused by a variety of circumstances. Among the causes so operating may be mentioned, in the first place, a large tumor mass located in the pyloric orifice, acting like a cork or ball-valve; secondly, the rigidity which the walls undergo in scirrhus and in colloid carcinoma; finally, the effective contractions of the scirrhus, whereby a considerable resistance is offered against the advance of the gastric contents. These disturbances may be increased by a large variety of inflections and dislocations resulting from adhesions, as well as by the weight of the accumulating gastric contents. Incontinence of the pylorus may occur contemporaneously with stenosis, but it may also exist in a very severe degree without stenosis where the cancerous ulceration has destroyed more or less of the pyloric ring. These changes at the pylorus are important because they are very frequent, for, as is evident from what we have said concerning the

various types of carcinoma, the pyloric antrum is the most frequent seat of cancer formation. According to the statistics of William H. Welch ("A System of Practical Medicine by American Authors," edited by William Pepper, vol. II, page 561), the frequency of carcinomata occurring at the pyloric region is 60.8 per cent.; at the lesser curvature, 11.4 per cent.; at the cardia, 8 per cent.; at the posterior wall, 5.2 per cent.; the whole, or the greater part of the stomach, 4.7 per cent. According to Orth, 60 per cent. of all gastric cancers invade the pylorus; 20 per cent., the lesser curvature; 10 per cent., the cardia; and the rest, the remaining parts of the stomach. As gastric carcinoma makes up 35 to 40 per cent. of all carcinomata, the great importance of pyloric cancer can be appreciated. In the mid-regions of the stomach the cancers are limited to a portion of the circumference, but in the vicinity of the two openings they frequently occupy the entire circumference in a ring- or girdle-shaped manner.

The growth of carcinomata occurs partly through simple peripheral extension, partly through daughter-nodules which develop at some distance from the main tumor, but sooner or later coalesce with it. These nodules evidently lie underneath the mucosa, which may be movable over them; hence, it may be assumed that they have arisen through infection by way of the lymph-channels. The frequent occupation of lymphatic vessels by cancer masses in the neighborhood of larger nodules argues in favor of this view. Concerning the secondary infection of lymph-glands, it may be stated that, with gastric carcinomata, it often happens that glands are diseased which do not receive their lymph from the direction of the stomach; such glands are the retroperitoneal. It is possible that this is caused by the cancerous impermeability of glands located higher up, which compels a return of the lymph-current. Following the current of the lymph, it has been found, by Orth and others, that the thoracic duct may be infected. Possibly, the infection of the left supraclavicular lymph-glands occurs in connection with the transportation of cancer cells through the lymph of the thoracic duct. The lymph-vessels of the diaphragm may be entirely filled with cancerous masses, and may disseminate the elements of the disease to the pleural cavity, bronchial glands, and lungs. In 25 per cent. of all gastric cancers secondary nodules are contained in the liver, and are transported there by direct extension after "adhesive invasion" with the lymph-current from the porta

hepatica and transportation by the blood-stream, the latter mode being by far the more plausible. The metastases may occur through minute particles which are not retained emboli, whereas in other instances emboli can be demonstrated in the larger vessels, certifying that such emboli should originate from the stomach, because the gastric veins are roots of the portal vein, and cancerous invasion of the veins of the stomach is conceded. The spreading of gastric cancer to the esophagus, duodenum, spleen, pancreas, and intestines occurs by direct extension along the paths that are either normally present or newly formed pathologically. Participation of the peritoneum has its foundation in the direct extension of the carcinoma into the gastric serosa; when, however, the peritoneum is once invaded, the rest of it is not affected by continued simple extension of the growth, although this may occur with colloid carcinomata, but by dissemination, which means the falling of tiny particles into the peritoneal cavity (carcinoma seed, as it were) and their attachment in suitable places (at first in the deepest portions of the peritoneum, in the rectovesical and rectouterine pouch). It is evident that gravity is an element in the spreading of peritoneal carcinoma. The ulcerations of gastric cancers depend partly upon ichorous degeneration and suppuration and partly upon the digestive influence of the gastric juice, which occasionally causes perforation of the stomach. The vessels of the stomach and of the spleen may be affected by an inflammatory gangrenous ulceration, which may lead to dangerous hemorrhages, but these cases are infrequent. Cancerous ulcerations are very similar to the simple peptic ulcer, from which they may be distinguished only by the presence of the tumor wall, which, if absent, enhances the difficulty of distinction between the two. If, indeed, cancer masses are found in the surroundings of such an ulceration, the question may be asked, Has the ulceration arisen from a carcinoma, or has a simple gastric ulcer been secondarily affected by cancer transformation? Here the clinical history, as well as the examination and analysis of gastric contents, may give the desired information. Rosenheim has shown that normal or supernormal hydrochloric acid secretion persists in the carcinoma which has secondarily developed from an ulcer; but when the carcinoma is the primary growth, the hydrochloric acid is permanently absent at an early stage in the disease.

Anatomically, it may be stated that when the ulceration has a

regular bowl-shaped appearance, and is on every side surrounded by tumor masses, even where no cancer masses can be found in its base, the carcinomatous tumor was undoubtedly the primary, the ulceration the secondary, object; and, reversely, when a simple ulcer, accompanied by all typical peculiarities, presents a thickening only at one side, the latter tumor mass must be regarded as secondary and the ulcer as primary. The remaining gastric mucous membrane sometimes shows insignificant alterations, which agrees with clinical observations as to gastric cancers remaining latent. In other cases a pronounced chronic inflammation is present, particularly in the immediate vicinity of the tumor masses or ulcerations. A hypertrophy of the musculature is frequently apparent, which partly depends upon alterations in the mucosa and partly upon a pyloric stenosis, the latter being responsible for the condition of the gastric contents, because it provokes dilatation and its consequences. The loss of secretion and the admixture of blood with the gastric contents is directly traceable to the cancerous infiltration. The hemorrhages may arise by ulcerative disintegration, as well as from rupture of the small vessels in the villous cancer proliferations.

What is the source or basis of the primary development of cancer formation? According to prevalent views, all gastric carcinomata do not originate from the connective tissue of the submucosa, as was formerly believed, but from the mucosa, and particularly from the glandular, or surface, epithelium of the same, the cells of these carcinomata having great similarity to the various cells of the mucosa. All gastric carcinomata are therefore epithelial tumors. We owe to Waldeyer the first exact investigations concerning the beginning of cancer formation, which have been confirmed later by other researches. According to him, the process begins with an enlargement and hypertrophy of a group of ten or twelve glands, which, breaking through the muscularis mucosa, enter the submucosa. The cells of these gland-ducts react differently to staining reagents, being colored much deeper by them, and filling the lumen of the gland in an irregular manner. A further step is that the connective tissue of the mucosa, and particularly of the submucosa, undergoing a transformation into granulation-tissue, advances and is pushed up against the aggregations of epithelial cells, which are thus forced apart and inclosed in groups by the connective tissue, giving rise to the cancer

alveoli and cancer bodies (cancer cells). Accepting this as a general rule, the question arises, What causes this gland-group and the adjoining connective tissue to enter upon this abnormal growth? Cohnheim says that abnormal conditions of primitive germinal tissue are present here, a remnant of unused primitive cells from which the proliferation starts. This is a hypothesis which can admit of no proof, since after the proliferation has occurred it is impossible to obtain any knowledge of the condition of the locality that existed there prior to the proliferation. But even admitting Cohnheim's theory, we must ask, Why do these embryonic cells suddenly begin to grow after many years? There must evidently be some other incentives to growth.

There is undoubtedly some disposition toward the development of gastric cancers with advancing age. What the nature of this predisposition is we do not know. There seems to be no predisposition of sex, for both sexes are attacked with equal frequency. The pronounced tendency which the structures of the pylorus exhibit toward cancerous infection attracts attention to the mechanical relations there existing. Hauser has made some interesting observations on the development of cancers from simple peptic ulcers. He has shown that the gastric secretory glands at the edges of healing ulcers undergo a proliferation which may be augmented to a cancerous neoplasm, and he seeks the explanation for this process in an increased supply of nutritive material to the glands, and in a reduction of the resistance of the adjoining tissues in consequence of an ulcerative and cicatricial process.

As frequent as primary carcinomata are, just so rare are the secondary. Secondary cancers may arise in the stomach by direct extension from the immediate surroundings. In this manner a cancer might extend to the gastric walls from the pancreas, liver, and lymphatic glands. Clinically, the most important of the primary carcinomata is the esophageal, which, when it is located at the cardia, may invade the stomach. Reversely, the extension of gastric cancer into the esophagus is really more frequent. There is another kind of extension of esophageal, lingual, and facial carcinomata to the stomach, which is not transmitted by the lymph- or blood-channels but by a direct implantation of cancer cells upon the mucosa. Klebs was the first to report three of such cases, and Beck has investigated a case, concerning which he assumes, on the strength of his microscopical preparations, that the loosened parts of the esoph-

ageal cancer had fastened themselves in the gastric glands. The new nodules which were thus formed were flattened epithelium carcinomata, composed of the typical pavement-epithelium of the esophagus. This raises the interesting question whether the new tumors arise solely and exclusively from the implanted tumor cells, or whether these cells produce a kind of infection of the local cells upon which they fall, so that the latter are converted into pavement-epithelium cancer cells. Klebs assumes the latter view, but Beck leaves the question undecided. In the place occupied by the tumor that he investigated, no gastric cells were observable, and also no transition forms to pavement cells. The secondary cancers of the peritoneum, already described, arise in a similar manner, namely, by the falling of cancer particles into the peritoneal cavity. Reversely, it has been observed that an implantation carcinoma may arise upon the gastric serosa from a deeper portion of the abdominal cavity. Orth describes a case in which the inner mucous membrane of the pylorus showed a typical cylindrical cell carcinoma, while the serosa of the same viscus revealed a pronounced colloid nodule as large as a walnut, which could not have arisen in any other way except by implantation from a colloid carcinoma of the cecum. Another mode of secondary cancer formation is that of metastasis by way of the blood-vessels, these secondary neoplasms corresponding to the primary tumors in structure, and are recognizable, according to Grawitz, as secondary by their circumscribed character. These secondary forms are rare.

The theory of infection for the origin of gastric cancers would not explain the great variety of the histogenesis of the carcinoma. In accepting the existence of a "cancer-producing microbe," one would have to assume that this organism could produce a transformation of connective tissue into epithelium, or that it regularly produced proliferation only in one kind of tissue, namely, the epithelial. A pathogenic micro-organism with these qualities is unknown at the present time. In the formation of metastases, only the transported cancer cells keep on proliferating in the new organ, while the tissue of this organ either does not participate at all, or only to a small degree, in the formation of the new cancer nodule. In the transportation of tuberculous tissue, however, it is this tissue which breaks down, and the new tuberculous focus develops from the invasion of the transported tubercle bacilli, which not only cause a disease of the epithelium, but also of the

remaining tissues (connective tissue, bone, etc.) with which they come in contact. Transplantation of carcinoma into animals has very rarely succeeded, whereas inoculations of infectious diseases are generally successful. Another explanation of the development of cancer has been attempted in the so-called "irritation theory," which is based upon the susceptibility of the two openings of the stomach to greater irritation during digestion than other parts; these portions are consequently most frequently affected (60 to 70 per cent. of all cases); but satisfactory proof that this irritation may cause cancer *per se* is wanting. We have elsewhere stated the percentage of cancers occurring at various decads of life: According to the statistics of Welch, Brinton, and Lebert, three-fourths of all cancers occur from the fortieth to the seventieth year, and from the thirtieth to the seventieth year 95 per cent. of all gastric cancers manifest themselves. So far as we know, only one case of congenital cancer that was limited to the stomach has been reported (Wilkinson). There has been one case of congenital cancer combined with carcinoma of other organs (Widerhofer). We found in the literature on this subject a case of gastric cancer in a child five weeks old (Cullingworth). Three other cases in children somewhat older are reported by Scheffer. (The subject of the etiology of cancer is reviewed in an interesting article by Roswell Park, *N. Y. Med. Record*, volume LII, No. 1, July 3, 1897.)

Heredity.—It is generally accepted that the predisposition to cancer may be inherited. According to Fleischer, the life insurance companies in Germany have increased their premiums for candidates in whose family gastric carcinoma has been observed. (Napoleon I, his sister, and his father died of gastric carcinoma.)

Geographical Distribution.—The geographical distribution of gastric cancer is very irregular, for while it is very rare in some countries,—as in Turkey, Egypt, and the tropics,—it is said to be very frequent in Thüringen, in Suabia, in Normandy, and in Switzerland. The causes of this unequal distribution are unknown. In Egypt gastric cancers are unknown (Griesinger), but gastritis and enteritis are of common occurrence. This seems to show that a genetic relation between gastritis, enteritis, and carcinoma does not exist. According to Eichhorst and Haeberlin, two per cent. of all deaths in Switzerland are caused by gastric cancer. From mortuary statistics, Tanchou ("Rech. sur le Traitement Med. des Tumeurs du Sein," Paris, 1844) estimates the frequency of gastric

cancer as compared with that of all causes of death at 0.6 per cent.;* Virchow, at 1.9 per cent.; Wyss, at 2 per cent.; and D'Espine, at 2½ per cent. In 8468 autopsies, chiefly from English hospitals, Brinton found gastric cancer recorded in 1 per cent. of the cases. Gussenbauer and von Winniwarter found gastric cancer recorded in 1½ per cent. of the 61,287 autopsies in the Pathological Anatomical Institute of the Vienna University. From an analysis of 11,175 autopsies in Prague, Welch found gastric cancer in 3½ per cent. of the cases.

Welch has collected and analyzed, with reference to this point, the statistics of death from all causes in the city of New York for the fifteen years from 1868 to 1882 inclusive, and reported that of 444,564 deaths during this period, cancer of the stomach was assigned as the cause in 1548 cases and cancer of the liver in 867 cases. Some, at least, of these so-called cancers of the liver may be reckoned as gastric cancers. This would make the ratio of gastric cancer to all causes of death about 0.4 per cent., and nearly 1 per cent. (0.93 per cent.) if only the deaths from twenty years of age upward be taken, gastric cancer hardly ever occurring under that age. It is a fair presumption, also, that in New York not over 1 in 200 of the deaths occurring, at all ages and from all causes, is due to cancer of the stomach, and that about 1 in 100 of the deaths from twenty years of age upward is due to this cause.

The following table (by William H. Welch, *loc. cit.*) gives the age in 2038 cases of gastric cancer, obtained from trustworthy sources and arranged according to decads:

Age, . . .	10–20	20–30	30–40	40–50	50–60	60–70	70–80	80–90	90–100	Over 100
No. of Cases.	2	55	271	499	620	428	140	20	2	1
Per cent., .	0.1	2.7	13.3	24.5	30.4	21	6.85	1	0.1	0.05

From this analysis we may conclude that three-fourths of all gastric cancers occur between forty and seventy years. The absolutely largest number is found between fifty and sixty years, but, taking into consideration the number of those living, the liability to gastric cancer is as great between sixty and seventy years.

* Tanchous statistics are based upon an analysis of 382,851 deaths in the Department of the Seine (see Welch, *loc. cit.*, p. 532).

Nevertheless, the number of cases between thirty and forty years is considerable, and the occurrence of gastric cancer even between twenty and thirty is not so exceptional as is often represented, and is by no means to be ignored. The liability to gastric cancer seems to lessen after seventy years of age, but here the number of cases and the number of those living are so small that it is hazardous to draw positive conclusions.

Location.—The following table gives the situation of the tumor in 1300 cases of cancer of the stomach (from article by Wm. H. Welch, *loc. cit.*):

Pyloric Region.	Lesser Curvature.	Cardia.	Posterior Wall.	Whole or Greater Part of Stomach.	Multiple Tumors.	Greater Curvature.	Anterior Wall.	Fundus.
791	148	104	68	61	45	34	30	19
60.8 %	11.4 %	8 %	5.2 %	4.7 %	3.5 %	2.6 %	2.3 %	1.5 %

From this table it appears that three-fifths of all gastric cancers occupy the pyloric region; but it is not to be understood that in all of these cases the pylorus itself is involved. In four-fifths of the cases the comparatively small segment of the stomach represented by the cardia, the lesser curvature, and the pyloric region, is the part affected by gastric cancer. The lesser curvature and the anterior and posterior walls are involved more frequently than appears from the table, inasmuch as many cancers assigned to the pyloric region extend to these parts. The fundus is the least frequent seat of cancer. In the cases classified as involving the greater part of the stomach the fundus often escapes.

Frequency.—According to Jos. D. Bryant, of New York, malignant disease is on the increase in this country. The death-rate from cancer in New York City was 1.82 per cent. from 1874 to 1884, but from 1884 to 1894 the death-rate from cancer was 2.17 per cent. of the total mortality (Jos. D. Bryant, the "Wesley M. Carpenter Lecture," *New York Med. Journal*, May 18, 1895). Haeberlin (*Deutsches Archiv f. klin. Med.*, 1889, Heft 3 and 4) gives the percentage of cancer of the stomach from 1877 to 1886 as 4.1 per cent. This writer has called attention to the fact that in Switzerland also gastric cancer is on the increase; his figures, showing the death-rate from cancer of the stomach for 1000 inhabitants, are

the following: 1877, 0.61 per cent.; 1878, 0.66 per cent.; 1879, 0.72 per cent.; 1880, 0.77 per cent.; 1881, 0.85 per cent.; 1882, 0.87 per cent.; 1883, 0.85 per cent.; 1884, 0.84 per cent.; 1885, 0.90 per cent.; 1886, 0.99 per cent. In England the proportion of deaths from cancer to the total mortality rate was 1 in 129, in 1840. This had risen to 1 in 28 in 1880. The death-rate from cancer is now about four times as great in England as it was fifty years ago. The published figures of the Registrar General's report indicate that the mortality from cancer in the years from 1870 to 1890 has increased 53 per cent. in England. These facts are alarming and should stimulate the most diligent search for the cause of this disease.

(B) SARCOMATA.

The sarcomata of the stomach are also classified into two groups according to their origin; namely, primary and secondary. The latter are by far the more rare. A single exception to this rule is the lymphosarcoma. The primary sarcoma of the gastric wall may develop from any place within the organ, but the greater curvature seems to be preferred, at least by such development as that in which tumor nodules (myosarcoma and fibrosarcoma) are formed, and in which no extensive lateral infiltrations are met with.

There is a disposition on the part of some authors (H. Schlesinger, *Zeitschrift für klinische Medicin*, Bd. XXXII, Supplement-Heft, p. 179) to separate the lymphosarcomata, on account of their different anatomical relations, from the other sarcomata. The point of issue of the latter group is either the muscularis or the submucosa, the mucosa not being diseased primarily. It may, however, become injured in the further progress by the arching forward of the tumor toward the interior of the stomach, resulting in lesions of the mucosa; in a purely mechanical way, from pulling and stretching; or it may become ulcerated toward the inner gastric cavity. In some cases the tumor may arch toward the peritoneal cavity. In the center of the sarcomatous nodules, particularly in the center of myosarcomata, processes of softening and disintegration, even of a purulent nature, may occur and give rise to septic peritonitis. These tumors possess a spherical or an irregularly knotty form, and are attached by either a broad or narrow basis; they sometimes attain vast dimensions (Brodowski described a myosarcoma weighing 12 pounds). Metastases in

neighboring organs, particularly in the lymphatic glands, greatly modify the anatomical picture, duplicating as to appearances the original tumor. The tumors that have been observed so far are: Spindle-cell sarcoma (Hardy, Weissblum, Habershon, Tilger, Malvoz); angiosarcoma (Bruch); myosarcoma (Virchow, Kosinski, Kolisko, Brodowski); and fibrosarcoma (Tilger, Ewald, Dreyer). The majority of the round-celled sarcomata that have been described (Virchow, Cayley, Legg, Berry, Shaw, Drost, Rasch) are properly classed with the lymphosarcomata. W. Fleiner ("Lehrbuch der Krankheiten der Verdauungsorgane," 1. Theil, *vide* Magensarcom, S. 295 und 311) has clinically observed one case of lymphosarcoma and one case of round-celled sarcoma, and made histological studies of the same from the autopsies; and H. Schlesinger has given the clinical history and undertaken the histological study of two cases of lymphosarcoma and one of round-celled sarcoma of the stomach (*loc. cit.*).

The primary lymphosarcomata of the stomach seem to be rarer than the secondary form. In some cases the infiltration is limited mostly to the pyloric region, causing a rigid thickening of this part (Török). In other cases it occurs in enormous infiltrations extending over the whole stomach, giving to the inner surface the appearance of a coarse swelling; it may also have the appearance of a uniform infiltration. The mucous membrane may be preserved for a long time in lymphosarcomata, but ulceration is not impossible. The spreading of the disease, as is usual with a lymphosarcoma, occurs, by the lymphatic channels, those lymphatic glands that are nearest becoming diseased first, then the adjacent organs, and, lastly, the peritoneum. Sometimes no metastases occur, as in one case of Fleiner's; more frequently lymphosarcomata are found as secondary neoplasms in the stomach after primary tumors in other organs. In the cases of Kundrat ("Ueber Lymphosarcomatose," *Wien. klin. Wochenschr.*, 1893, No. 12) the original infected areas of origin were the neck, pharynx, gums, and even the rectum.

According to other authors (Beck and Schepelern), the cecum, and, in another case, of Maier's, the retroperitoneal lymph-glands, were the points of origin. The gross anatomical picture is that of infiltrations in single folds in the form of thick plump wheals, or in the form of swellings as thick as the thumb, which ulcerate or even cicatrize, yet seem to spare the mucosa for a long time. The apparently normal parts of the stomach which lie remote from the neo-

plasm may show a diffuse, uniform, round-celled infiltration (Schlagenhaufer and Redtenbacher), limited to the mucosa and submucosa, the glands themselves being compressed, but yet distinctly preserved. In a case of Maass' the tumor had developed in the submucosa, the secretory layer was atrophied, and the glands were strongly infiltrated with small round cells. The secondary sarcomata of a non-lymphosarcomatous type are, as a rule, only found accidentally at the autopsies, since they develop only at a late stage of a universal sarcomatosis, when the signs and symptoms are totally obscured by others that are more severe, and because the nodules have acquired no considerable size. There are no etiological factors of importance known. The growth is comparatively rapid and vigorous, and Schlesinger could determine, by palpation, extreme increase of size within a few weeks. Primary lymphosarcomata may occur at any age. Statistics thus far are too limited to decide whether or not sex is an etiological factor of importance. *Clinically*, it may be said that the beginning of the disease is a very slow and insidious one, and, in many cases, not marked by prominent gastric symptoms. In other cases, anorexia, a feeling of fullness, pressure, extension, sour eructations, and a decomposed taste in the mouth, together with progressive emaciation, attract the attention of the patient to his condition. In many cases of secondary sarcoma no complaint referable to the stomach is made. The appetite may be good for a long time; hemorrhages may be absent in the incipient stages because the neoplasm has not yet taken on ulceration. Gradually the symptoms become severer, the anorexia becomes pronounced, and vomiting is more frequent; the vomit smells sour and is decomposed, and occasionally has the appearance of coffee grounds. Admixtures of a coffee-ground nature were present in the stools in all of Schlesinger's cases, which were mostly accompanied by diarrhea. Sometimes a neoplasm can not be demonstrated even in the later stages; but when a tumor is palpable, it is painful to pressure and behaves similar to carcinomatous gastric tumors regarding motility. Dilatation may exist, according to Kundrat and Schlesinger, without stenosis of the pylorus, and a general tetany may eventually result exactly as in other cases of pyloric stenosis. If the lymphosarcoma is located at the pylorus, it is distinctly palpable and causes secondary dilatation with motor insufficiency. Fleiner, Schlesinger, Dreyer, Maass, and Hammerschlag, found absence of HCl and presence of lactic acid, with

enormous numbers of yeast and other bacteria but no sarcinæ. H. Schlesinger, in two of his cases (*loc. cit.*), found the long, immotile bacilli, which were pronounced by Kauffmann and W. Schlesinger to be characteristic of lactic acid fermentation (see illustration, p. 119). There seems to be a constant enlargement of the spleen with primary as well as with secondary sarcomata and lymphosarcomata. This is a rare occurrence with the carcinomata, and might therefore be of value in a differential diagnosis. Von Leube cautions against making a diagnosis of gastric sarcoma. In one case of universal sarcomatosis of the skin he found a genuine epithelial carcinoma in the stomach.

From what has been said, it is evident that no important clinical factors can be drawn from the local signs and symptoms, nor from the general condition of the blood, urine, and the general state of health, for the establishment of differential diagnosis between carcinoma and sarcoma of the stomach. Regarding the prognosis and treatment, therefore, we refer to these headings as they are considered under carcinoma of the stomach.

Symptomatology.—What is said here on symptomatology has reference to all neoplasms of the stomach. In a small number of cases the development of malignant growth of the stomach remains entirely latent, because every typical gastric symptom is wanting until death. As far as we have had occasion to observe, the first symptoms of a gastric carcinoma are those of chronic gastritis.

Most of these patients state that, up to the time of their disease, they enjoyed a very good, sound stomach. The first dyspeptic complaints are those of pressure and fullness in the gastric region, eructations, anorexia, nausea, vomiting, cardialgia, anomalies in the secretion, absorption, motility, and coated tongue. Disturbances of sensibility are not felt until the neoplasm has reached a certain size, thereby exerting pressure on the sensory nerves of the stomach; or when, by its ulcerations, or by the irritation of the digestive juices, these nerves have been exposed. The patient has a sensation as if a stone were lying in his stomach, sometimes complaining of unpleasant feelings of emptiness, which come both a varying time after eating as well as on an empty stomach, and not rarely continue an entire day, so that the patient is constantly reminded of his stomach, which he was not aware of formerly, and is placed in that characteristic despondent and melancholy mood frequently met with in gastric sufferers. These sen-

sations may increase to actual pain, which, however, are not so severe as in ulcer. Eructations are present in the beginning of gastric ulcer as well as later on, either bringing up air or small particles of the gastric contents, which have a bitter taste; but later on, when, in consequence of achylia, the stagnating gastric ingesta ferment more and more, the eructated gas may have a disgusting, decomposed odor and taste.

Pyrosis may be present, and is, as a rule, accompanied by excess of organic acids. Singultus occasionally accompanies the eructations, and is observed most frequently with carcinoma of the cardia. In rare cases (Ebstein and Eichhorst) there may be a tetany of the constrictor muscles of the pharynx, which is said to be caused reflexly from the stomach, and may prevent ingestion of food. The frequency of nausea and vomiting depends upon the location of the tumor in the stomach; they are never absent when the neoplasm is located at the cardia or pylorus. The nature and chemical condition of the vomited matter depend upon the time of the emesis and the degree of secretory disturbance as well as upon the extent of the gastritis, and may consist of more or less altered ingesta, decomposed food remnants, mucus, blood, or bile. Advanced decomposition is not observed until a late period of the growth. The vomited masses are rich in bacteria and contain the Oppler-Boas bacilli, which are characteristic of lactic acid fermentation, though not pathognomonic of cancer (p. 119). The tongue nearly always has a brownish-yellow or grayish-white coating, but may be quite clean after profuse vomiting. The taste is said to be pasty, bitter, or offensive in the last stages, and salivation and thirst are increased. The loss of appetite belongs to the earliest symptoms, with particular dislike for meat at all stages of the disease. In some patients complete anorexia alternates with bulimia. As a rule, appetite remains fair as long as there is any gastric secretion, or as long as the motility remains fairly good.

Disturbances of Peristalsis, Secretion, and Absorption.—These are caused by chronic gastritis, anemia, or direct extension of the neoplasm into the mucosa and muscularis of the stomach. The disturbances of motility are either due to destruction of the muscularis or invasion of the gastric neoplasm, or to stenosis at the pylorus. We do not believe that they are traceable to the induced gastritis, because in chronic gastritis, according to very careful observations, the motility in the majority of cases is not very seriously inter-

fered with. Accordingly, we find that in three to four hours after the test-breakfast, or eight to ten hours after a full test-dinner, when the stomach normally should be empty, an abundance of food remnants is contained in it. In cases where the neoplasm is not located at either orifice of the stomach, the motility remains good for a long time, and even in the absence of secretion of HCl, the vicarious digestion of the intestines is sufficient to make up for the loss of gastric digestion and to avoid emaciation.

If it is desired to test motility in gastric carcinoma by means of our method,—that is, by the "deglutable india-rubber stomach-shaped bag,"—it is wise not to distend the bag too much; in fact, in cases of advanced cancer, the use of any intragastric instrument except the stomach-tube for this purpose is unjustifiable, because, in our experience, the stomach-tube, with the help of previous test-meals, has been found perfectly sufficient to ascertain the condition of the motility in this disease. The loss of secretion in gastric cancer was first discovered by von der Velden (*Deutsch. Arch. f. klin. Med.*, Bd. XXIII, p. 369, 1879). The great diagnostic value of the absence of free HCl in the gastric contents in malignant neoplasm is to-day universally admitted. There are other diseases (atrophic gastritis and achylia gastrica) in which free HCl is wanting, but it is one of the most constant symptoms of gastric cancer. Only in the carcinoma that arises from an ulcer do we find HCl present, and this is, in our opinion, explained by the fact that ulcus carcinomatosum is, in the great majority of cases, reported a very localized affection with little or no disseminating infiltration. We should not conclude our study of the secretory function by merely testing for free HCl. In all critical cases an artificial digestion experiment should be made with egg-albumen discs, as described in the first part of this work, and the amount of the HCl deficit determined. The Jaworski method should not be neglected in testing for the gastric ferments. About 200 c.c. of a five per 1000 solution of HCl are poured into the stomach, after it has been previously cleansed, and a quantity redrawn in twenty minutes; if this does not digest egg-albumen after a further addition of HCl, and no rennin-zymogen is contained in it, then the glandular activity is completely extinguished. The state of the resorption is very much reduced, according to Eichhorst, Zweifel, Wolff, and others. Capsules of three to five grs. of iodid of potash, which should give the iodin reaction in the saliva fifteen

minutes after they are swallowed, do not, as a rule, give this reaction before one hour to one and a half hours have expired.

The effects of gastric carcinoma on the blood can be easily recognized by blood examinations and staining of blood preparations for the microscope, as described by Dr. Whitney in the section devoted to this matter. With the progressive cachexia, the color of the face and of the external mucous membrane becomes pale or yellowish. There is persistent insomnia, and, as a consequence of the hydremia and the impoverished condition, the vessel walls become more permeable, producing the frequent edemas at the ankles and other dependent parts which sometimes invoke suspicion of nephritis. The body weight may be reduced 30 to 40 pounds in from one to two months. The urine is greatly diminished in quantity, concentrated, and highly colored, as a result of the impaired absorption, frequent emesis, and edemas. The reaction is neutral or even alkaline, particularly if, by methodical lavage, the acids and acid salts have been largely removed. If constipation exists, an excess of indican is found in the urine; this is conveniently tested for by adding to ten c.c. of the urine an equal amount of concentrated hydrochloric acid and one c.c. of chloroform. One drop of calcium hypochlorite solution is then added, and the mixture shaken. During the ensuing oxidation the indigo which is formed is dissolved in the chloroform; a strong bluish discoloration is a pathological indication. If the urine is highly colored, or contains much albumin, it is advantageously cleared of this product by treatment with plumbic acetate, and the filtrate used as just stated.

Von Jaksch demonstrated the presence of acetone in the urine, and Maixner discovered peptone in the urine of 12 cases he examined, and his results were confirmed by Parganowski. Maixner attributes the peptonuria to impairment of the ability of the gastric membrane to change peptone back into albumin, while Parganowski believes that the formation of the peptone takes place in the disintegrating cancerous tissue. The urine also gives a Burgundy-red color with chlorid of iron, which is probably due to diacetic acid. The feces frequently contain undigested muscle-fibers and egg-albumen taken in the food. There is usually very pronounced constipation, which is caused by two principal factors, namely, the greater amount of work that the intestine is called upon to perform and the impaired peristalsis.

Special Symptoms.—*Hematemesis.*—The hemorrhages from gastric carcinoma are, as a rule, not abundant, although they are quite frequent. They are most frequently observed in carcinoma of the pylorus and of the lesser curvature. As hemorrhages which are not copious do not easily lead to emesis, there is sufficient time for changing of the blood pigments into hematin, which is uniformly mixed with the gastric contents, so that when they are eventually vomited they present the appearance of coffee grounds or dark chocolate, which is, according to Brinton, a vomit that is observed in about 42 per cent. of the cases, and is, therefore, an important sign in gastric cancer. This kind of vomit may also occur in chronic passive congestion of the stomach and in ulcer. In chronic gastritis one rarely meets with coffee-ground vomit, but the ingesta show simply streaks or points of blood. If the coffee-ground admixture in vomit occurs frequently or daily for weeks, then it becomes an important pathognomonic symptom of gastric malignant neoplasm.

The Occurrence and Determination of Tumor.—In all cases of suspected carcinoma of the stomach the examining physician should carefully and systematically go through the routine of inspection, palpation, percussion, and artificial distention of the stomach by air or gas. When an abdominal tumor is formed by the dilated stomach itself, the diagnosis, in the majority of cases, can be made by a simple inspection. To determine the presence of peristalsis in dilated stomachs, rarely anything else than close inspection is necessary.

There are two conditions in which the stomach itself may form a palpable tumor by chronic contraction: one is where the organ shrinks in consequence of occlusion of the esophagus, and can be felt as a narrow firm ridge lying below the left lobe of the liver; the other condition is known as *cirrhosis ventriculi*, and is the result of chronic hyperplasia of the walls, with subsequent contraction of the lumen. In very rare instances it might be caused by diffuse carcinomatous infiltration. The infiltrating scirrhous carcinoma of the stomach, even at the autopsy, may not be distinguishable microscopically from cirrhosis of the organ. In this instance we are concerned most directly with the nodular and massive tumors of the stomach.

As three-fifths of all tumors occupy the pyloric region, and as the normal pylorus is palpable through the abdominal walls, these

tumors should not escape diagnostic palpation. For, when the stomach is filled with food or water, distended by gas, or in a state of atony, the pylorus is always below the edge of the liver. In some cases where the abdominal walls are not too thick, it is possible to feel and recognize the normal pylorus as a small, transversely placed ridge, which varies its position with respiration. Personally, we have frequently been able to grasp the normal pylorus between the fingers of the left hand, and, by massage and compression of the fundus by the right hand, we have been able to recognize the escape of air and liquid ingesta through the pyloric ring. The detection of the location of the pylorus is easier in dyspeptic patients than in the normal state, because in cases in which it becomes important to recognize the pylorus there is generally considerable emaciation of the abdominal walls, thus facilitating palpation. Alternating relaxation and contraction of the pylorus may sometimes be felt, but this phenomenon is very rare. Osler, in his "Practical Monograph on the Diagnosis of Abdominal Tumors" (D. Appleton & Co., 1894) sums up the leading points concerning the solid tumors of the stomach in the following terms: "Though only a small section of the stomach is available for palpation, a very large proportion of all tumors of the organ may be felt, owing in part to their greater frequency at the pyloric portion, and in part owing to the frequent depression of the organ." He gives an account of 24 cases, in all of which a tumor or induration was detected. In the majority of the cases no trouble was experienced in determining whether or not a tumor was in the stomach. Excessive mobility of a pyloric growth and extensive infiltrating masses in the epigastric region were the only conditions causing trouble in any of the cases of his series. As other forms of tumor of the stomach (those that we have referred to in the sections on Malignant and Benign Tumors) are rare, a palpable tumor in the stomach may, as a rule, be considered a carcinoma. One can not exclude carcinoma even when a palpable tumor is absent, because 20 per cent. of all cases escape objective demonstration during their entire clinical history (Fleischer). Among these cases we must reckon the cancers affecting the cardia, the lesser curvature, and part of those affecting the posterior wall. In order to instruct ourselves concerning the typical peculiarities of a tumor, and to avoid confounding it with neoplasms of adjacent organs, repeated and thorough examinations are necessary. The

patient should be placed in a horizontal position, his knees should be flexed and his mouth kept open, and palpation should be careful and gentle, because energetic and rapid palpation does more harm than good, as it produces an annoying tension of the abdominal muscles. We are in the habit of evacuating the stomach by lavage, and also the entire intestinal tract by a purge, prior to palpation in order to bring about the most favorable conditions for this examination.

Occasionally, a tumor will be evident upon simple inspection as a round or oval prominence, but the results of palpation are more reliable. The cancer can be felt as a hard, uneven, sharply circumscribed, nodular tumor. When the growth has extended more toward the interior of the stomach and brought about diffuse infiltrations, one feels a more uniform and frequently only an indistinct resistance toward the depths of the epigastrium or right hypochondrium. In the beginning of a carcinomatous growth at the pylorus, its smooth contour will not permit of a sure differentiation from benign hypertrophy, especially as the consecutive symptoms of stenosis, gastritis, and dilatation are coincident with both. In this case the only means of reaching a decision is by watching the further progress of the tumor. When the stomach is filled with food and gases the tumor is less distinct on palpation. If the tumor mass rapidly disintegrates, without perforation, it may permanently disappear. Persistent and stubborn reflex cramp of the pylorus may give the impression of carcinoma. In a case reported from Leube's clinic, in a man aged thirty-six years, who had a dilatation and a distinctly palpable tumor in the umbilical region, an operation was decided upon, but when von Heinecke and Leube re-examined the man under chloroform anesthesia, the tumor had disappeared; the patient was consequently not operated upon, and was reported living in good health twelve years after this experience.

Position and Movability of the Tumor.—Five-sixths of the normal stomach is located in the left half of the abdomen; therefore, gastric tumors are generally palpable in the epigastrium a little to the left—very rarely to the right—of the median line, and above the umbilicus. Later on, the tumor sinks downward more and more and can be demonstrated below the umbilicus, particularly if it be a pyloric neoplasm. If no adhesions exist, it may even sink down into the pelvis. When there are adhesions near the locality of the tumor it is immovable, but if they are absent the tumor may be

moved to and fro to some extent. In the article referred to, Osler gives some clear illustrations of the positions into which gastric tumors can be moved. Changing the position of the body brings about a moderate movement in the growth. In order to ascertain whether a tumor belongs to the stomach proper, the changes in form and position of the organ, which are caused by filling the same with water or distending it with air, are very helpful. If on filling the stomach with water the tumor lies within the area of dullness thus artificially produced, and if on subsequent distention of the stomach with CO_2 a tympanitic resonance can be made out above and below the tumor, it belongs to the stomach. On filling the colon with one to two liters of water the gastric tumors rise upward, and may hide behind the liver and sternum. It is generally stated that gastric tumors, in the majority of cases, do not move with respiration, but that tumors of the liver do. There are many exceptions to this rule. If there are any adhesions to the liver, spleen, or diaphragm, stomach neoplasms participate in the respiratory movements of these organs. If the tumor is of considerable size it moves downward with respiration, because it can not get out of the way of the descending diaphragm. The percussion note over the tumor is most frequently of a dull tympanitic character. It has been stated that hepatic tumors give only a dullness on percussion, and that this may serve to differentiate them from the stomach tumors; this however, is a misleading sign, because large gastric tumors will give dullness on percussion, and if a hepatic tumor is located at the margin of the liver, or if a few loops of intestine or the colon lie between it and the abdominal wall, a tympanitic percussion note will result. Pulsations may be evident in tumors that are adjacent to the celiac axis or superimposed upon the aorta. If the latter is compressed by the tumor, the tonicity of the crural pulse is diminished, and the epigastric growth may mimic an aneurysm.

Differentiation of Gastric Tumors from those of Adjoining Organs.—(*a*) *From Splenic Tumors.*—As gastric cancers rarely occur at the fundus, their differentiation from splenic tumor is rarely called for, and is seldom difficult. The spleen is movable with respiration; the stomach only exceptionally. In splenic tumor we have dullness on percussion, absence of dyspeptic symptoms, and disturbance of gastric function. In gastric tumor we have a tympanitic resonance on percussion and disturbances of secretion and

motility. Splenic tumors can be very often mapped out and found to be ascending back of the ribs.

(*b*) *From Tumor of the Liver.*—Hepatic tumors move with respiration, and frequently it is possible to grasp the gastric tumor and separate it from the liver. The contours of the liver should be determined, if possible; for with hepatic tumors the liver is generally enlarged and sensitive to pressure, and the surface is frequently uneven. Phenomena of disturbance of hepatic circulation, such as icterus and ascites, denote liver tumor. It is true that dyspeptic symptoms may be secondarily caused by malignant disease of the liver; but then they present themselves later in the disease, with gastric cancer; dyspeptic symptoms are among the very first.*

(*c*) *From Malignant and other Tumors of the Gall-bladder.*—Carcinoma of the gall-bladder and accumulations of gall-stones may be confounded with pyloric carcinoma. The latter brings on gastric dilatation and grave anomalies of function, which are absent in affections of the gall-bladder. Cancers of the gall-bladder are not secondary to cancers of the stomach, as a rule. If, however, the gall-bladder tumor presses upon the pylorus, and also causes stenosis and dilatation, as we had occasion to observe at an autopsy in the Maryland General Hospital, December, 1896, the differentiation from gastric neoplasm is practically impossible. The assertion that hydrochloric acid is still secreted when the carcinoma is in the gall-bladder and not in the stomach, is not supported by sufficient evidence, because passive congestion concomitant with gall-bladder and hepatic tumors often brings about loss of gastric secretion.

(*d*) *Carcinoma of the pancreas* has frequently deceived clinicians in the diagnosis of abdominal tumors. Its immovability during respiration and palpation might suggest pyloric carcinoma; pancreatic tumors, however, frequently cause stasis in the portal vein and pronounced icterus, while dyspeptic disturbances and loss of HCl, which are early and prominent symptoms of gastric cancer, are absent.

(*e*) *Tumors of the Omentum and Peritoneum.*—The differentiation of these tumors from gastric carcinoma is difficult when symptoms of disturbed gastric digestion are present and the tumor does not exceed the limits of the stomach. As a rule, these tumors, being

* The liver itself may give the signs and symptoms of a tumor (see chapter on Enteroptosis).

secondary, are not so sharply circumscribed as gastric tumors. Ascites is rarely absent. The original source—the primary tumor in some other organ—should, if possible, be found out. Sometimes disease of the stomach may be excluded by known analytical methods, and then the diagnosis becomes possible.

(*f*) *Tumors of the colon*, as a rule, sink downward, because the colon is very movable, unless (very rarely) adhesions form with the abdominal wall. By alternately filling the stomach with water and air, and subsequently evacuating it again, it may be demonstrated that the tumor is independent of the stomach. When the colon is filled with one to two liters of water, the tumor rises but very slightly, if at all; while tumors of the stomach, on filling the colon, generally ascend, and may disappear behind the liver or sternum. Tumors of the anterior wall of the colon become more distinct when it is filled with water, while those of the posterior wall become less distinct. A stenosis, as a rule, appears promptly, and the colon becomes tremendously expanded in front of the constriction. Osler (*loc. cit.*) has reported a carcinoma of the cecum and colon, with enormous secondary enlargement of the liver, and extensive secondary nodules were scattered through the lungs. His case is very instructive, as the intestinal symptoms were absent, thus illustrating the ease with which an erroneous diagnosis may be made.

(*g*) *Duodenal carcinoma* can scarcely be separated from pyloric carcinoma. The occurrence of a tumor in the vicinity of the umbilicus, the cachexia, the consecutive gastric dilatation, and (if the duodenal carcinoma should ulcerate) the coffee-ground vomit—all these signs may be present in cancers of either locality. If free HCl can be demonstrated in the gastric contents, or its secretion can be caused after methodical lavage, the other symptoms of neoplasm might possibly be referred to the duodenum. Carcinoma of the duodenum, as Ewald and Riegel have shown, may be combined with atrophic gastritis, so that absence of HCl may, in these cases, occur, which absence is not directly caused by a gastric carcinoma; hence, the diagnosis is largely a matter of chance. It is evident that a thorough knowledge of gastric cancer and the application of all physical and chemical methods of diagnosis are necessary if we wish to be approximately correct in our diagnosis. In all cases of doubtful differential diagnosis exploratory laparotomy should not be deferred until the loss of strength of the patient contra-indicates the operation.

Cachexia.—As the gastric cancer progresses, anemia and emaciation increase to a pronounced cachexia. The color of the skin becomes grayish-white or yellow; it may appear wrinkly, atrophic, and exfoliating. Frequently an annoying pruritus is present. The body weight becomes less and less; the more digestion is disturbed, the severer the vomiting, and the more the passage of chyme into the intestine becomes obstructed. The blood grows poorer in red blood-corpuscles and in hemoglobin, and a state similar to pernicious anemia may result. Pronounced diminution of the hemoglobin is such a constant accompaniment of gastric cancer that it can almost be excluded in case the amount of hemoglobin is equal to 60 per cent. or more (Häberlin-Eichhorst). The amount may sink to 40 and 30 per cent. In one case of Eichhorst's it sank to ten per cent. Accidental sounds about the heart and signs of cerebral anemia or of moderate edema have been observed. Ascites is a consequence of secondary metastases in the liver or peritoneum, or of thrombosis in the portal vein; but toward the end of life it may be caused by great cardiac asthenia and hydremia. Thrombosis of the main vessels of one leg has been observed, and constitutes a very fatal sign. The pulse is mostly accelerated and the body temperature subnormal. Fever is a rare occurrence in gastric cancer; if it does occur, it is traceable to auto-intoxication with septic products formed in the ulceration or degeneration of the cancer.

Coma carcinomatosum is a complication of symptoms similar to the coma of diabetics, and is accompanied by a peculiar dyspnea. The respirations are strong and deep, and generally attended with a groaning sound in expiration. The rate of respiration is either normal or moderately increased. The temperature is either normal or subnormal. There is no evidence of disease of the lungs or air passages. It does not usually appear until anemia is far advanced. This kind of coma was first described by Petters and Kaulich, and later by von Jaksch (*Wien. med. Wochenschr.*, 1883, p. 473). This coma is probably a consequence of auto-intoxication by products of abnormal decomposition. (See literature of this subject in Albu, "Autointoxicationen des Intestinaltractus," Berlin, 1895, p. 105.)

As we have stated in the pathology of carcinoma, swellings of the peripheral lymph-glands are not rare. A hard swelling of the supraclavicular glands was considered typical by Friedreich and Henoch. If severe vomiting ceases suddenly, a breaking down of the cancerous infiltration of the pyloric region may be inferred, by

which communication with the intestines may be restored, or it may be due to excessive muscular insufficiency.

Carcinomatous Ulcer (Ulcus Carcinomatosum).—We have already stated, in the section on the Pathology of Gastric Ulcer, that atypical cell-proliferation may develop from a benign gastric ulcer, which thereafter entirely assumes the character of a carcinoma. Rokitanski and Dittrich were the first to describe this condition, but Hauser ("Das chronische Magengeschwür," Leipzig, 1883) gave the most accurate histological description of it. In 1891, Kollmann (*Berlin. klin. Wochenschr.*, 1891, 5 and 6) reported 14 cases, to which, up to the present time, as far as we know, about 14 more can be added from literature. In this form of carcinoma the gastritis is, at the beginning at least, limited to the immediate neighborhood of the ulcer, or it may be entirely absent. Accordingly, the functional disturbances are less, and secretion may be normal, or we may even have hyperchylia. Very late in the course of this type the remainder of the mucosa may suffer from cancerous infiltration or gastritis, just as is the case with the ordinary gastric carcinoma, and then the functional disturbances become more pronounced. The diagnosis of carcinomatous ulcer can be made if a tumor can be recognized, together with normal or excessive secretion of HCl and a progressive cachexia. Of course, the previous history, which gives an account of years of gastric pain,—whereas cancer patients have, as a rule, when they present themselves not suffered so long,—is a valuable factor in the diagnosis. If vomiting of blood or melena occurs in the clinical history, the diagnosis becomes probable, but it is difficult to distinguish between carcinomatous ulcer and the tumor-like induration of a large simple ulcer. It is also difficult to distinguish the carcinomatous ulcer from hypertrophic stenosis of the pylorus. Sometimes the recognition of secondary metastases in the liver, or other signs of cancerous dissemination, such as ascites and peritoneal carcinosis, may be deciding factors. In one of our cases there was no tumor to be felt, only symptoms of cachexia and of gastric ulcer. For literature, see Rosenheim, "Zur Kenntniss des mit Krebs complicirten runden Magengeschwürs," *Zeitschrift f. klin. Medizin*, Bd. XVII, S. 116; also Boas (*loc. cit.*), second edition, 1895, pp. 188 and 189, and Hemmeter, *New York Med. Record*, vol. 52, No. 11, September 11, 1897, p. 365.

Perforations.—When the carcinoma perforates into other hollow

organs, or exteriorly, life may be maintained for a short time, but perforation into the pleural or pericardial cavities, or into the lungs, rapidly leads to death. Gastrocolic fistulæ cause very rapid emaciation, because the ingesta pass directly into the colon, in which very little digestion and resorption occur. With this kind of perforation, portions of excrement may be vomited. Bronchitis, traumatic pneumonia, and pericarditis may accompany the disease. Tuberculosis may be combined with gastric carcinoma.

Diagnosis.—The majority of authors say that fragments of the cancer rarely occur in the vomit or are brought up in the tube. In fact, it appears that the finding of carcinomatous particles is considered an accident. It is probable that this occurrence is said to be so rare, not because these fragments do not occur in the vomit or wash-water, but because they are not methodically and systematically looked for. The great importance of an early diagnosis of carcinoma justifies the clinician in going to some trouble in order to find these fragments. We are in the habit of feeding all suspected cases for forty-eight hours by the rectum. Thereafter, the stomach is washed out with normal salt solution; for this purpose we use a stomach-tube, which, though quite soft, is provided with a sharp chisel-like edge around the lower opening. This tube in being moved about in the stomach is much more likely to dislodge surface particles of the neoplasm than the tubes ordinarily used. There is every reason why we should intentionally attempt to secure cancer particles from the stomach just as they are secured by curetting from the uterus. We have been able, in this manner, to find particles of the neoplasm in the wash-water after it has been permitted to settle in a conical glass for about six hours, or after the solid particles were brought down with the centrifuge. When the sediment in the bottom of the glass is first examined by a low power, and afterward by the higher power of the microscope, cells in a state of mitosis can frequently be found. Previous to the rectal feeding, we wash out the stomach thoroughly in order to avoid confounding cancer particles with particles of meat, etc., retained with the ingesta. If this method is systematically followed, we believe that cancer particles will be more frequently found; nor should we always deny the existence of carcinoma when we find no fragment giving the typical histological structure of these neoplasms, as described under their pathology.

Whenever we find pieces of mucosa in which the glandular

ducts are elongated and dilated, and the cells present numerous karyokinetic figures and forms of mitosis, and when asymmetrical and hypochromatic forms are found, the possibility of the existence of carcinoma must suggest itself, even when typical carcinoma cells are absent; particularly when the interstitial tissue is considerably broadened, and shows much small round-cell infiltration in addition to above changes.

Whenever cancer is suspected the wash-water should be obtained from the fasting stomach in the morning, before any food is taken; preferably, the contents should be drawn by the expression method without dilution, and any cellular detritus brought down by the centrifuge. In speaking of Rieder's pioneer work in this direction, George Dock ("Cancer of the Stomach in Early Life," *American Journal of the Medical Sciences*, June, 1897, p. 655) expresses himself as follows: "It was therefore a matter of great interest when Rieder (*Deutsches Archiv für klin. Med.*, Bd. LIV, H. 6, p. 544) reported a case in which he made a diagnosis of malignant disease of the peritoneum and pleura from finding numerous cells in the exudates, showing indirect nuclear division." The patient was a woman of forty years. "Section showed sarcoma (carcinoma?) of the peritoneum, probably secondary to malignant disease of the ovaries." In the fluids obtained during life, cells were found which were remarkable "in the first place, on account of the differences in size and shape of the individual cells. Often there were indentations and constrictions, sometimes buddings. In many cells there were one or many vacuoles, often so large that the nucleus was pushed to one side, sometimes hardly visible. The nuclei varied in size and number." The examination of the stained cells showed large numbers of cells in a state of indirect division, and especially cells with atypical mitoses.

"The most remarkable features of the sediment are presented by the great number of karyokinetic figures. These are especially common in cells from 12 to 18 mm. in diameter. The protoplasm of these cells is usually more homogeneous than that of others. Vacuoles sometimes occur, and in rare cases the protoplasm may be very much degenerated. Mitoses are so numerous that every field contains one or more. Often two to five can be seen in a small field. Various stages of nuclear and cell division are present. The most common is that of the equatorial plate. The spirem and the monaster are uncommon. The metaphase is not so easily

recognizable, partly on account of the obscurity of many of the figures. The anaphase is common."

Cells containing more than one nucleus, and with the nuclei in different stages, are also common. In these cells one or more of the nuclei are in the resting stage, and one, or sometimes more, in various stages of indirect division and sometimes showing an atypical figure.

The mitoses found, so far as they can be studied by the chromatin alone, show all the common abnormalities. Thus, we find hypo- and hyperchromatic nuclei, the latter being rare. Giant mitosis may be represented by the tripolar figure. Asymmetrical mitosis is not easy to recognize on account of the imperfect preservation of the chromosomes in many cases. The examples of mitosis in multinuclear cells resemble often the figures given by Krompecher ("Ueber die Mitose mehrkerniger Zellen, und die Beziehung zwischen Mitose und Amitose," *Arch. für path. Anat.*, Bd. CXLII, p. 447).

The interesting history of atypical mitosis can only be touched on here. Eberth (*Archiv für path. Anat. und Physiol.*, Bd. LXVII) was the first to describe division into four parts, but his statements were at first discredited by Flemming and Strassburger. Later, however, Arnold (*ibid.*, Bd. LXXXIII) found multiple karyokinesis in carcinoma. He thought the process might result in polynuclear cells. Since then a great deal of work has been done on this subject, much of it being excited by the ingenious speculations of Hansemann. From an examination of the work done so far it appears that atypical mitoses are found in various pathological conditions, not only in new growths like cancer and sarcoma, but also in benign tumors and in regenerations; in short, "in all tissues of strong reproductive activity and when there is active mitosis" (Strobe). They are also found in tissues irritated by various poisons, such as quinin, chloral, nicotin, etc., or in tissues exposed to high temperature (Galeotti). In cancer all observers find them in great richness and variety, but the view that the presence of even a large number of pathological mitoses in a tissue justified the diagnosis of cancer is gradually being abandoned. As the literature is quoted in the works of Hansemann (*Arch. für path. Anat.*, Bd. CXIX, p. 299, Bd. CXXIII, p. 356, Bd. CXXIX, p. 436; "Studien über die Specificität den Altruismus und die Anaplasis der Zellen," Berlin, 1893), Strobe (*Beiträge zur path. Anat.*, Bd. XI,

XIV), Cornil (*Journal de l'Anat. et de la Physiol. Norm. et Path.*, 1891, tome XXVII, p. 97), and Galeotti (*Beiträge zur path. Anat.*, Bd. XIV, XX), it is not necessary to give a complete bibliography here (George Dock, *loc. cit.*).

The conclusion is justifiable that although the presence of a large number of cells in stomach contents showing mitosis is not pathognomonic of carcinoma, nevertheless it is very significant, and should stimulate further clinical investigation toward positive demonstration of existence of malignant gastric neoplasm.

We have found in one case, four weeks before the tumor at the pylorus was palpable, portions of gastric mucosa, in which the glandular ducts were very closely packed, containing numerous leukocytes and showing a marked atypical appearance of the glandular epithelia, differing from the normal gland-cells by intense pigmentation of the nuclei and much darker staining of the protoplasm. Such appearances in fragments should stimulate further careful and frequent examinations. Sooner or later, in our experience, a fragment will be obtained which will give the typical structure of carcinoma.

Concerning the significance of the Oppler-Boas bacillus we have already spoken in the first part of this work. We can confirm the opinions of the authors quoted, that this organism is a very important diagnostic sign in this disease. We have thus far examined 16 cases of gastric carcinoma, and found the organism present in 14. Lactic acid is a valuable sign of intragastric fermentation, due to stagnation from dilatation and stenosis. It is not pathognomonic of gastric cancer, because it may not be present in excess even in cancer, provided the gastric peristalsis is unimpaired; and again it may be present when the pyloric stenosis is due to a benign obstruction. However, as the majority of gastric cancers destroy the motility and cause obstruction, lactic acid, notwithstanding the exceptions reported (William S. Thayer), is a valuable diagnostic sign. (See "A New Test for Lactic Acid in Gastric Contents," J. P. Arnold, *The Journal of the American Med. Association*, August 21, 1897. This is a modification of the chlorid of iron test, but not so accurate as that given on page 160.) The value of an early diagnosis, upon which the success of any possible operation must depend, is based on: (1) the recognition of tumors; (2) the finding of cancerous particles in the wash-water; (3) demonstration of the Oppler-Boas bacillus; (4) the excess of lactic acid;

(5) the absence of HCl and ferments; (6) the occurrence of hematemesis and melena; (7) the loss of motility and presence of gastrectasia; (8) the general symptomatology and anamnesis.

After a short résumé of the diagnostic and important points of the various types of gastric carcinoma, we will proceed to the details of the medical treatment. For the surgical treatment we refer to the section devoted to that subject.

Résumé and Synopsis of Main Diagnostic Factors in Carcinoma Ventriculi—(Cancer of the Stomach).—Gastric cancer is characterized by progressive cachexia, which distinguishes it from all other chronic affections of the stomach. It is not sufficient for diagnosis and therapy to diagnose the simple presence of cancer, since these neoplasms vary so greatly according to their location at the cardia, the curvatures, and the pylorus, and in so many important symptomatic and diagnostic points that they compel separate consideration. It is expedient to consider these malignant tumors under three headings, viz.:

(1) Carcinoma of the cardia, (2) of the body of the stomach, *i. e.*, the curvatures and the fundus and walls, (3) of the pylorus.

Carcinoma of the Cardia.—*Signs and Symptoms.*—Complaints of an uncomfortable feeling of a foreign body and of pressure above the gastric region, particularly after the ingestion of food. Sensations of pain are not contemporaneous with swallowing of food, but occur independently. On ingestion of food a sensation as if the same becomes clogged, or is caught before it reaches the stomach; patients imagine that copious draughts of water give relief, most likely because this can pass through the stenosis caused by the neoplasm. Another important symptom is vomiting, which is not actual gastric vomit, but the retching up of mucus and a few food particles. The cause of these regurgitations of masses of mucus is the formation of a large dilatation of the esophagus above the stenotic carcinoma of the cardia. In this esophageal diverticulum or dilatation the food is caught, retained, putrefies, and is eventually vomited up again. There is also a septic catarrhal esophagitis present at this place. Liquid or semi-liquid substances may for a long time be able to pass, while relatively solid substances give rise to the difficulties stated. Later on, as the stenosis increases, liquids can not pass either, and loss of appetite and strength goes on uninterruptedly.

If an obstacle to the passage of the sound can be ascertained at

the entrance to the stomach in a person over thirty years of age, the diagnosis of cancer of the cardia becomes certain. In all such suspected cases only a soft elastic tube should be used for explorative sounding. In two cases in private practice we were enabled to establish the diagnosis by microscopical examination of small portions of the carcinoma that were brought up with the sound. These neoplastic fragments are occasionally found in the eye of a lower opening of the sound, and they constitute a definite criterion. In one of the above cases the diagnosis was confirmed by Dr. M. Einhorn, and in the other by autopsy. In addition to the sounding and the cancerous fragments, the following signs are of diagnostic importance :

1. Percussion of the region over xiphoid cartilage is very painful.

2. On the sound, blood will frequently be found mixed with the extremely fetid mucus, and at times nests of cancer cells.

3. On placing a stethoscope over the epigastrium, normally two deglutition sounds can be heard. One is synchronous with the beginning of the act of swallowing, and the other is heard from seven to twelve seconds later. Now, in carcinoma of the cardia the second deglutition sound, which signifies the entrance of liquid into the stomach, may be much delayed or absent entirely; this sign is of importance *per se*.

4. Supraclavicular swelling of the lymph-glands, if palpable, supports the diagnosis also.

5. Lauenstein asserts that there is a systolic murmur audible in the epigastrium, due to pressure of the tumor upon the aorta. According to Boas this is an inconstant sign.

Duration of the disease is six to nine months after the first symptoms are manifested; death occurs as a result of gradual exhaustion, marasmus, aspiration pneumonia, secondary carcinomata in the liver and other organs, and intercurrent hemorrhages.

Differential diagnosis from chronic gastritis is difficult in the beginning of the cancer, as in both the presence or absence of hydrochloric acid is no criterion; but as the cancer progresses the sound will settle the doubt in locating the stenosis. From esophageal ulcer the cardia carcinoma is differentiated by the fact that pain is immediately associated with deglutition of food, by the age of patient (see tables of ages at which ulcer and cancer are most frequent), by the hematemesis and the bloody stools of ulcer. Ulcer of the esophagus is extremely rare in comparison to cancer.

From diverticulum the cardia carcinoma is differentiated by the following facts: Diverticulum is frequent in the upper third, rare in the lower third, of the esophagus. The permeability of the gullet will be more variable than in cancer, because the sound will often skip the diverticulum. In the latter there will rarely be pain, and the marasmus will not be so progressive and rapid. From cardiospasm, or cramp of the cardia, the carcinoma is differentiated by the occasional free passage of the thickest tubes in the neurosis, which occurs almost exclusively in neurasthenics. Nutrition is not so much damaged as in cancer.

If tuberculosis or syphilis be present, one must think of the possibility of the neoplasm being caused by these diseases.

Treatment of Cardia Carcinoma.—So long as there is no cure possible, this must be palliative. During the time that deglutition still brings liquid food into the stomach the sufferer must be carefully fed on highly nutritious liquid diet—liquid eggs and wine, as described in the diet of gastritis, beef-tea, soups of fluid potato or pea purée in bouillon, Leube-Rosenthal beef solution, von Mehring's Kraft chocolate, egg-nog. When pain was great we have found that chloral hydrate, grs. xv, t. i. d., not only relieved it but acted as a local disinfectant in the diverticulum above the stenosis. Boas recommends iodid of potassium in doses of 15 gm. three times daily as aiding in keeping the esophagus from closing up as soon as it would otherwise. Arsenic is said to effect the same prolonged permeability.

In one case we succeeded in keeping the esophagus open for six months by intubating with an inelastic tube four inches long and as wide as an ordinary Ewald tube. The tube was removed every ten days and replaced. Patient lost no weight in those six months, but even gained. Death was caused by aspiration pneumonia, during a period in which the tube was left out in order to rest the esophagus from the stout cord by which the tube was connected with the mouth, and which was usually tied around the patient's neck.

When deglutition is impossible, the only thing left to be done is gastrostomy. If the patient can be persuaded to undergo this operation, it should be done before marasmus proceeds too far, as it then prolongs life and the shock of the operation is better borne.

This operation consists in making an opening into the stomach for the purpose of feeding the patient by passing food directly into

the organ. F. Kaiser (in Czerny, *Beitr. z. operativ Chirurg.*) collected 31 gastrostomies; of these, 28 died of the immediate results of the operation. Zesas (*Archiv f. klin. Chirurg.*, Bd. XXXII, S. 188) reported 131 cases from literature, mostly esophageal cancers, which in their stenosing effects are identical with those of the cardia. Among these only 19.5 per cent. recovered sufficiently from the operation to call this a success. A cure is out of the question; gastrostomy a palliative measure only. (See pp. 356, 357.)

Carcinoma of the Body of the Stomach.—(*Pylorus Cancer of the Fundus, Anterior or Posterior Walls, the Curvatures and Pylorus.*)

Subjective Signs.—1. Sudden abrupt beginning of the disease, striking an apparently healthy organ.

2. Loss of appetite in 90 per cent. of cases.

3. Aversion to meat.

4. In stenosing pyloric cancer there is much thirst.

5. Frequent eructations, which, when there is dilatation, can be very offensive.

6. Pressure in the beginning, pain later on.

7. There is frequent vomiting, which is more copious in pyloric cancers because of the accumulations from the dilatation.

Frequently the vomit has a coffee-ground appearance, and the hemin test (referred to in part 1) proves the presence of blood. The state of the bowels is variable. The vomit contains, as a rule, no hydrochloric acid, but excess of lactic acid and Oppler-Boas bacilli.

Objective Signs.—On inspection, palpation, and percussion a tumor can be made out in at least 50 per cent. of the cases.

Tumors of the pylorus do not move with the respiratory movements unless attached to the liver; tumors of the curvatures generally show distinct respiratory movements.

Examination of Stomach Contents.—The results will be characteristic in most cases and evince—

1. Grave interference with the motility.

2. Suppression of secretion.

3. Products of stagnation dependent upon these.

The disturbances of peristalsis are due most likely to a direct invasion of the muscularis by cancerous proliferation. The simplest way of testing the motor disturbance is to cleanse the stomach thoroughly by lavage in the evening, giving a test-supper thereafter and examining the following morning, when, normally, the stomach

should be empty; but in carcinoma much food and mucus, with absence of hydrochloric acid and presence of lactic acid, is found in 88 per cent. of the cases in our experience. In carcinomata that have arisen from old ulcers there is claimed to be a secretion of hydrochloric acid until the last stages of the disease. This assertion of Rosenheim's is not always correct, as we have shown twice this winter at our clinic. If the glandular layer is invaded, secretion must cease, no matter whether the carcinoma arose from an ulcer or not. Lactic acid is tested for by Uffelmann's reaction; in carcinoma there is an excess in from 86 to 90 per cent. of the cases. Demonstration of the long, base-ball-bat-shaped Oppler-Boas bacillus is, according to Kaufman, Schlesinger, and Riegel, a very important sign. There should always be a careful lookout for histological evidences, such as bits of the growth in the wash-water and vomit; this clinches the diagnosis.

Secondary symptoms are anemia, cachexia, and edema of the ankles in 15 to 20 per cent. of the cases. The urine contains excess of nitrogen excretion, indican, and peptone. Latent cancers may occur; they are very rarely observed, however, at the autopsy.

Ulcus Carcinomatosum.—The diagnosis is made from a history of ulcer, with years of gastric pain, not a sudden and abrupt beginning, and the presence of hydrochloric acid, even hyperacidity. A previous history of hematemesis and blood in the stools points to origin of the carcinoma from ulcer. Simple uncomplicated ulcer may cause a tumor-like thickening, simulating cancer; here the analysis of gastric contents may even show excess of lactic acid, owing to motor insufficiency and cicatricial stenosis, and the diagnosis then becomes difficult, as is also the differential diagnosis of ulcus carcinomatosum from simple hypertrophic stenosis of the pylorus. Fortunately such states without any other important signs are rare.

Treatment.—There is no successful medicinal treatment for this disease. Life may be prolonged by a suitable diet, as nutritious as possible and adapted to the individual conditions. A highly nutritious proteid, carbohydrate, and fatty diet should not be interdicted so long as the motility is good and the patient's strength can be upheld by intestinal digestion. Where there is stagnation owing to pyloric obstruction, the carbohydrates and fats must be diminished. The best tonic for the stomach is daily lavage, even where there is not

much stagnation; but where the latter is marked and accompanied by fermentation, antiseptics may advantageously be added, such as boracic acid, 20 to 30 : 1000 H_2O; salicylic acid, 3 : 1000 H_2O; sodium benzoate, 10 to 30 : 1000 H_2O; resorcin, 10 to 30 : 1000 H_2O; thymol, 5 : 1000 H_2O; lysol, 1 to 2 : 1000 H_2O; hydrochloric acid, 4 to 5 : 1000 H_2O. It is always well to get the stomach clean by simply using warm salt solution, and to finish the lavage by a last irrigation with one of the disinfectants, of which we prefer hydrochloric acid.

A tonic which has been serviceable in our experience and which will arouse appetite and promote digestion in the invaded organ, if this is at all possible, is the following:

℞. Extract. condurango, ... 45.0 c.c. f℥xij
Strychnin sulphatis, ... 0.021 c.c. gr. ⅓
Hydrochloric acid, dil., ... 12.0 c.c. f℥iij
Elixir gentianæ, . . . q. s. . . . 180.0 c.c. f℥vj. M.

Sig.—Take f℥ss in f℥ij aquæ, after meals, through a tube.

When there is much anemia, the following formula has our preference in this disease as well as in ulcer:

℞. Solution of iron and manganese (Parke, Davis & Co.), f℥vj
Liquor potassi arsenit, ... ♏xlviij. M.

Sig.—f℥ss, t.i.d.

Constipation is best met by large colon irrigations or with the fluid extract or active syrup of cascara sagrada. (Clinton.)

Diarrhea must be met by salol, bismuth salicylate, or benzonaphthol. Opiates are not advisable for this symptom.

For pain, hot external cataplasms and 20 to 30 drops of compound spirit of ether should be first tried. If severe, codein, gr. ¼, extract. Belladonnæ, gr. $\frac{1}{6}$, in f℥j of peppermint water, generally relieves it, and may be repeated if requisite. The pain is rarely so intense as to require hypodermic injections of morphin. Lavage systematically and scientifically employed seems to prevent pain; it certainly prolongs life and sometimes apparently works wonders for these patients.

For the diet in gastric carcinoma we refer to the chapter on Dietetics, page 231.

Fifty gm. of rich milk or a glass of tokay, a few crackers, and chocolate are permissible foods, also young pigeon, partridge, and prairie chicken. If the motility is good, one must not be too severe

on the patient's desire for food; many cases can live and gain strength on an ordinary nourishing diet when it is not retained too long in the stomach; under these circumstances mutton chops and broiled beefsteak, finely minced, may be allowed.

Surgical Treatment.—As already mentioned, gastrostomy is a palliative operation for malignant tumor of the cardia and esophagus, to permit of direct introduction of food by establishing an opening between the stomach and the abdominal wall. In carcinoma of the pylorus another palliative operation is practised, where it is impossible or inexpedient to remove the growth, under the name of gastro-enterostomy. This consists in the establishment of a new communication between the stomach and the small intestine, thus allowing the chyme to reach the intestines without passing the pylorus.

The *radical* operations are *resections* of the pylorus and excision of the tumor, no matter where it may be situated in the stomach. These operations are contraindicated if metastases are detectable in other organs, by the presence of great anemia or cachexia, by the large size of the tumor, or if there are adhesions to other organs. The detailed descriptions of these operations belong to text-books on abdominal surgery:

See "Surgery of the Alimentary Canal," by A. Ernest Maylard; P. Blakiston, Son & Co., Philadelphia, 1896.

"Abdominal Surgery," by J. Greig Smith, published by P. Blakiston, Son & Co., Philadelphia, 1896.

"Surgery by American Authors," by Roswell Park, vol. II, chap. 8, published by Lea Brothers, Philadelphia.

"System of Surgery," by Fred. S. Dennis, vol. IV, p. 217.

"Abdominal Surgery," by M. H. Richardson and Farrar Cobb.

"A Text-book of Abdominal Surgery," by Skene Keith and G. S. Keith.

Fred'k Treve's "Manual of Operat. Surgery," vol. II, p. 405.

Franz Kœnig, "Lehrbuch d. speciel. Chirurg.," Bd. II, S. 281.

Penzoldt and Stintzing's "Handbuch d. speciel. Therapie," vol. IV, p. 444. ("The Operative Treatment of Gastric Disorders," by Prof. von Heinecke. Erlangen.)

Diagnostic Résumé and Treatment.—Cancer of the stomach, which in by far the larger number of cases is primary, is, like all cancers, a disease of advanced age, so that about 75 per cent. of all the cases occur between the ages of forty and seventy. According to Häberlin, the number of fatal cases a year in 1000 people is, in the forties, 0.1; in the fifties, 0.46; in the sixties, 1.35; in the

seventies, 2.67; in the eighties, 3.31. But one must not rely too much upon age, since, according to statistics, ten per cent. of all cases of carcinoma of the stomach come in the thirties and about two per cent. in the twenties.

A further factor in the development of carcinoma of the stomach is heredity, either cancer in general, or cancer of the stomach in particular, may often be proved in the ascendance. Figures are of little value in expressing the relation.

Of the other favoring causes, only ulcer of the stomach has been determined with certainty. Hauser has proved directly and histologically the transition from ulcer to cancer. The statistics of Häberlin show that in more than seven per cent. of the cases carcinoma appeared after ulcer. Among the etiological factors we also find recorded traumatism, corrosions, and infections. They are probably only collateral accompaniments, or coincidents.

Concerning the seat of gastric carcinoma, we have already given the statistics of Welch, Brinton, Häberlin, and others, under the pathology.

Occurrence and Detection.—With the exception of the cases that arise from ulcer, cancer of the stomach, as a rule, develops gradually. Among the phenomena are symptoms of more or less severe dyspepsia, gradually ranging to the most decided ones of chronic gastritis, which, almost without exception, accompany every carcinoma of the stomach. Therein lies a great hindrance to timely detection. If, however,—and this especially in older individuals,—the symptoms of chronic gastritis appear without any distinct cause, and in a stomach previously entirely sound, and get worse constantly, even with a mild diet, and are increased by pains and vomiting even before food is taken (with a jejune stomach), so that in a few weeks rapid emaciation, with an extraordinary sallow complexion, become pronounced features, carcinoma of the stomach may be suspected.

What are the characteristic and most important diagnostic signs of gastric carcinoma? Examination with the stomach-tube is to be made, and the reaction for free acid in the contents of the stomach which have been brought up one hour after the trial breakfast is to be tried. In any case, the treatment with lavage is to be instituted, and with it the most frequent repetition of the test for free acid. In this connection it is well to make examination of

the juices after several trial meals, and eventually with means inciting to the secretion of acids (orexin), but, of course, always at a time when, under normal circumstances, free acid ought to be present. Protracted absence of free hydrochloric acid, even though it may occur in other diseases of the stomach, speaks, in the method of procedure indicated, with great probability for cancer, since in more than 90 per cent. of the cases HCl was found absent. Frequent presence of hydrochloric acid argues against cancer. Repeated examination with the tube brings us other signs of carcinoma, which, by other observations, are not,—or at any rate not so easily,—obtained. Occasionally a particle of the cancer is found in the vomited masses, and the diagnosis is made sure. This may happen more easily with lavage in the wash-water from carcinoma of the cardia, when it also may be found in the eye of the tube. During methodical lavage the coffee-ground vomit is seen earlier and more frequently than if we depend upon the vomiting, and, further, in the latter case it is often poured away before the physician gets a chance to examine it. In case of carcinoma one does not need to fear hemorrhage in probing, provided it is done carefully. Finally, one may also use the contents of the stomach obtained with the tube for the quantitative and qualitative determination of lactic acid. (See page 160.)

An approximately simple calculation for this purpose has been recently published by H. Strauss, of Riegel's Klinik (*Berlin. klin. Wochenschr.*, 1895, No. 37). The abnormal presence of lactic acid, according to Boas and others, occurs in 70 per cent. of the cases of cancer. It is true that it has also been shown by him and other authors to exist with gastritis and hypertrophic benign stenosis of the pylorus, which somewhat diminishes its value. But still, as it appears at present, in connection with other signs it is valuable for an early diagnosis, even though its absence does not argue against carcinoma and was especially observed in carcinomatous ulcer. It will therefore be possible in most cases to fix the diagnosis with a great degree of probability, even before a tumor is palpable, when elderly persons previously sound grow rapidly worse in spite of suitable treatment and when cachectic symptoms appear quickly, when the absence of free hydrochloric acid continues, or when there is vomiting of coffee-ground masses. Since ordinarily the tumor can only be felt when

the cancer has reached a certain size and lies in an especially favorable position, the diagnosis by recognition of a tumor is generally no longer an early diagnosis. The examination in chloroform narcosis must be brought in at a comparatively early date to facilitate palpation, and with a sufficient degree of insensibility it will indeed very much facilitate the detection. Distention with air through the stomach-tube, renders a tumor at the front wall or at the pylorus more distinctly recognizable, and gives information concerning the size of the stomach. Distention of the intestine in narcosis by means of air is also brought in as an aid to the diagnosis. If it is not possible to feel a tumor, and if, in spite of this, one is convinced that a neoplasm does exist, one should propose an exploratory incision, with eventual further operative procedures if the prospects warrant immediate good results. If carcinoma should follow apparently in the course of gastritis or ulcer, the diagnosis becomes more difficult than if it is developed in an apparently healthy stomach, for then the symptomatology, the state of the secretions, and the proof of the presence of lactic acid or hemorrhage, are of much less value. Then the diagnosis requires the greatest circumspection. Since the detailed description of all the possibilities does not suit the compass of the work, we will here only refer again to the fact that, with rapidly increasing emaciation of the patients, the physician must not rest until he has found the causes in a carcinoma, or in another factor, such as stenosis of the pylorus. In cases in which the carcinoma causes no symptoms, or only very indefinite ones as regards the stomach (for instance, in the case of people advanced in age), the diagnosis is, of course, impossible and treatment not so important. If there is a palpable tumor in the region of the stomach, we have the problem of determining that the same is really a new formation belonging to the stomach. From the therapeutic standpoint, one is to avoid confounding it with tumors which either need not be or can not be operated. Among the former are to be mentioned the normal head of the pancreas, which with severe emaciation might be mistaken for a carcinoma; lymphatic glands, which are felt as small smooth nodules alongside of the spinal column and may be quite harmless (Leube); tumor of the spleen, which can not be grasped from above; movable kidney, which is smooth and which gives the kidney shape. Of the non-operable tumors, or only exceptionally operable, we should exclude

cancer of the liver, which, without the characteristic gastric symptoms of the stomach, causes the liver to appear enlarged and much distended, or causes nodules to appear on the palpable lower edge (see the extension of the cancer from the stomach to the liver). Gall-bladder and omental carcinomas are chiefly to be excluded from diagnosis by the absence of the conspicuous stomach symptoms, and the latter by the want of respiratory movability and by the presence, generally, of ascites. Carcinoma of the mesenteric glands is, under some circumstances, not to be distinguished from that of the stomach, as Penzoldt recently observed in a case which had, in addition, violent hemorrhages and stomach symptoms. The differential diagnostic points from tumors of the duodenum, colon, and neighboring organs have already been considered (pp. 521–523).

DIET FOR GASTRIC CARCINOMA (*Rosenheim*).

8 A. M.—One cup of tea with milk or a farinaceous soup, eventually with a little wheat bread.

10 A. M.—Toast, sardelles, caviar, perhaps also oysters, with good red wine, sherry, or Madeira.

1 P. M.—Bouillon or soup (flour, rice, sugar, and tapioca soups), eventually with addition of peptone, or Leube-Rosenthal's meat solution. White meat or game, or beefsteak from finely scraped beef, or jellies with gravy, or calves-feet. Vegetables. Potato purée, finely chopped spinach, well cooked asparagus.

Stews: Stewed apples, pears, prunes (without hulls).

Drinks: Red wine, water with cognac.

4 P. M.—Meat peptone, chocolate or cocoa with cakes.

7 P. M.—Bouillon and soup from leguminous flour.

For further diet for gastric carcinoma see p. 231.

Treatment of Loss of Appetite.—Of the so-called stomachic remedies condurangorinda enjoys a great reputation. We prefer the officinal fluid extract of condurango. The other stomachics and bitter tonics used are the tinctures of colombo, gentian, cinchona, etc., likewise hydrochloric acid, which, however, does not always agree with the patient. Orexin generally has no effect, but an attempt with 0.2 to 0.3 gm. of orexin basicum should be made. Also, lavages of the stomach with decoctions of hops and quassia wood, according to Kussmaul and Fleiner, may be used with advantage. Washing the stomach remains the best means for exciting the appetite.

Treatment of Vomiting.—Against vomiting we recommend:

small quantities of ice, ice-cold water containing carbonic acid or champagne, a few drops of chloroform, tincture of iodin, creasote, morphin subcutaneously or as a suppository, cold bandages on the epigastrium. If it is a consequence of stagnation of foods in the stomach, lavage is the most efficacious treatment. If the vomited matter has a foul smell, and foul belching is present, one may add thymol (0.5 per cent.), boric acid (two to three per cent.), salicylic acid, resorcin, chloroform (0.5 per cent.), to the wash-water. The treatment of *hemorrhages* is the same as for gastric ulcer.

Treatment of the Pain.—Steam vapor, bandages, and poultices, hot cloths or plates have only a temporary success. If the pains are very violent, one can not avoid the subcutaneous injection of morphin, but care must be exercised on account of starting the morphin habit. (See p. 535.)

Treatment of Constipation.—This very frequent and troublesome symptom must be eliminated, if possible, by large colon irrigations (one liter), by injections of water with the addition of soap, turpentine, castor oil, etc., which increase their effect, or by the injection of glycerin. Only when this is of no avail must recourse be had to the vegetable purgatives (*e. g.*, Extr. aloe, Extr. rhei. comp., āā 3.0; adde Succ. liq., q. s., ft. pil. 30. M. One to two pills at bedtime). Saline purgatives are justly objected to, since they weaken the patient to a remarkable degree. For the same reason, Penzoldt, Ewald, and Lebert declare that drinking cures at Carlsbad and other saline springs are not advisable. This prohibition is generally very hard for those patients who have placed all their hope on a sojourn at the springs. The advice of Lebert (quoted by Ewald), to let them drink small quantities of the mineral water at home, is very practical, for generally it is without success, and the patient will then willingly give up a trip to the springs. So the treatment in a nutshell is lavage, tonics, rest, the most highly concentrated and nutritious food, whenever it is too late or impossible to operate.

Prognosis.—If the diagnosis can be made early, and operative treatment gives fair prospects of immediate good results, there is, as we have seen from the statistics given in the chapter on Surgical Operations (pp. 336 to 357), some hope of prolonging life. But if an operative interference is impossible (see the contra-indications, p. 350) or refused, the disease must prove fatal. Careful dietetic and mechanical treatment may, in individual cases, prolong life for

several months. In cancerous neoplasms that do not affect the orifices, the immediate danger is not so great. We have ourselves reported a case in which a positive diagnosis of malignant tumor could be made from a cancer particle that came up in the wash-water and in which a tumor was diagnosed by Da Costa and Musser sixteen months ago. At this date (September, 1897) the patient is still doing well, is free from pain, and enjoys her diet.

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TABLE OF DIFFERENTIAL DIAGNOSTIC POINTS.

	Cancer.	Gastric Ulcer.	Nervous Gastralgia.	Hyperchlorhydria.	Gastritis (Chronic).
Duration.	Six months to one year.	Indefinite.	Attacks rarely over three or four days, but recurring.	Long duration.	1. Indefinite. 2. Long duration.
Sex.	No marked differences between the two sexes.	More frequent in women (2 : 1).	More frequent in women.	More frequent in men.	More frequent in males.
Age.	Middle age and advanced life.	More rare in youth; frequently increasing progressively from puberty to an advanced age.	Most frequent between the ages of eighteen and thirty-five.	Met with in all periods of life, except in youth, when it is quite rare.	A disease of mature age.
Tongue.	Almost always thickly coated.	Dry and red, showing a white stripe in the middle, or smooth and moist, or slightly furred.	Presents a normal appearance.	Is either clean or slightly furred.	Gray-white coating; shows impression of teeth frequently.
Sensations.	Feeling of oppression; drawing and pain of variable character. Later, pain in shoulder.	Burning in the stomach; circumscribed boring pains frequently radiating to the back.	Variable sensations in the stomach, at times hot and at others cold.	Sensations of heat and burning; distention; pyrosis.	Pressure, fullness, distention.
Appetite.	Appetite, as a rule, very poor.	Appetite not impaired, although patients, as a rule, eat less on account of resulting suffering.	Variable; in the intervals good.	Often increased.	Absent, as a rule.
Epigastric Pain.	The pain is less intense in character, but more steady; there are seldom free intermissions. during which no distress is felt in gastric region.	Quite intense; appears shortly after meals; grows severer on pressure; disappears at the end of digestive period; perfectly free periods more frequent than in cancer.	The pain appears without regularity, and is not in any way dependent upon the meals; is relieved by pressure, and shows intervals of several days' duration which are perfectly free from pain.	The pain appears about one or three hours after meals, and disappears after partaking of some food, especially meat, milk, or eggs, or after the administration of bicarbonate of sodium.	May be present, but not regularly pronounced. It is rarely actual pain; only tenderness of diffuse character. Lancinating pains in atrophic gastritis only.

Regurgitation.	No water-brash; pyrosis quite intense.	At times present; frequently water-brash associated with pyrosis.	Not present.	Water-brash and pyrosis quite frequent.	Frequently present.
Belching.	As a rule, present, and very often associated with a disagreeable, even fetid odor.	As a rule, absent; if present, without any bad odor.	Variable.	Excessive.	Frequently copious eructations.
Fever.	Fever rare. When present only seen toward the end of life. Temperature may be subnormal.	Slight febrile movement, but only in the presence of adhesive inflammations caused by the perforation of the ulcer, or in connection with larger hemorrhages.	No fever.	No fever.	Rarely fever; temperature sometimes subnormal.
Taste.	Very often bitter and sour.	Nothing abnormal.	Normal.	Normal.	Pasty, decomposed, sour, bitter.
Hematemesis.	Vomiting of blood occurs; the quantity is relatively small, the color ordinarily coffee-brown. The blood appears in a decomposed condition; frequently a fetid odor. The vomiting often recurs, with short intermissions.	Vomiting of a large quantity of blood, either clear red or of a coffee-ground color. Blood is also found in the stools. A repetition of the hematemesis may occur on the following day, but if once arrested it does not reappear for quite a long period.	No vomiting of blood.	No vomiting of blood.	Absent.
Secretory Function.	1. HCl, as a rule, absent or highly decreased. 2. Lactic acid, as a rule, excessive. 3. Ferments absent.	1. HCl, as a rule, increased. 2. Lactic acid absent. 3. Ferments increased.	1. HCl variable. 2. Lactic acid absent. 3. Ferments normal.	HCl increased. Ferments increased.	Arrested, except in gastritis acida.
Vomiting.	The vomiting, as a rule, occurs not after meals, but once or twice a day, or once in two days, the quantity being often very large.	Appears in some cases soon after meals, if at all.	Shows no regularity in appearance.	No vomiting.	Vomiting frequent with alcoholic gastritis; vomitus matutinus.
Perforation.	Perforation occurs only in the last stages of the disease.	Perforation might take place after a short period of illness.	No perforation.	No perforation.	No perforation.

TABLE OF DIFFERENTIAL DIAGNOSTIC POINTS (*Continued*).

	Cancer.	Gastric Ulcer.	Nervous Gastralgia.	Hyperchlorhydria.	Gastritis (Chronic).
Tumor.	Tumor frequently palpable, presenting, as a rule, an uneven surface; is painful to pressure and easily movable.	No tumor; rarely, however, if the ulcer is near the pylorus the latter becomes thickened, and can be felt as a smooth, lengthy body.	No tumor.	No tumor.	Tumor may result in the hyperplastic form by thickening of the stomach walls.
Complexion Cachexia.	Complexion sallow and yellow; skin dry; marked cachexia.	Complexion commonly fresh, but anemic after severe losses of blood.	Complexion pale; in the intervals normal.	Complexion pale; mostly normal.	Pale; malnutrition.
Stool.	Variable; may contain blood.	Constipation; may contain blood.	Normal.	Constipation.	Constipation, alternating with diarrheas.
Urine.	Concentrated, dark, neutral or alkaline; contains indican and acetone, sometimes peptone; preformed and ethereal sulphates increased.	Quantity reduced; indican increased.	Quantity may be increased.	Frequently neutral or alkaline; phosphates increased.	Reduction of urates and phosphates; total acidity lessened.
Tissue Fragments.	May be found in wash-water, showing structure of neoplasm.	Absent, as a rule.	Absent, as a rule.	May occur, showing glandular hypertrophy in two-thirds of the cases.	Fragments may occur after lavage, showing chronic inflammation.
Improvement.	None.	On suitable treatment, marked.	Improvement prompt on treatment.	Marked on treatment.	May occur.
Characteristic Microscopic Signs.	Oppler-Boas bacilli.	None.	None.	In two-thirds of cases fragments showing proliferation of glands.	Leukocytes; fragments showing chronic inflammation.
Dorsal Pain Points.	Diffuse; not characteristic.	Very characteristic; located at twelfth thoracic vertebra, one inch to the left of spinal column.	No characteristic dorsal pain.	No characteristic dorsal pain.	No characteristic dorsal pain.

CHAPTER V.

STOMACH DISEASES CAUSED BY INFECTIOUS GRANULOMATA.

The above-named affections have, almost exclusively, a purely pathological significance. Among the infectious granulations reported as affecting the stomach are tuberculosis, syphilis, abdominal typhus, glanders, and lymphadenoma. We will consider gastric tuberculosis and syphilis separately.

Typhoid neoplasms and ulcerations of the stomach are very rare; even more unusual than tuberculous ulcerations. The medullary swelling of lymph-glands, however, as well as the ulcers arising therefrom, have been described as occurring in the stomach (Orth, "Specielle pathol. Anatomie," Bd. I, p. 714).

Concerning the occurrence of glanders in the human stomach, only one observation is on record, viz.: Bollinger (O. Wyss), "Rotz," "von Ziemssen's Handb.," Bd. III, p. 482, 1876.

Leukemic and aleukemic lymphadenoma, as occurring in the human stomach, have been reported several times. ("Lymphadenoma," Cornil and Ranvier, "Manuel de l'Histolog. Patholog.," p. 294). This neoplasm occurs in the deeper part of the true mucosa and in the submucosa, but sends prolongations into the outer layers. Lymphadenoma and glanders may ulcerate on the inner surface of the stomach.

TUBERCULOSIS OF THE STOMACH.

The gastric mucosa has almost entire immunity from bacterial infection. As the intestines are the seat of frequent infection, when there can be no doubt that bacteria have entered through the esophagus and stomach, the immunity of the last-named organ must depend upon some peculiarity in its structure or secretions. Tubercle bacilli are not affected by the acid gastric juice, as has been proved by Falk (*loc. cit.*) and Frank (*loc. cit.*), who demonstrated that the growth of the bacilli could not be retarded by the gastric secretion. This does not imply, however, that these bacilli can grow

in the gastric juice. The normal stomach, as a matter of fact, is not favorable to bacterial development. The gastric immunity may further be accounted for by the scarcity of lymph-glands in the gastric wall. In the intestines lymphatic nodules are abundant, and they bear some definite relation to the formation of tubercle. The occurrence of smaller and larger tuberculous foci in the stomachs of adults and children proves that the gastric immunity can not be complete. Undoubtedly a number of conditions must simultaneously co-operate to bring about a tuberculous invasion. Prominent among these are: (1) A lessened resistance or reduced vitality of the gastric mucous membrane. (2) A diminished secretion or absence of HCl. (3) An altered state of the blood. (4) The presence of tubercle bacilli.

A number of the tuberculous gastric ulcerations that are reported have developed, not from a direct invasion of the bacilli into the mucosa, but from an invasion into the serosa, occurring from circumscribed or diffuse peritonitis.

Gastric tuberculosis occurs in two forms: (1) Miliary tuberculosis of the wall of the stomach, a not uncommon type; (2) tuberculous ulceration of the stomach, an extremely rare occurrence. Miliary tuberculosis of the stomach occurs simultaneously with the eruption of tubercle throughout the organism. It is usually found to exist with a miliary tuberculosis of the intestines and peritoneum. These cases may strongly resemble intense attacks of typhoid fever. The author, while physician-in-charge of Bay View Asylum, Baltimore, observed two cases of acute miliary tuberculosis which were diagnosed as typhoid fever. Even the characteristic rose-spots were present. This was at a time when microscopic examination for tubercle bacilli was not in vogue. The necropsies were made by Prof. William T. Councilman, revealing acute miliary tuberculosis.

The immunity of the stomach from tuberculosis was shown in the experiments of Orth (*loc. cit.*). By feeding rabbits with tubercle bacilli he obtained intestinal tuberculosis seven times, and gastric tuberculosis but once. In a case of tuberculosis of the esophagus, reported by Dr. S. Flexner (*loc. cit.*), although extensive destruction of the esophagus existed, and the pleural cavity had been opened, and though millions of tubercle bacilli must have been taken into the stomach, no account of any gastric tuberculosis was given.

Clinically, gastric tuberculosis is without much significance. It is, as a rule, not diagnosticated. Tuberculous ulcerations are found most frequently in the pyloric part, and Orth describes isolated miliary tubercles occurring in the vicinity of ulcerations. In rare cases tuberculous ulcerations have been reported to produce fatal symptoms by disintegrating and eroding a gastric artery, or by perforating the gastric wall. The demonstration of the tubercle bacillus in a gastric ulcer was first made by Coats (*loc. cit.*). Matthieu and Rémond (*loc. cit.*), Musser (*loc. cit.*), and Serafini (*loc. cit.*), in their cases, also succeeded in proving the presence of tubercle bacilli. Kühl examined for the tubercle bacillus in four cases from the Pathological Museum of the University of Kiel, but could demonstrate it positively in only two of these, which were recent cases. The other two were older specimens, having been in the museum a long time. A large number of the reported cases of tubercular ulcers are doubtful, either because no microscopic examination was made at all, or the authors failed to stain for the bacillus. Such cases are those of Paulicky (*loc. cit.*), Hebb (*loc. cit.*), Chvosteck (*loc. cit.*, 4 cases), Lange (*loc. cit.*), Barlow (*loc. cit.*), Quénu (*loc. cit.*), and Bignon. The earliest reported case of tubercular ulcer is Litten's (*loc. cit.*), which showed an isolated ulcer on the anterior gastric wall, with typical giant cells and caseating tubercles. Letorey (*loc. cit.*) recently reported a case, and gave an analysis of 21 cases. In 1887, Marfan reviewed the subject, and collected 14 authenticated cases. The disease is most frequently found in males. In 19 cases collected by Letorey, in which the sex was stated, it occurred 16 times in males and three times in females. The ulcers are usually single. In a case from Professor Osler's clinic, however, at the Johns Hopkins Hospital (parts of this stomach were presented to the author for study through the kindness of Dr. S. Flexner, who performed the autopsy), there were numerous ulcers of various sizes. In this case the intestines were also the seat of numerous ulcerations penetrating to the muscular coat. The stomach showed 118 to 120 losses of substance over the entire organ, but most thickly on the anterior aspect near the greater curvature. Hermann Dürck (*loc. cit.*) observed four cases of undoubted tuberculous ulcer in 900 autopsies at Munich. Frerichs and Litten have reported cases in which the tuberculous ulceration was limited to the stomach, the intestines being intact. The sizes of the ulcers vary from a pin's head to five cm. in diameter. Musser

(*loc. cit.*) has reported a case of tuberculous ulcer 3 x 1 inches in extent; and in one of the cases of Dürck (*loc. cit.*), occurring in a child ten years old, there existed an ulcer exceeding in size that of a German 5-Mark piece (somewhat larger than a silver dollar). Secondary tuberculous changes may extend to the stomach through perforation resulting from caseating neighboring lymph-glands. This is generally rapidly followed by purulent processes in the glands. When the peritoneum of the stomach becomes involved in a general peritoneal tuberculosis, the posterior wall of the organ, which is probably the most protected, is either entirely free, or, at any rate, it is much less affected than the anterior wall.

Habershon (*loc. cit.*) assumes that infection of the gastric mucosa occurs by way of the vascular channels. He does not believe in a direct infection of the mucosa because of the acidity of the gastric juice. Klebs ("Tuberculose," published by Leopold Voss, 1894, p. 80) assumes that tuberculous new formations occur on the basis of pre-existing gastric ulcers. A critical review of the literature is given in an interesting report of multiple tuberculous ulcers of the stomach (three cases, by Alice Hamilton, M.D., *Johns Hopkins Hospital Bulletin*, April, 1897).

The bibliography, though extensive, is not quite complete, and we have, in the following, added cases which have come to our notice. In these three cases tubercle bacilli were demonstrated by the Ziehl-Neelsen method of staining. Dr. Hamilton inclines to the opinion of Klebs, that gastric erosions, or previously existing losses of substance, constitute the portals of entry for the tubercle bacillus. The facts in the second case indicate that many small erosions of hemorrhagic origin pre-existed in the stomach, some of which became invaded with tubercle bacilli swallowed with the sputum. Perforation not infrequently occurs. It was present six times in the 14 cases reported by Marfan—three times through a tuberculous gland. In eight of Letorey's cases the presence of a gastric tuberculosis was suspected during life and confirmed at the necropsy. Death by perforation peritonitis resulted in a case reported by Paulicky (*loc. cit.*). Most frequently death results from advanced tuberculosis in other organs. In three of the cases death was caused by severe hemorrhage following an erosion of a blood-vessel through the ulcerative process. In the critical consideration of the subject by Dr. Hamilton (*loc. cit.*) it was found that the authentic literature contained 15 undoubted cases, and nine more,

which were probable, but not proven. While there is a disposition for development of tuberculosis in the intestine, there are numerous cases reported where ulcers existed in the stomach, the intestines being wholly exempt. The deepest ulcers, when found multiple in the stomach, do not extend beyond the muscularis mucosa, and the infiltration of the mucous membrane extends little further than the actual ulceration. These facts are very evident in the sections kindly presented to the author by Dr. S. Flexner, and which were taken from the cases reported by Dr. Hamilton. Superficial small ulcerations still showed vestiges of glands, but without any recognizable distinction between oxyntic and chief cells. Epitheloid and lymphoid cells were profusely scattered throughout the remnants of mucous membrane. We were unable to detect well-defined and typical tubercles. The free surfaces of the ulcers were in a state of necrosis, covered at times by a homogeneous, finely granular matter. The deeper layers were in a state of comparative preservation. Tubercle bacilli were present in small numbers, both on the free surface of the ulcers and among the remnants of the glands. Letulle (Anatom. Société Paris, 1893; also abstracted in *Centralbl. f. allgem. Pathologie*, Bd. IV, 1893, p. 760), in 108 autopsies on undoubted cases of pulmonary tuberculosis, found but one case of tuberculosis of the stomach. The organ presented ten submucous nodules as large as peas, containing giant cells and a few tubercle bacilli.

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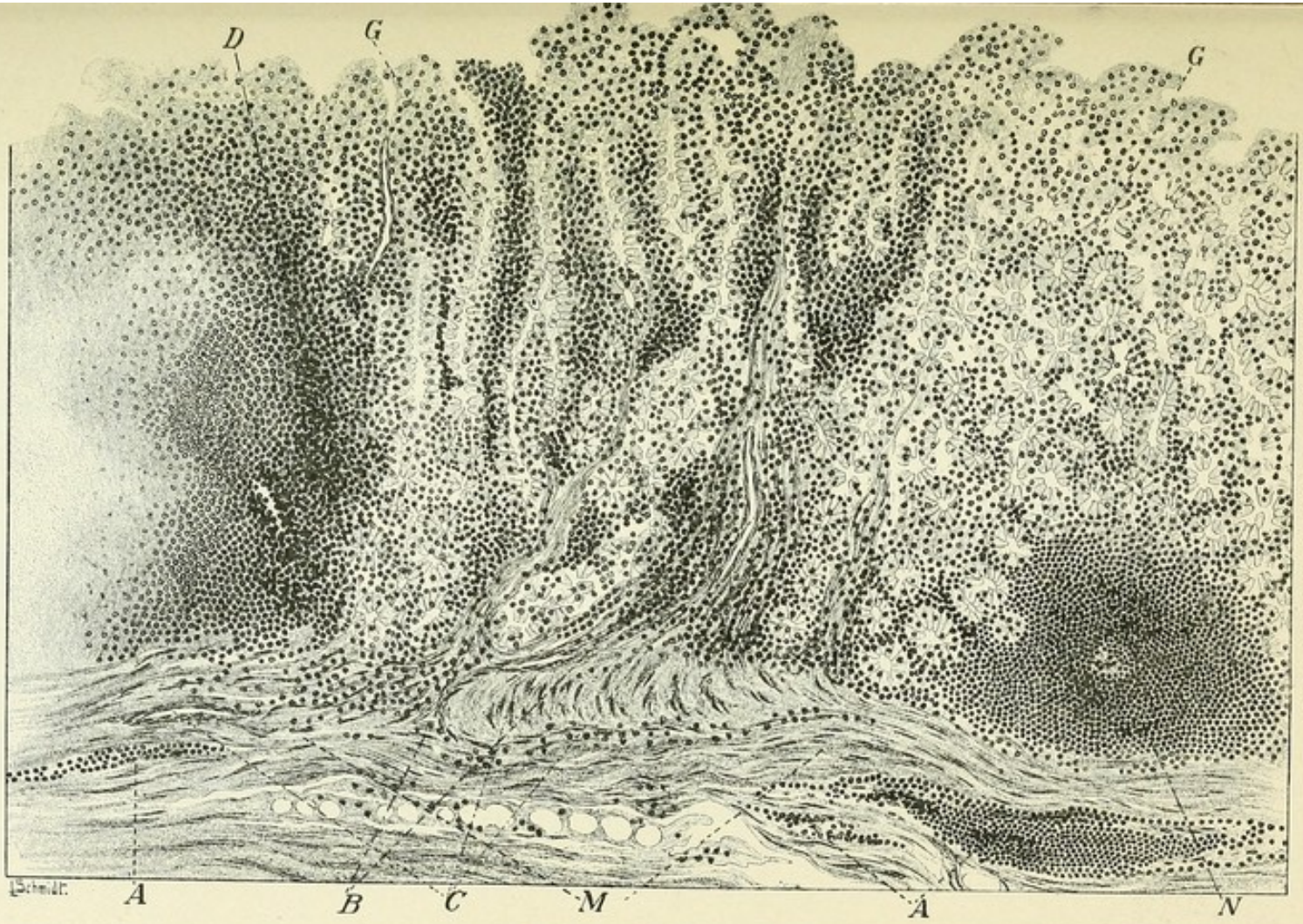
SYPHILIS OF THE STOMACH.

Pathological changes caused by syphilis occur in the stomach in two main forms, or gummata: (1) The syphilitic ulcer; (2) the syphilitic neoplasms. To these we may add a probable third form, (3) the diffuse syphilitic gastritis. While the first two forms are due to direct syphilitic disease, the third form may be due to indirect syphilis; for instance, to the formation of countless syphilitic gummata, forming nodules barely visible to the naked eye in the mucosa and submucosa. This third form, in the majority of cases, is probably due to what Chiari terms indirect syphilis; that is, due to circulatory disturbances, passive congestions, hemorrhages, etc., produced by syphilitic disease of other organs, especially of the liver.

Gastric disturbances are observed in individuals affected with syphilis, even at an early prodromal stage of cutaneous eruptions.



SYPHILITIC GASTRITIS.

Showing degeneration and loss of the superficial columnar epithelium and that of the vestibules and upper gland ducts. Enormous small round-celled infiltration, having forced asunder the fibers of the muscularis mucosæ at *A* and at *N* and *D*, has accumulated in large nodules extending from the muscularis mucosæ upward through the remnants of the glands to the surface. To the left of the illustration the gummatous nodule has broken down into a pale homogeneous non-staining granular matter. At *B* large bundles of muscle fibers are forced up into the glandular layer by the distorting infiltration; within these muscle bundles are contained numerous small arterioles at *C*. The remainder of the gland ducts, with cells in a state of degeneration, are shown at *G*. *M*. Muscularis mucosæ.

These patients may develop all the symptoms of acute gastritis, with a feeling of pressure, fullness in the stomach, loss of appetite, nausea, etc. The symptoms of acute or subacute gastritis may be accompanied by gastralgia, coated tongue, headache, and actual vomiting. These symptoms can not be pronounced as syphilitic, because other etiological factors can not be excluded; one is therefore disposed to assign the gastric symptoms to better known causes. Acute diseases of the neighboring organs (liver, pancreas, and spleen) are not so rare in syphilis as was assumed not long ago. Jullien (*loc. cit.*) describes attacks of vomiting, colic, and diarrhea in the course of recent syphilis; and, according to Fournier (*loc. cit.*), bulimia is, in rare cases, a symptom in severe forms of lues. This condition is observed more frequently in women than in men, and occurs between the third and sixth month of the disease. According to this author, it is sometimes associated with polydipsia. In a consideration of the previous gastric diseases, we have seen that the nerves react in a very sensitive manner to certain anomalous states of the blood—anemias, etc. In lues we have characteristic reduction of the erythrocytes and of the percentage of hemoglobin. We might trace the symptoms described to these blood conditions in preference to ascribing them to disease of the gastric mucosa.

Chronic Gastritis Due to Syphilis.—This is a rather frequent and important syphilitic affection, and is one of the main causes of the poor state of nutrition in luetics. It is, as a rule, associated with characteristic syphilitic disease in other digestive organs (spleen, pancreas, and liver). Histologically, it may be found to be a simple chronic gastritis, or else combined with gummata or gummatous ulcers, and is then a phenomenon of the later stages. Syphilitic chronic gastritis, in the absence of gummata or gummatous ulcers, does not differ pathologically from ordinary chronic gastritis. The cases that are described by Virchow (*loc. cit.*) were due to chronic passive hyperemia caused by circulatory disturbances, and, in our opinion, present nothing characteristic of syphilitic gastritis. Chronic syphilitic gastritis may develop from repeated attacks of the acute form, just as with non-specific chronic gastritis. Relapses need not necessarily be due to syphilis.

If characteristic syphilitic lesions exist in the liver, kidneys, spleen, pancreas, or intestines, the chronic gastritis should, in our opinion, be attributed to syphilis. In tertiary syphilis the remark-

able malnutrition is due to a chronic luetic gastritis. The clinical phenomena of luetic gastritis are not different from the non-specific inflammations of the stomach.

Diagnosis.—It is conceded by gastro-enterologists that iodids and salts of mercury have a deleterious effect upon the gastric functions in normal individuals. If, therefore, the symptoms of gastritis develop in a confirmed luetic, and improve upon the administration of iodid of mercury, getting worse when the mercury is discontinued and improving again when the drug is resumed, the diagnosis of chronic syphilitic gastritis is logical.

We have observed symptoms of acute gastritis in a child eleven years old, daughter of a man who had contracted syphilis while he was a soldier in the German army in the Franco-Prussian war, 1870–1871. The father of this child has had maniacal attacks, in which he had to be restrained. Once a month he has typical epileptic convulsions, which may last, with short intervals, for ten to twelve hours, with foaming at the mouth, involuntary evacuations, etc. He admits the original infection, and gives a correct history of primary and secondary syphilis. The child recently developed a huge gumma of the lower jaw, which assumed the dimensions of a goitre. The gastric symptoms were incessant vomiting and gastralgia. By treatment with mercurial inunctions the stomach symptoms disappeared in the course of two weeks. The efficacy of this treatment was all the more fortunate since the child could retain nothing on its stomach. Two years ago the author treated another child of this man (his wife, by the way, has had four miscarriages), for gastralgia, nausea, eructations, and vomiting, by a saturated solution of iodid of potassium. The child took as much as 40 drops of the saturated solution of KI three times a day, with evidences of distinct improvement, the symptoms subsiding entirely at the end of two weeks. Tullio (*loc. cit.*) reports improvement and cure of severe chronic gastritis by iodid of mercury given internally. The patient became worse when the mercury was discontinued. Non-syphilitics were made dyspeptic by taking iodid of mercury. The following conclusions appear to us to be logical. When digestive disturbances, resembling those of gastritis, occur in a syphilitic, and other etiological factors can be excluded, the diagnosis of syphilitic gastritis is correct if the phenomena disappear under anti-syphilitic treatment. The diagnosis, then, depends upon the evidence of undeniable syphilis as a cause, and the disappear-

ance of gastric symptoms under anti-syphilitic treatment. Professor S. Flexner (pathologist to the Johns Hopkins Hospital) presented us with the stomach of a syphilitic negro, who had gummata in the following places: (1) Frontal bone, extending into the meninges and frontal cerebral convolutions; (2) one in the liver; (3) one in the spleen; (4) three in mesenteric glands; (5) one in the testes and epididymis. The author was present at the autopsy, which was made by Dr. Flexner, but did not see the patient prior to his death. After hardening in formol, the sections were stained in orange G. and hematoxylin. All the sections, no matter from what portion of the stomach they were taken, showed an intense diffuse gastritis. At first we failed to find characteristic evidence of lues. The surface of the mucosa was covered with finely granular elevations, quite evident to the naked eye. The surface cylindrical epithelium was lost entirely. There was a very characteristic endarteritis and thickening of the vessel walls, producing occlusion of the lumen (endarteritis obliterans). Throughout the mucosa and submucosa were countless miliary nodules, about the size of a pin's head, composed of apparently densely packed collections of small round cells. Some of these nodules exhibited themselves in the submucosa, but in that situation they were rare. The majority of them rested upon the muscularis mucosæ, and thence extended upward into the glandular layer, pushing apart, compressing, and distorting what was left of the gland ducts. Some of the small round-celled infiltrations resembled normal lymph-glands of the stomach. The majority of them were, however, larger than the gastric lymph-glands, extending from the submucosa to the surface of the mucous membrane (see illustration, Plate IX). One of our artists (Mr. Louis Schmidt) has given a graphic illustration of the condition present. In many places collections of round cells had forced asunder the fibers of the muscularis mucosæ, splitting apart this layer, which usually runs along in one stratum. In some places the muscularis mucosæ was seemingly torn apart and forced either downward into the submucosa, or upward into the mucosa, in large bundles, by infiltrating masses of round cells. The longitudinal layer of muscle-fibers was similarly split up by enormous collections of small round-cell infiltration. The fibers of the muscularis mucosæ normally ascend into the glandular layer and surround the gland tubules. We have, however, never seen these muscle-fibers ascending in such masses as in these specimens. In places,

the entire glandular layer was replaced by a mass of small round cells. To the left of the illustration is represented one of the miliary nodules, showing a gradual breaking down or softening at the side. Although at first inclined to consider this whole process due to indirect syphilis, caused by passive congestion due to the luetic hepatitis (a large gumma being present in the liver), the finding of nodules, as large as a pin's head (which showed signs of softening), suggests that we may possibly be dealing with minute miliary gummata. Chiari (*loc. cit.*) reported 243 autopsies on undoubted syphilitics; 145 were hereditary and 98 acquired syphilis. His conclusions are the following: (1) Pathological changes caused by syphilis really occur in the stomach; (2) they may be direct syphilitic changes, or owe their origin indirectly to syphilis; (3) the direct syphilis of the stomach is a great rarity, and is either a gummatous process or a simple inflammatory infiltration; the latter occurs only in the hereditary form; (4) the indirect syphilitic affections of the stomach are due to circulatory disturbances caused by syphilis of the other organs, especially of the liver, or else they are due to gastric hemorrhages occurring interstitially as phenomena of a syphilitic-hemorrhagic diathesis; (5) gummous processes in the stomach are characterized by presence of gummatous tissues; they are first developed in the submucosa, and enter the other layers from there; (6) syphilitic gastric ulcers may be caused by disintegration and autodigestion. The cicatrices in the stomach demonstrated by Cornil (*loc. cit*) and Weichselbaum (*loc. cit.*) could be attributed to syphilis only if gummatous tissue were present, or other non-ulcerating gummata.

Only in three cases could Chiari designate the changes as direct syphilis—one gumma in a case of hereditary syphilis, one gumma in a case of acquired syphilis, and one in diffuse inflammatory infiltration of the mucosa and submucosa in hereditary syphilis. His percentage of gastric syphilis was 1.2 per cent. of the total material of 243 sections,—1.3 per cent. in hereditary syphilis and 1.2 per cent. of acquired syphilis.

Syphilitic Ulcers of the Stomach.—In a study of the subjoined literature, authentic cases of syphilitic gastric ulcers are not so scarce as one might presume. Galliard (*loc. cit.*) and Cruveilhier (*loc. cit.*) were disposed to believe in a causative relation between simple gastric ulcer and syphilis. Among 100 cases of gastric ulcer, Engel could trace a syphilitic history in ten per

cent. T. Lang (*loc. cit.*) stated that 20 per cent. of gastric ulcers occur in syphilis. Ewald expresses himself with doubt on this subject. "It must remain questionable," he says, "in two diseases as common as those under discussion, whether we are dealing with cause and effect, or with accidental coincidents." Frerichs, Drozda, Murchison, and Chvostek found scars in the stomach, coincidently with general syphilis. Gastric ulcers may occur in syphilitics from necrosis of the mucosa, due to specific endarteritis, or to disintegration and breaking down of a gumma. In 1838 Andral (*loc. cit.*) concluded that a gastric ulcer in his clinic was due to syphilis because it was cured by mercurial treatment. Rosanow (*loc. cit.*) described a case in a soldier who had suffered from gastric ulcer for eight years and had been treated by him for two months by typical ulcer treatment. The cardialgia, however, continued, and was associated with pains in the lower extremities. The patient showed no signs of lues. He was cured in forty-seven days by treatment by inunctions and iodid of potassium. It is necessary to distinguish between typical simple, round ulcers occuring in syphilitics, and gummatous ulcerations, with gastritis. According to Wagner (*loc. cit.*) and Klebs (*loc. cit.*), all ulcers found in the stomachs of syphilitics have arisen from gummata, but Galliard (*loc. cit.*), Lang (*loc. cit.*), and Mauriac (*loc. cit.*) differ from this opinion, and hold that the syphilitic gastric ulcers need not necessarily arise from gummata.

Neumann (*loc. cit.*) asserts that syphilis is more often the cause of gastric ulcer than has been hitherto believed; furthermore, that gastric ulcer and syphilis do not all develop from gummata, but have the same manifold etiology as the non-specific ulcer. They may develop from erosions, which are very frequent in syphilitics, from endarteritis, diminution in the amount of hemoglobin, and reduction of the alkalinity of the blood, increase and disintegration of the leukocytes. These states are characteristic of lues, and are accepted as etiological factors of round ulcers also. To these might be added hyperacidity and bacterial infection causing necrosis. In a case of Fauvel's (*loc. cit.*), the stomach showed chronic gastritis and several ulcers. In a case of Capozzi's (*loc. cit.*), numerous ulcerations of the mucosa extended from the cardia along the greater curvature to the pylorus. In a case of Oser's, the patient was affected with a syphilitic papulous eruption and psoriasis pal-

maris, the gastric mucosa was injected and permeated by numerous hemorrhagic erosions.

The *symptoms*, *course*, and *termination* of syphilitic gastric ulcers are not different from the non-syphilitic. In a case of Rosanow's (*loc. cit.*), the gastralgia occurred only at night, and from this the author diagnosticated the probable syphilitic nature of the ulcer. Bartumeus (*loc cit.*) speaks of nightly vomiting occurring with syphilitic ulcer.

Diagnosis.—When other etiological factors—such as tuberculosis, alcoholism, chlorosis, and the manifold causes which we have enumerated in the article on ulcer—can be excluded, and undoubted syphilis can be established, the diagnosis of the syphilitic origin of gastric ulcer might be made, although not with certainty. The diagnoses of Andral (*loc. cit.*), Hayem (*loc. cit.*), and Marc (*loc. cit.*), were based upon the curative effect of antisyphilitic treatment.

Prognosis.—Wagner (*loc. cit.*), Lanceraux, and others, report cures of syphilitic ulcers by iodid of potassium. The conditions after the reported cures were similar to those existing after the cures of non-specific ulcers. Stenosis of the stomach and chronic gastritis, following syphilitic ulcers, have been observed by Cornil, Capozzi, Wagner, Fauvel, and Klebs. The direct cause of death in autopsies of cases of syphilitic gastric ulcers hitherto reported was plainly attributable to pathological states in other organs, such as tuberculosis, amyloid and fatty degeneration of various viscera, dropsies, and edema. Some of the cases reported as syphilitic are doubtful. This is our opinion of the case reported by Zavadski and Luxenbourg (*loc. cit.*), in which no characteristic syphilitic lesions are described, as the endarteritis and the small round-cell infiltration may occur in chronic gastritis of a non-luetic character. The patient, a medical student, had denied luetic infection.

Syphilitic Neoplasms of the Stomach.—The percentage of gastric gummata occurring in syphilis has already been stated in the results given from 243 autopsies on syphilitics performed by Chiari (*loc. cit.*).

Gastric gummata have been described by Galliard (*loc. cit.*), Cornil (*loc. cit.*), Birch-Hirschfeld (*loc. cit.*), Chiari (*loc. cit.*), Wagner (*loc. cit.*), Klebs (*loc. cit.*), and Lanceraux (*loc. cit.*). Cornil's case, which may be regarded as typical, was that of a woman who had gummata both in the liver and stomach. That in the stomach

was located on the lesser curvature, and had the appearance of a flattened reddish tumor, two to five cm. in diameter. The gastric gummata reported by Chiari (*loc. cit.*) were sharply circumscribed elevated swellings. They occur together with gastric or intestinal ulcers or cicatrices, and develop in the submucous layer as dense, compact, felt-like masses, formed of fasciculi of connective tissue infiltrated with small round cells. From the submucosa they advance into the serosa and mucosa. The mucosa is thickened, smooth, and glistening, and of a pale yellow color. The muscularis and the serosa are also thickened. The condition of the gummata will vary with the stage in which they are found. In one case Cornil found three gummata, respectively two, three, and five cm. in diameter, in the neighborhood of the pylorus. The mucosa over these gummata was thinned out and adherent. Lanceraux (*loc. cit.*) found an ulceration 30 cm. in diameter in close proximity to the pylorus on the lesser curvature. The case was that of a man sixty-six years old, with many manifestations of syphilis. The ulceration had destroyed the gastric wall—thinned it down to a very delicate lamella. The nodule was in a state of fatty disintegration, and apparently had prevented a perforation by its own structure. Birch-Hirschfeld's case occurred in a newborn infant, with skin syphilis and a gumma in the liver and lungs. In the pylorus was a slightly elevated thick area, as large as the palm of the hand. It was of a whitish color and of tolerably firm consistence, formed of granulation tissues infiltrated with masses of small round cells. Weichselbaum (*loc. cit.*) described two ulcers and one cicatrix at a spot where the transition of the fundus into the pyloric part occurs. One of the ulcers had a triangular shape and was 12 mm. long. This occurred in a man twenty-five years old, with syphilitic manifestations of the cranium, nose, throat, larynx, and liver. Another case of Chiari's, a child three weeks old, with pemphigus syphiliticus, swelling of the inguinal glands, and fissures in the lips, tongue, and penis, showed in the lungs numerous nodules as large as peas, some as large as hazelnuts. There was an induration in the hilus of the liver, and a similar callosity on the common duct, and also on the cystic duct. The wall of the gall-bladder neck was strongly infiltrated. The gastric mucosa showed five plate-like gummata. The gastric gummata generally soften, break down, and ulcerate; this has been the cause of assigning all syphilitic ulcers to

the breaking down of gummata. The ulcer which arises from a gumma is a loss of substance that is smaller in the true mucosa than in the submucosa. The simple perforating gastric ulcer is a loss of substance that is greatest in the true mucosa, becomes smaller in the submucosa, and still smaller in the muscularis.

This gives the simple gastric ulcer the characteristic terraced appearance, which is never seen with a gummatous gastric ulcer. The edge of the simple gastric ulcer is not undermined, but has the appearance as if cut out with a punch. The edge of the gummatous gastric ulcer, however, is irregular, angular, and rolled up. The surroundings, the walls, and the floor of the simple gastric ulcer exhibit no pus and no necrotic tissue elements; perhaps some slight hemorrhagic infiltration is observable, if a previous hemorrhage has occurred. The gummatous gastric ulcer is covered by a yellow, tough, gelatinous deposit. In the surroundings one frequently finds gummata. Old and extensive simple ulcers may closely resemble gummatous ulcers on account of the fibrous thickening of the edges. The occurrence of gummata in other parts of the digestive tract or organs may then decide the nature of the gastric neoplasm. Gummata of the stomach, according to Neumann, are manifestations of late lues. The cases of Birch-Hirschfeld (*loc. cit.*) and Chiari (*loc. cit.*), however, were inherited syphilis.

Diagnosis.—These lesions do not give symptoms sufficiently characteristic to make their clinical recognition possible. In pronounced syphilitics, with palpable hepatic gummata and stenotic symptoms in the stomach, antisyphilitic treatment may possibly give some clue regarding the nature of the gastric neoplasm.

Hemorrhage from the Stomach as a Result of Syphilis.—This is an extremely rare occurrence. Hayem reports a case of grave hematemesis which baffled the usual treatment, but ceased after the administration of iodid of potash. Gastric hemorrhage may occur as a result of intense passive congestion, caused by obstruction of the portal circulation. Hiller (*loc. cit.*) reports a case of a man thirty-nine years old, who admitted having acquired lues in 1868. In the night from the 3d to the 4th of December, 1881, he vomited large quantities of blood, and passed blood by the stool. On the 5th of December, the vomiting of bright red blood was repeated; he also had three passages that were black with partially digested blood. The gums and uvula were covered with

numerous radiating scars; the pharynx showed two recent scars. In the nasal partition there was an irregular, deepened ulcer with callous edges. For several months there had been a purulent offensive discharge from the nose. There was decided enlargement of the spleen and liver. Several uneven prominences were palpable on the surface of the liver. The diagnosis of syphilis of the liver was made, with passive congestion in the spleen and stomach. The patient recovered under antisyphilitic treatment. It is impossible to decide in these cases whether the hemorrhage comes from an ulcer, from hemorrhagic erosions, or from disease of the blood-vessels.

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CHAPTER VI.

BENIGN TUMORS OF THE STOMACH.

Myomata.—Fibromata.—Lipomata.—Polypi.—Myxomata.—Papillomata.—Lymphadenomata.—Pedunculate Tumors.—Foreign Bodies.—Gastroliths.

The stomach may be the seat of a great diversity of tumors. A neoplasm that can be determined by palpation, however, is, as a rule, a carcinoma. Benign tumors are very rare, and their clinical history does not present any great interest. But occasionally they may become the cause of errors in diagnosis. This reason obliges us to say a few words about them, as well as of other foreign bodies and gastroliths which are liable to occur in the stomach.

Myomata, lipomata, papillomata, and lymphadenomata have been found in the stomach.

In acute toxic gastritis caused by the ingestion of corrosive sublimate, calcareous masses may develop in the depths of the mucous membrane. In comparing simple chronic gastritis with gastric tuberculosis, we have shown that in the former of these diseases the glands were capable of undergoing a cystic degeneration more or less pronounced. Aneurysms of vessels in the walls of the stomach have also been described.

Polypi.—Papillomata arising from the mucous membrane sometimes form very well developed villosities in the pyloric region. In their interior one finds a very fine fibrillous network, formed by the prolongations of branched cells; their surface is covered with cylindrical epithelium.

Polypi may develop from myomata, lipomata, fibromata, and papillomata. They vary in size from that of a pea to that of a walnut. They may be pedunculated or attached by broad bases. The term polyp is only descriptive, and not so important, anatomically, as the terms for other gastric neoplasms. In the structure of polypi at times the connective tissue, at others the glandular element, predominates. They might, therefore, be classed logically among the fibromata and adenomata. They may present smooth,

warty, or villous surfaces, the latter resembling the surface of a raspberry or at times of cauliflower.

Villous growths presenting warty surfaces and a papillomatous structure should be logically classed among the fibromata; they are covered by a single layer of cylindrical cells.

Mucous Polypi.—Cornil (*Gaz. des Hôpitaux*, 1864, No. 20) has brought to light two cases of mucous polypi which had not manifested any symptoms during life. In one of these cases the red, mammillated, in places slate-colored, stomach, presented eight vegetations, from the size of a grain of wheat to that of a bean, which had their seat in the vicinity of the pylorus. These vegetations were soft, rosy, and injected with blood; their surface was irregularly mammillated, and they were formed exclusively at the expense of the mucous membrane. In the other case there existed only one pedunculated polypus, rounded like a cauliflower, and as large as a hazelnut. This tumor, formed at the expense of the mucous membrane and of the submucosa, was very vascular at its center. Lambl (Prag. *Beobachtungen aus dem Franz Joseph Kinderspital*, 1860, p. 376) has described a tumor as large as a pigeon's egg, extending three cm. along the fundus of the stomach, and covered by the mucous membrane, which had become thin; no sign had revealed its existence during life. Debove found a mucous polypus by means of the tube, in a patient suffering from nervous dyspepsia.

Rokitansky attributed the formation of these tumors to chronic gastritis; they would thus develop around the glands of the papillæ in unusual numbers and sizes. Wilson Fox also admits the part played by inflammation in the genesis of these polypi. Canus Govignon ("Polypes de l'Estomac," Thèse Paris, 1883) attributes a certain influence to alcoholism. These polypi, whatever may be their structure, are somewhat rare. The first case was pointed out by Cruveilhier ("Atlas dé l'Anatomie," XXX livraison, Fig. 2, p. 2); the stomach, the drawing of which he gives, contained ten pedunculate excrescences, one of which obliterated the pylorus. Andral (*Clinique Medicale*, tome 11) in one case discovered laminated structures analogous to the mucosa of ruminants. Ripault (1833), Mercier (1887), Castilhes (1843), Barth (1845), Richard (1846), Leudet (1847), Barth (1849), have reported a certain number of cases of the same character. Ebstein (*Arch. p. Anat. u. Physiol.*, 1864) has collected all these observations, and has added 14 cases which he had himself obtained from 600 autopsies. Of the 24

cases thus collected, 15 occurred in men and eight in women; in one case the sex of the patient is not mentioned. The frequency of these tumors increases after forty years; in half the cases they are isolated; in one case there were 50 of them, and in two cases there was a number varying from 150 to 200. Their form is variable: they are rounded, club-shaped, cylindrical, ramified; their color depends on their vascularization; frequently they are pigmented. The mucous membrane which covers them is sometimes entirely smooth, sometimes villous, or thickened; they are usually located at the pylorus, and their size is in inverse ratio to their number.

Lipoma (Murray, "Fatty Tumor in Wall of the Stomach," *Pathol. Tr.*, vol. XI, 1890).—The lipoma is also very rare. Starting from the submucosa, it sometimes makes a projection toward the gastric cavity, pushing back the mucous membrane which continues to cover it, but grows thinner in proportion as the tumor increases; sometimes it pushes aside the muscular fibers and succeeds in making a hernia under the serosa. A large tumor of this kind might cause digestive troubles by the dragging of its weight on the wall of the stomach, but the rareness of these cases interferes with an exact knowledge and description of their symptoms. Orth has observed lipomata growing from the serosa in a pendulous manner (*loc. cit.*, S. 717).

Myoma.—The myoma develops in the interior of the muscular layer, gradually projects under the mucous membrane, and occasionally ends by forming polypi,—sometimes isolated, sometimes in numbers. These tumors do not differ in a histological point of view from those which may be found, for example, in the uterus, but their size rarely exceeds that of a pea or a cherry. Their development is not accompanied by any symptoms, and they are rarely discovered except at the autopsy. (Myoma, see Virchow–Onkol, III, 126.)

Symptoms.—The symptomatology of these tumors is variable. Sometimes they become ulcerated, and Rondeau (*Presse med. Belge.*, No. 18, 1881) has pointed out a case where their presence was revealed by serious hemorrhages; naturally, the observer was not able to diagnose the cause of this hematemesis. At other times, as in the case of Cruveilhier's patient, the tumor may obstruct the pylorus and cause a dilatation of the organ. Bernabes (*Rivista Clinica di*

Bologna, Juillet et Auot, 1882) had, in this way, the opportunity of observing a woman seventy years old who, for a long time, had vomited a few hours after meals, and experienced sharp epigastric pains, in the absence of characteristic symptoms. At the autopsy there was found a polypus of six or eight cm. implanted on the anterior surface of the stomach, five cm. from the pylorus. Five other smaller polypi were scattered on the pyloric antrum and along the greater curvature (Bruman, *Th. de Paris*, 1883; Breissaud, *Arch. Gen. de Med.*, 1885; Marfan, *Th. de Paris*, 1887; Menetrier, *Arch. de Phys.*, 15. Fevrier, 1888).

Lymphadenoma.—The stomach may also be the seat of lymphoid tumors. These likewise constitute a pathological rarity. Pitt ("Pathol. Trans.," vol. XI, 1890) has reported one case, and states that he has been unable to find more than 17 in the literature on the subject. The patient had succumbed to phenomena which were all attributed to a tumor of the lungs and to an empyema of the left pleura. Soft nodules were scattered over the stomach and the intestines which had perforated the mucous membrane, or had made greater or lesser projections at its surface; histologically, all the characteristics of lymphadenoma were exhibited. The spleen, the mesenteric, and the bronchial ganglia were invaded; the liver and the kidneys were not affected, however. The neoplasm, develops in the mucous membrane and the submucosa, and forms tumors projecting into the cavity. On the other hand, at times the serosa is first attacked. The muscular stratum then becomes more or less affected by distention, and a dilatation of the organ becomes evident. In other cases the tumors which project into the gastric cavity become ulcerated, and the patient succumbs to a hematemesis, as in Reimer's observation (*D. Arch. f. klin. Med.*, Bd. XXXIII, p. 632, 1879). To these phenomena are always added a diarrhea of varying seriousness; but when the tumors remain limited to the stomach, health may be little affected by it. Anatomically, one finds around the base of the tumor a hardening of the mucous membrane; the glands affected are in fatty degeneration, while at their periphery there exists a characteristic reticulated tissue. The degenerated glands finally disappear, and there remains nothing more than the reticulated tissue of the tumor.

Pedunculated **adenomata**, attaining the size of an apple, have

been observed, which were composed exclusively of tortuous, irregularly dilated gland tubules. Tumors may occur in the stomach as well as in the intestines, which anatomically and clinically must be considered cancers, and having a pronounced glandular structure are designated as destructive or *malignant adenomata* or *adenocarcinomata*. The case reported by Pitt (*loc. cit.*) is suggestive of this type. They have been considered under the malignant tumors.

Cysts.—Retention cysts of the gastric glands occur in "gastritis polyposa" and polypoid hypertrophy. Ruysch ("Adversaria Ana," tom. III, p. 1, Dec., 1732) described a gastric dermoid cyst containing hair. Engel–Reimers (*D. Arch. f. klin. Med.*, XXIII, p. 632, 1879) describe a multilocular lymphangioma occurring in the outer gastric wall beneath a chronic ulcer of the lesser curvature. This cyst contained a milky liquid, produced by stasis of lymph in consequence of occlusion through inflammatory processes in the vicinity of the ulcer. Albers ("Erläuterungen," IV, p. 151) mentions a cyst 2¼ inches long found on the lesser curvature in a child.

Foreign Bodies. — Foreign bodies are, in certain cases, capable of imitating a tumor, both by the subjective symptoms which they cause, and by the deception to which they give rise on palpation. A patient of Baillarger's (*Union Med.*, No. 48, 1874) had kept in his gastric cavity for six years a zinc fork; an epileptic, cited by Foville (*Gaz. hebd. de Med. et de Chir.*, No. 18, 1874), had swallowed 28 dominoes; an ecclesiastical patient thus preserved his rosary in his stomach for a time. Labbé (*de l' Acad. des. Sciences*, 21. Avril, 1866) extracted a fork by gastrotomy. A sailor ("Med. Chir. Transact.," vol. XII, p. 72), cited by Ewald, in imitation of a juggler swallowed 35 small knives, and only succumbed a long time afterward to digestive troubles; there were found, at the autopsy, 32 blades, more or less corroded, 30 in the stomach and two in the intestines. It is unlikely, however, that such objects as these could produce the signs of tumors.

Schönborn (*Berl. klin. Wochenschr.*, No. 17, 1883, und *Arch. f. klin. Chirurgie*, Bd. XXIX, p. 609, 1883) has reported an observation in which a *gastrolith* (movable tumor) was discovered in a girl fifteen years old, occupying the left half of the abdomen, it was easily pushed back under the left edge of the ribs, and very painful, both spontaneously and on palpation. The patient grew thin, would not

tolerate any food, and her state became so serious that it was decided to perform a laparotomy, after having hesitated for a long time between the diagnosis of a movable kidney and that of movable spleen.

The opening being made, the stomach was found distended; it was cut into, and a mass of 281 gm. was found, formed by a network of short hairs, and molded to the form of the gastric cavity. The patient then confessed that four years before she had swallowed the hair in order to "make her voice clear." After this case, Schönborn made a careful collection of the known cases, and found seven of them, the oldest of which dates back to 1777. These cases include six women and one boy; none were insane. All these subjects had died; some from peritonitis through perforation, others from uncontrollable vomiting. One case ended in hematemesis, and Russel (*Med. Times and Gazette*, June 16, 1869), who published it, reports that the tumor weighed four pounds, seven ounces, was 12 inches long, five inches broad, and four inches thick. Never before had there been digestive troubles, and it had been supposed that it was a tumor of the spleen. In the observation of Inmann (*Med. Times and Gazette*, July 3, 1869), the mass of hair was equally large. Best (*Brit. Med. Jour.*, Dec. 11, 1869) reported a case of a woman thirty years old who, for sixteen years, had complained of pains after meals, and had frequent vomitings, occasionally streaked with blood. For six years the pains had been almost intolerable, and hindered the patient from giving herself to any occupation. At the epigastrium a movable tumor was discovered, not sensitive to pressure, smooth, hard, extending from the right hypochondriac region to the left of the umbilicus, the prolonged palpation of which caused emesis. Peritonitis from perforation ended the case. The stomach and the esophagus were filled with a quantity of hairs, some of which were from 10 to 12 inches long, and which all together weighed 30 ounces. The patient had acquired a habit of swallowing her hair fifteen years before. Since Schönborn's notice, Kooyker (*Zeitschrift f. klin. Med.*, XIV, p. 203, 1888, also *Weekbl. v. d. Nederl., Tydsch v. Geneesk*, December, 1887) has reported the case of an individual fifty-two years old who, after having presented phenomena of cachexia, with hematemesis, succumbed at the end of three years of sickness. During life a tumor the size of a small apple had been felt at the epigastrium; and displacement of the spleen, a floating kidney, a cancer of the stomach, and a cancer of the colon

were suspected, one after the other. At the autopsy a renal-shaped foreign body was found, 18 by 8 cm., weighing 885 gm.; two other masses were also discovered, the size of a hen's egg. On examination with the microscope, these bodies showed some grains of starchy matter and some vegetable cells, some of which contained chlorophyl, but no trace of animal substances. This case is analogous to that of Capelle's (*Jour. de Med. de Bruxelles*, Fevr., 1861), who treated a woman forty-three years old for a tumor of the stomach, who had been ill for a long time. There were emesis, intense gastric pains, and constipation. The tongue was white, palpitations frequent, the pulse small and weak. Under the xyphoid cartilage was found a hard, immovable tumor as large as a pigeon's egg, which disappeared when vomitings, more violent than others, had caused the expulsion of a foreign body of nine c.c., half softened, and formed exclusively of vegetable debris. The patient recovered. Lastly, Bollinger (*Münchener med. Wochenschr.*, No. 22, 1891) published the case of a girl sixteen years old in whom there existed a hairy tumor which caused death by inanition. A malignant tumor had been suspected, and there was found in the stomach and in the dilated duodenum, a tumor 55 cm. long by 11 cm. broad, and 28 cm. in circumference, formed by 500 gm. of hairs, which measured each about ten cm.

The presence of foreign bodies in the stomach may give rise to the following signs and symptoms: The organ dilates, becomes displaced (Russel); the mucous membrane atrophies; the pylorus may become expanded through muscular efforts to pass out the foreign body, which will act as a ball valve when expulsion is impossible; the peptic secretions disappear; the erosions allow the escape of blood in more or less abundance, and the patients usually succumb to a cachexia, since alimentation in the end becomes impossible.

Therapeutic measures are useless in these cases. An exploratory laparotomy is the only rational procedure.

Erlach removed a myoma from the stomach weighing 5400 gm. (*Centralbl. f. allgem. Patholog.*, Bd. VI, 1895, p. 240). In the same journal (vol. VI, p. 717) Hansemann is reported as having found four peculiar tumors in the stomach: (1) A myoma with cystic degeneration; (2) a sarcoma with hyaline degeneration and containing large calcareous bodies; (3) a tumor of myxomatous nature; (4) a tumor composed of finely fibered connective tissue,

inclosing hollow spaces which contained cells in a state of fatty degeneration, simulating the cortical substance of the adrenal bodies. Professor Julius Schreiber reported a case of phytobezoar composed of the fibrous roots of a plant (Schwarzwurzel) which is a popular remedy for all sorts of ailments in Germany. The patient was a female peasant forty-five years old. The tumor very much resembled a floating spleen or malignant neoplasm. The diagnosis was correctly made and the woman successfully operated upon by von Eiselsberg (*Mittheil. a. d. Greuzgebieten d. Medizin u. d. Chirurg.*, Bd. 1, 1896, S. 729).

CHAPTER VII.

MOTOR INSUFFICIENCY.

Gastric Atony or Myasthenia.—Gastrectasia (Dilatation of the Stomach).

There is no uniformity in the classification of the various forms and degrees of abnormal enlargement of the stomach.

The defective function in these cases is not commensurate with the size and capacity, but with the tonicity of the peristalsis. A very large stomach (megalogastria) may have a perfect motor function, and a very small stomach may have a defective motility.

Boas recognizes a *mechanical insufficiency of the first degree*, which is a myasthenia or atony of the gastric muscularis in which the ingesta remain in the stomach too long, but finally are completely moved out into the intestines. There is no absolute retention of food, but simply a delay in the expulsion. Boas calls the fully developed dilatation *mechanical insufficiency of the second degree.*

Riegel differentiates:

1. *Simple atony, or insufficiency of the stomach.*
2. *Atonic or typical ectasia, or dilatation.*
3. *Secondary ectasia, or pyloric stenosis with ectasia (dilatation).*

Naunyn speaks simply of motor insufficiency, and Rosenbach of mechanical stomach insufficiency. Schreiber (Boas, *Archiv f. Verdauungskrankheiten*, Bd. II, S. 423), in attempting to select a designation which should signify the most constantly present condition of all these morbid states of motility, and one which should unite them all around itself, reached and suggested the term *stasis stomach* ("Stauungsmagen"), with permanent digestion or permanently digesting stomach. Besides being a cumbersome circumlocution, the term does not even include all conditions of this type, for in Boas' mechanical insufficiency of the first degree, and in Riegel's simple atony—conditions which we are convinced really do exist—there is certainly no permanent digestion.

Permanent digestion goes on in fully developed dilatations with

impaired peristalsis as long as hydrochloric acid and ferments are secreted. But as there undoubtedly are long-standing dilatations with complete achylia gastrica, or loss of secretion (Einhorn), there can be no digestion in them. The fact that the food is over-retained in them does not imply that it is digested; only in dilatations that show hydrochloric acid and ferments can we speak of permanent digestion. The efforts of Schreiber to establish Reichmann's chronic secretion as a complication of dilatation with retained food products and permanent secretion caused by stimulation of the retained food, are very convincing. We shall speak of the pathogenesis of gastrosuccorrhea, or Reichmann's disease, under the Nervous Affections of the Stomach. It is impossible, however, to invent a term which shall comprise the important features of all types of motor and mechanical insufficiency, and probably as clear a classification as any is one based on Riegel and Boas, as follows:

1. Simple gastric atony, or motor insufficiency, or myasthenia without dilatation.

2. Atonic dilatation (motor insufficiency due to relaxation of the gastric walls) without pyloric stenosis.

3. Secondary dilatation (motor insufficiency due to pyloric stenosis).

It is self-evident that these conditions may have widely different causes; the one common sign is not the retention of food nor permanent digestion, but the impaired motility.

Etiology.—Two kinds of cases may occur: either the atony of the gastric wall is not due to a mechanical obstacle,—in this case nothing will oppose the free course of the contents, and they will only linger in the stomach because the latter is really incapable of ejecting them from its cavity in proper time,—or the atony will be due to a pyloric stenosis; the muscular tonicity will have been overcome by an impassable obstacle, the fibers exhaust themselves in contending with an excessive resistance, and the dilatation may then be considered as following on existence of the obstacle. In the first case the etiology is variable, and arises finally from a defect in the nutrition of the muscular layer; in the other case it is purely mechanical.

DILATATION CAUSED BY A MECHANICAL OBSTACLE.

Intrinsic causes of opposition to the passage of stomach contents into the intestines, and of such a resistance to the contractions of

the stomach that it dilates, are, first of all, the constrictions of the pylorus. These are generally the result of anatomical alterations, viz., cancer, cicatrices, circular ulcer, or muscular hypertrophy of the pyloric sphincter.

Nauwerk (*loc. cit.*) was one of the first to draw attention to hyperplasia and hypertrophy of the pyloric sphincter as a cause of dilatation. A spasm of the pylorus, which can be compared to a spasm of the sphincter of the anus, can just as well determine an occlusion. This spasm, which has been admitted by authors for a long time, for reasons a little theoretical perhaps, has been demonstrated since gastric surgery has permitted the more direct exploration of this organ. Martin (*loc. cit.*) has reported a case in which a pylorus large enough to admit the passage of two fingers brought on a considerable dilatation by its spasmodic constriction, consequent upon a circular ulcer accompanied by considerable hyperacidity. Landerer (*loc. cit.*) is said to have proved the existence of a congenital pyloric constriction analogous to the congenital mitral constriction described by some authors. He collected ten such observations, and claimed that this orifice, large enough during infancy, might undergo an arrest of development and remain very small, while the stomach grows larger with age; a serious dilatation would result from these diverging effects.

However, the obstacle need not necessarily have its seat in the tissue of the pylorus itself. In the chapter on Benign Tumors we have described the possibility of a polypus inserted in the gastric cavity and capable of bringing about a dilatation by becoming fixed, more or less, in the intestinal orifice, and thus causing its occlusion, acting like a ball-valve. Deiters (*loc. cit.*) has collected, from the Anatomical-Pathological Institute, of Greifswald, a large number of observations, in which congenital malformations, abnormal foldings, diverticulæ, and atresia had provoked dilatations by constricting the intestine in the immediate vicinity of the pylorus. An anatomical lesion of the duodenum—the cicatrix of an ulcer, for example—would produce the same effects by diminishing the caliber of the passage.

The causes of extrinsic origin which have been observed to effect compression of the pylorus or duodenum are very numerous. Among these are peritoneal adhesions, circumscribed or not, the results of former inflammations. Fibrous bands issuing from a

gastric cicatrix may so distort the normal location of the pylorus (although not situated in the pylorus itself) as to compel the duodenum to describe an abnormal course.

Inflammations originating in the liver and the pancreas may be the starting-point of similar anatomical modifications. The head of the pancreas, so intimately connected with the duodenum, may become cystic or cancerous, and cause a duodenal stenosis by compression, with following dilatation of the stomach. A congenital displacement of the duodenum would bring about the same disorders (Cechini). Biliary concretions, by dilating the diverticulum of Vater, or by compressing the intestinal wall, may produce a compression of the duodenum sufficient to bring about gastric dilatation; Grundzach has recently reported a case of this kind. Landau (*loc. cit.*), Bartels (*loc. cit.*), Warnek (*loc. cit.*), Mueller (*loc. cit.*), Litten (*loc. cit.*), and other authors have studied the relations of dislocation of the right kidney and of gastric dilatation. Similar studies have been made in an interesting work by Bruhl, and Mathieu has also recently reported new cases (Société Médicale d'Hôpitaux). Patients presenting this coincidence of movable kidney and dilatation of the stomach are usually young girls or women of the working class, who are in the habit of fixing their skirts at the level of their hips, and lacing tightly, causing an external constriction, which is shown by the presence of a permanent furrow. Fleiner (*loc. cit.*, p. 211) gives a number of graphic illustrations on these malformations. Men who wear a belt or strap may produce the same results. Lud. Knapp ("Wanderniere bei Frauen," Berlin, 1896) associates the frequency of floating kidney with abnormalities in the pelvic organs in women, causing constant dragging on the kidneys by means of the ureters. The right kidney may become displaced forward and inward, pressing upon the fixed, descending portion of the duodenum, which is situated between the hilum of the kidney and the vertebral column. Such partial obliteration of the intestine would bring about a slower and more difficult evacuation of the contents of the stomach. As food would remain much longer in the stomach, there would result, first, an increase of activity and a slight hypertrophy; then, later, a muscular relaxation, distention of the walls, and a dilatation of the cavity of the organ. We shall treat the effects of floating kidney more fully in the chapter on Enteroptosis.

Ewald (*loc. cit.*) and Pertick (*loc. cit.*) have gathered together a

certain number of cases in which a hernia of the floating portion of the duodenum, or of the first part of the jejunum through a laceration in the mesentery, or a diverticulum of these portions of the intestine, has brought about an impediment to the normal course of the ingesta, and caused, in the end, a gastric dilatation.

ATONIC DILATATION.

Gastric atony is a condition of reduced or lost tonicity of the musculature. It is a state of sub- or hypotonicity, also very aptly designated as gastric *myasthenia*.

Dilatations resulting from this state which seem to be primary may be acute or chronic. The first kind, which are very rare, have for their cause either a traumatism or a surgical intervention (laparotomy), or else a serious infectious disease; Hilton Fagge (*loc. cit.*), Bartels (*loc. cit.*), Montaya (*loc. cit.*), and Lepoil (*loc. cit.*) have cited examples in which typhoid fever seems to have played the part of the chief cause. In this case the dilatation seems to be due to the loss of tonicity of the musculature of the stomach and of the abdomen. In other cases the origin of the evil is an excess of food, an error committed so frequently by convalescents after they have been confined to one diet for a long time.

Chronic forms of atonic dilatations are dependent upon a great number of factors. Those addicted to excessive indulgence in food suffer first with distention of the stomach; then, later, with dilatation. This phenomenon is comparatively frequent with persons into whose ordinary diet enters a large quantity of liquids; such is the case with excessive beer drinkers, the beer acting not only mechanically by its volume, but also through the irritating and poisonous substances with which it may be adulterated. Debove (*loc. cit.*) has drawn attention to the drawbacks of prescribing milk in considerable quantities, and has cited, among others, a case of circular ulcer cured by the daily allowance of eight liters of milk; but an enormous dilatation of the stomach resulted. In the chapter on Acute Gastritis we have pointed out that overfeeding produces a certain amount of gastritis. The dilatation is produced under this double influence of the inflammation and of the distention; without the addition of the first of these causes, megalogastria alone would occur.

Simple chronic gastritis may result in a considerable atrophy of the muscular fibers of the stomach, which may lead to dilatation of

the stomach. The same may be said of hyperchylia, provided that it is one of the forms where a hyperacid secretion causes a prolonged stasis of the amylaceous substances ; such cases have been collected by Mathieu and Rémond, under the name of dyspepsia with organic hyperacidity and stasis. In other cases muscular atony is the result of a prolonged retention in the stomach of undigested food, with fermentation thereof, when hydrochloric acid is absent. Drawn out by a weight more or less considerable, and distended by the gases that are developed in the putrefying mass, the muscular fibers gradually lose their elasticity. The dilatation found in consumptives, in chlorosis, etc., is due solely to chronic gastritis, which is caused by alterations in the blood, the results of these diseases. In diabetes both the chronic gastritis and superabundance of food co-operate in the alteration of the walls, and may finally lead to amyloid and colloid degenerations of the muscular fibers.

Atony of purely nervous origin, concerning which the French writers Germain-Sée (*loc. cit.*) and Mathieu (*loc. cit.*) have published numerous researches, is held by them to be a consequence of crises appearing to indicate the successive and alternating intervention of a particular state of spasm and of atony of the gastro-intestinal tract. These crises are produced by an occasional and general cause, such as sad emotions, mental shock, neurasthenia, etc.

The atonic form of dilatation was first recognized, toward the end of the last century, by John Peter Frank (*loc. cit.*), who separates it distinctly from the forms caused by stenosis. The atony due to neurasthenia can be just as well brought about by lesion of the central or peripheral nervous system, and the dilatation will then depend on a deep-seated alteration either of the central organs, or of the peripheral nerves. Bouveret, Dujardin-Béaumetz, and Glénard have represented general ptosis of the abdominal organs as the expression of a particular diathesis, a condition of relaxation of the tissues with smooth muscular fibers, and have suggested that there is a dilatation depending upon this general state. The dilatations resulting from nephroptosis are included in this class by Glénard, Debove, and Rémond. (These states will be more fully considered in the chapter on Enteroptosis.)

Pathological Anatomy.—Having already considered the pathological histology of the various causes of dilatation, viz., neoplasms, benign and malignant cicatrices, chronic interstitial gastritis, etc., etc., the pathological anatomy of the dilatation, *per se*, is simple.

At the autopsy of a subject dead from cancer of the pylorus, for instance, one finds the abdomen filled by a voluminous sac, which comes down more or less near the pubes. This sac, which represents the stomach having lost all its normal relations, and excessively dilated, may contain enormous quantities of liquid, and the ancient authors, who knew only the extreme cases, have cited extraordinary examples of this. (The history of the subject is given by Penzoldt, "Die Magenerweiterung," Erlangen, 1875.) Plempius (*loc. cit.*) is said to have seen a stomach that held nine pints of liquid; Stengel mentions a stomach containing 12 "measures"; Schurig, a stomach containing 48 liters; Henricus ab Herr found a stomach which filled the whole of the abdomen. (Penzoldt, *loc. cit.*) Portal (quoted by Ewald and Pick) states that the stomach of the Duke of Chausnes, one of the greatest "gourmands" of his time, had a capacity of 4½ liters.

The walls of the gastric sac have become thin; in general this thinness is found in all the coats, and microscopically one finds an atrophy of the mucosa; at the same time, the muscularis is now composed only of isolated bunches of muscular fibers separated by the connective tissue. When the dilatation is caused by an obstruction at the pylorus, hypertrophy of the muscular wall is, as a rule, produced first; then interstitial sclerosis comes on, little by little, submerging the true elements, and the final atony of the wall is due to the disappearance of the contractile fibers. An apparent hypertrophy, through exaggerated proliferation of the connective tissue, sometimes masks the actual atrophy of muscle fibers in these cases. The muscular hypertrophy continues very long in the pyloric region, where it also attains its maximum point. The increased resistance and thickening of the walls sometimes results from ulcer, and may simulate a tumor (Rémond).

A dilated stomach may present variable forms due to the action of the special cause. If a cicatricial or scirrhous constriction causes the cardia and the pylorus to approach each other, the stomach will be pyriform; but if the same lesion has plowed a transverse furrow, more or less deep, on the wall, a dilatation in the shape of an hour-glass will be produced; but the symptoms do not differ from those caused by occlusion of the pylorus.

Symptomatology.—The *tongue* is, in most cases, coated by necrobiotic epithelium, mucus, and retained food debris, the breath frequently being very offensive; there is generally a stomatitis,

glossitis, or gingivitis present. We have spoken fully of the various states of the tongue on page 432.

State of the Appetite.—The appetite is normal at the beginning, but when the disease is developed it may be lost, or become considerably decreased. Some patients, for instance, will not need more than one meal a day. In other cases, since the stomach merely plays the part of a reservoir with no outlet, and the foods being no longer evacuated from the stomach into the intestine, digestion and absorption can not occur. In rare instances the patients may be tormented with hunger, and they are in an analogous situation, so far as effects are concerned, to that of persons seized with an impassable stenosis of the esophagus. They try to satisfy their appetite, and, yielding to the solicitation of hunger, actually may present bulimic phenomena. In reality it is not hard to understand this difference, which depends practically on the nature of the obstruction to the course of the foods; anorexia is observed chiefly in cancerous patients and those seized with chronic gastritis, while a cicatrix of a circular ulcer may have obliterated the pylorus without bringing about very serious interference with the special sensibility of the stomach.

Pyrosis.—The regurgitation of a certain quantity of very acid or alkaline ingesta often accompanies the eructations which pass through the cardia, causing intense pyrosis.

Eructations and Gaseous Discharges.—In motor insufficiency of the first degree, the gastric heaviness and the distention give way little by little, and, if the patient takes his meals at regular intervals, his stomach at last empties itself and his pains disappear. But, in motor insufficiency of the second degree, generally the distress ceases only when more or less copious emesis has relieved the gastric cavity of the foods which have burdened it, sometimes for more than twenty-four hours.

To this feeling of fullness are added disgusting gaseous discharges, often very fetid. The alimentary contents, in fact, are liable to set free, in considerable quantity, many different gases. We have cited some examples of these cases in the chapter on the Gases of the Stomach, and Kuhn (*loc. cit.*) has published an interesting study of this subject. The principal gases are carbonic acid, hydrogen, oxygen, nitrogenhydrogen-sulphid, and carbonic dioxid. Whenever there is stasis, their presence may be verified by directly extracting the gas from the stomach by the tube, or by

setting free the gastric contents in a closed vessel, after having extracted them artificially. The causes of this gaseous development are supposed to be the presence of fungi resisting the antiseptic action of the hydrochloric acid even when present in excess, the fungi having been isolated and cultivated. These fermentations, which are very frequent, are modified by salicylic acid or saccharin. Boric acid, carbolic acid, creosote, and chlorin water have no effect except in doses which are incompatible with their therapeutic use. The great quantity of liquid contained in the stomach facilitates the development of anaerobic germs, giving rise to products of fermentation more complex and perhaps more poisonous.

Pain.—The pain of dilatation is not marked; the uncomfortable sensations are those of pressure, fullness, and distention. Naturally, if cancer or ulcer is coexistent with dilatation, pain will be a prominent symptom.

Vomiting.—In motor insufficiency of the second degree, the attacks of emesis are quite characteristic. They are not so frequent as they are at certain stages of the development of cancer or of ulcer, and are generally separated from each other by a variable but comparatively long time, and it is rarely that they occur at the time of the maximum of digestion. The following is the course which matters usually take: For one or two days a patient has suffered, after each meal, from a sensation of growing uneasiness, and from a feeling of weight at the epigastrium, more and more painful; then, suddenly, often toward the middle of the night, he is seized with very abundant vomitings, after which he can enjoy a little rest.

The vomited material is sometimes composed of several liters of a mixture of solid food, drinks, and mucus. The quantity of vomited matter is a first-rate symptom of dilatation, and allows it to be distinguished, for instance, from simple displacements of the stomach. Chronic gastritis, cancer, etc., may also give rise to slight hemorrhages, and in this case the blood, very much modified, remains a long while in the stomach; the same phenomenon can be recognized in dilatation.

Boas has pointed out that the persistent presence of bile and of pancreatic fluid, of which the characteristics have been given, is an indication of stenosis of the duodenum, and is a valuable symptom of dilatation following on the compression of this part of the intestine by a dilated gall-bladder or a movable kidney, for instance.

The vomited matter will have a more offensive smell the longer it has remained in the stomach. Later on in the disease, when the walls are distended, the vomiting comes on at greater intervals, the odor of substances vomited becomes more revolting, and then the emesis is rarely sufficient to evacuate the stomach, the feeling of relief, which at first followed, is no longer experienced. Sometimes the vomitings cease after they have been very frequent —a grave sign of exhaustion.

Symptoms of auto-intoxication from dilatation have been described by Al. Pick (*Wien. klin. Wochenschr.*, 1892, No. 46), Boas (*loc. cit.*, p. 73), and J. Friedenwald (*Med. News*, 1893, Dec. 23). A most exhaustive account of the anto-intoxication with motor insufficiency will be found in the works of Albu (*loc. cit.*) and Bouveret (*loc. cit.*).

In dilatation through an organic cause, the disturbance of the general state will vary with this cause. Thus, it is observed that in cancerous patients the dilatation is accompanied by the most evident cachexia. In ulcer, Reichmann's disease, and chronic gastritis, it will be coincident with an emaciation more or less marked, but no cachexia.

In the case of children, Comby and Moncorro have attributed to dilatation caused by overfeeding a certain part of the development of rachitis. The latter author also considers it to be the cause of certain convulsions, of insomnia, of ring-worms, of urticaria, and of bronchitis.

Constipation.—Constipation is frequent and obstinate, and not only are the stools rare, but the quantity of substances evacuated is also much less than in the normal state. This is a very valuable indication, for it shows the approximate amount of food which passes into the intestines. Thus Kussmaul had already been able to establish the prognosis of the patients attended by him according as, in treating the dilatations, the normal course of the food substances was re-established or not. The latter state indicates an incurable stenosis of the pylorus. Putrefactive diarrhea may alternate with constipation.

Gastrorrhexis—(Rupture of the Stomach).—Newmann (*loc. cit.*), Buist (*loc. cit.*), and Hofman have reported rupture of the stomach and sudden extravasation of its contents into the peritoneal cavity. Rupture may occur after a very sudden, acute dilatation, or in the last stage of one of long standing. The tear generally occurs near an old cicatrix. A case reported by Chiari (*loc. cit.*) had a cicatrix

near the lesser curvature, through which the tear occurred after overindulgence in food. In a case observed by Hofmann (*loc. cit.*), in which a rupture of the lesser curvature had taken place, no other cause but food engorgement was assigned.

State of the Urine.—The quantity passed in twenty-four hours may be reduced to 500 c.c. Boas makes use of the daily quantity for an approximate estimate of the degree of dilatation.

1st degree. Quantity of urine in twenty-four hours, 1500 to 1000 gm.
2d " " " " " 1000 to 500 "
3d " " " " " 500 gm. and less.
The urine is generally alkaline, the chlorids diminished.

The urine is frequently modified in quantity and in quality. The patients are in a state of chronic inanition, and the urea is therefore necessarily diminished. The stomach absorbs little liquid, as the constant thirst by which these patients are tormented testifies; and the dilatation brings about a deficient urinary secretion. Lastly, when the dilatation accompanies an excessive secretion of hydrochloric acid, and the latter is thrown out, either by frequent vomitings or by frequent lavage, the urine becomes alkaline. We have already sufficiently developed this special urology on page 382.

Nervous Phenomena.—Erb found increased galvanic and faradic irritability of all accessible motor nerves with the exception of the facial. The increase of the galvanic irritability of the nerves is a more constant symptom than the increase of faradic irritability. Von Frankl-Hochwart found the latter to be normal at times. Trousseau found that tetany could be caused by compression of the main nerve trunks or of the principal blood-vessels of the limbs, so that the arterial and venous circulation was impeded. When this compression was kept up for two or three minutes, the tetany began, but would cease when the pressure was relieved. Chvostek discovered an increase of mechanical irritability of the nerves in the extremities and also of the facial nerve in particular. This irritability became evident on tapping the nerves lightly with a percussion hammer or with the finger. This mechanical stimulation brought on rapid instantaneous twitchings in the muscles supplied by those nerves. On passing over the face from the temporal regions down to the chin with the finger, distinct twitchings occurred in the muscles supplied by the facial nerve, because this stroke of the finger exerted an irritation on all branches of that

nerve (Fr. Schultze). Tetany of gastric origin has been considered on page 362.

General State of Health.—The general state of health is more deeply influenced by the cause of the dilatation than by the dilatation itself. Neurasthenia often causes an atony of the muscle fibers of the stomach, the consequences of which can not but have a marked influence on the nutrition of the patient and encourage and develop in their turn a secondary neurasthenia. Diabetes, chlorosis, and great pyrexias, which may have caused the atony, provoke general disorders also, and it is difficult to distinguish from among these disturbances that which belongs properly to gastric atony.

Cardiopulmonary Symptoms.—These have been considered in the chapter on the Influence of Gastric Diseases on Other Organs, where we have dwelt on the effects of distention of the gastric cavity by gases hindering considerably the functions of the diaphragm and disturbing the action of the respiratory apparatus and of the circulation, more or less in different cases. Dyspneic phenomena, or modifications in the sound of the heart and in the rhythm of the pulse, are most frequently met with.

Mattheides (*loc. cit.*) has gathered together a number of cases in which he observed a sensation analogous to that of globus hystericus in patients afflicted with dilatation. He called attention to the fact that this sensation was aggravated when the stomach had sunk and diminished; on the other hand, when it had risen, he had concluded that the displacement of the stomach so often accompanying the dilatation of this organ was the cause of this sensation of globus, through the dragging which occurs on the esophagus. Schmidt (*loc. cit.*) is said to have verified, by a laparotomy, the existence of these anatomical disorders in a patient who had previously complained of the sensation of globus. The connection between the two is not at all satisfactorily proven nor even significant.

Percussion and Palpation of the Stomach.—As Osler (*loc. cit.*) emphasizes, the diagnosis is often possible by inspection. Percussion and palpation allow us to ascertain the limits of the lower edge of the greater curvature, and thus to appreciate the degree of the ectasia. The percussion should be performed with the patient standing up, and again when lying on his back. The measured ingestion of a certain quantity of water will allow one

to estimate the atony of the wall, and will at the same time furnish exact data on the displacement of the lower edge of the organ, which has become heavy. The stomach may be distended by CO_2, and the colon by water, thus facilitating the differentiation between the two. Other authors have proposed to perform the operation inversely, and percuss the stomach made heavy by a certain quantity of water, while the colon is distended by gas (Ewald). It is true that these precautions would make mistakes very difficult. Professor Osler holds that when the distended stomach is outlined on the wall one can usually follow its delineations with the eye, and, of course, much better by percussion. In the *Philadelphia Medical Times* for May, 1891, Pepper reports a case of dilatation caused by scirrhus of the pylorus in which there was a visible peristalsis.

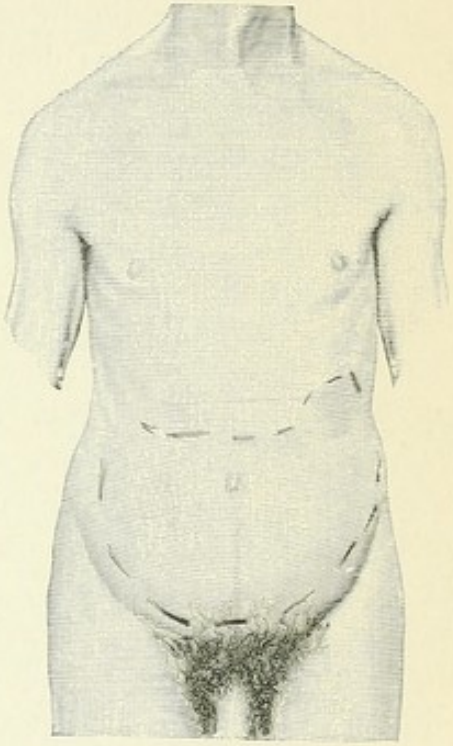


FIG. 39.—DILATATION OF STOMACH.—(*Eichhorst.*)
Outline obtained by percussion.

The gaseous distention has also the advantage that it allows the distinction to be made between true dilatation and a simple displacement of the organ.

By palpation the splashing sound can be investigated. This is easy to perceive when the stomach, the pylorus of which is constricted, is full of those liquid masses already mentioned in connection with the vomiting. But when the dilatation is not very marked, the splashing becomes less clear, and Debove has recently shown that the intestines, when half distended by gases, are capable, under the influence of movements communicated by the fingers, of producing a sound so like that of the gastric splashing as to make the distinction very difficult. Chomele (*loc. cit.*) had already drawn attention to this cause of mistakes, and to that which depends on the presence of liquid and gas in the large intestine: "The splashing in the stomach," says he, "might be confounded with a similar sound of which the large intestine is sometimes the seat, which can be produced by the lateral movement of the body,

but still more easily by the pressure of the hand on the regions occupied by the colon." It is met with especially in subjects who have recently received an injection, and those who have been seized with serous diarrhea. The knowledge of these conditions and the particular source of the gastric splashing sound are sufficient to distinguish it from intestinal splashing. Jaworski (*loc. cit.*) has reported four cases of very audible splashing sound even when, on introducing the probe into the stomach, he had been unable to withdraw any liquid whatever. The author has observed this fact in a number of cases. It is not, therefore, a reliable sign.

For determining the location of the greater curvature, Thiébaut (*loc. cit.*), of Nancy, has devised an instrument which consists of a probe through which slides a thread with a leaden weight. The probe is long enough to reach the cardia, and the quantity of thread taken by the leaden weight before it arrives at the bottom of the stomach allows one to measure the vertical dimension of the gastric cavity.

We have described the methods of procedure based on the employment of salol, oil, iodid of potassium, etc. (p. 72), designed to determine the state of the motor functions and of the absorption of the mucous membrane. In dilatation they give information of varying value, but inferior to that furnished by exploration with the sound. We have already stated the signs by which one can recognize atony of a muscular wall: either presence of debris of food in the morning before breakfast, or the prolonged stay in the gastric cavity of a test-meal. The Hemmeter gastrograph is a graphic method of obtaining motor records from the human stomach, and the results obtained therewith are generally reliable. (Plates III and IV, p. 76.)

Test-meals.—The gastric cavity should be washed out on the evening of the day before a test-meal is given. The substances extracted by this preliminary lavage are sometimes very abundant, and having the same composition as those vomited, generally become separated, when allowed to stand, in three layers: an upper one, frothy and turbid; a middle one, liquid; and a lower one, composed of alimentary detritus of all kinds, or simply of amylaceous substances, as occurs in the case of patients suffering from hyperacidity. Organic ferments and sarcinæ will be discovered, and all the series of products which can, normally or abnormally, be contained in the stomach. If the motor insufficiency is caused by malignant neoplasm, the Oppler-Boas bacillus will, as a rule, be found in this

material. In the morning before breakfast, the gastric cavity, which has been cleansed the evening before, may again contain the normal products of secretion, or material which is rich in organic acids. The digestion of the test-meal will generally be slow, and, especially in cases of cancer, it will be impossible to detect free hydrochloric acid. In other patients a normal or exaggerated state of secretion of hydrochloric acid will be found. When the normal HCl is absent the filtered gastric contents will show excess of lactic and butyric acid. If the motor insufficiency is due to alcoholism, acetic acid will be a prominent constituent.

Diagnosis.—Dilatation or motor insufficiency of the second degree may have to be differentiated from atony, or myasthenia from gastroptosis and physiologically large stomach or megalogastria. However, no sign, unless it is in the presence in notable quantity of food in the stomach before breakfast, is pathognomonic. Bugge (*loc. cit.*) recommended the following operation: he determined, by percussion, the patient standing up, the lower edge of the stomach, and drove the needle of a hypodermic syringe above the discovered limit. If the liquid extracted was acid he concluded that the stomach had been reached. It is evident that this procedure is not without danger, and is not even accurate.

We prefer the simple exploration by means of the sound, associated or not with artificial gaseous distention of the stomach. These methods, in fact, suffice to distinguish a dilated from a displaced stomach, and from a naturally large stomach. But if they do not, the method of the author will leave no room for doubt. (See p. 76.)

Gastroptosis (displacement of the stomach) will be fully considered in a special chapter on that subject. The pylorus may be displaced, and freely movable below the epigastric region, without in reality causing any gastric disturbance. By palpation and percussion the greater curvature of the stomach can be made out below the umbilicus. One might then be very much disposed to suspect a dilatation, but on setting free carbonic acid gas in the gastric cavity, one sees not only the greater, but also the smaller curvature outlined under the skin, and the outline of the whole stomach can be traced on the abdominal wall, following the limits of the corresponding resonant zone by percussion.

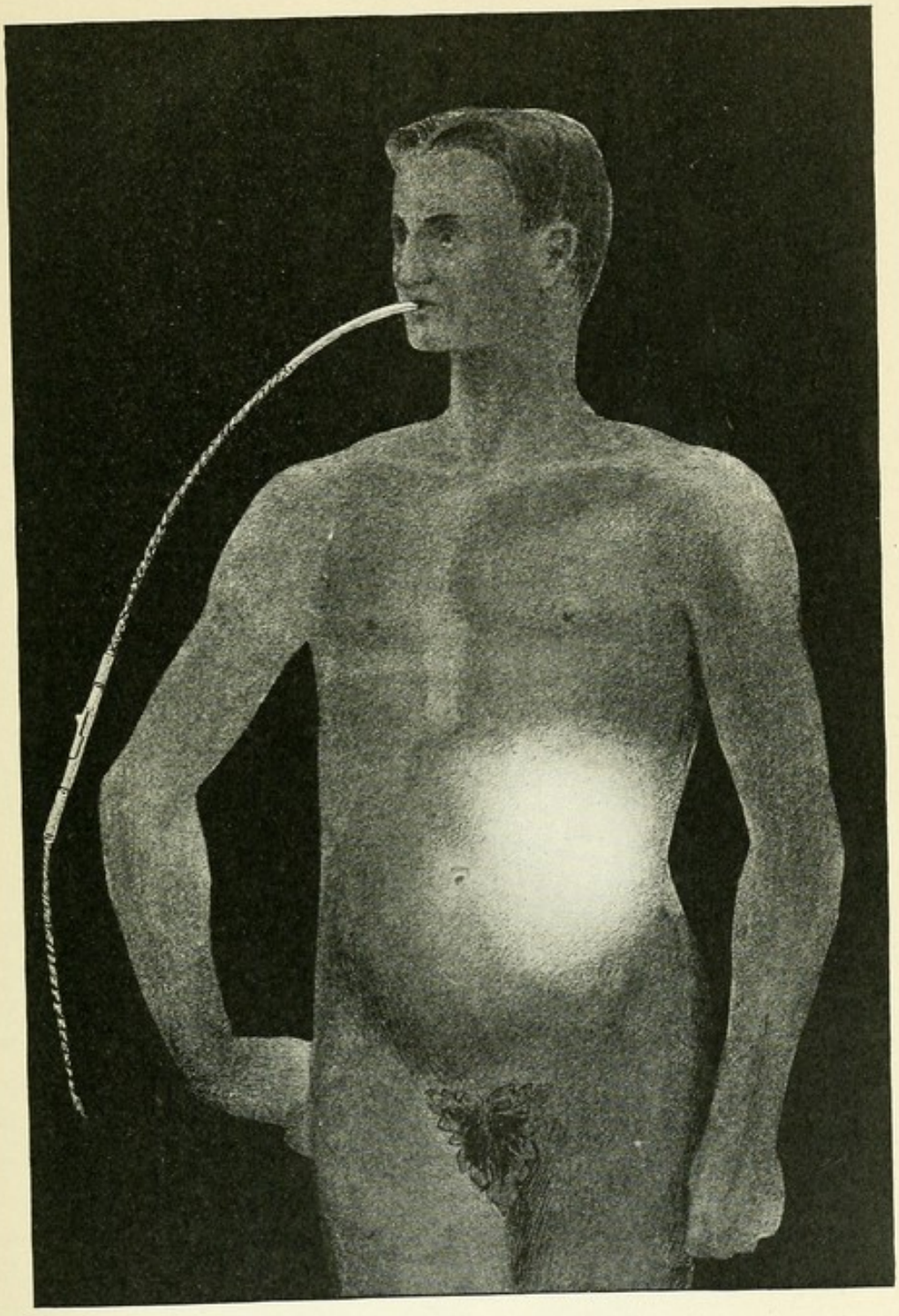
The points of difference between a dilatation and atony are the following: In the morning, before food has been ingested, the

dilated stomach contains an accumulation of putrefactive products and food material, showing either excess of lactic and fatty acids or, when these are absent, abnormal amounts of HCl. In atonic dilatations and in simple atony the stomach is, as a rule, entirely empty in the morning,—in rare instances it may contain trifling amounts of food, but not in a state of decomposition. In atony the bowel evacuations are less likely to be so few in number and small in amount. In atony the total quantity of urine voided in twenty-four hours is normal or only slightly reduced. In dilatation the amount of urine is subnormal, concentrated, and contains diacetic acid and aceton, which are very rarely found in atony.

In megalogastria we are not dealing with a diseased stomach, and hence a differentiation is unnecessary. Tetany occurs only with dilatation, but gastric vertigo is frequent in atony. The differentiation between a dislocated and a dilated stomach is facilitated by the clinical history. In a dislocated stomach the motor function is frequently normal, and hence we find that emesis, if it occurs, does not bring out such very large amounts as in dilatation. Diuresis and thirst are normal in gastroptosis; in dilatation thirst is intense, but on account of the regurgitation or vomiting of fluids diuresis is subnormal. No matter where a stomach may be located within the abdomen, or how large it may be, it does not become abnormal until the motor function is interfered with. A Leube test-meal or our double test-meal (see p. 111) will instruct us concerning these points. (For methods to ascertain the location, size, and capacity of the stomach see pages 97 to 104.)

The differential diagnosis between gastroptosis or prolapsus of the stomach, gastrectasia or dilatation, and megalogastria or large stomach (giant stomach), can be facilitated by distending the organ with air or gas, but even here the success of inspection, palpation, and percussion will depend upon the thickness and resistance of the external abdominal wall. Where there is very little or no emaciation, it is by no means easy to palpate through the abdominal wall. Then, again, much gas escapes into the intestine when the stomach is distended by effervescent mixtures.

But by means of the stomach-shaped intragastric rubber bag, which has been described on page 76, or by Einhorn's electrodiaphane (p. 102), it is possible to make the differential diagnosis without much difficulty. By the Hemmeter apparatus, which was originally designed to obtain records of the gastric peristalsis, it is



GASTRECTASIA.

Transillumination (electrodiaphany) of the stomach. The organ is filled with 600 c.c. of water, and has sunk downward to the left. The electric lamp has just reached the fundus—on being pushed further the intensest part of the transillumination would appear below the umbilicus.

also possible to measure the capacity of the stomach by determining the amount of air required to distend it within the stomach (see p. 101 on this subject). An intragastric rubber balloon was first used for this purpose by Schreiber, of Königsberg. This will at once enable one to diagnose a dilated stomach from one which has prolapsed but retained its normal capacity. Einhorn's diaphane is a practical method for demonstrating these two conditions to the eye. The Roentgen rays are also available for the same purpose, as demonstrated by the author (Hemmeter, "Photography of the Human Stomach by the Roentgen Rays," *Boston Medical and Surgical Journal*, 1896). The greater curvature may be outlined by photographing a metallic spiral electrode that has been introduced and made to apply itself along the greater curvature, according to suggestions first made by Wegele; or the stomach may be photographed by our method, which consists in distending the stomach by an intragastric stomach-shaped bag, the inner surface of which has been previously coated by a deposit of plumbic acetate or nitrate of silver, which is poured into the bag in a saturated solution and allowed to dry on the inside. The bag is then introduced and distended by air, filling out exactly the entire stomach. The thin coating of plumbic acetate cuts off the Roentgen rays sufficiently to obtain a photograph. This method is troublesome, and can be satisfactorily executed only in hospital practice. For private practice the Einhorn electrodiaphane is most expedient, as it permits a diagnosis to be made by inspection. Debove and Rémond ("Maladies de l'Estomac," p. 87) state that this method is difficult of execution, and imposes much suffering upon the patient. From what we have seen almost weekly with Einhorn's apparatus, we differ emphatically from these observers, and believe a further experience with the apparatus will effect a change in their opinion.

Diagnosis of the Cause.—The cause is more difficult to detect than the dilatation itself. In this connection we refer to what has been said in the consideration of ulcer carcinoma, benign neoplasms, etc., and their respective diagnosis.

The anamnesis, the examination of the substances vomited, and the results furnished by test-meals will provide the principal data. The clinical history differs, in fact, considerably, according as one finds a dilatation of cancerous origin or one caused by ulcer or gastritis. The ingestion of poisons and their effects have been considered

under Toxic Gastritis. The corrosive poisons frequently effect a cicatricial contraction of the pylorus. If hydrochloric acid is present in normal or excessive quantity, one can eliminate cancer almost with certainty, especially if the substances vomited contain, at the same time, bile. If the bile is always wanting in the substances vomited, or in the gastric contents either before or after eating, one will be led to think of constriction of the pylorus; the latter will probably be of cancerous origin if the hydrochloric acid is wanting at the same time.

The constant presence of bile and of pancreatic juice in the stomach would be a proof that the dilatation is a consequence of a stenosis of the duodenum, which may result from a movable kidney, a fibrous adhesion, gall-stones, pancreatic neoplasm, etc.

The author has devised a method by which a stenosis of the pylorus and of the duodenum can be accurately determined (Hemmeter, "Intubation des Duodenum," Boas, *Archiv f. Verdauungskrankh.*, Bd. II, S. 85. See also this volume, p. 54).

Dr. F. Kuhn, who belongs to Riegel's school (Giessen), has also devised a method for sounding the pylorus, which is, however, a development of a revolving spiral sound first invented by Dr. F. B. Turck, of Chicago. The Turck-Kuhn method is very ingenious and simple, but owing to the revolving of the spiral sound within the stomach, it is a hazardous method in cases where we should suspect open ulcers or carcinoma, since the intragastric revolutions of the sound can readily bruise or tear the ulcer or neoplasm and set up hemorrhage or lead to perforation.

The accuracy of these results, it is true, is by no means absolute, but in practice, in associating them with other data furnished by the elements of the diagnosis of each gastric affection, one arrives at a sufficient approximation.

Prognosis.—The evolution of motor insufficiency varies according to the cause; when it is a case of simple atony of recent date, a proper treatment, of which we shall speak again further on, may bring amelioration rapidly, and even cure. But when it is a case of dilatation with atrophy of the muscular coat, especially when there exists an impassable obstacle at the pylorus, the cure is impossible, except sometimes by operation. The treatment still relieves the painful phenomena, but the inanition makes progress from day to day, and the patient succumbs gradually, unless one of the complications that we have mentioned appears and hastens the end.

Malformations of the Gastric Cavity.—As dilatation is, in reality, a deformity of the stomach, a certain number of malformations and changes of form may be appropriately considered in this connection.

According to Debove and Rémond, *atresia* of the gastric cavity results from diminution of work by the organ, through insufficiency of alimentary contributions. Inanition and constriction of the esophagus or of the cardia will thus have been the first cause of this atrophy. In other cases it is a cancerous infiltration extending over the whole wall, or a chronic gastritis with hypertrophy of the submucosa and of the connective tissue (linitis plastica), or a fibrous, deforming peritonitis, which will have played the same part. The caliber of the stomach thus narrowed sometimes does not exceed that of the intestine. This condition we have referred to in the chapter on Hypertrophic Gastritis. When the upper digestive paths are open, attacks of emesis occur which appear as soon as the quantity of food exceeds the very small volume of the stomach, the small caliber becoming still more evident when one comes to distending it with carbonic acid, or expanding it with the intragastric rubber bag. If stenosis of the esophagus or of the cardia exists, the passage of the probe becomes impossible, and the symptoms of these constrictions assume enough importance to obscure entirely those which might be furnished by the state of the stomach.

The Hour-glass Stomach.—This type of deformed stomach has been frequently reported. We quote the following cases from Rémond (*loc. cit.*). Stoker (*loc. cit.*) has published one case where the stomach, divided into two parts by a congenital furrow, had never, during life, presented any functional disturbance. Iago (*loc. cit.*) has related the story of a patient who succumbed when forty-two years old, after having presented for ten months uncontrollable vomitings; on the examination of the abdomen, there was found underneath the liver a soft tumor, which had been taken for the right kidney, displaced; there existed no tumor at the level of the pylorus; emesis took place without pain, and was not preceded by regurgitation; there was no cachexia to be found. At the autopsy there was found a stomach presenting two dilated sacs, which communicated by a closed narrow passage situated about the middle of the organ; the index finger could not pass this constriction. The cicatrices which had produced this deformity must have been

caused by a former disease, which appeared at the age of thirty and was characterized by hematemesis and acute pains. A patient fifty years old, observed by Luigi Mazotti (*loc. cit.*), experienced such intense pains after meals that she used to squirm on her bed, and only found relief after having vomited everything that she had just taken. At the autopsy the stomach was found divided into two parts, the upper one vertical, the lower one directed horizontally toward the right side; a narrow passage was situated between the two parts. The lower portion of the stomach had made a complete circle, and the contracted point was exactly the center around which this rotation had occurred. The upper part of the stomach was distended by gases; the lower one was empty and joined to the abdominal wall by adhesions. When the viscus had been replaced in its normal position, it was found that neither the orifices nor the wall presented any modification, and it was impossible to discover the cause of this displacement. In another case, Chiari (*loc. cit.*) suspected a cancerous constriction of the pylorus in a patient who, in reality, had an intussusception of the stomach into the duodenum. Fleiner (*loc. cit.*) has described and pictured a number of gastric deformities caused by tight lacing (*Schnürmagen*). In some of his types the pylorus and cardia are so compressed and brought near to each other by the lacing as to be almost in juxtaposition.

Further particulars concerning similar cases can be found in the recent memoirs of Bauermeister (*loc. cit.*) and Saundby (*loc. cit.*) and in the theses of Kern (Inaug.-Dissert., Berlin, 1881) Chiari (*Wien. med. Wochenschr.*, No. 42, 1890), Bauermeister (Inaug.-Dissert., Halle, 1790). Von Hacker, in his monograph ("Magenoperationen," etc., published by Braumüller, Vienna), gives a number of illustrations of stomachs divided into three parts by cicatrices or adhesions. (Tilger, *Virchow's Archiv*, Bd., CXXXIII. See Bibliography at end of this chapter.)

Treatment of Motor Insufficiency of the First Degree (*Gastric Atony or Myasthenia*).—*Prophylaxis.*—The muscularis of the digestive organs may be weak by inheritance. Chlorosis, anemia, tuberculosis and cholelithiasis, exhausting hemorrhages, infectious diseases, typhoid, malaria, diphtheria, influenza, may bring on myasthenia; and frequent and rapidly consecutive births may, by causing increase of space in the abdominal cavity and loss of tone to the abdominal muscles, lead up to gastric atony. Bad chewing, hasty eating and deglutition, and defective teeth predispose to atony. The

treatment in all cases must seek and adapt itself to removal of the cause. Anemia must be treated by proper food, peptonate of iron, extract of bone marrow, and arsenic. In women with gastroptosis and atony, the abdominal muscles must be strengthened and supported by proper bandages. The treatment proper includes diet, hydropathic and electric procedures, massage, and medicines.

Diet.—Patients with gastric atony must eat frequently, but very little at a time. As water is not absorbed from the stomach, the quantity of liquids must not exceed one to 1½ quarts in twenty-four hours, including all drinks, coffee, soups, etc. When there is a craving for more liquid than this, it should be introduced by enema.

Our experience with the frequent and persistent administration of milk as observed in milk cure sanitariums in Germany is discouraging. We believe this treatment to be a useless dietetic experiment in gastric atony.

The special diet must be selected according to the state of the gastric secretions. If there is hyperacidity, a vigorous beef and mutton diet, with limited carbohydrates, hard- and soft-boiled eggs, ham, tongue, oysters, duck, and deer in every form is recommended; of vegetables, we allow carrots, spinach, soft-boiled turnips, beans, peas, cauliflower. Potato, macaroni, rice, and farina gruel are permissible. If the hyperacidity is the cause of the atony, we favor restriction of proteid diet and a preponderance of amylaceous food, according to principles laid down elsewhere (pp. 188, 189). We are strict concerning the use of alcohol, and where a trial proves that it injures digestion, we generally forbid claret, rhine wine, and even small amounts of beer. But where a trial with light wines demonstrates their beneficient action, about two ounces of wine with each meal may be permitted. Whenever the hydrochloric acid is diminished, the lighter meat varieties,—chicken, pigeon, birds,—fish, and boiled sweetbreads or calves' brains, should be allowed only, but a larger amount of carbohydrates conceded. The special diet is stated more explicitly in the chapter on Dietetics (p. 228).

Constipation is a serious cause and constant accompaniment of gastric atony; it must therefore receive the most undivided attention. Purgatives should be used only as a last resort, and the main reliance placed on diet. A pint of cold water, preferably Bedford Magnesia Spring Water, in the morning on an empty stomach, black (rye) or graham bread, abundance of vegetables,—turnips, carrots, asparagus, tomatoes, rhubarb plant, beans, peas, lentils,—noodles,

macaroni, barley, sweet compotes, plums, figs, apples, currents, cranberries, cider, buttermilk, kefyr, sour milk, honey. When sweetening is desired, milk-sugar should be preferred to cane-sugar. The use of these articles very rarely fails to bring about regular passages without medicines (see p. 236). Whenever a drug is positively unavoidable, we prefer cascara sagrada. In pronounced atony, constipation can not be treated by this diet only, because it increases the weight of ingesta.

The hydropathic treatment consists in cold morning sponge baths, cold wet packs, and *Priessnitz* bandages to epigastrium. We are in the habit of ordering a daily bath in severe neurasthenic myasthenia which contains three per cent. chlorid of sodium and two per cent. sodium bicarbonate. Temperature of bath 98° F.; to remain in twenty minutes. When taken in the evening this bath favors sleep.

Electric Treatment.—Intragastric application with the Einhorn electrode within the stomach; the faradic current is applied up and down over the spinal column and over the abdominal muscles. The constant current is applied in the same manner in the strength of 20 milliamperes and for about ten minutes. Systematic massage, both general and local, over the stomach is an important adjuvant. (See chapter on Electrical Treatment, pp. 288–295.)

Medicinal.—This form of treatment should be as limited as possible. The most approved tonic for the motor function is strychnin.

℞. Strychnin sulphatis, ... 0.021 gm. gr. ⅓
Elixir gentianæ cum ferri chloridi, ... 180.0 c.c. ℥vj. M.
Sig.—f℥ss, t. i. d.

Where the hydrochloric acid is deficient it must be supplied; where it is excessive it must be neutralized by the following:

℞. Magnes. ust., ... 15.0 ℥ss
Bismuth carbonic,
Natron bicarbonat., ... āā . . . 5.0 ℥j + gr. xv
Strychnin sulphatis, ... 0.1 gr. iss. M.
Sig.—One-half teaspoonful one hour after meals.

Creosote and orexin are claimed by competent authorities (Pick and Penzoldt) to be able to excite the peristalsis; the latter may be used where there is anacidity or subacidity.

Lavage.—As a rule, one will be able to get along without lavage in the first stage of motor insufficiency. But where the food re-

mained in persistently over time, we have seen improvement of muscular tonicity follow the rapidly alternating cold and warm intragastric douche (p. 286). This exerts a powerful and stimulating effect also on the secretion when it is defective; when the latter is excessive, the douching should be carried out with alkalin water.

Treatment of Motor Insufficiency of the Second Degree. (*Fully Developed Dilatation*).—This may be considered under three headings: (1) Dietetic, (2) Medical, (3) Surgical.

The diet is essentially based on the same principles as in simple myasthenia; the amount of liquid permissible must not exceed 1500 c.c. in twenty-four hours. With exaggerated vomiting and pains, we would recommend exclusive feeding by the rectum for fourteen days. A specified diet list for both simple atony and pronounced dilatation will be found on pages 226 to 229. It is impossible to treat the latter form successfully without *lavage;* this is not only a palliative measure of great value, but in cases of atonic dilatation due to muscular weakness, and not dependent upon mechanical obstruction, it may even be able to effect a cure, when combined with other means presently to be described.

The first washings are carried out with pure warm water, but the last ones are done with solutions adapted to the chemical and septic states present in the organ. For instance, if there is great excess of hydrochloric acid and fermentation by sarcinæ and yeast, sodium biborate or bicarbonate should be added, as these salts are not only antacid, but, with regard to these organisms, antiseptic. If there is butyric or lactic acid fermentation, boracic acid, three per cent., salicylic acid, 0.3 per cent., creolin or lysol, 10 to 15 drops to a quart, should be used; but the stagnation can not be prevented from recurring by these means unless the motility is improved by other treatment.

Electricity should be employed externally and internally, as described in a previous chapter devoted to this subject (p. 288).

Massage undoubtedly improves the gastric musculature, but should only be used on days when the stomach has been washed out, because the mechanical compression may force stagnating masses into the intestines, thus spreading the putrefaction. Abdominal bandages properly adapted and applied have proved a valuable palliative measure. Hydrotherapeutic applications are indispensable, and should be used as described in the paragraph devoted to the consideration of that treatment (p. 295).

Medicinal treatment has a twofold object: (1) To promote the motor function; (2) to prevent, so far as possible, gastric fermentation and decomposition. The only drug in which we have any faith for improving gastric peristalsis is strychnin sulphate; it should be given in heavy doses, not less than $\frac{1}{24}$ of a grain for adults, t. i. d.

Boas combines strychnin with an antifermentative in the following manner:

℞. Strychnin sulphatis, ... 0.0022 gm. gr. $\frac{1}{30}$
Codein phosphoric, ... 0.03 " gr. $\frac{2}{3}$
Bismuth salicylici basic, ... 0.5 " gr. viiss. M.
SIG.—One powder taken after each meal.

F. Kuhn has proposed salicylic acid, 0.5 gm. a dose, salicylate of sodium, 15 to 30 grains, saccharin and sodium benzoate, of each from 10 to 30 grains a dose, to counteract gastric fermentation. Carbolic acid was first used by Naunyn for the same purpose. When there is marked lactic or butyric acid fermentation, there is not a better agent than hydrochloric acid to counteract it; 20 to 30 drops of the dilute form in ℥ij of water, through a glass tube. Among other remedies that are recommended are: Salol, naphthol, betanaphthol bismuth, and betanaphthol bismuth benzoate, or benzonaphthol, hydrochloric and carbolic acid. Bouchard is very enthusiastic concerning antifermentative treatment of gastrectasia, but it is certain that this treatment alone, without lavage and proper diet, is fallacious.

Dujardin-Béaumetz employs:

℞. Bismuth salicyl.,
Magnes. usta.,
Sod. bicarb., ... āā 10 150 gr. M.
SIG.—To be divided into 30 powders; one powder after meals.

Our formula for gastric fermentation, particularly when associated with putrid diarrhea, is:

℞. Betanaphthol. bismuth. benzoatis, ... 8 ℥ij
Bismuth salicylatis, ... 8 ℥ij
Magnesiæ ustæ, ... 8 ℥ij
Saccharin, ... 1 ℥j
Menthol, ... 8 ℥ij. M.
SIG.—To be divided either into 12, 24, or 36 powders to suit the indications; if there is much fermentation, it should be divided into 12 powders, and one given three times daily. Otherwise it should be divided into 24 powders, and one given every three hours.

Ewald's formula for prevention of gastric fermentation is the following:

℞. Resorcin resublim., ... 5.0
Bismuth salicyl.,
Pulv. rad. rhei,
Natrii sulphur, ... āā 10.0
Sacch. lact., ... 15.0. M.
SIG.—Make a powder; a knife-blade full twice daily.

When HCl secretion is lost, dilute HCl should be administered, according to the formula given on page 450; if HCl is not well tolerated, pancreatin should be tried according to the principles given on page 451.

For improving the appetite, strychnin, orexin, HCl, and lavage, are the most approved means of therapy.

For vomiting, lavage is the most efficacious treatment, but if it fails resorcin, gr. ij in ℥ss of chloroform water, or a hypodermic injection of morphin and atrophinæ sulphatis will be called for. As a rule, menthol and chloroform do not disappoint when used for the relief of vomiting. The following formula is practical:

℞. Mentholi, ... 1.0 gr. xvj
Chloroform, ... 1.5 gtt. xxiv
Elixir simplic., ... q.s. 60.0 f℥ij. M.
SIG.—f℥ij every hour.

Insomnia must sometimes be treated, as these patients imperatively need rest; for this purpose chloral, grs. xv, per enema, is most advisable. Correction of hyperacidity will often induce sleep. Sulphonal and chloral combined, of each eight grains, will produce a more lasting sleep than if either is used alone. Trional is recommended for the same purpose by Boas.

Surgical Treatment.—The operations that have been suggested for the relief of motor insufficiency vary according to the object to be accomplished. Motor insufficiency from simple atonic dilatation may be relieved by reducing the size of the stomach by excision of a piece of the same,—an operation known as gastroplication or gastrorrhaphy (Weir).

If the pylorus is stenosed by a simple cicatrix or hyperplastic sphincter, Loreta's digital divulsion of the pylorus is an operation which, judging from the statistics, is an unsafe and unreliable procedure. The pyloroplastic operation of von Heinecke-Milkulicz, which Boas terms the ideal surgery for the relief of pyloric stenosis of a benign nature, produces more permanent results.

Gastro-enterostomy and resection of the pylorus, as well as gastrorrhaphy, an operation originated by Dr. Heinrich Bircher, a Swiss surgeon, will come under consideration. The indications for these operations, and their technic, are subjects concerning which the reader must be referred to the chapter on Gastric Surgery. The larger portion of dilatations are undoubtedly due to some obstacle to the exit of the chyme (ischochymia, as Einhorn calls it), and it is rational to presume that purely medical means can not effect a permanent cure of these conditions. But the obstructions or obstacles to the chyme are not all found in the stomach itself, for in the account given under the etiology, distended gall-bladder, gall-stones impacted in the diverticulum of Vater, floating kidney, duodenal cicatrices and neoplasm, peritoneal adhesions, etc., have been referred to, and all of these give their separate and distinct indications for operation.

DIET FOR MOTOR INSUFFICIENCY OF THE FIRST DEGREE —ATONY—MYASTHENIA (*Boas*).

	Calories.
8 A. M.—100 gm. of milk, 50 gm. of toast, 30 gm. of butter,	401.2
10 A. M.—50 gm. of wheat bread, 30 gm. of butter, 60 gm. of scraped beef,	415.2
12 M.—150 gm. of boiled beef, 50 gm. of potato purée or macaroni,	439.3
3 P. M.—100 gm. of milk, 50 gm. of Zwieback,	401.2
7 P. M.—100 gm. of cold ham or beef, 150 gm. of wheat bread, 30 gm. of butter,	557.5
Total,	2214.4

About three ounces of good port wine or claret may be allowed during the day.

DIET FOR MOTOR INSUFFICIENCY OF THE SECOND DEGREE —PYLORIC STENOSIS—MYASTHENIC DILATATION (*Hemmeter*).

	Calories.
8 A. M.—100 gm. of Mosquera's beef chocolate, or 50 gm. of tea with 50 gm. of milk, sweetened with saccharin, no sugar, 50 gm. of toast,	195.5
10 A. M.—100 gm. of scraped lean beef,	437.0
30 gm. of toast,	77.7
10 gm. of butter,	71.3
Total,	586.0
12 M.—150 gm. of roast beef,	320.7
50 gm. of potato purée,	63.7
Total,	384.4

In place of the potato purée, the same of spinach, carrots, peas, or beans may be allowed in the same quantity.

	Calories.
2 P. M.—50 gm. of cream,	107.3
4 P. M.—100 gm. of tea or coffee with milk, no sugar, but saccharin, 50 gm. of toast,	195.5
7 P. M.—100 gm. of broiled white or yellow perch or oysters,	71.75
50 gm. of wheat bread,	129.0
10 gm. of butter,	71.3
100 gm. of cream,	214.0
9 P. M.—50 gm. of cream,	162.3
Total,	1885 15

In atony and dilatation, as well as in carcinoma, experience is the best guide for enlarging and varying the diet. Every new article of diet must at first be tried with great caution; if liquids are well tolerated, they may be increased, and soups allowed for the noon meal. The daily lavage should at times be undertaken at hours when a test-meal can be secured thereby, which will incidentally instruct the physician concerning the digestibility of new foods and, what is more important, the state of the motor function.

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CHAPTER VIII.

ENTEROPTOSIS—GASTROPTOSIS.

There is a very extensive piece of work concerning the diseases of the abdomen in the fifth volume of *Virchow's Archiv*, in which this subject of dislocations is treated at great length in connection with two other questions, which, we believe, have not been sufficiently emphasized; namely, first, in connection with partial chronic peritonitis, and, secondly, in connection with the acute diseases of the mucous membranes, especially with diphtheric forms.

Partial peritonitis of the female sexual organs, and especially their appendages, comes under consideration in connection with dislocation of the transverse colon and stomach. In almost all adults partial states of dislocation of the viscera, and especially of the intestines, occur so frequently that more people have this displacement than a normal location of the intestines (Virchow, *loc. cit.*). The French authors are justified in assuming the great frequency of these dislocations. Undoubtedly, the majority of all civilized peoples have a certain deviation in the location of their intestines, or, in other words, some slight degree of enteroptosis. While it is undoubtedly true that the majority of these dislocations are due to sinking,—that is, ptosis, or a descensus of the intestines,—nevertheless the contrary is also found. There are also dislocations which move upward, in which, for instance, the splenic flexure (flexura linealis) comes to a position above the spleen immediately next to the diaphragm, and others in which the hepatic flexure moves upward far under the liver. This upward distortion of the intestines should also receive careful consideration. It is evident, however, that every decided change in situation of this sort—especially if it is at the same time accompanied by kinking, or if considerable deviation in the direction of the intestines occurs (or, as it has been expressed by Virchow, when "anomalous flexures" arise)—must bring about an interference with the passage of the contents of the intestines; and therefore nothing is more fre-

quent than to find, at the necropsy, collections of fecal matter just at these angles and flexures, or that accumulations of gases occur while adjacent parts of the intestines are contracted. Thus we get a picture in which much contracted, one may even say spastically contracted parts of the intestines alternate with much dilated portions. It must be emphasized that the colon is the main seat of difficulty, and the dislocation which occurs most frequently is a lowering of the transverse colon, which often sinks under the navel and sometimes even to the true pelvis, and then forms a V-shaped loop, or one with two parallel legs. The next most frequent point is the iliac flexure, which may show all the possible varieties of descents and displacements toward the right. The two large flexures in the upper part of the abdomen—the hepatic and the splenic—are third in the order of frequency of dislocations. The cecum may also be drawn into similar displacements, and may sometimes move under the liver, and at other times sink down to the true pelvis. These states are relatively frequent, although little attention has been given to these very chronic conditions, because it is not known to what extent certain symptoms in life are connected with them, and, as a rule, no one dies from these displacements.

In the treatise by Virchow, before mentioned, it was distinctly proven that anomalous adhesions frequently occur simultaneously with these states; for instance, growing together of the intestines with each other—that is, the various curves and loops among themselves, and at other times with the adjacent organs. The hepatic flexure of the colon to a great extent becomes connected with the gall-bladder and the whole apparatus of the evacuating gall passages; and, on the other hand, the splenic flexure comes into close connection with the spleen and the diaphragm, and the iliac flexure with the sexual organs, especially in women. There are a number of reciprocal relations which establish beyond a doubt that tractions must result from them, such as pulling of the various parts among one another, which under certain circumstances may develop injurious effects.

The relation of partial peritonitis to visceral dislocations is more difficult to understand, and in this respect two relations are to be distinguished; namely, a primary one, in which peritonitis occurs earlier, and a secondary one, in which, conversely, the peritonitis is

caused by the dislocation and by the other processes going on within the adherent part of the intestines.

Concerning the first, we have a clear example in the recognized cases of circumscribed peritonitis, which are caused by processes starting from the gall-bladder (perihepatitis, peritonitis cystica), where adhesions are formed within the environment of the gall passages; this is followed by a shifting of the parts among each other, since the adhesive masses gradually contract and the retraction proceeds further and further. On the other hand, secondary peritonitis is much more difficult to prove in cases where one is confronted with the completed process; one can only recognize it where fresh processes still exist. These recent processes exist mostly in those cases where a severe disease of the mucous membranes, extending to the peritoneum, causes a bending or kinking of the intestine.

Virchow was the first to draw attention to these facts in studies on dislocations with so-called diphtheritic dysentery, which was, formerly, as a rule, considered a disease of the rectum (proctitis) until it was ascertained that dysentery, similarly to other forms of intestinal inflammation, may appear distributed in a variable manner, so that the foci of inflammation are separated by long stretches of normal mucous membrane. Entirely normal sections are succeeded by new areas of very severe disease, so that one may distinguish a sort of interrupted localization. The questions were raised, How did this interruption arise? how did it come about that new areas of disease are formed at intervals? Virchow suggested that the anomalous flexure, just as the normal, is in itself a motive for localization, in that it brings with it a retardation in the passage of the contents of the intestines, which contain injurious substances that react upon the mucous membrane, and from which the irritative process is developed. It is exactly the same thing which we see in stenoses, where further disturbances arise above the obstruction, or with incarcerated herniæ, where the inflammation develops in the intestinal part above the incarceration, and sooner or later extends to the peritoneum.

These consequences of partial enteroptoses due to acute inflammations are easily recognized. In this way severe secondary peritonitis may arise (*Archiv für pathologische Anatomie u. Physiologie*, 1871, l.ii, 34).

From these views we may infer that enteroptosis is not an

anatomical process which is connected with constant clinical symptoms. On the contrary, the symptoms will probably be very manifold, according to the special pathological circumstances which occur in various cases. Accordingly, the disease in each case will, to some degree, come under different categories, and one may not bring diphtheria or colitis, developed in anomalous flexures, into the same category as dislocation pure and simple; these are two very different things (Virchow). We shall, therefore, have to decide not to call the enteroptosis an entity, but to divide it into several groups of diseases when it is to be regarded symptomatologically and therapeutically. We are dealing here with a very common and, if we could count the cases, an almost normal series of deviations, which only from time to time become the origin of severer symptoms.

Leshaft has shown that the size and fullness of the abdominal organs, as well as the condition of the abdominal integument, are important for the reciprocal conduct and relation of the intra-abdominal organs.

Every abnormal fullness or increase in size of one of these organs, as well as a decrease in the power of resistance within the compass of the abdominal integuments, will produce a change in location,—a sort of ptosis, in the sense of Glénard. It has been proved that a descent of the transverse colon, especially the right flexure, carries with it a lowering of the stomach and a descent of the right kidney. It is evident that the transverse colon does not descend spontaneously (idiopathically) without pathological causes, so that there can not well be a primary enteroptosis; it is caused by etiological factors, such as abnormal adhesions, change of contents (coprostasis), anomalous flexures (Virchow), tumors, etc., by which the transverse colon is pulled down, and with it, secondarily, the stomach and small intestine, and eventually the kidney, especially on the right side. Therefore most enteroptoses, if not all, are of secondary nature.

Dislocation of the Kidneys.—Litten, in 1887, published a monograph concerning the relation of diseases of the stomach to changes in location of the right kidney, in which he emphasized the facts: "Concerning the anomalies of location of the kidney, one must sharply distinguish dislocation and movability." Although both irregularities often occur together and simultaneously, there is no organic necessity for the same; rather,

both processes may appear entirely independent of each other, although in many cases one finds a dislocated and a movable kidney in the same individual. Litten distinguishes the following relations concerning the location and movability of the kidney: First, a simple dislocation of the same; this is more frequently congenital than acquired. The congenitally dislocated kidney is found more frequently on the left than on the right, and with approximately equal frequency in men and women. Often both organs are dislocated. Not taking into account the most frequent form of dislocation, the so-called horseshoe kidney, in which both organs are united into one, the dislocated kidney is found either close under the bifurcation of the aorta, or above the promontory of the sacrum, or, finally, above the sacro-iliac synchondrosis. With the change in location there is almost always connected an anomaly of the origin or course of the renal artery, while the suprarenal capsule more frequently remains in its place and does not follow the kidney to which it belongs. Congenitally dislocated kidneys are almost always fixed in the place of their dislocation. An exception to this is the movable horseshoe kidney.

Acquired dislocations of the kidneys are chiefly due to pathological enlargements of neighboring organs (spleen, liver, pancreas, suprarenal capsule), and are found higher or lower than the normal and nearer to or further from the vertebral column. The pressure of articles of clothing (such as corsets, belts, girdles, etc.) seems to have considerable influence, by which the liver, and with it the kidney, is pushed down. Consequently this anomaly of location is found less frequently left than right—more frequently in women than in men. By the sinking of the liver—*e. g.*, in consequence of hydatid cysts—the kidneys may be completely turned around, as a result of which one may feel the inner edge with the hilus upward, the convex edge downward, or pointing in some other abnormal direction. Most frequently in this form of dislocation one finds the kidney pushed downward and inward; *i. e.*, toward the median line. This form of dislocation of the kidney, acquired late in life, may become movable; it is almost always replaceable if it has not become fixed in its new location by secondary inflammation. While one might describe the above-mentioned forms as dislocation of the kidney with and without movability, we now come to the forms in which the movability plays the main rôle, dislocation the secondary kind—movable

kidneys with and without dislocation. Here we have to distinguish two main classes: (1) the wandering or floating kidney; (2) the movable kidney.

The *floating* kidney is distinguished by the mesonephron, a mesenteric fold fastened to the kidney, which generally consists of two plates, between which the organ is held and with which it is surrounded. The presence of this anomaly is always to be traced back to a congenital disposition of the peritoneum, with consequent stretching of the renal vessels. Generally one finds at the autopsy in these cases that all the folds, protrusions of the peritoneum and mesenteries, are abnormally long and lax, and the foramen of Winslow is very wide, corresponding to the laxity of the lesser omentum and the ligamentum hepatico-duodenale.

If one uses this anatomical arrangement for the classification, and does not designate at will every excessively movable kidney as a "floating or wandering kidney," as is frequently done from a clinical point of view, it is evident that every floating kidney, in the sense adopted by Litten, must be congenital. It will hardly be possible, therefore, during life to distinguish these two processes from each other; on the other hand, there will also be cases in which a very short and tight mesonephron will restrict the extent of wandering of the floating kidney. A simultaneous permanent dislocation of the kidney may be present, but not necessarily; it happens, occasionally, that a floating kidney in the course of time becomes fixed and permanent in any abnormal location by means of perinephritic processes, so that in spite of its mesentery it is no longer movable. Litten has reported a considerable dislocation in a wandering horseshoe kidney, where one portion of the organ lay in the right inguinal region, while the opposite part lay upon the horizontal ramus of the right os pubis. The organ could be pushed about within the widest limits, and it also moved spontaneously, causing unpleasant sensations to the patient. In this intraperitoneal position of the kidney the organ seems to lie immediately under the abdominal integuments, where one can not only palpate it, but can sometimes also recognize its contour distinctly through the abdominal integuments. Percussion over the organ produces in these cases a distinct dullness, not a tympanitic sound, as is the case with the kidney situated extraperitoneally.

The *movable* kidney is distinguished from the normal solely by an excessive movability, which is revealed to a greater or

less degree (not taking into account respiratory movability) in changes in the position of the body. Often this anomaly is accidentally recognized in manipulations instituted for the examination of the abdomen. A dislocation of the kidney can and often does exist simultaneously with the above-mentioned condition, but it does not necessarily have to exist. We find the right kidney more frequently movable than the left (the proportion being 15 to 1), and the anomaly is more frequent in women than in men (the proportion being 85 to 16). The degree of movability seems to be chiefly dependent upon the varying laxity of the part of the peritoneum descending in front of the kidney, as well as upon the abundance or absence of the perinephritic or subperitoneal adipose tissue in the region of the loins, and the greater or lesser power of resistance of the intra-abdominal organs, including the abdominal integuments.

One can often feel the kidney so distinctly that it may be palpated with anatomical exactness, although it lies extra-peritoneally, separated from the anterior abdominal wall by intestinal loops; consequently it gives forth a decided tympanitic sound, although one can press it very close to the abdominal wall. The degree of dislocation is just as variable as the degree of movability; in most cases we find only slight dislocations from the normal position, downward and inward. In by far the greater number of cases this condition is acquired, especially in the years between twenty and forty.

Etiologically, the following factors seem to have the greatest influence: The disappearance of the fat in the adipose capsule in which the kidney is held—through this the kidney becomes movable in this capsule; the disappearance of the perinephritic adipose tissue, through which the kidney, and also its fat capsule, are moved out of place; further, the laxity of the peritoneum, the increase in the weight of the liver, together with the respiratory displacement of the same, prolapsus of the uterus and of the vagina, carcinoma and retroflexions of the uterus, herniæ, weakness and laxity of the abdominal walls, and, above all, the much-discussed enteroptosis. Also heavy lifting, coughing, pressing, repeated pregnancies, vomiting, as well as traumatism and violent agitation, are given as causes. The most frequent cause for the movability of the right kidney seems, however, to be lacing with corsets, belts, and girdles. Fleiner (*loc. cit.*) gives several illustrations of this effect. Thus, von

Fischer-Benzon, in his dissertation from the pathological institute at Kiel, states that in 21 cases of movable kidney there was found in 11 cases a furrow in the liver due to lacing.

By lacing a decided pressure is exerted upon the lower part of the thorax, by which this is greatly narrowed and the organs lying within it compressed. The liver, being the largest and least compressible of these organs, will suffer especially. The liver, and the kidney closely connected with it, are pushed down, and the latter must participate in the respiratory excursions of the liver.

H. Schmid (Penzoldt and Stintzing's "Handbuch d. Therapie," Bd. VI, S. 345) regards the renal vessels as the most important attachments of the kidney—if the vessels are abnormally long, an essential support is lost. L. Knapp (*loc. cit.*) looks upon uterine displacements and consequent dragging upon the ureters as a frequent cause.

Since the respiratory movements of the diaphragm are communicated to the kidney, the respiratory movability of the kidney may be regarded as physiological. The question is: Can we prove this respiratory movability of the kidney, and under what circumstances? This depends especially upon the various individual conditions in the person to be examined.

Method of Palpating the Kidneys.—The chances of feeling the respiratory movability of the right kidney will vary according to the relaxation and the degree of resistance of the abdominal integuments, the control and experience of the patient in breathing, and according to the manual dexterity of the examiner. It is especially important that the individual in question inspire deeply; this can be learned easily enough by practice. The examiner himself must not cause any pain or tension of the abdominal integuments by his manipulations. It will then be possible in many cases, by means of the bimanual method of examination, to feel the kidneys, especially the right one, as it is more easily palpable. It would, however, be entirely false to believe that one could only prove the respiratory movability of the kidneys in females, and especially in multipara. On the contrary, it may be shown with the greatest distinctness in men and girls, and even in small children. In bimanual examination, in order to feel the right kidney the left hand is placed immediately in the rear, under the edge of the ribs on the right side, while the tips of the fingers of the right hand similarly placed

together take the corresponding position at the lower arch of the ribs on the same side; on gradual downward pressure one feels a larger or smaller part of the organ between the fingers during deep inspiration. The deeper the inspiration, the greater the portion of the kidney which appears, until with forced inhalation one may sometimes feel the whole organ pressed out under the arch of the ribs, and can, with the greatest distinctness, examine it by palpation between both hands. If the fingers of both hands are pressed together, the kidney escapes from the hands, and the person examined feels a slightly unpleasant sensation, and sometimes a decided sensation of pressure or pain, or a distinct jerk. Naturally, the abdominal walls must not be too adipose, otherwise palpation is impossible. The knees and thighs of the patient must be flexed. This escaping of the kidney from the hands upon pressure is very characteristic.

With this method of examination one may get a precise conception of the size, consistency, and thickness of the organ; possibly neoplastic formations, lobulations, irregularities, even granulations and processes of shriveling, and especially increase in consistency, size, and diameter. The looser the abdominal integuments, and the more completely the condition of enteroptosis is developed, the more favorable are the conditions for palpation. If the kidney can not be palpated when the patient is in the dorsal position, we have frequently succeeded in palpating it by placing the patient on her hands and knees in bed. The examiner stands on the left side of the bed and patient, facing the head. Both arms are passed around the patient's body; the right hand is inserted beneath the lower edge of the liver while the left seeks to meet it by pressure from a point about two inches above the umbilicus.

Gastroptosis.—Diseases of the stomach are frequently connected with dislocation and movability of the kidney. Ewald and others are of the opinion, supported by observation of numerous successive cases, that the frequency of gastrectasia with dislocation of the right kidney was due always to an etiological connection. The view taken by Quincke, Nothnagel, and Leube is that both pathological processes occur indeed very often side by side, without any causal connection necessarily existing between them. The changes of position (after descent) of the stomach in consequence of so-called enteroptosis and dislocation of the right kidney, are not the

classic gastrectasias which develop in consequence of mechanical obstructions at the pylorus (new formations, cicatrices of ulcers, distortions in consequence of adhesions, compression, obliteration, kinking, etc., page 231 of the Proceedings of the German Congress for Internal Medicine, 1887), but consist in the insufficiency of the pylorus, with deep location and dilatation of the stomach, because the pylorus and the duodenum frequently retain a normal position. If one does not take into account the gastrectasias which are the result of mechanical causes, there still remains a very large number of functional disturbances of the stomach, which lead further to insufficiency of the musculature and to dilatation and low position of the organ. To this class are assigned:

1. Disease of the musculature of the stomach in consequence of protracted chronic gastritis.

2. Excessive exertion of the stomach in consequence of too much and unsuitable indigestible food.

3. Abnormally slow peristalsis with retention of food, as well as the abnormal decomposition of the retained ingesta, with excessive formation of gases.

As a result of these various chronic pathological conditions of the stomach, each of which singly forms only a link in the whole chain, a dilatation of the stomach with attenuation of the walls finally develops, and later a sinking down of the organ and descent of the right kidney, with abnormal movability. The function of the stomach is seriously injured in many directions, especially the motor power, which under some circumstances ceases entirely.

Litten states that he has observed a great number of individuals, especially females, where one could recognize, even six or seven hours after taking a cup of milk or coffee, a very distinct and widespread splashing sound, reaching down below the navel.

In such individuals, who had, under the supervision of the physician, taken 200 gm. of milk and coffee mixed at seven o'clock in the morning, and were then kept under constant observation, during which they took in absolutely no liquids, there could still be heard a loud splashing between two and three in the afternoon, and by means of the siphon 170 gm. of liquid were recovered. Such conditions (called by the French "dyspepsie des liquids") concern only liquid food, while solid foods are digested, though much more slowly than normally. It was thus possible to effect a nearly

normal digestion of discs of albumin placed in the gastric juice pumped out of the stomach. These observations of Litten are more intelligible in the light of von Mehring's experiments (see part first), according to which it is certain that no absorption of water takes place from the stomach even normally; and for the resorption of substances that can be taken up by the mucosa, a return secretion of liquid takes place from the mucosa into the stomach. So that it sometimes happens that after six or eight hours more liquid is drawn out of the stomach than was ingested. The digestive power varies in these cases with the state of HCl; it may be fairly good and it may be entirely absent. There are undoubtedly cases of gastroptosis in which the dislocated stomach functions normally.

The complexity of symptoms which lies at the bottom of the so-called Glénard's disease is so frequently traced to nervous influences exclusively, that it seems worth the while to look for a distinct pathological substratum for the disease. Glénard believes he has discovered it in the primary lowering of the flexura coli dextra. This flexure, he argues, may sink by the giving way of the naturally weak ligamentum coli hepaticum. The transverse colon is thus pushed out of place, and, instead of running transversely to the descending colon, it now ascends diagonally from the lower right to the upper left part of the abdomen. In place of the junction characterized by the ligamentum pylori colicum, there arises a kinking, with consequent obstruction to the course of the feces, etc. Beyond this obstruction the colon contracts its walls, becomes thickened, and it can be palpated as a rigid cord, which Glénard calls the "*corde colique transverse.*" At the same time a downward distortion of the stomach occurs, sinking of the mesentery, and sometimes a nephroptosis or hepatoptosis, or, in one word, a displacement of all of the abdominal organs. Ewald has questioned the frequency of splanchnoptosis, as given by Glénard, and does not consider his diagnostic signs for enteroptosis sufficient. The pulsations of the aorta, and the feeling of the transverse colon, which the latter considered characteristic signs, Ewald replaced by better indications, such as injection of air into the stomach and colon. Further, Ewald does not agree with Glénard concerning the explanation of the manifold complaints; he does not trace them back to the mechanical or *stenotic* phenomena, but to reflex, torsional phenomena and to venous stases. The

"*corde colique transverse*," he argues, is nothing else but the pancreas, which can be palpated through a lean abdominal integument. But essentially he recognizes, with Glénard, Cuilleret, and other French authors, and with the German Chlapowski (the only author who had, up to the date of Glénard's publication, written about the disease), that enteroptosis is a genuine, characteristic, "clinical entity."

The displacements of the intestines, which arise through circumscribed peritonitis, through obstruction of the feces, etc., were described by Virchow as early as 1853. Glénard and Ewald have made reference to the entirely uncomplicated, one might say the purely gastric and intestinal, cases.

Landau does not think it correct to be content with the diagnosis of the movable kidney, floating liver, and kinking of the transverse colon, but he demands that the etiological diagnosis be also given in order not to come to the incorrect conclusion that we have to do with independent diseases. Enteroptoses, etc., are secondary states and should, for diagnosis and treatment, be taken in the same way as roseola with typhus or syphilis, in which one is certainly not content to diagnose roseola, but adds typhus or syphilis with roseola.

Now, which are the diseases that cause a sinking of one or more of the abdominal organs? All those which absolutely or relatively increase the capacity of the abdomen, and thus allow the large intestine, fastened by the relatively long mesocolon, to sink, according to its weight. The slightest or first degree, for instance, may be said to exist where we meet with a light inguinal, femoral, or umbilical hernia. In this case all the intestines not in the hernial sac may sink through pulling and dragging. Since the portions of the intestines which were formerly in the peritoneal cavity have come out, the capacity of the abdomen has been relatively increased.

It is true, splanchnoptosis is not a necessary consequence of large ruptures. Where the elasticity and contracting power of the abdominal integuments are very great, they may counteract the increase in volume by tonic contractions. A number of cases are reported in which almost all the intestines lay in ruptures, and in spite of this the kidney, liver, and uterus were approximately in normal positions. The abdominal integuments were contracted tightly inward and were concave (Landau).

Of far greater importance for the origin of all kinds of enteroptoses is a second great category of diseases, namely, all wasting diseases, which produce a quick consumption of the fat of the body and also disturbances in the nutrition of the abdominal integuments. Thus, after typhoid fever, scarlet fever, and other infectious diseases, we may easily recognize splanchnoptoses of all kinds, such as dislocation of the kidneys and of the liver, etc. It is evident that the transverse colon must be lowered also when the liver and kidney can be felt to have descended, for the transverse colon, lying under these organs and connected with them directly and indirectly, can not retain its position under these conditions. To prove the descent of the transverse colon in such cases, the method of the injection of air into the stomach and into the rectum (Ewald) is not always necessary, nor the very doubtful result of direct palpation of the transverse colon, as given by Glénard.

Chronic diseases, such as phthisis, produce exactly the same effect as acute diseases, and Landau holds that primary nervous dyspepsia, primary chronic gastritis, duodenitis, etc., are frequently not the result of enteroptosis, but, by means of the disappearance of fat and weakening of the abdominal integuments, *causes* of it. In individuals which we may examine to-day, and in whom we can prove no sinking of the liver and kidney, if they should acquire an ulcer of the stomach or nervous dyspepsia, with disturbances of nutrition, we may be able to show prolapsus of the liver or kidney later in the course of the disease.

Among the patients observed by us,—sufferers from disturbances of nutrition, nervous dyspepsia, emaciation, etc.,—in which the splanchnoptoses mentioned were demonstrated, there were surely many cases in which the disturbances of nutrition caused the ptoses, and not the reverse.

Relaxation of the abdominal muscles with or without pendulous abdomen is a frequent cause of splanchnoptosis. Sinking of the kidneys and liver develops in women from whom large ovarian tumors or myomata have been removed by laparotomy. Other cases of pendulous abdomen are acquired by repeated births, following close on one another, and by unsuitable treatment during confinement.

Similarly, splanchnoptoses will appear in individuals who suffer from ascites, and who have been punctured repeatedly, and in whom the abdominal muscles have finally become paretic on account of ab-

normal fullness and distention. The typical form of pendulous abdomen does not always arise in these cases. It suffices that the support of the abdominal viscera, which is formed by the abdominal integuments, becomes insufficient; then the intestines follow the law of gravitation and descend the length of their mesenteries, and when the abdominal integuments have become distended and inelastic, the peritoneal folds of the liver and kidney relax also, and these follow the law of gravity and sink because one of their main supports, namely, the intestinal mass, has been withdrawn. That which is principally and etiologically of prime importance in these particular cases, therefore, is not the sinking of the transverse colon, but disease of the abdominal integuments.

In a large number of splanchnoptoses we have had the experience that the determination of the etiological factor is of the greatest importance in the treatment. Floating kidneys were for a time ignored, and then again were considered a terrible disease, dangerous to life. There was a time—and it has not been so very long ago—when the movable kidney was regarded as an indication for its removal, and it was in fact extirpated. This was, in a certain sense, a consequence of the opinion that the movable kidney as such was a disease *sui generis*, or, to speak with Glénard, a "morbid entity," which made life a burden. It could also happen similarly with the prolapsed transverse colon, if we take its case as a morbid entity and accuse it of causing the host of nervous symptoms, etc. It is therefore not only scientific, but also practical, to combat this idea of a "morbid entity," and to oppose the hypothesis that enteroptosis is an individual disease. Of course, we do not wish to deny that the falling of the intestine and the abdominal organs may bring unpleasant circumstances with it, or, to be more exact, that in individuals who have a floating kidney (an enteroptosis) one observes manifold and unpleasant symptoms, among which those of nervous dyspepsia are most marked. But we must again emphasize that it is not the sinking of one organ, but a more general, more important state which causes this disease with all its complications,—the falling of the intestine, as well as a host of nervous complaints, venous obstruction and the like,—namely, the relaxation of the abdominal integuments, the decrease in irritability and the attenuation of the muscles, the loss of elasticity of the skin and of the peritoneum, etc. Naturally, from these secondary states caused by pendulous abdomen, further very severe diseases may sometimes arise.

There are five places especially at which stenosing phenomena may appear through temporary kinking:

(1) At the pyloric part of the stomach, or where the duodenum passes over from the superior horizontal part into the vertical portion, which is tightly joined to the spinal column.

(2) At the entrance of the jejunum into the duodenum, at the duodenojejunal flexure (E. C. Perry and L. E. Shaw, "Diseases of the Duodenum," Guy's Hospital Reports, 1893, p. 171).

(3) At the transition of the small intestine into the fixed portion of the cecum.

(4) At the transition of the transverse colon into the descending colon, which is comparatively tightly fixed at the posterior lateral abdominal wall, and is further attached high up into the left hypochondrium by the phrenocolic ligament (Phœbus). The left flexure of the colon normally forms a right angle which, however, becomes an acute angle with pendulous abdomen.

(5) In some cases, when the hepatic flexure of the colon has not sunk, stenoses may develop in it at a corresponding place, as described in No. 4 (Landau, on "Pendulous Abdomen," p. 82, *et seq.*).

The floating kidney is to be put semiotically on a level with enteroptosis; it is not a disease *sui generis*.

While the sinking of the kidney was formerly regarded as a rare disease, it is now recognized universally as a very frequent condition. Sufficient emphasis has already been laid on the possible sources of error in the diagnosis. A surprisingly large number of movable kidneys are diagnosed by otherwise very skilful diagnosticians, when they are really dealing with cases of descent of the liver. The best way to palpate the kidney is by the bimanual method of palpation given by Jenner and Freund. All later contributions to the subject by other authors have added nothing essentially new to the bimanual method. If we, therefore, regard the movable kidney, just as the sinking of the intestines and of the liver, to a certain degree as a secondary consequence of the relaxation of the abdominal integuments, we must, however, admit that the movable kidney on its part, though caused secondarily, may itself often be the cause of very severe diseases.

The dangerous sequences of floating kidney are hydronephrosis, intermittent hydronephrosis, and incarceration of the kidney (first described by Dittl). This dangerous condition is largely ascribable to compression of the renal veins.

Landau agrees with Glénard in recommending almost verbatim the same method of treatment which he has used for enteroptosis and pendulous abdomen. It is very simple, and consists in strengthening the lax abdominal integuments, and in providing, to a certain extent, artificial abdominal integuments. Bandages are useful in the treatment of floating kidney. The abdominal bandages suggested by Landau, Ewald, and Glénard do not differ materially. A large number of the patients suffering from movable kidney are still tortured by the use of bandages with padded plates, which are supposed to keep the kidney fixed after replacement, which they rarely do.

Diagnosis.—It is evident that movable kidney may be undoubtedly recognized in all cases where we can fixate it between the fingers and can thus determine exactly its entire configuration. Secondly, the diagnosis is comparatively simple in those cases where we indeed may not determine the configuration, where we discern only a smooth movable body, often movable only within narrow limits, but where we are aided by the communications of the patient, that suddenly, after bodily exertion, this body was felt. But there are cases where this information of the patient is lacking, where we can no longer make the diagnosis from palpation with certainty, and here mistakes are possible. The chief error, according to our experience, is that one has to do with a small lower part of the liver, separated from it by a strong furrow due to compression, which part, on account of the depth of the furrow, seems to be movable. Guttmann has seen such cases repeatedly in autopsies, which during life were regarded as movable livers. Osler (" Lectures on the Diagnosis of Abdominal Tumors," p. 97) gives an illustration of a tongue-shaped prolongation of the anterior margin of the right lobe with the gall-bladder projecting below it.

Since by means of the bimanual examination, which has been described in detail, we are, at least in a number of cases, able to feel the lower end of the right kidney during inspiration, it is not difficult to exclude movable kidney. And since movability occurs most frequently with the right kidney in more than 80 per cent. of the cases, therefore, by finding the right lower edge of the kidney in bimanual palpation, the error of mistaking a portion of the liver (separated from it by a furrow) for the kidney might be avoided.

Proving by means of percussion that the kidney is not in the place where it belongs, is very unsatisfactory. It seems, indeed, in

some cases, that the dullness is less on that side where the kidney is wanting, than on the opposite side where the kidney is present. But in most cases of movable kidney there was no difference in the resonance in the region of the loins on either side. We therefore attach no value to the results of percussion in dislocated kidney, and the same opinion of the use of percussion holds good in diseases of the kidney in general. Only in isolated circumstances it may aid the other methods of examination,—*e. g.*, with great swelling of the kidneys,—but in most cases percussion of the kidneys is entirely worthless, and hence it is not considered a diagnostic method.

Frequency of Dislocated Kidney.—The statements of various clinicians concerning the frequency of floating kidney vary considerably. Lindner stated that it was the most frequent abnormality of the female body, and that one woman in five to seven was afflicted with the trouble. Edebohls gives 18 per cent., Mathieu 27.1 per cent., Fischer-Benzon 17 to 22 per cent., John Schmitt 10 per cent. (New York, *Mediz. Monatsschrift*, March, 1891). Ludwig Knapp ("Wanderniere bei Frauen," Berlin, 1896) gives five per cent.

As far as our clinical material permits us to judge (148 examinations from hospital and private practice), the rate for Baltimore is six per cent. The right kidney is dislocated more frequently than the left, the proportion being 15 to 1, and bilateral dislocation was found only once.

Reversal of the Location of the Viscera (*Situs viscerum inversus*).—In this state the stomach lies normally on the right side, the liver on the left. The site of all of the viscera is exactly reversed; there is a *situs inversus*, however, in which the heart is found in the normal position, at the same time the location of all other organs is reversed.

Vertical position of the stomach is an anomaly frequently associated with atony, which is attributed to tight lacing. It becomes important clinically only when the motor function is interfered with, in which case the treatment is the same as that given for motor insufficiency. The diagnosis of vertical stomach should present no difficulties when the methods stated on pages 97 to 104 are employed.

Ewald's assertion of the extraordinary frequency of movable kidney is the more astonishing because in autopsies one does not often find movable kidneys. Guttmann in reporting his experience,

resting upon about 8000 autopsies at Berlin, which for the most part he himself noted down, stated that in these autopsies the floating kidney was not frequent. His experience agrees with that of the pathological institute of the Charité, Berlin. Landau, in his monograph on the floating kidney, states that in the Charité floating kidneys were found very rarely at autopsies. We may add, however, that in a horizontal position the movable kidney sinks back to its normal position, and is, therefore, liable to be overlooked in the necropsy; but the movability must persist, and this must attract our attention when the kidney is taken out. An experienced dissector will notice at once in taking out the kidney whether it is normally fixed or whether it is movable at the place in question.

Symptoms of Gastroptosis.—The symptoms are brought on by the gastric and intestinal atony and the mechanical disturbances caused by the descent of the organ. We have observed a number of cases of gastroptosis that presented no digestive symptoms whatever. The dyspeptic symptoms that are most common are: pressure, fullness, distention, and pain (gastralgia), coming on at irregular intervals, and independently of the digestive act or the quality and quantity of the food. A sensation of heat or burning at or below the umbilicus is at times described. Eructations, nausea, vomiting, and pyrosis may be complained of. Chronic constipation is a typical accompaniment; flatulence and occasional attacks of diarrhea alternate with constipation.

When there is an evident coloptosis, a very stubborn membranous dysentery is, as a rule, present, being no doubt caused by abnormal kinking and stenosing flexures in the course of the large intestine.

The *quantity of the urine* may be very variable and depends upon the permeability of the ureters. Absolute obliteration of the ureter by kinking may produce oliguria or anuria, which may be followed by profuse urination when the ureter becomes straightened.

Nervous Symptoms.—The typical clinical picture of aggravated neurasthenia is frequently associated with these cases. The manifold pains complained of during bodily exertion are referable to drawing and tugging upon the nervous apparatus supplying the dislocated organs. Intense lumbago is a most frequent sign.

According to Kuttner, Litten, and Ewald, four distinct phases or

degrees of nephroptosis may be differentiated by palpation: (1) A respiratory movability without dislocation. (2) Respiratory movability with slight anterior dislocation,—one-third to two-thirds of the kidney can be palpated; this is termed a "dislocation of the first degree." (3) Respiratory movability with close approximation to the anterior abdominal wall. The kidney is palpable in its entirety, and can be easily moved about; this is termed a "dislocation of the second degree." (4) The dislocated kidney is firmly adherent in its abnormal position.

The *spleen* has been very rarely found dislocated in splanchnoptosis, Glénard having observed splenoptosis only twice in 148 cases.

Hepatoptosis can be recognized by a lowering of the area of hepatic dullness. There are several degrees of liver displacement: (1) A portion of the liver projects beyond the arch of the ribs into the abdomen, and the upper border is correspondingly lower. (2) The larger portion of the liver projects into the abdomen; the liver dullness above the edge of the ribs is reduced to a narrow zone or entirely absent. (3) The entire liver is located within the lower abdomen.

Coloptosis.—Descent, displacement with consequent local stenosis, and dilatation of portions of the colon, can only be recognized by distending the colon with air or water. The air is usually forced in through a long colon tube (Langdon) by means of a double bulb pump, or 600 c.c. to one liter of warm water are gradually allowed to run in under gentle pressure. Normally, a distended area is palpable and even visible two or three inches above the umbilicus; the ascending and descending colon can be recognized as two arching elongated prominences about three inches to either side of the umbilicus. When the colon is prolapsed, the transverse portion is found touching at the symphysis pubes, or even within the pelvis. We have frequently observed that a prolapsus of the colon was recognizable by the distention which had occurred through gaseous fermentation, and artificial distention was unnecessary. When a prolapsed colon is distended with warm water it changes its position with the attitude of the patient. Thus a colon that rests on the symphysis will rise to its normal location when the pelvis is elevated and the thorax depressed. If this does not occur, the colon is adherent in its abnormal location.

Electrodiaphany is an excellent method of diagnosing the course and location of the colon. We have frequently used it for that

purpose (see pp. 103 and 104), for which it is as convenient and accurate as for recognizing gastroptosis.

Gastroptosis can occasionally be recognized by inspection; the methods described in chapter XI, pp. 97–104, if systematically carried out, can leave no possible doubt regarding the existence of this dislocation. The differentiation between prolapsus and dilatation is facilitated by electrodiaphany. If the stomach is dilated, not prolapsed, the transilluminated area will exhibit respiratory movability. Boas and others have observed gastric dilatation in conjunction with gastroptosis. This combination, in our experience, is very rare. Ewald, Litten, and Bartels assert that dilatation of the stomach frequently occurs with nephroptosis or hepatoptosis, this is not confirmed by Boas; myasthenia, with over-retention and stasis of ingesta, is frequently observed, though not in all cases of prolapsus of the stomach. But we agree with Boas that the typical clinical picture of classical dilatation is rare in connection with gastroptosis.

Analysis of the gastric contents in gastroptosis, though yielding no practical aids to diagnosis, is of utility in selecting a proper diet. The results of such chemical analyses are variable.

The **course of gastroptosis** is a protracted and generally a chronic one. Great feebleness, abnormal sensations of pain, compression, or of cold and hot auræ, indisposition to exertion, and faintness are among the most common symptoms in this most variable clinical picture. Severe disturbances of nutrition and anemia unfailingly appear as the digestive distress continues.

The points of differentiation between falling of the stomach and dilatation and atony have been considered in the chapters on these diseases. The differential diagnosis between the symptoms of gastroptosis and nervous dyspepsia is difficult, sometimes requiring all the ingenuity of an experienced diagnostician. This is principally because the symptoms of both states are occasionally identical, and because gastroptosis is often associated with nervous dyspepsia. The state of the peristalsis is normal in nervous dyspepsia, but in gastroptosis there is, as a rule, a myasthenia, with over-retention of ingesta.

Treatment of Gastroptosis.—*Prophylaxis.*—The frequency of gastroptosis and enteroptosis in the female sex demands that the physician should emphatically oppose tight lacing, or any garment that constricts the waist; the dresses should be so constructed as to be supported from the shoulder. We have already

spoken of this under the heading of acute gastritis. If possible, the modifications in dress should be made in accordance with the rules of fashion. It will do no good to oppose the unrestricted domination of this tyrant of the female sex, without clear indications of the benefit to be derived. We have already emphasized that a properly constructed corset need not necessarily work harm, but may eventually be useful by the support it gives to the back and breasts. The dresses should, then, be supported from the shoulders. It is necessary to do this before the enteroptosis is developed.

The relaxation of the abdominal muscles must be prevented by well-applied bandages used after confinements, and worn for several months. The bowels must be kept regular. Massage, electricity, and cold-water applications may contribute to a vigorous abdominal musculature, but the most effective means of strengthening the abdominal muscles, and one which we do not find considered in the text-books on the subject, are abdominal gymnastics.

We will briefly describe two of the most practical methods of training the abdominal muscles.

No. 1.—The patient places himself on a couch, or on a blanket spread on the floor, clothed simply in his or her underwear; the hands are placed at the side; the exercise begins by slowly raising the limbs from the couch to a vertical position in the air, keeping them there for thirty seconds, then very slowly letting them return to the horizontal position of rest. The secret of this maneuver is the slow execution of it. With one hand on the abdominal muscles, the patient may feel the tightening and rigidity which occur in the act of raising the limbs. This exercise should be repeated ten times.

No. 2.—The second exercise should be carried out in the following manner: The patient places himself flatly on a blanket on the floor, with his feet inserted under a bureau or piece of heavy furniture of any kind; both hands are placed steadily at the side of the body. The patient now must slowly bring the trunk of his body into an erect position, and when this has been reached, the trunk is just as slowly replaced to a position of rest on the floor.

Both of these exercises are quite similar; in No. 1 it is the trunk which is fixed and the lower limbs are slowly moved up and down, and in No. 2 the lower limbs are fixed and the trunk is moved up and down. These exercises should be carried out systematically

and methodically ten times every morning and evening. Sandow's book on training will instruct those desiring information on this very useful subject.

Rapid emaciation must be avoided. Physicians are nowadays frequently consulted by thin people desiring to get fat, and by fat people desiring to become thin in the most convenient manner. Rapid falling-off and loss of fat, when undertaken as a cure for obesity, is in our experience a hazardous undertaking. The fat is not only lost from the trunk and extremities, but the internal organs are deprived of their normal incasing and imbedding of fat, which constitutes their support, so that cures tending to reduce the weight of the body should only be conducted under the supervision of a capable physician.

The Mechanical Treatment.—The mechanical treatment consists in applying a properly selected and adapted abdominal bandage. There is no one particular bandage that will suit all cases. The bandages should have their main support and resting-places upon the crests of the ilium, symphysis pubes, and spinal column. From here the strength of the bandage is secured by broad pieces of metal or whalebone inserted into the linen, leather, or rubber parts of the bandage. These bandages must be fitted to the nude figure, must fit perfectly, and be worn day and night. Boas recommends the bandage of Landau and Bardenheuer. Prolonged rest in a horizontal position on the back favors restitution of the abdominal viscera to their normal position. In cases of great weakness, therefore, with emaciation, the Weir Mitchell rest cure is one of the most effective means of treatment.

Constipation is best combated by proper diet. We recommend compotes of fruit, such as figs, prunes, apples, pears, plums, and sweet grapes. Buttermilk and kefyr favor normal evacuation. Sugar of milk, ℥ss three times daily, is also efficacious (see p. 236). As gastroptosis predisposes to dilatation, it may occur that the foods are retained an abnormally long time within the stomach. In these cases lavage will be indispensable.

When the symptoms of atony and motor insufficiency are pronounced, local intragastric douches with alternating cold and warm water are very effective in restoring partial tonicity to the muscular walls. Treating the abdominal muscles by massage and the faradic current is of some utility in patients that are too feeble to undergo the abdominal gymnastic training. These means of treat-

ment may be applied also in those cases that have not the will-power to persist in such abdominal gymnastics, but electricity and massage can not effect the permanent improvement which we have observed as a result of abdominal gymnastics.

Floating kidneys, according to Bachmeier (*Wien. med. Presse*, 1891, Nos. 19 and 20), may be replaced best in the following manner: The patient is placed on his back in bed, the physician taking a chair facing him; both hands are placed on the right side of the patient, under the anterior arch of the ribs; the hands are then pressed gently and firmly toward the posterior and superior parts of the abdomen. While this pressure is exerted, the finger-tips must make constant shaking and trembling motions.

Diet.—In enteroptosis and gastroptosis the diet should be as nourishing as possible, and adapted to the state of motility and secretion. If the condition of the digestive organs will permit, attempts should be made at increasing the adipose tissue. Distinct diseases of the gastric mucosa contraindicate a large food supply. Experience has taught us that one of the best treatments for floating kidney is that which causes an increased deposition of fat. Fat is best introduced in diet in the form of fresh butter, rich gravies, and cream. For further particulars of nourishing diet we refer to the section on dietetics. In our sanitarium for digestive diseases, we have found the schedule on pp. 240 and 241 useful in nephroptosis.

Medicinal Treatment.—Medicines are sometimes unavoidable for the treatment of constipation. Our favorite remedy for this, where it becomes necessary, is the active syrup of cascara sagrada (Clinton Pharm. Co.), or large colon irrigations with warm water which will also benefit the membranous colitis. Where patients can take it, the time-honored castor oil is a good and harmless remedy. Of other laxative remedies, we favor rhubarb, magnesia, senna, and podophyllin. Aloes, jalap, and scamany do not act well, nor do the very drastic purgative waters, such as the Hunyadi Janos and Rubinat Condal. We have seen more lasting results follow a persistent use of Bedford Magnesia Springs water, which is rather mild in its purgative qualities.

Glénard recommended magnesium and sodium sulphate to combat the effects of auto-intoxication, but Boas, after a prolonged trial of these remedies, asserts that he has observed detriment to

result from them. Our formula for combating auto-intoxication in gastroptosis is the following :

R.	Betanaphthol. bismuth., ...	4.0	ʒj
	Resorcin. resublim., ...	4.0	ʒj
	Strychnin sulphatis, ...	0.02	gr. ⅓
In anacidity—achylia—dilute HCl, ʒiv, should be added.			
	Elix. gentianæ, ...	180.0	fʒvj. M.

Sig.—One tablespoonful three times daily.

In addition to these, the salicylate of bismuth, salicylic acid, chloroform water, and betanaphthol have been recommended. They are made more efficacious if combined with strychnin and an adapted diet.

It is conceivable that methods of surgical procedure may in future be devised for replacing a prolapsed stomach, by attaching it partly to the diaphragm and partly to the retroperitoneal fascia in such a manner as to avoid kinking or stenosis.

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CHAPTER IX.

NEUROSES OF THE STOMACH.

Gastric neuroses may be classified as follows:

I. Motor.
II. Sensory.
III. Secretory.

Under each one of these we may distinguish

(*a*) States of excitation.
(*b*) States of depression of nervous influences.

I. Neuroses of Motility, or Peristalsis.
- (*a*) *Irritative states:*
  - (1) Cramp of the cardia, or cardiospasm.
  - (2) Cramp of the pylorus, or pyloric spasm.
  - (3) Cramp of the entire musculature, or gastrospasm.
  - (4) The peristaltic unrest of Kussmaul.
  - (5) Nervous eructation.
  - (6) Nervous vomiting.
- (*b*) *Depressive states:*
  - (1) Insufficiency of the cardia, including rumination and regurgitation.
  - (2) Insufficiency or incontinence of the pylorus.
  - (3) Atony or insufficiency of the entire gastric musculature (gastroplegia).

II. Sensory Neuroses.
- (*a*) *Irritative states:*
  - (1) Hyperesthesia.
  - (2) Gastralgia.
  - (3) Bulimia and polyphagia.
- (*b*) *Depressive states:*
  - (1) Acoria.
  - (2) Anorexia.

III. Neuroses of Secretion.
- (*a*) *Irritative states:*
  - (1) Hyperchylia, hyper- or superacidity.
  - (2) Supersecretion or gastrosuccorrhea, gastroxynsis.
- (*b*) *Depressive states:*
  - (1) Hypochylia or subacidity.
  - (2) Achylia gastrica or inacidity.

Nervous Dyspepsia.

General Considerations.—All diseases of the stomach in which no pathological anatomical change can be demonstrated in the organ are classed as neuroses. Hitherto we have considered only diseases that were based upon an actual alteration in the structure of the stomach. Neuroses, then, are idiopathic diseases of the gastric nerves, with absence of histological changes that can be demonstrated in the tissues. The gastric nerves can be affected, it is true, in the course of gastritis, ulcer, carcinoma, and dilatation, by the changes in the deeper layers of the stomach brought about by these diseases. A large portion of the dyspeptic disturbances, as well as of the anomalies of secretion and motility, are attributable to injurious influences exerted upon the nerves in the course of these diseases. These nervous affections which accompany changes in the gastric structure are known as secondary symptomatic nervous disturbances. It is very probable that anatomical changes may lie at the foundation of many neuroses, but up to the present time they escape our microscopic technic. For instance, in more than half the cases of hyperacidity a proliferation of the oxyntic cells, or of the glands as a whole, has been ascertained by Hayem, Einhorn, Cohnheim, and the author. It is more than probable, also, that atrophy is present in from one-half to two-thirds of the cases of achylia gastrica. With further progress and improvement of our methods of staining and hardening the number of gastric neuroses will become more and more reduced.

The histological changes in the mucosa accompanying hyperchylia and achylia are not caused by the nervous condition, but constitute a primary affection independent of the neurosis. This is made probable by the fact that proliferation of oxyntic cells and of gland tubules can be found at the autopsy, in the stomachs of individuals who have never shown any symptoms of nervous diseases or neurasthenia, and also because proliferation has been found in fragments of mucosa gained from the wash-water of perfectly normal individuals so far as any neuropathic state was concerned.

When the gastric nervous apparatus is the primary seat of the disease, it is called a primary neurosis, but when the disease of the gastric nerves is reflexly excited from the central nervous system, or from other organs, such as the intestines, liver, spleen, and kidneys, it is called a secondary or reflex neurosis. Neuroses may cause secondary anatomical alterations in the stomach ; for instance, if anacidity is associated with impaired motility, we may have a gas-

tritis develop from decomposition of a stagnating ingesta. When hyperacidity, or supersecretion, causes a persistent spasm of the pylorus, a dilatation may result producing the same symptoms. Disturbances of the sensory nerves of the stomach may extend to the bowels and bring about the so-called neurasthenia or nervous dyspepsia of the intestines; with persistent nervous atony of the stomach, the motility of the intestine frequently begins to suffer also. This, apparently, is a direct extension of the nervous trouble to the intestines. In achylia gastrica, when the antiseptic effect of the HCl is missing, an excessive putrefaction of the intestinal contents with abundant formation of gases is sometimes noticed, so that the intestinal wall becomes very much expanded, and an atony can arise in this manner. It is well known that strong psychic impressions and emotions like anger, aggravation, fright, fear, and sadness, as well as excessive joy, can completely suppress the appetite. In very excitable people these emotions may even cause eructation, nausea, and severe gastralgia. These nervous disturbances mend rapidly, as a rule, when the mind has been quieted, but when the emotional excitement was great, and frequently repeated within a short time, particularly in very excitable, neuropathic individuals, a lasting neurosis may develop.

Gastric neuroses which are the result of functional or anatomical diseases of the nervous central organs, or of hysteria and neurasthenia, may be so masked that the fundamental disease may be completely submerged, and be little regarded by the patient, and is not discovered by the physician until after a careful study of the case has been made. In sclerosis of the posterior columns of the spinal cord (tabes dorsalis) a train of gastric symptoms has been first described by Charcot under the name of "crises gastriques." They are described as intense cramp-like pains occurring suddenly in the midst of comparative well being, and radiating toward the abdomen and back; they are usually followed by copious vomiting. The vomit at first consists of food, and later of mucus, bile, and duodenal secretions, and may occur several times in the same day, frequently every hour. These attacks appear and disappear very rapidly, and are separated by long intervals of perfect freedom from gastric disturbances. Ewald emphasizes that these attacks may occur in tabes so early in the disease that the fundamental affection can not at all be diagnosed because all typical symptoms are wanting. Sensations of a boring and burning character and severe gastralgia

may be present in tabic patients years before the spinal disease is recognizable. In other anatomical diseases of the nervous central organs, in myelitis due to compression, meningitis, brain tumors, and after powerful concussions of the brain and spinal cord, nervous gastric disturbances appear. In the progress of certain diseases of the medulla, repeated nausea and vomiting may be attributed to irritation of the vomiting center. Reflex neuroses of the stomach may occur from disease of the neighboring organs, as, for instance, from the liver, intestines, bile passages, spleen, and peritoneum, as well as from the kidneys, sexual organs, and heart. In biliary colic gastric symptoms are rarely absent. We generally find that gastralgia, nausea, vomiting, eructation, and anorexia are present during the passage of gall-stones, and rapidly disappear as soon as the stone has passed through into the intestine. The gastric complications of cholemia and cholelithiasis do not disappear so rapidly, because the excretion of the foreign materials in the blood of cholemia, and the correction of anatomical changes in cholelithiasis require some time. Renal colic in a similar manner may cause dyspeptic symptoms. Cases are reported in which the renal symptoms were so masked by the gastric that the diagnosis of ulcer was made. This may very readily occur when the nephritic pains radiate toward the back and shoulders like those of ulcers. Gastric symptoms occurring as a sequence to uremia are the result of a direct or indirect irritation of the central nervous organs, and also of the gastric nerves by retained products of catabolism. In a case of contracted kidney with chronic uremia in our recent practice, the patient suffered exclusively from gastric symptoms, but test-meals showed that the motor and secretory functions were normal, and that, therefore, the dyspeptic complaints were disturbances of the sensory nerves of the stomach. Diseases of the sexual organs, in both sexes, but particularly in women, may bring about reflex gastric neurosis. Kretschy and Fleischer have found that the physiological process of menstruation may cause disturbances of function in the stomach, which naturally are also met with in a more exaggerated form in dysmenorrhea, in the diseases of the uterus and ovaries, and during pregnancy. Whatever may be the etiological culmination of factors in *vomiting of pregnancy*, there is no better explanation offered up to the present time than that it is a reflex neurosis of the stomach induced by the expansion of the

uterus and irritation of the sympathetic fibers caused thereby. R. Frommel (Penzoldt and Stintzing's, "Handbuch d. specielle Therap. innerer Krankheiten," Bd. IV, p. 440) has obtained very good results in this disease with basic orexin, a medicine which acts mainly in nervous affections, and rarely in anatomical alterations of the stomach.

Gastric neuroses are much more frequently seen in women than in men. Women and girls of the better classes constitute the prevailing number of those affected with gastric neuroses. The recognition of pure gastric neuroses and whether they exist as idiopathic independent diseases, or are in some causal relation to a pre-existing disease, like those we have mentioned, is often a problem presenting great difficulties. The stomach is an organ that is very rich in nerves, and is inclosed in a widely connected network of fibers which bring it into close connection with other vital organs. Concerning the anatomy and physiology of the gastric nerves, we may say that the innervation of the organ is carried out above all by the vagi. The left vagus spreads over the cardiac portion and the lesser curvature, and forms the anterior gastric plexus with fibers coming from the abdominal sympathetic. The right vagus supplies mainly the liver, pancreas, spleen, kidney, and small intestine, and a small part of its branches reaches the posterior wall of the stomach. Anastomoses from the abdominal sympathetic with branches of the right vagus form the posterior gastric plexus. The vagi also enter into the formation of the celiac or solar plexus. Branches from the solar plexus form the so-called superior coronary plexus of the stomach lying along the lesser curvature, while branches coming from the hepatic plexus and running along with the right inferior coronary artery form the inferior coronary plexus of the stomach. These four plexuses are united into a great network by connecting communicative branches. In the pyloric portion, the beginnings of the large and important intestinal plexuses can be demonstrated. The intestinal sympathetic nerves form a network with close meshes in the submucosa as well as in the muscular layer. In the broadened points of union of this nervous, network are found numerous ganglion cells. The Meissner network supplies the muscularis mucosæ, and the mucosa and the intermuscular plexus of Auerbach supply the muscular layers with very fine branches. Openchowsky has demonstrated large

masses of ganglion cells not only at the pylorus, but all along the fundus and cardia in the serosa; these ganglia are in communication with the large vagosympathetic fibers.

What we know of the physiology of the gastric nerves is, up to the present time, very limited. Experimental physiology has as yet not cleared up gastric innervation. It is not even known whether secretion is influenced by the vagus nerves, nor by what nerve routes the sensation of hunger is transmitted. The influence of the vagus and sympathetic fibers on peristalsis is also quite unknown. Although we do not know the paths of vast secretory motor and absorptive impulses, nor of sensation, it is generally assumed on clinical grounds that each of these functions is represented by different nerves, and we therefore accept the existence of special nerves for motion, sensation, secretion, and absorption. Clinical experience has confirmed this assumption because peristaltic, sensory, and secretive disturbances may exist by themselves.

All gastric neuroses may show considerable variation in their course. Thus the contents of the stomach may one day show inacidity or achylia, and on the next day show hyperchylia. In the same manner we may find motor insufficiency alternating with peristaltic unrest. Although the neuroses may exist singly as individual diseases, we are confronted, as a rule, with combinations of various disturbances. Thus we may find that hyperacidity and gastrospasm are associated with each other, that hyperesthesia will be combined with vomiting, and that superacidity will be present in atony. These diseases may develop pronounced attacks at periods when the stomach is resting. The intensity of the attack is very frequently entirely independent of the quantity or quality of the food, but the effect of psychic influences is generally unmistakable. Neuroses may exist side by side with organic diseases of the stomach, but as a rule they are part of the symptomatology of neurasthenia or hysteria. The symptoms of a general neurosis are rarely absent; that is, the characteristic changes of the psychic disposition, the lassitude, irritability, feeble memory, indisposition to work, insomnia, neuralgia, migraine, vertigo, polyuria, weakness of the bladder, and a varying pain. All these neuroses which we have mentioned are really not individual diseases, but rather symptoms; but, as these symptoms generally occur with a certain independence and are disturbances peculiar to themselves, it will not be unlogical to call the complex anomalies by the name of one

symptom, so that we will speak of cardiac spasm and nervous eructation and hyperacidity as diseases peculiar to themselves (neuroses *sui generis*), bearing in mind, however, that we are simply describing symptoms.

CARDIOSPASM (Cramp of the Cardia).

The etiology of spasm of the ring musculature of the cardia agrees fully with that of cramp of the pylorus. In the great majority of cases the cramp of the cardia represents a secondary disease, which may appear with hyperesthesia and very strong irritation of the mucous membrane of the cardiac region; further, with abnormal dilatation of the stomach through air and gases, as well as with caustic action upon the mucous membrane present with ulcer and ulcerating carcinoma of the cardia; hence, it is produced by the same causes as cramp of the pylorus. Much more rarely the spasm of the cardia is due to a genuine symptomatic or idiopathic neurosis of the motor apparatus, which is characterized by an increased irritability. It is observed as a partial symptom of hysteria and neurasthenia, simultaneous with other nervous disturbances, which may facilitate the recognition of the neuropathic basis of this form of cramp. Whether cramp of the cardia as a pure neurosis of the motor apparatus is a functional impairment of the peripheral motor nerve apparatus, or whether it is of central origin, is at present still a debated question. We may distinguish two forms of cramp of the cardia:

(1) Acute cramp, which, appearing rather suddenly, often spasmodically, is generally only of short duration.

(2) Chronic cramp, which is a very stubborn and serious disease.

One of the most frequent causes of the rare form of secondary cramp, is dilatation of the stomach by air and gases. An abnormal dilatation of the stomach by air, which may finally bring about a cramp of the cardia, is only found in those persons who have the nervous habit of swallowing large quantities of air. If the air is not soon removed through eructation, it keeps on collecting in the stomach and expands on becoming warmed, so that finally a considerable dilatation of the stomach is produced, and with it cramp of the cardia, which is, perhaps, always complicated with cramp of the pylorus. Ewald and Fleischer have had opportunity to examine repeatedly cases of intentional swallowing of air. Fleischer's case was a girl who had been practising it as a kind of

sport for many years. The stomach was constantly dilated, even in a jejune state, and felt like an air-pillow. The rounding of the region of the stomach was plainly visible, even through the clothing. The elastic stomach-tube introduced into the esophagus met an obstruction at the cardia, which was not easily overcome, even after the insertion of the tube into the stomach. When the outer end was put into water, numerous bubbles of air escaped through it; but even with a strong external pressure in the region of the stomach one could not succeed in removing all the air. The resistance in this region continued, though to a less degree. With repeated thorough palpation, it was discovered that the cramp of the cardia and pylorus, occurring intermittently for many years, had caused a hypertrophy of the musculature of the stomach; and it was this condition, probably, which prevented the formation of a more severe atony and ectasia of the stomach, for the lower limit of the stomach was only slightly below the normal.

The stomach may also be dilated by a very copious intragastric formation of gases to such a degree that a cramp of the cardia arises; and this happens very easily when the formation of gases is very rapid, as is observed sometimes with protracted stagnation of the ingesta in the stomach, as a result of atony, ectasia, chronic gastritis, advanced stenosis of the pylorus or duodenum, as well as with primary and secondary cramp of the pylorus, when the contents of the stomach are subject to fermentation and decomposition. If the gases can not pass over into the intestine, they continue to collect in the stomach, so that it finally becomes very much dilated. The region of the stomach becomes arched, and troublesome sensations of pressure and tension appear in the same. If the stomach presses the diaphragm upward, and if the latter in turn presses on the lower part of the lung and the heart, dyspnea, precordial oppression, palpitation of the heart, and asthma dyspepticum may result. With this there sometimes exists great prostration, rapid, soft pulse, and headache. That these very often dangerous symptoms are really caused by the dilatation of the stomach and not, perhaps, by auto-intoxication—that is, by products of fermentation and putrefaction of the chyme—is evident from the fact that the symptoms cease quickly when the cardial closure is finally broken (the pylorus, on account of its very much stronger ring musculature, offers a much greater resistance to the passage of the gases into the intestines), when the spasm

relaxes, or a tube is introduced into the stomach, and the air or gases have an opportunity to escape outwardly.

On the other hand, the cramp of the cardia may also be primary, and the dilatation of the stomach (pneumatosis) may be secondary. If the cramp of the cardia sets in immediately after the meal, and if the eructation of the air which is swallowed during the meal with the food and liquids is prevented, then the stomach may also become abnormally distended. Since the neuromuscular apparatus of the cardia is mechanically irritated with strong distention of the stomach, the cramp is thereby continued, and, therefore, the cramp probably lasts longer during digestion than with an empty stomach. Just as the removal of air and gases from the stomach through eructation is sometimes made entirely impossible by a primary or secondary cramp of the cardia, so the removal of the contents of the stomach by vomiting may also be made impossible. Even the strongest efforts at vomiting bring up nothing, and patients may be much tormented by fruitless muscular exertion. When this is of long duration and frequent repetition it may bring about atony of the stomach in consequence of overexertion of the musculature. If the cramp is caused as a secondary or reflex neurosis by hyperesthesia, strong irritation, or ulceration of the mucous membrane of the cardia, it sometimes produces a painful feeling of contraction in the region of the cardia, which may radiate toward the breast, the back, and to the region of the heart. It has been asserted that the cramp which appears as a pure neurosis of the motor apparatus may also cause the same sensations of pain, but this is not very probable. At least the observations with that form of cramp of the pylorus which is not dependent upon the states of disease before mentioned, but depends simply upon a pure neurosis of the motor apparatus, argue against it, since the latter is not accompanied by pain. The pains described are not a constant symptom, and, therefore, may not be used as a factor in a differential diagnosis of the two forms of cramp. Since with an entirely empty stomach the acute spasm produces no symptoms, it may for some time remain latent, and is sometimes recognized only by accident when from some cause a tube is introduced into the stomach which meets an obstruction in the region of the cardia, or when food or drink is taken by the patient during the cramp, deglutition being then somewhat hindered. Chronic cardiospasm gives much more significant symptoms.

Besides the symptoms mentioned previously, very severe complaints from deglutition appear. The patients have the unmistakable feeling that some of the food becomes stuck before it reaches the stomach. If, in spite of this, the meal is continued, the lower part of the esophagus is filled with food, and after some time the patients, with great exertions, sometimes succeed in bringing it up again in almost an unchanged condition. That it comes from the esophagus and not from the stomach, is shown by the absence of the free hydrochloric acid in the same. The second deglutition sound (auscultation over the lower part of the sternum) is always lacking, and in its place a low rippling noise may be heard, which probably arises from the circumstance that the cardia is not completely closed, and liquids pressed on may still flow into the stomach. With protracted duration of the malady, the ingesta remaining in the lower part of the esophagus may exert such a pressure upon its walls that a diverticulum may be formed which prevents the taking in of food. The nutrition of patients may be much reduced, so that, especially when the patients are advanced in age, the suspicion may arise that a carcinoma of the cardia is developing, which, as we know, can also cause a stenosis of the cardia as well as the formation of a diverticulum.

Fortunately, chronic cramp of the cardia is a very rare malady; it may exist for months, even years.

Prognosis.—The prognosis of the acute primary or secondary cramp of the cardia is, on the whole, favorable, especially when it is recognized in time, and if one is successful in rapidly removing its fundamental causes,—swallowing of air, formation of gases in the stomach, hyperacidity, hypersecretion, atony, hyperesthesia, etc. On the other hand, its prognosis is always to be made with caution, especially when there has already been formed a diverticulum of the esophagus which hinders the passage of food.

Diagnosis.—In order to distinguish acute primary and secondary cramp, one must learn, above all, if one of the states of disease before mentioned which can produce cramp of the cardia, can be detected. With repeated thorough investigations one generally succeeds in determining the cause of the secondary cramp. If it is due to hyperesthesia, to a strong irritation, or to loss of substance of the mucous membrane of the cardia, then pains in the region of the cardia often draw attention to this manner of origin, and the introduction of a tube into the stomach will then

cause pain also. But if a decided cause for the cramp can not be discovered, if it recurs periodically, if it is always of rather short duration, and if other nervous troubles are present, then probably there exists a symptomatic functional neurosis; and if all these signs are lacking, then there is an idiopathic functional motor neurosis, but this is a very rare occurrence.

The diagnosis of the chronic cardiospasm leading to a permanent closure of the cardia is more difficult, because it may easily be mistaken for carcinoma or malignant stenosis of the cardia, as well as for a diverticulum of the lowest part of the esophagus, which, however, is rare.

Advanced age, anemia, and cachexia, appearing at a time in which the passage of food is not yet hindered to a great extent, argue for carcinoma of the cardia. In the food brought up by regurgitation, as well as in the examination with the tube, one often finds traces of blood, and in some few cases particles of carcinomatous structure (in the aperture of the tube). In most of the patients, free hydrochloric acid is wanting in the contents or food that may be drawn from the esophageal diverticulum. The more the stenosis increases with the progress of the carcinoma, the thinner the tubes that must be used, in order to pass. Cicatricial stenosis of the cardia is less frequent, a diverticulum of the lowest section of the esophagus much rarer, than carcinoma of the cardia. Both diseases are not connected with any particular age. The nutrition of the patients is decreased only when the passage of the food is very much impeded. In the formation of diverticulum traces of blood in the contents of the tube are generally lacking, and with stenosis they are very rare. With diverticulum, tubes of different thicknesses sometimes penetrate into the stomach at the first attempt, at other times only after many fruitless endeavors, according as the stomach is full or empty. With stenosis, when there is much difficulty in deglutition, only thin tubes can penetrate. In both maladies there are no anomalies of secretion in the stomach. With carcinoma, as well as with stenosis of the cardia and with diverticulum, the difficulties of deglutition increase very gradually, while in the case of cramp they generally come to an acute stage in a short time. The chronic cramp, which is very rare, may appear at any age. The general nutrition suffers only after protracted duration. Traces of blood can neither be

found in the examination with the tube nor in the food that is eructated.

The examination of the contents of the stomach in cases of simple cramp of the cardia shows nothing abnormal. With intermittent relaxation of the cramp, the complaints of deglutition cease temporarily, and a rigid tube may be pushed into the stomach without meeting with any resistance; neither of these phenomena are observed with carcinoma and stenosis, except in the rare case of disintegration of the carcinoma. An important distinction of the cramp consists in the fact that thick, rigid tubes overcome the obstacle at the entrance of the stomach much more easily than thin tubes (Boas). Fleischer has made the same observations repeatedly with spastic stricture of the urethra, which is generally a result of hyperesthesia of the mucous membrane of the urethra. If in this case a thin catheter is introduced, its point, with moderate pressure, irritates only one spot of the mucous membrane, and by this a cramp of the musculature is produced (or a previously existing one is increased), which is so severe that it may easily be mistaken for an organic stricture. A wrong diagnosis is, however, easily avoided if the mucous membrane is first anesthetized with a five per cent. cocain solution. Then, after a short time, one can push the catheter further. If a much thicker catheter is introduced, the broader point of the same will exert an even pressure upon the whole mucous membrane at the point in question, which is not so irritating. Probably the sensory nerves are then for a time paralyzed, and the cramp abates. Very likely the same conditions obtain in the probing of the esophagus. If after protracted duration of a cramp a diverticulum of the esophagus has been formed, considerable quantities of food may be retched up at one time, and a thick tube will then pass the obstacle at the entrance of the stomach, the facility of the passage depending on the fullness of the diverticulum, and sometimes the passage is accomplished only after many unsuccessful endeavors.

Therapeutics.—The patient must abstain from all injurious influences. The abnormal conditions which, according to experience, produce cramp of the cardia, must be removed. Those who swallow air must be cautioned against the bad effects of the habit. With strong dilatation of the stomach through air and gases, in consequence of fermentation and stagnation of the contents, the air and gases should be removed as quickly as possible by

the introduction of a rather thick, rigid tube, and a more copious formation of gases must be prevented by methodical lavage of the stomach, often with the addition of antiseptics. The diet must, for some time, consist of milk, and later of various meats taken in a minced form. Other nervous disorders must receive suitable treatment. One of the best methods of treatment for cramp of the cardia is the introduction of firm, thick tubes, which are permitted to remain in position for some time. Sometimes the cramp ceases entirely after sounding one or more times. If the spasm is the consequence of hyperesthesia of the mucous membrane of the esophagus and cardia, the sensibility is blunted by frequent soundings.

In very stubborn cases of hyperesthesia with cardiospasm it is advisable to apply a solution of cocain hydrochlorate to the mucous membrane just before the meal, in order to prevent the appearance of the cramp. For this purpose one had best use a small sponge, saturated with a three per cent. solution of cocain, and fastened to the lower end of an open, rather thick, firm tube, with rounded edges, by means of a strong silk thread brought through the tube to its upper end. After introducing the tube into the cardia, the cocain solution is forced out of the sponge by pulling the silk thread, or by blowing air into the tube, and the mucous membrane may thus be anesthetized. Another way of accomplishing this is with the Einhorn intragastric spray, by which the lower part of the esophagus and the cardia may be sprayed with cocain and menthol. With chronic cardiospasm also, the methodical introduction of firm tubes is the most successful remedy. The effect may be aided by external or internal galvanization (the anode in the tube). According to an interesting observation of Boas, solid foods are sometimes introduced more easily than liquid ones. Before meals the foods lying in front of the cardia are to be removed as completely as possible, especially when a diverticulum has developed.

In both acute and chronic cardiospasm we have obtained the most permanent relief by the galvanic current. The length of the esophagus is determined by methods devised by Penzoldt (*loc. cit.*) and Isert Perl, and a rather large spiral electrode (Stockton's) is introduced to a distance compelling it to be in or near the cardia; the anode is placed on the cervical region, the cathode in the cardia, and a current of 25 milliamperes is turned on for ten minutes. Then the same procedure is repeated with the anode on the epigastrium. In all cases where there is any doubt about the differential diagnosis

between cardiospasm and carcinoma of the cardia, the patient should be examined under anesthesia. If the passage becomes readily permeable to the sound under narcosis, carcinoma can be excluded.

In November, 1896, we had a girl patient, aged ten years, under treatment, where the diagnosis was difficult because the tube inevitably became caught above the cardia. As the child could swallow nothing but water, she became extremely emaciated. For weeks we supposed we were confronted with an esophageal diverticulum. The child was fed through a tube which at first we only succeeded in passing under anesthesia, but later also, when the patient was conscious, she was cured by electricity, the symptoms disappearing entirely.

Naturally, the inspection of the esophagus with the esophagoscope would decide most of such cases. (See p. 175.)

PYLORIC SPASM (Pylorospasm, Cramp, Convulsion, Spasm of the Pylorus).

Cramp of the ring musculature of the pylorus is brought on by entirely different causes: it may appear with hyperesthesia, with very strong chemical irritation of the mucous membrane of the pylorus by means of hydrochloric acid (hyperacidity, supersecretion), by excess of organic acids, as well as with dilatation of the stomach by gases (as a reflex neurosis), and finally also after the caustic action of toxic substances, and further as a secondary disease accompanying ulcer and ulcerating carcinoma of the pylorus. While the existence of a secondary cramp of the pylorus is generally recognized, strange to say the existence of a primary cramp of the pylorus, caused by an independent motor neurosis, restricted to the pylorus alone, is still generally denied. If one grants, however, that the insufficiency of the pylorus may appear also as a genuine motor neurosis, due to a decrease of the irritability of the motor nerve apparatus of the pylorus, there is no reason to deny entirely the occurrence of a primary cramp of the pylorus, which is due to an abnormally increased irritability of the motor nerves, even though this be rare. Indeed, Stiller, one of the most competent judges of neuroses of the stomach adduces a primary cramp of the pylorus for the explanation of peristaltic unrest of the stomach. It is true the detection of the same, as well as that of the secondary spasm, is very difficult, since the most

important result of the same—namely, an increased peristalsis of the stomach—can not be proved with normal location and size of the stomach except by Hemmeter's or Einhorn's method.* However, one may assume, with probability, a primary cramp of the pylorus, if after the exclusion of the before-mentioned causes (secondary cramp of the pylorus), as well as of organic disease of the stomach, the reaction for salicyluric acid in the urine or iodin in the saliva occurs much later than under ordinary circumstances, after the introduction of 1.0 salol or 0.1 iodoform into the stomach with the test-breakfast. The diagnosis becomes probable also when atony of the stomach appears without any assignable cause. The results of a primary cramp of the pylorus are the same, naturally, as those of the secondary. Since contents of the stomach can not pass into the intestine during the entire duration of the spasm, there must result a stagnation of the ingesta and a protracted burdening of the stomach, which often causes atony of musculature, which can also become very easily exhausted through the energetic exertions for overcoming the increased resistance at the pyloric orifice. If the neurosis is very stubborn, the atony may pass over into a pronounced dilatation, particularly if the stagnating ingesta decompose rapidly and the atonic stomach is abnormally distended with gases.

Therapeutics.—If a primary cramp of the pylorus is suspected, a digestible, non-irritating diet is to be prescribed (see pp. 219, 220); every immoderate burdening and dilatation of the stomach through very abundant meals, which might heighten the irritability of the motor nerves, is to be avoided. With this the bromids, preferably the bromid of strontium, in liberal doses (3.0 (45 grs.) to 5.0 (75 grs.) per die), extractum belladonnæ (0.02 ($\frac{1}{3}$ of a gr.) to 0.03 ($\frac{1}{2}$ of a gr.), and codein phosphate (0.02 ($\frac{1}{3}$ of a gr.) to 0.03 ($\frac{1}{2}$ of a gr.)) are to be prescribed. Electricity is a valuable adjuvant to the treatment, and should be used in the same manner as indicated for cardiospasm. Spraying the pylorus with cocain and menthol is a satisfactory treatment. Under narcosis the pylorospasm relaxes. The

* In a singular case of periodic pylorospasm occurring in a hysterical female regularly at the menstrual period, we obtained a record with our triple intragastric bag which may be characteristic of these cases. This bag (*N. Y. Med. Jour.*, June 22, 1895) records the pyloric, fundic, and cardiac peristalsis separately on three tambours on the kymograph (see p. 81), and in this case the pyloric pen showed great spastic contractions, and tenesmus lasting from three to five minutes before they relaxed.

pylorus may be catheterized by the author's method of intubating the duodenum. This method constitutes a ready means of recognizing pylorospasm. Kuhn's method (*loc. cit.*) is available for the same purpose.

GASTROSPASM (Convulsions of the Stomach).

Gastrospasm is a neurosis in which the musculature of the stomach is so strongly contracted that the whole organ may become hardened like a board, and may be recognized by palpation as a resistant mass through the lax abdominal integuments. It is a very rare disease. Whether it ever occurs as an independent genuine neurosis of the motor apparatus, or whether, as is generally supposed, it occurs only as a secondary nervous affection, with hyperesthesia of the sensory nerves of the stomach, or as a consequence of a cramp of the pylorus, combined with hypertrophy of the musculature of the stomach, is still an open question. In the very rare cases observed thus far, the single paroxysms of gastrospasm invariably lasted but a short time, and the quick intermission might be sufficiently explained by the enormous overexertion of the musculature during the attack. The treatment is the same as for hyperperistalsis (Kussmaul).

GASTRIC HYPERPERISTALSIS (Peristaltic Unrest (Kussmaul)—Tormina Ventriculi Nervosa).

General Considerations.—Peristaltic unrest is the name given by Kussmaul to a state of the stomach first described by him, which is characterized by the appearance of extraordinarily rapid contractions of the stomach, following close upon one another, which appear especially after meals, continuing also during the day and sometimes through the night with an entirely empty stomach. This excess of peristalsis brings about very disagreeable sensations of heaving to and fro, of unrest, and contractions in the region of the stomach which, without being really painful,—as, for example, the so-called cramps of the stomach with cardialgia,—may nevertheless annoy the patient very much. Only when there is an ectasia or a dislocation of the stomach present simultaneously, as was the case in all the observations up to date, are these abnormally strong contractions of the stomach to be seen and felt externally through the lax abdominal integument as distinct undulatory motions. The peristaltic waves generally from the fundus to the pylorus; that is, from left to

right. Besides the peristaltic motions, there were observed, in a small proportion of cases, antiperistaltic motions also; sometimes the latter were observed to exist alone, but this is rare (Schütz, Cohn, Glax). If the size and location of the stomach are normal, the objective symptoms are lacking entirely, and only the subjective complaints, particularly the feeling of unrest, are evident. Sometimes, also, peristaltic unrest of the small intestine co-exists with that of the stomach.

Increased irritability of the motor nerves of the stomach is looked upon as the cause of peristaltic unrest.

Etiology.—An abnormally increased activity of the stomach may be brought about by very different causes:

(1) As a reflex process, through hyperesthesia of the sensory nerves of the stomach.

(2) By a very strong stimulation of the mucous membrane of the stomach by HCl (hyperacidity, supersecretion), by organic acids, the result of an abnormal fermentation of the contents of the stomach, by gases which distend the stomach to a considerable degree.

(3) With an advanced stenosis of the pylorus and of the duodenum, and, finally, it may be due to an increased irritability of the motor nerves, and may thus be the result of an independent functional neurosis.

The question arises, Which of the motor nerves of the stomach take part in the merely functional illness in the case of peristaltic unrest?

While Stiller, who has had a large experience in the domain of neuroses of the stomach, traces back peristaltic unrest to a cramp of the ring musculature of the pylorus, other authors explain it by an increased irritability of those motor nerves which innervate the musculature of that region of the stomach lying between the cardia and the pylorus.

In severe cases the anomaly of function is probably extended over all the motor nerves of the stomach, since with cramp of the pylorus alone, the peristaltic motions are not so intense as with peristaltic unrest. Fleischer had opportunity of convincing himself of this in a case of dilatation of the stomach in which the greatly contracted pylorus could each time be felt distinctly through the lax abdominal integuments.

With normal size and location of the stomach the peristaltic motions are not visible in the epigastrium, in spite of the pres-

ence of peristaltic unrest, on account of the thickness of the abdominal integument and because a part of the stomach is under the liver; it is thus very probable that many cases escape detection. Sexual excesses, repeated emotions, an unsuitable mode of living, general nervousness, as well as anemia, increase the disposition to peristaltic unrest.

Symptomatology.—If a stomach which is dilated or dislocated downward is seized with peristaltic unrest, the symptoms are, in decided cases, so characteristic that they may not easily be overlooked or mistaken. The very strong contractions of the muscles, repeating quickly, can be distinctly seen and felt as undulatory motions, especially when abdominal integument is relaxed. If the stomach, at the same time, contains liquids and gases, the peristaltic waves are often accompanied by strong gurgling noises which can be heard at some distance. These undulatory motions, caused by restless action of the muscles, generally run in the direction from fundus to pylorus; that is, from the left above to the right below, more rarely also in the reverse direction of right to left. In some cases only antiperistaltic waves have been observed. By the contraction of the muscles, the fundus of the stomach may at times be distended to the size of a child's head, so that it strongly arches the abdominal walls.

After a time the elevation sinks, to appear in another region of the stomach. At the height of the contraction of the muscles there may be a slight circular constriction or furrow seen in the middle of the stomach, dividing the organ into two nearly equal parts, so that it temporarily assumes the shape of an hourglass.

Since the muscular undulations can be observed only with a dilated or dislocated stomach, they naturally extend beyond its normal location; if peristaltic unrest of the small intestine exists simultaneously, the undulations extend also over a part of the hypogastric region, and even with an empty stomach a rolling and rumbling noise, originating in the intestines, can be heard.

While energetic contractions of the stomach hasten the execution of its normal functions, an excessive peristalsis has a directly injurious effect, and causes manifold disturbances of digestion. Patients frequently complain of a lack of appetite, belching, nausea, and vomiting. If the peristaltic unrest is very stubborn, the patients may suffer a loss in nutrition, so that the suspicion seems

justified that malignant neoplasm is developing. If the peristaltic unrest continues also through the night, the state of mind is generally much depressed, because patients are constantly reminded of their stomach and their disease; any neurasthenia which may be present is often considerably increased. If the small intestine is also the seat of active peristaltic unrest, intestinal gases and liquid contents sometimes regurgitate into the stomach. The eructations are then very foul-smelling, and often feculent masses are vomited, which may, exceptionally, even contain scybala.

The demonstration of scybala in vomited matter indicates that the peristalsis of the colon, which generally is not concerned in peristaltic unrest, is considerably increased. In spite of peristaltic unrest of the small intestine, very stubborn constipation and meteorism often occur, because the colon is, as a rule, pacific during these enteric contortions.

With normal size and location of the stomach, the objective signs of peristaltic unrest are wanting, and sometimes the disagreeable sensations of unrest in the region of the stomach constitute the only subjective symptom of the disease.

Prognosis.—If the peristaltic unrest is the result of a genuine, independent, or symptomatic neurosis, the prognosis on the whole is favorable. When injurious substances before mentioned are kept away, and the primary diseases—neurasthenia, anemia—can be removed, the peristaltic unrest, as a rule, soon recedes with suitable mode of living, diet, and with methodical use of electricity.

Diagnosis.—In order to diagnose that type of peristaltic unrest which is an independent, genuine motor neurosis with certainty, it is necessary first to exclude those other diseases which also cause an increased peristalsis of the stomach. The so-called cramps of the stomach with cardialgia are accompanied with more or less severe boring, gnawing, or cramp-like pains, and, therefore, are generally easily recognized. Whether the increased peristalsis is the result of a very strong irritation of the mucous membrane of the stomach by hydrochloric acid, organic acids, or by gases which distend the organ to excess, may generally be easily determined by a repeated chemical analysis of the stomach contents; further, the gastralgia sometimes ceases entirely when the stomach has been emptied by the tube and is thoroughly cleansed (which is not the case with peristaltic unrest). The increased peristalsis of dilata-

tion resulting from stenosis of the pylorus or duodenum is also arrested by lavage. The diagnosis is very difficult with normal size and location of the stomach. In these cases the author's method of graphically recording the motor functions by the deglutable stomach-shaped bag is, perhaps, the only reliable means of settling the differential diagnosis between peristaltic unrest of the stomach and that of the intestines. In fact, in all neuroses of motility the intragastric stomach-shaped bag gives most valuable information of the nature and intensity of the peristalsis (see pp. 76–82).

One may suspect peristaltic unrest when the symptoms recede rapidly after methodical application of electricity, and when other nervous disturbances occur coincidently. In the distinction of peristaltic unrest of the stomach from that of the intestine one should ascertain whether the rolling and rumbling is still audible with an empty stomach, and whether the peristaltic motions can also be perceived outside of the limits of the stomach. If, with an empty stomach, every splashing noise is constantly absent, and if the sounds appear again shortly after drinking water, this argues for peristaltic unrest of the stomach. If dilatation or dislocation of the stomach can be excluded, the visible peristaltic motions are to be ascribed to the intestines.

Leube has described cases in which loops of small intestine were evidently pushed up between the stomach and the abdominal wall while in active peristalsis.

Sedatives, like the bromids, opium, and belladonna, are said to exert a more controlling effect on the intestinal than on gastric hyperperistalsis.

Therapeutics.—The sufferer must be urged to keep away from injurious influences, such as sexual excesses, mental shocks, and overexertions, etc., and lead a quiet, regular mode of life. If the peristaltic unrest is a partial or resultant effect of a decided neurasthenia or anemia, a protracted sojourn in the country, in the mountains, at the seashore, and hydropathic procedures (cold rubbings, baths), will influence favorably the nervous system and the composition of the blood; the anemia must also be fought by a strengthening, easily digestible diet (scraped meats), and by iron and arsenic preparations.

In severe cases in which the peristaltic unrest continues through the night, resting in bed and a mild diet (milk, soups) are recommended, and cold bandages or packings of the stomach should be

tried, and if these do not relieve, then warm cataplasms. Every immoderate loading of the gastric walls, as well as every severe dilatation of the stomach by means of gases, is to be strictly avoided, in order not to increase the irritability of the motor nerves. Kussmaul attained very favorable results by the internal and external applications of electricity. In the former case the anode, by means of the tube, is inserted into the stomach, partially filled with a small quantity of a physiological common salt solution (0.6 per cent.), and then slow rubbing motions are to be made with the cathode externally in the region of the stomach; in external galvanization the anode is used for the last-mentioned movements, while the cathode is placed on the sternum.

Of the medicines, sodium, ammonium, or strontium bromid, —in doses of three to five gm. (45 to 75 grs.) three times in twelve hours,—extract of belladonna, dose, 0.008 to 0.013 gm. (⅛ of a gr. to ⅕ of a gr.), or codein phosphate, dose, 0.02 to 0.03 gm. (⅓ of a gr. to ½ of a gr.), are to be recommended. The bromid of strontium, 20 grs., four times daily, has our preference. Exclusive feeding by the rectum for one week is more effective when combined with rest in bed and the use of the bromids. In a persistent case of peristaltic unrest we obtained very good results from salicylate of sodium ℈j, with bismuth subnitrate, gr. xvi, three times daily.

NERVOUS ERUCTATION.

This is a frequent symptom in hysteria, neurasthenia, and allied neuropathic conditions. It is said to be particularly frequent in the sexual neuroses. The belching up of tasteless or offensive gases is a frequent symptom in most gastric diseases. In fact, it occurs at times in every normal person, and then consists of the sporadic expulsion of air that has been swallowed with the food, or of CO_2 that has been taken in with beverages, or has been formed by fermentation of the food. The pathological condition which occurs in neurasthenia consists of the explosive evacuation of tasteless gas in large quantities. The attacks are usually paroxysmal, and the gas that is expelled is generally air which is not formed in the stomach but which has been swallowed. Every time air is eructated from the stomach the closure of the cardia must be opened, and with a frequent repetition of this, a permanent relaxation of the cardia may develop. The muscular development of the pylorus

being much stronger, this orifice is not so easily opened by gas. We have known nervous individuals, particularly hysterical patients, to belch up air during the entire day, and often during the night. Air can be aspirated into the stomach when the cardia is relaxed and the esophagus is closed, either in consequence of a negative thoracic pressure when the vocal cords are closed, or because the lumen of the stomach expands and dilates under nervous influence. On the other hand, some nervous patients have the bad habit of intentionally or unconsciously swallowing air until the stomach is expanded, when the same air is eructated with explosive violence. In one of the patients of Cartellieri (*Wiener allgemeine med. Zeitung*, 1885, S. 3), 2500 eructations occurred in one hour. Some patients have dyspeptic symptoms, while in others digestion is not disturbed. We have personally known a neurasthenic colleague who could eructate whenever called upon to do so. It is probable that in these cases the air that is swallowed does not reach his stomach, but gets no further than the upper part of the esophagus, when it is again expelled. Oser has explained the aspiration of air into the stomach, assuming that it acts on the principle of an elastic balloon,—the contraction of the longitudinal muscle enlarging the gastric lumen and thereby sucking in air, and the circular muscle contracting it again and thereby expelling it. This would not explain all cases, because in some hysterical patients the eructation is so rapid and uninterrupted that there seems to be no time left for swallowing air in this manner. It is probable that a clonic spasm of the pharyngeal muscles may exist here, persistently pressing air into the esophagus, which eventually reaches the stomach, but generally is expelled from the esophagus. (Bouveret, *loc. cit.*, "Aerophagia.") Esophageal eructation and vomiting may be produced by hysterical patients at will. Cartellieri asserted that his patient (*loc. cit.*) had no time to swallow air during the attack, and Ewald raises the question whether these attacks are really nervous eructations, or only simulate them.

Pneumatosis.—This is a condition of the foregoing disease, in which the stomach is abnormally expanded with air, producing a sensation of unpleasant distention and dyspnea. When the air escapes into the mouth or intestines the torturing feelings cease. The suffering may be permanent or only periodical, and has been

attributed to a spasmodic closure of the cardia and pylorus. The dyspnea that occurs in these cases has much similarity with the "asthma dyspepticum" of Henoch. Pneumatosis may be easily recognized by an inspection and percussion of the inflated stomach, which, of course, should be differentiated from a possible distended transverse colon. In many cases persistent constipation will be found to be an etiological factor, for in these cases the pneumatosis rapidly improves when the bowels become regular. A possible gastric dilatation and atony must be excluded.

Treatment.—The patient and his attendants must be instructed that the eructation and the pneumatosis is largely a habit, and that by close observation of the patient he or she can be interrupted in the act of swallowing air. Penzoldt cured a patient of this kind who had been uninterruptedly swallowing and eructating air, by making him keep his mouth open for a half hour, as it is impossible to swallow air when the mouth is open. The eructation ceased entirely and the patient became convinced that the swallowing of air was the cause of his suffering. The explosive eructations of hysterical patients are best treated by methods directed toward the psychic condition of the case. Quincke has seen cures by introducing a thick soft stomach tube and permitting it to rest for a while in the esophagus. The cases that depend upon aspiration by alternate expansion and contraction of the stomach, are, in our experience, benefited by the intragastric application of the galvanic current. The neurasthenic foundation of the disease should receive careful attention,—thus nervous eructation and pneumatosis have been repeatedly cured by a course of surf bathing. Cold sponging and massage are very useful aids in treatment. Among the drugs that have been recommended are small and frequently repeated doses of arsenic, belladonna, or atropia, hypodermic injections of morphin, and cocain. Boas obtained good results from the following pill:

℞. Extract. physostigmatis, ... 0.13 grs. ij
Extract. belladonnæ (Alc.), ... 0.25 grs. iv
Strychnin sulphate, ... 0.03 gr. ss. M.
Fiant pill, No. XX.
Sig.—One pill three times a day.

Spraying the pharynx with solutions of cocain and menthol, and the internal administration of bromid strontium are available thera-

peutic measures. Neurasthenia depending on uric acid diathesis frequently causes nervous eructations, for which salicylate of sodium is of more value than the bromids.

NERVOUS, HABITUAL, OR REFLEX VOMITING.

In the classical experiments of Magendie, the stomach of an animal was replaced by a pig's bladder, and after tartar emetic was injected into the blood, the contents of the bladder were vomited; this experimentor, accordingly, concluded that the stomach had nothing to do with the act of emesis, but that it was brought about by action of the abdominal muscles. Tintani, however, showed later on that the experiment of Magendie no longer succeeds when the cardia still remains intact and is not cut away; therefore, the cardia, inasmuch as it can prevent vomiting, must be concerned in the act of emesis, which was found to consist of firm closure of the pylorus, opening of the cardia, while powerful peristaltic and anti-peristaltic waves traveled over the stomach. The main force for emesis is then furnished by the abdominal muscles, which are energetically assisted by the contractions of the stomach itself. There are three forms of nervous vomiting: (1) The cerebral or spinal vomiting (also known as central vomiting), which is caused by direct or indirect stimulation of the vomiting center in the medulla oblongata from other irritated foci in the brain and spinal marrow. (2) Nervous vomiting, occurring as a symptom of hysteria or neurasthenia. (3) The reflex vomiting, in a more restricted sense, brought about by reflex irritations from various other organs in the body.

Cerebral vomiting is a frequent symptom in organic diseases of the brain and its membranes, particularly when they are associated with circulatory disturbances or changes in intracranial pressure occurring more or less suddenly. It has been observed in acute inflammatory processes, like encephalitis and meningitis, also with cerebral abscesses, tumors, and focal diseases. It may result from acute anemia or hyperemia, and after concussion of the brain. It is said to occur also with vivid emotional affections, and after intoxication by opium, chloroform, ether, nicotin, and also in uremia. Spinal vomiting in the sequence of diseases of the cord is rarer, but it is quite frequent in exophthalmic goiter and in tabes dorsalis, in which it occurs in form of the gastric crises, first described by Charcot. In a majority of cases the gastric crises

are accompanied not only with severe vomiting, but with gastric hyperesthesia and hyperchylia. According to Leyden, the vomiting may be absent entirely. The attack begins without any prodromal symptoms. In the midst of well-being the patients complain of intense spasm-like pains in the stomach, particularly in the epigastrium, which radiate to the sides and to the back. The face is pale, the pulse is small, soft, and rapid, there is vertigo and palpitation of the heart. The bowels are constipated, the appetite is lost, and thirst is great. At the same time there is great prostration and weakness and a clinical picture of severe collapse. Although the patients drink large quantities of water, they pass very little urine, partly because the water is again vomited, or because it can not enter the intestines on account of an existing pyloric spasm. The abdomen is much retracted on account of clonic contractions of the stomach and intestines, as well as of the abdominal wall. Very soon copious vomiting begins. First food, then bile, mucus, and particles of blood are vomited. The reaction of the vomit shows hyperacidity. After abundant vomiting transient relief is generally experienced, and in light attacks this may be the end of the crisis; but in severe attacks the vomiting may occur hourly, continuing to the evening, with very short intermissions. During the night the attacks generally cease, to return again on the following day. This course of symptoms may repeat itself in eight to ten days, by which time the debility of the patient is very great. The suffering may cease just as rapidly and suddenly as it came. The vomiting stops, the appetite improves, and the general condition of the patient slowly convalesces. If gastric crisis occurs in a case of advanced tabes, its recognition is not difficult, but when it occurs as one of the very first symptoms of tabes, the correct diagnosis may be very difficult. In that case we must test the patellar reflexes, the reflexes of the pupil, Romberg's symptom, and inquire concerning the existence of lancinating pains. In case the connection with tabes is not established, gastric crises may be mistaken for gastroxynsis, hemicrania, or hyperacidity. Hemicrania, intense one-sided headaches, are rarely complained of in gastric crises. The gastric pains in hemicrania are insignificant. Gastroxynsis occurs only in men, after severe mental exertion or after well-known toxic influences, and is always incited by certain opportune and traceable causes, which is not the case with gastric crisis.

Periodical Vomiting (Leyden).—This is a combination of symptoms in which the prominent feature is vomiting that returns in regular intervals. In some cases the day of the paroxysm of the attack may be predicted with tolerable accuracy. The attacks begin without any marked prodromal symptoms, in the midst of apparently good health. Gastralgia may introduce the attack or may follow it. The appetite is lost, pulse small and frequent, tongue coated and dry. The patients may have intense headache and even slight delirium. The clinical picture is very similar to that of the gastric crisis. The character and the reaction of the vomit is essentially the same. The duration of the attack varies between twenty-four hours and fourteen days; some patients have lancinating pains in the extremities, in place of gastralgia. Toward the end of the attack the vomiting gradually ceases, and the remaining complaints slowly disappear. The characteristic of periodical vomiting is that the attacks occur at certain definite intervals, of from two to ten weeks. The repetition occurs with great regularity, and the disease may last many years.

The *prognosis* is therefore a very doubtful one. The distinction from gastric crisis is made by the typical periodicity, and the presence of great hyperacidity of the vomit in the crisis. Periodical vomiting appears occasionally as a primary, iodiopathic neurosis of the vagi. It has been known to occur with hydronephrosis, with floating kidney, diseases of the uterus and ovaries with intestinal entozoa, and nicotin poisoning.

Nervous Vomiting in the Course of Neurasthenia and Hysteria.—This vomiting is found more frequently in hysteria than in neurasthenia. If it occurs in neurasthenia it is associated with marked sensitiveness of the lower thoracic and the upper lumbar vertebræ to the electrical current (M. Rosenthal). The patients frequently complain of severe pain in the gastric region, pointing to a hyperesthesia of the sensory nerves, which may probably be the cause of this kind of vomiting. Stiller gives the following points which are characteristic of vomiting of nervous origin: (1) The facility of the emesis. (2) The independence of the quality and quantity of the ingesta. (3) The capriciousness with which very bizarre articles of diet are frequently retained to the exclusion of others. (4) Sometimes the elective vomiting of certain substances which seemingly are separated from the mixed chyme. (5) The carelessness with which

the patient endures the habitual sickness. (6) The tolerance of the body to the effect of inanition caused by the habitual vomiting, even when the metabolism is much reduced. (7) The extraordinary influence of the slightest external and internal causes that act on mood or temperament. (8) The frequent occurrence of emesis when no food has been taken and the stomach is apparently empty. (9) The presence of other nervous symptoms alternating or contemporaneous with the vomiting. To these Boas adds (10) the absence of important secretory or motor disturbances. In some of these cases the vomiting occurs almost every day, occasionally after each meal. In other cases the attacks occur at longer or shorter intervals, either spontaneously or after severe influences exerted upon the psychic sphere. Cases have been repeatedly observed in which only the liquids have been expelled, and in others only the solids. Sometimes the vegetable and carbohydrate foods are vomited and proteid food retained, or vice versâ. Nausea and retching are absent in the vomiting of hysterics, which occurs without any exertion.

Juvenile vomiting rarely occurs by itself, but is rather an expression of a dyspepsia developed in school-children as a result of mental overexertion. The symptoms are the following: dyspeptic complaints, gastralgia, vomiting, great pallor, dilatation of pupils, slowing of the pulse, constipation. In all of these cases improvement follows when the children are removed from school and allowed to rusticate in the fresh country air (Leyden, "Ueber period. Erbrechen," etc., *Zeitschr. f. klin. Med.*, 1882, Bd. IV, S. 605).

Reflex Vomiting in a More Restricted Sense.—Strictly speaking, vomiting is most always a reflex act, and the separation of other forms of vomiting from reflex vomiting is justifiable only on didactic grounds (Fleiner). There is hardly an organ which could not produce this form of vomiting when it is in a state of irritation. The peripheral irritations which cause this reflex vomiting are, among the first, those which strike the sensory and motor nerve-endings in the esophagus, the posterior pharyngeal wall, the epiglottis, the soft palate, and the root of the tongue. All organs that are supplied by branches of the vagus—so particularly the abdominal organs—may, under pathological conditions, excite an attack of reflex vomiting. It may occur as a result of constipation, meteorism, lead colic, irritations by foreign bodies, and intestinal parasites. It is one of the first symptoms of strangulated hernia, and of conditions of

irritation in the peritoneum. Abscess of the liver, perityphlitis, renal and hepatic colic are associated with reflex vomiting. It has been known to occur by the invasion of the ascaris lumbricoides into the ductus choledochus. Emboli in the kidney, liver, pancreas, and spleen, floating kidney, and severe concussion or contusion of any abdominal organ have been known to cause reflex vomiting. Diseases of the female sexual organs are a prolific source of this form of emesis. It is not the severe anatomical diseases of these organs that most often cause these attacks, but preferably the slight, inconsiderable affections. Normal menstruation and pregnancy are occasionally accompanied by emesis. The so-called pernicious vomiting of pregnancy may be caused by a variety of conditions, although its pathogenesis is still obscure. When vomiting is uncontrollable in a female in whom the evidences of pregnancy are unmistakable, the embryo should be removed and the uterus curetted before prostration becomes too great. Fleischer states that this should be done in order to save the life of the mother and eventually that of the child (*loc. cit.*, p. 977); the pernicious vomiting of pregnancy occurs, however, at such an early period in our experience that the child would not be viable. Every form of severe vomiting, when it continues for a week or more, will eventually produce hematemesis from local ischemias produced by the convulsive gastric contractions during the emesis. In a case that died at the Maryland General Hospital in April, 1897, the young woman, who was undoubtedly pregnant, and who refused to be curetted, vomited and purged blood in the second week so that the practitioner who presented her for admission stated that she had undoubtedly a gastric ulcer. At the autopsy, which occurred three weeks after the beginning of the attack, an embryo between two and three months old was found within the uterus, while nowhere in the stomach could a lesion be found excepting large ecchymoses, some of them attaining the size of a five-cent piece, and scattered over the entire surface of the stomach. Displacements of the uterus, pelvic exudates, parametritis, inflammations and ulcerations of the uterine mucosa, myomata, and ovarian diseases may cause reflex vomiting, which is much rarer with the diseases of the male sexual organs. Nevertheless, it is occasionally observed with injury or inflammations of the testicles and in epididymitis. Chronic inflammation about the nasal mucous membrane, polypi, and hyperplasia of the upper air-passages have been recorded as producing the disease. Eichhorst

has repeatedly observed reflex vomiting in certain individuals on hearing very shrill tones. Von Troeltzsch has called attention to the fact that irritation of the external auditory canal may cause emesis.

Prognosis of Nervous Vomiting.—The prognosis will vary with the fundamental causative disease. Gastric crises, when they occur in advanced tabes, may cease entirely after a time and remain away, although the fundamental disease continues and even becomes worse. Periodical vomiting, which occurs after insignificant disturbances in other organs, which get well without difficulty, may stubbornly persist after the fundamental disease has been cured. The prognosis is favorable wherever the causes can be recognized and completely removed.

Diagnosis.—The majority of the forms of nervous vomiting can be determined after an exhausting examination of the entire body, the urine, the blood, and gastric contents. A careful study of the previous history is indispensable. Nervous vomiting, as we have already said, may be in rare cases an idiopathic vagus neurosis (Leyden), but in most cases some palpable cause for the vomiting can be detected. Prominent among these are dislocated kidneys, hydronephrosis, uterine and ovarian diseases, entozoa, and nicotin poisoning. A careful examination of the fundus of the eyes, of the ears, and of the nose, mouth, pharynx, and larynx should never be omitted.

Treatment.—Whenever possible, the treatment must be directed to the underlying causal disease. Only when the various morbid states of other organs which have been mentioned in the etiology and symptomatology can be excluded after application of all methods of diagnostic technic, only then is a purely symptomatic treatment justifiable. Hysteria and neurasthenia are to be met by hydropathic procedures, or by absolute rest, abstention from mental and emotional excitement, and a sojourn in the mountains or at the seashore. In most cases the greatest possible rest, and strict avoidance of psychic disturbances will be indispensable. Gastralgic pains and hyperesthesia can be relieved by a hot cataplasm on the stomach, but the application of the galvanic current anode on the stomach, or in the stomach, and cathode alternately on the sternum or spinal column will be more efficacious. The internal gastric douching with warm water, and, as we have found, spraying of the inside of the stomach with solutions of menthol and cocain, are also generally followed by cessation of the pain. In vomiting

of pregnancy, rectal feeding and six grains of the basic orexin in a gelatin capsule three to four times a day should be tried first, but curetting of the uterus should not be delayed too long. The following formula we have found efficacious in the treatment of this form of vomiting of the non-pernicious type:

℞.	Cerii oxalatis, ...	4.0	gr. lx	
	Cocain hydrochlor., ...	0.20	gr. iij	
	Menthol, ...	0.80	gr. xij	
	Bismuth salicylatis, ...	4.0	℥j	
	Elixir simpl., ... q. s.	180.0	f℥vj.	M.

SIG.—f℥ss on an empty stomach four times daily.

The idiosyncrasy of the patient concerning diets should be carefully studied. A priori, "no diet can be suggested that shall be universally applicable to all cases." The ingestion of liquids must, as a rule, be very much limited, and thirst relieved by colon enemata. If every meal is vomited it is best not to permit the ingestion of larger quantities of food, but simply to give nourishment in very small quantities—iced milk, champagne, cold tea or coffee, or egg-albumen with brandy, or clam bouillon in tablespoonful doses. Superacidity must be treated according to principles laid down in the chapter on this subject. When there is abnormal hyperesthesia of the stomach, it is well to feed the patient by rectal enemata for about a week to ten days according to methods described in the chapter on Dietetics. The most effective sedative that we have is morphin, particularly the hypodermic injection of ¼ of a grain together with $\frac{1}{100}$ of a grain of atropin sulphate. The following suppositories are useful where we do not wish to create an adaptation to morphin:

℞.	Extract. belladonnæ, ...	0.20	gr. iij
	Codein phosphatis, ...	0.80	gr. xij
	Butyr. cacao, ... q. s.		

Supposit. No. XII.

SIG.—Insert one suppository every two hours during the attack.

When the nervous vomiting persists, even during the night, the bromids, together with chloral hydrate, are of approved efficacy. We are in the habit of giving 30 grs. of bromid of strontium with 15 grs. of chloral in peppermint water, repeated every three hours until sleep supervenes. We have also found the following combination to be a reliable means of combatting this neurosis:

℞. Menthol, ... 1.0 grs. xv
Cocain hydrobromatis, ... 0.4 grs. vj
Aquæ chloroformi, ... 120.0 f℥iv
Spir. vini. gallic., ... 60.0 f℥ij.

SIG.—One tablespoonful three times a day. M.

In the treatment of the gastric crises Boas has found that iodid of potassium and bromid of sodium exert very favorable influences in diminishing the frequency and intensity of the attacks. The use of the constant current, with the anode within the stomach and the cathode over the spinal cord in the cervical region, is generally followed by a very marked, palliative effect. Chloroform, three to five drops given on sugar, ammoniated tincture of valerian, 25 drops p. r. n., and the compound spirits of ether, 15 to 20 drops p. r. n., might be tried, but in our experience they are rarely efficacious.

INSUFFICIENCY OR INCONTINENCE OF THE CARDIA.

Incontinence of the cardia, due to paresis or paralysis of the motor nerves of the ring muscle, is a comparatively rare malady, though somewhat more frequent than that of the pylorus. It appears either as an independent disease, or as a partial or resultant phenomenon of other neuroses. The relaxation of the cardia produces an effect directly opposite to that of cramp of the cardia. While the latter, as is well known, prevents the removal, by eructation, of the air and gases introduced into the stomach during meals, as well as that of liquid or solid contents of the stomach, insufficiency of the cardia, on the other hand, much facilitates it. When the relaxation of the cardia is accompanied by an increased irritation of those motor nerves which innervate the dilator of the cardia, so that by the spasm of the latter the esophageal orifice of the stomach is actively enlarged, energetic peristalsis of the stomach, with the additional influence of the abdominal pressure, will raise portions of the gastric contents into the esophagus, and even into the mouth.

The firmness of the cardial closure, even under normal circumstances, seems to vary greatly in different individuals. While many persons vomit only with difficulty,—since the resistance at the esophageal orifice of the stomach is greater, so that they have to make use of almost all known remedies and devices in order to overcome it,—and eject only a portion of the contents of the stomach,

others vomit with exceeding ease, and they succeed, with only a moderate contraction of the abdominal muscles, in emptying the stomach entirely through vomiting. In one and the same individual the closure of the cardia may vary at different times. At times small quantities of ingesta come up again into the mouth after eating, while at other times, with the same food and the same fullness of the stomach, this does not occur.

If only small quantities of the contents of the stomach come up into the mouth, now and then, this does not yet constitute an abnormal condition; the latter exists only then when large quantities of ingesta come up after eating, and when this is repeated frequently, almost regularly every day for a considerable period of time. If the masses which come up are expectorated, the whole process is called regurgitation; but if, on the other hand, they are swallowed again, it is called rumination (merycism, remastication), even when the regurgitated foods are not chewed again, which is observed in a small number of patients.

Concerning the causes of regurgitation and rumination, opinions still differ greatly at the present time. Some authors trace both conditions to a permanent relaxation of the cardia, but the presence of the deglutition sounds is an argument against a permanent incontinence, according to Ewald; other authors assume a temporary insufficiency of the cardia, and still others, in addition, an increased irritation of the motor nerves—and eventually also of the sensory nerves—of the stomach. According to M. Rosenthal, regurgitation and rumination are caused by an increased irritability of the vagus, and with this also an increased irritability of the motor nerves leading from it and supplying the dilator cardiæ, which causes a spasm of the latter, and through this an active enlargement of the esophageal orifice of the stomach. Whether the disease is of central or peripheral origin is at present also impossible to decide.

Regurgitation.—At a longer or shorter period after meals large quantities of the liquid and solid contents of the stomach are at first involuntarily brought up again into the mouth and are then expectorated. With protracted duration of regurgitation the patient generally learns how to facilitate the ascension of the ingesta by means of rather severe contractions of the musculature of the abdomen. According as regurgitation takes place in the first or second period of the digestion of the stomach, the regurgitated food particles have either the same taste as in eating, or they

taste sour (HCl) or bitter (peptone). Regurgitation is not easily mistaken for vomiting, since the sensations of nausea experienced before and after the latter are entirely lacking with regurgitation. Regurgitation takes place without any especial exertion on the part of the patients, and is easily distinguished from the retching forth of foods previously eaten, in cases of stenosis or the formation of diverticula in the esophagus—that is, from the rising up of the same into the mouth, when the diverticulum is full and runs over. The difficulties of deglutition, the result of sounding the esophagus, as well as the constant absence of hydrochloric acid in the regurgitated masses, make a sure differentiation of the two first-mentioned diseases possible. Most patients are able to suppress regurgitation, but in a few cases they do not succeed in this, however much they try. If copious quantities of the contents of the stomach are regurgitated in quick succession and are expectorated, the general nutrition may suffer considerably; but generally these patients have a very hearty appearance. Other nervous disturbances—signs of hysteria or neurasthenia—may be present coincidently. With protracted duration, regurgitation may develop into rumination.

The prognosis is generally favorable with regurgitation, since the state of nutrition remains good, and if disturbances of nutrition appear, the patient often can be induced to swallow the ingesta which has risen into the mouth and to energetically prevent its coming up again.

Therapeutics.—Regurgitation is promoted by hasty eating and quick swallowing of insufficiently chewed foods, especially when the latter are of difficult digestion; a diet should, therefore, be prescribed which is easily digestible, and the patients should be directed to eat slowly and chew thoroughly. Gladstone's suggestion is to give each morsel of food one bite or grind for every tooth in the mouth,—*i. e.*, 32,—before it is swallowed. It is best for the patients to eat in the company of such persons whose good opinion they value, so that they avoid expectorating the ingesta which has risen into the mouth, and will rather swallow it again, endeavoring to combat regurgitation to the utmost of their power. If indications of hysteria or neurasthenia can be shown, these diseases are first to be treated. In stubborn cases the swallowing of small pieces of ice is recommended (Alt), which may reflexly induce the musculature of the cardia to contract. In addition, mas-

sage of the epigastrium, internal and external galvanization and faradization, as well as internal administration of strychnin nitr. (0.003 : 0.006) are indicated. The galvanic current should be applied in the same manner as in cardiospasm.

RUMINATION OR MERYCISM.

Patients afflicted with this neurosis return the ingesta from the stomach through the esophagus back into the mouth sooner or later after they have been swallowed. This is not only done without nausea, but apparently with a certain enjoyment. The raised food is rechewed, and either swallowed again or expectorated. This occurs habitually several hours after meals and without the least exertion. Rumination in the human subject has been known for a long time. Fabricius Ab. Aquapendente described the disease in 1618. As the knowledge of physiology of older practitioners was very limited, and they had no conception of the functions and mechanism of gastric digestion, the most peculiar hypotheses were developed in explanation of this very interesting neurosis. It was firmly believed that ruminants descended from parents with horns, or that they at least had a horned father, or had been nursed from the udder of ruminating horned animals. It was also believed that the stomach of human ruminants was divided into several sections by partitions, as we find them in cattle. After it was found at autopsies that ruminants possessed stomachs like all other human beings, the profession gradually accepted the neurotic explanation of the malady.

Etiology.—A neuropathic constitution is a frequent factor in the development of rumination. Heredity seems to have some effect in the matter, as ruminating fathers have been known to have ruminating children. The element of imitation and suggestion can, however, not well be eliminated under this question of heredity. Freund and Körner have described a case in which two children developed this habit in imitation of their ruminating governess. The disease seems to be more frequent in men than in women. It occurs in all classes of society and at all ages. The following are some of the causes assigned to rumination. Sexual excesses, masturbation, fear, terror, anger, psychic irritations, and the very hasty deglutition of badly masticated food, particularly when it exists exclusively of vegetables, injury to the epigastrium, achlorhydria, obstinate constipation, and gastro-enteritis. It is claimed by Dehio

that whooping-cough may be followed by this disease; this is very plausible, because pertussis brings on frequent vomiting, and thereby an incontinence of the cardia. The majority of ruminating patients belong to the class of neurasthenics, and hysterical, hypochondriacal, epileptic, anemic, choreic, and idiotic individuals. The disease has been studied by Bourneville and Séglas, Dehio, Alt, Boas, Bear, Ducasse, Decker, Einhorn, Oser, Pönsgen, Johannessen, Lebert, M. Rosenthal, and E. Singer.

In the *N. Y. Med. Record* for June 12, 1896, Dr. H. A. Minassian reports an interesting case of merycism with achlorhydria and hyperperistalsis which was cured by hydrochloric acid, exclusion of fluids, and exercise of self-control. In the same journal for July 10, 1897, Andrew Halliday, M.B., a physician of Nova Scotia, who personally has the power of regurgitation and rumination at will, gives the analysis of his own stomach contents: Forty-five to sixty minutes after a Ewald test-meal the total acidity was 45 to 55. The free HCl 0.124 to 0.1604 per cent. His motility seems normal. These two cases suffice to show the variability of the state of secretion and motility in merycism.

Symptomatology.—The regurgitation of ingesta from the stomach into the mouth is usually at first voluntary, but later on involuntary. The rumination differs from simple regurgitation, in that the raised food is expectorated in the latter disease, but is swallowed again in the former. The ascent of the food from the stomach causes a pleasurable sensation to these patients, and they assist the act by bringing into effect the pressure of their abdominal muscles. In severe cases the rumination occurs after every meal, and lasts either only for the first hour or for five or six hours. The condition of the secretory function is variable. Jürgensen found no free HCl, Bear and Boas found subacidity, while Alt demonstrated hyperacidity in one of his cases. In some patients secretion was found to be normal. In one of the cases observed by us we found that the state of the secretion varied, as expressed in the chemical analysis of the raised masses according to the time after the ingestion in which they were regurgitated. If they were regurgitated immediately after the meal they were alkaline, contained no free HCl nor ferments which, however, were present within forty-five minutes of the first ingestion of food. It is probable that many of the discrepancies concerning the state of the secretion, as stated by the various authors mentioned, can be ex-

plained on the same grounds. Alt has suggested a very interesting theory in explanation of the ruminating habit (*Berlin. klin. Wochenschr.*, 88, Nos. 26 and 27): he suggests that the object of the act may be the correction of defective chewing and insalivation of the food, and the hyperacidity caused thereby. Acting accordingly, Alt treated his patient with alkalies, and claims to have found that the case ruminated less frequently and by and by could suppress the habit. Boas (*Berlin. klin. Wochenschr.*, 1886, No. 831) has published a case of rumination with subacidity, and in this case improvement followed the administration of HCl. According to Einhorn, but 106 cases of this malady have been described up to 1896, which cases occurred chiefly among the professional and educated classes—physicians, lawyers, and philologists. This observation was also made in a report by Johannessen (*Zeitschr. f. klin. Med.*, Bd. x, S. 274). The following is a brief account of a case occurring in our private practice. M. F., age thirty-eight, mother has been a highly nervous woman, much afflicted with insomnia and neuralgia; father died at the age of fifty-six from Bright's disease. He was a very irascible and eccentric man. M. F. has had no severe disease, except gout four years ago. He is a pianist of exceptional ability, and has played in foreign countries as well as in the larger cities of the United States. Ordinarily and when in a quiet frame of mind he rarely ruminates, but when he gives instruction, particularly when he has to play at a concert, or at other times when he is emotionally excited or disturbed, he begins to raise food into his mouth, which he at first swallows for about two hours. He confesses that he chews the food, and actually enjoys it, but at the expiration of two hours the muscles of mastication become so exhausted that he can no longer chew the raised masses. He would then like to put an end to the ruminating, or rather to the rising of the food, but then can not stop it as it persists in coming up from his stomach. He then terminates the rumination by voluntarily evacuating his stomach through vomiting, which he accomplishes very easily.

The masses begin to ascend within ten to fifteen minutes after a meal, and are then alkaline. He is also aware that they begin to taste salty sour, as he calls it, forty-five minutes to an hour after the meal. The total acidity one hour and a quarter after a meal, as judged from the regurgitated masses, is 70°. Free HCl 30°, erythrodextrin present. Achroodextrin present. Gastric motility,

as determined by Hemmeter's method, is plainly exaggerated. Physical examination of the thoracic and abdominal organs negative. Urine negative. Examination of blood negative. On one occasion this patient had not ruminated for three weeks, when he had to fulfil an engagement at a concert. The author, for the sake of study, was present when he took his supper on the evening of this concert. Within fifteen minutes after the supper we observed, by the movements of his throat, he had begun his old bad habit, which kept up during the entire evening, and was plainly observable while he was performing at the piano during the concert.

Atony and dilatation may be present, together with rumination, and the state of the motility seems to vary as much as that of secretion. The general nutrition is not, as a rule, affected, although the disease may have existed a long time; but when the patients persistently spit out the ascending masses of food, instead of swallowing them again, or when severe disturbances of secretion and motility exist, the patients rapidly lose strength and weight. According to von Hacker and G. Singer, there is an insufficiency of the cardia and a dilatation of the esophagus immediately above the cardia, which has been caused by mechanical expansion, resulting from the regurgitation of large bits of food. This esophageal expansion has been demonstrated by these authors with the esophagoscope.

Prognosis.—This is not a serious disease, as the general nutrition remains good, and if the patient really does begin to suffer he may be relieved by a rational psychic and symptomatic treatment, particularly if the patient himself will aid the therapeutic measures by self-control. The *diagnosis* presents no difficulties whenever the physician can observe a patient in the act of rumination; regurgitation and emesis imply the spitting out of food, and are always associated with nausea or some other unpleasant sensation.

Treatment.—Medicinal treatment in this disease is of little value. The state of the secretions should be carefully determined, and subacidity or achylia corrected by the administration of HCl, and hyperchylia by the use of calcined magnesia and bicarbonate of sodium. Körner is enthusiastic on the value of small pieces of ice given directly after meals. The stomach-tube has been used for the lavage and artificial feeding, but the relief has been only temporary. The physician should, however, in all cases insist on slow eating and careful chewing; the food should be easily digest-

ible and largely composed of gruels and diet of a soft consistency. The patient should always take his meals in the presence of persons for whom he has considerable respect, and who understand to oppose the morbid habit with kindness and yet with severity. The success of the treatment will depend upon the will-power of the patient himself. Whenever the patient feels a desire to ruminate he should be prompted to resist the temptation with all the self-control at his command. He should be guarded against using the contraction of the abdominal muscles to assist the act. Pönsgen, Boas, and Einhorn report permanent cures resulting from such persistent autosuppression. A trial might be made with the intragastric use of the faradic and galvanic current. Hydropathic methods are sometimes useful. In one case observed by the author, in which every meal was persistently ruminated, we carried out rectal alimentation for twelve days, not allowing anything to enter the stomach during this time. Since then four months have elapsed and rumination has not thus far returned. We hesitate in attributing this recovery to the rectal alimentation, although, of course, it was impossible for the patient to regurgitate and ruminate when no food was contained in the stomach. However, the psychic effect of hospital treatment, the entirely new surroundings, and attendance by intelligent nurses, the constant rest in bed, may have contributed as much as the rectal feeding toward the recovery. In another case the author cured the patient by giving ten grains of quinin after each meal. The good result is not attributed to the antimalarial effect, but rather to the fact that the quinin rendered the food so disgustingly bitter that the patient suppressed the regurgitation. Rossier claims to have cured one case by muriate of morphin, and another by large doses of opium. In our experience these remedies have been useless.

INSUFFICIENCY OR INCONTINENCE OF THE PYLORUS.

It has been known for a long time that an insufficiency or incontinence of the pylorus may be caused by organic diseases of the stomach and intestines, by carcinoma and ulcer, by bringing about a partial or complete obliteration or carcinomatous infiltration of the annular muscle, so that the latter becomes incapable of functioning; or it may be caused by a stenosis of the duodenum leading to advanced dilatation of the initial part of the same. Attention was first called by Ebstein to those interesting, though very

rare, cases of insufficiency, which appear, in the absence of anatomical changes, genuine neuroses—paralysis of the motor nerves of the annular muscle. It had been previously observed by Ebstein as a concomitant phenomenon of myelitis due to compression, and also in hysteria and gout; but it may, perhaps, occur also as an idiopathic malady. If the muscular insufficiency is confined to the pylorus, then the foods and liquids, according to the degree of the insufficiency, either remain a very much shorter time than usual in the stomach, or enter the intestines immediately after their ingestion. The nutriments then are not at all digested in the stomach, or only to a slight degree, so that the whole, or almost the whole, burden of digestion falls upon the intestine. Since, however, repeated experiments upon human beings and animals have proven that the intestine, in normal conditions, may entirely make up for the lack of digestion in the stomach—and the experience gained from the treatment of persons that have undergone a resection of the pylorus confirms this, therefore, even with protracted duration of the pyloric insufficiency, disturbances of nutrition generally fail to appear, especially when an easily digestible diet is prescribed, suitable to the malady, and provided that the intestinal functions are normal.

The symptoms of pyloric insufficiency are, in brief, the following, viz.: If frequent vomiting and belching existed, these suddenly cease after the setting in of the insufficiency. If rather large particles of food get into the intestine, which mechanically irritate its mucous membrane more than usual, then the increased peristalsis may cause diarrhea. This may also be brought about by very cold or very hot foods or drinks, which are gradually warmed or cooled by the stomach, as the case may be, before their entrance into the intestine, when the closure of the pylorus is normal. If a quantity of air was swallowed with the foods, or if drinks rich in carbonic acid have been imbibed (beer, seltzer water, champagne), a very acute tympanites of the intestine may develop from the escape of air or carbonic acid gas.

According to Ebstein, one can not succeed in distending the stomach with the artificial production of carbonic acid gas in the organ by acid. tart. and sod. bicarb.; but Ewald, Boas, and other authors justly contend that this evidence is not conclusive; since, even when the musculature of the pylorus functions in a normal manner, every attempt at distending the stomach by means of

gases may remain unsuccessful, because the amount of the gases formed is too small. Further, with an empty stomach the pylorus (Kussmaul) is normally so relaxed that a portion of the carbonic acid gas may easily pass over into the intestine, without the presence of any real insufficiency. This source of error may, however, be removed by letting the patient eat a test-breakfast before the distention of the stomach (Fleischer), since then, under normal circumstances, the closure of the pylorus becomes so firm that the carbonic acid gas set free can not at once escape into the intestine; or if, instead of CO_2, air is forced into the stomach by means of a tube and a pump, increasing the supply according to necessity. If the air passes quickly into the intestine the inflated ascending colon appears as a thick swelling on the right side of the abdomen. For a proof of the purely nervous origin of insufficiency it is necessary to exclude the above-mentioned diseases of the stomach and intestine, and such organic diseases of the stomach as chronic gastritis, which probably bring about a serous infiltration of the annular muscle, and may lead to a temporary insufficiency (Eichhorst, Boas). If the insufficiency be due to a stenosis of the duodenum, the stomach may very well be distended by CO_2, or air, in spite of insufficiency. If, after finding out the lower limits of the stomach, quantities of water are introduced through the tube, and no dullness appears in the lower parts, while gurgling noises, before lacking, now become audible in the intestine, and if the intestinal loops, just before this, gave a tympanitic resonance, and after the introduction of water exhibit a muffled sound, insufficiency is to be inferred. After the introduction of one grain of salol, or 0.1 gr. of iodoform, with the test-breakfast, salicyluric acid can be shown in the urine after taking the former, and iodin in the saliva after taking iodoform, much sooner than with continence of the pylorus, as these chemicals enter more quickly into the intestine, and on account of the neutralization of the hydrochloric acid by the alkaline intestinal juice, they are immediately converted into soluble compounds which may be absorbed. (See pp. 73, 74.)

Insufficiency of the pylorus may be recognized by the author's method of intubating the duodenum (*Archiv f. Verdauungskrankheit.*, Bd. 11, S. 85), and by the spiral revolving sound of F. Kuhn, of Giessen, and of F. B. Turck, both of which may be used for sounding the pylorus. This operation was first performed by the author and also by Dr. Turck, and Kuhn's claims of priority of sounding the py-

lorus are unfounded. The interesting observation of a case of insufficiency, reported by Schütz, in which it was possible to distend the stomach by means of CO_2 but not by air, so that by the increased irritation of the mucous membrane of the pylorus by the carbonic acid gas a contraction of the annular muscle was brought about, but with the forcing in of air the stomach did not become distended, points to the fact that different degrees of insufficiency occur. If, as easily happens in a case of pyloric insufficiency, some of the contents of the intestine go back into the stomach causing dyspeptic complaints by the irritation of the mucous membrane and neutralization of the HCl, this can be recognized by testing for bile (p. 121).

Therapeutics.—If symptoms of irritation of the intestine—namely, diarrhea—are absent, only dietetic treatment is necessary. In order to relieve the intestine of its excessively burdensome task, we must prescribe easily digestible, well-prepared foods, which are to be carefully masticated, and are not to be taken too hot or too cold.

If, on the other hand, complaints such as diarrhea appear, we must attempt, in addition to the treatment of the causal disease, to get rid of the insufficiency by means of massage, internal and external galvanization and faradization applied directly to the pylorus by the author's method (p. 55), douches, and eventually also by giving strychnin, gr. $\frac{1}{30}$, t. i. d. If flatulence and constipation arise on account of the neutralization of hydrochloric acid which, as is well known, has a stimulating effect upon the peristalsis of the intestine, and, further, acts as an antiseptic, then the massage, as well as the galvanization, is to be applied also to the intestine, and dilute HCl should be administered in doses of 30 drops largely diluted and taken after meals through a tube. In all other indications the treatment must be directed to the cause.

ATONY OF THE STOMACH (Myasthenia Gastrica, Mechanical Insufficiency of the Stomach).

In the consideration of dilatation of the stomach, we have fully quoted the classifications of Riegel, Schreiber, Boas, Naunyn, and Rosenbach. By simple atony we mean that combination of symptoms in which there exists a disproportion between the peristaltic work the stomach has to perform and its real expelling muscular force. Objectively, the disease makes itself known by the fact

that the ingesta is retained in the stomach beyond the normal time, but although the muscular action of the organ is weakened, the food is eventually expelled into the duodenum. By this it is distinguished from the mechanical insufficiency of the second degree, the pronounced dilatation, in which the food is permanently retained in the stomach, and only exceptionally reaches the bowels. Every relaxation of the muscular wall that is not due to any pyloric or other mechanical obstruction may be justly designated as an atony. In simple atony the stomach is not considerably enlarged in the empty state, but only becomes so with increasing burdening of the ingesta, but in atonic dilatation the diseased organ remains in a dilated state even after it is empty. (1) This disease may occur as a typical, primary, idiopathic neurosis, as a consequence of persistent overloading the stomach with indigestible food, particularly with liquids. It may appear very suddenly as a transient affection, under the influence of violent emotional disturbances,—fright, anger, grief, etc., occurring in this way principally in neurasthenic persons. It is probable that gastric myasthenia may be inherited, and may be transmitted through several generations. It is generally referred to as the so-called " weak stomach " in some families. The abuse of alcoholic liquors, particularly of beer, and even of coffee and soups, has been assigned as a cause. (2) Myasthenia occasionally appears as a reflex neurosis evolved from other diseased organs; for instance, diseases of the liver, bile passages, peritoneum, intestines, kidney, and sexual apparatus. (3) It occurs as a secondary neurosis, constituting part of the symptoms of hysteria, neurasthenia, gastrospasm, cardiospasm, and pylorospasm. It has been observed as a complication of gastroptosis, nervous dyspepsia, of ulcer, and chronic gastritis. There are a number of intestinal affections which may be complicated or even cause atony. These are stenosis in the inferior horizontal portion of the duodenum, or stenosis of the jejunum, enteroptosis, and stenosis of the colon; passive congestions and enlargements of the liver, cholelithiasis, are definitely known to be etiological factors. Critically speaking, we designate only such cases gastric atony in which the organ retains its normal size when it is empty. As soon as the stomach remains permanently enlarged, even when it is empty, it is more logically classed with the motor insufficiency of the second degree, as atonic dilatation. As we have seen in the section referred to, Riegel makes a separate class for stenotic dilatation.

The final cause of simple atony, or myasthenia, is malnutrition, overstretching of the muscles and motor nerves of the stomach, or an early and progressed exhaustion after undue and improper exertions. Occasionally unknown neurotrophic influences may be responsible for the origin of atony. As secretion and absorption depend more or less upon energetic contraction of the gastric muscularis, they are in most cases interferred with in the absence of effective muscular tonicity. The gastric contents do not diminish in quantity as rapidly as they should, and in consequence of this the gastric wall is excessively expanded by the prolonged weight of food. If fermentation of the ingesta occurs with abundant formation of gases, the expansion will be still greater. The gaseous distention may secondarily produce spasm of the pylorus and cardia, thus adding another etiological factor to the causation. If the atony is very progressed and has persisted for a long time, it may develop into an irreparable dilatation, particularly if dietetic and hygienic regulations are disregarded. We have observed a number of cases of this kind, in which permanent dilatation was developed when long-standing gastric distress was left unheeded. Myasthenia—by diagnostic methods for judging the motility—may be found to be very pronounced, and still remain latent and unnoticed even by the patient for a long time.

The following is an example of this class of cases. Miss S., aged twenty-two, a well-built and apparently healthy girl, moving in the best circles of social life, complains of only one symptom, that is a severe headache, occurring two or three times of every week, and lasting for twenty-four hours. On being questioned about her stomach she asserts that her digestion is good, appetite excellent, and bowels regular. She eats all kinds of food, apparently without distress. On passing the tube in the morning, on an empty stomach, 200 c.c. of a slightly yellowish mucous liquid was obtained which shows free HCl by Congo paper. The next day she was directed to take the double test-meal (described on p. 111). One hour after the second meal the contents were drawn, and rice and egg of the early breakfast, which was taken six and a half hours before, were still present in her stomach, together with a considerable amount of mucus. Total acidity, 60; free HCl, 20. Subsequently the same state of affairs was found after other test-meals. The striking feature of this case is that, although there is a pronounced gastric atony, the patient is not at all conscious of it, and regularly

expresses surprise when she recognizes food in the lavage that has been taken eight to twelve hours before. It is very probable that such cases as this one would develop unmistakable symptoms of myasthenia in a very short time if left untreated. Superacidity and supersecretion may cause cardiospasm and pylorospasm, and subsequently gastric atony, by the irritation of the muscular structures at the orifices caused by the intense acidity. But, reversely, gastric atony may cause superacidity and supersecretion by the fact that the ingesta are retained in the stomach for an unduly long period, and thereby excite the gastric glands to stronger functioning. Stiller and Boas assert that gastric atony rarely develops into permanent gastric dilatation. The subjective symptoms of gastric atony are very similar to those of gastritis and insipient dilatation. The patients complain of pressure and pain in the head, the feeling of pressure and distention in the stomach, a premature sensation of fullness during eating by which the appetite becomes appeased very rapidly, very frequent eructation, and persistent constipation. The feeling of pressure is intimately associated with the ingestion of food. When the stomach is empty, the patient feels quite well. The headache is very frequently observed, together with the so-called stomach vertigo. We can agree with Boas that this so-called gastric vertigo (Trousseau, *Gazette des Hôpitaux*, 1862) is much more frequently found in atony and dilatation than in any other gastric disease. The feeling of pressure and distention may persist as long as there is food in the stomach: in recent cases, about one hour; in advanced cases, it continues from one meal to the other. One of the most frequent symptoms is eructation of air, which generally has the taste of the food that has been last taken. We have noticed that the most annoying sensations of pressure in the advanced cases are felt after breakfast, at a time when one would presume that they should be absent, since the stomach should have been rested during the night. The duration of the time after meal during which the eructations continue is generally a good indication of the extent and degree of the myasthenia. In some cases, however, we may be confronted with typical neurotic regurgitation and eructation, that has existed before the atony developed, and then this indication is invalid. If there is hyperacidity, the atony may be associated with attacks of vomiting, and pyrosis is generally present. The constipation is undoubtedly an expression of the general atony of the entire gastro-intestinal tract.

Objective Symptoms.—The most important distinguishing sign between simple atony and dilatation consists in the fact that the stomach, in the former, should be empty in the morning, when nothing has been taken since the previous supper; in other words, the jejune stomach of atony contains no food particles, while the stomach in a state of dilatation does contain them. The splashing sound in the epigastric region is absent in the morning with simple atony, but it is present in dilatation. The size and location of the stomach vary physiologically. A myasthenic stomach yields and distends with greater readiness when it is filled with water or air than a normal stomach. Boas asserts that even an atonic stomach may react more normally to the distending force of water and air in cases in which the superimposed layers are swelled and much thickened by inflammatory infiltration. The methods of investigation and diagnosis which we have found useful are, in addition to inspection, palpation, percussion, and auscultation, the distention of the stomach by air or carbon dioxid gas, the Hemmeter intragastric stomach-shaped bag and the gastrodiaphane. Very frequently the contour of the greater curvature may be recognized on the outside of the abdomen. Palpation may, in some cases, instruct us concerning the limits of the organ, and enable us to separate it from adjacent organs. The so-called splashing sound may be elicited by permitting the patient to drink a glass of water, and then, placing the palm of the left hand firmly over the right hypochondriac region, and by gently tapping the epigastrium with the right hand, the sound is generally very evident if atony is present. In most cases of gastric atony a splashing sound can be heard with binaural stethoscope on shaking the stomach from the outside. Dehio has given a very expedient method for judging the elasticity of the gastric walls by means of gradually increasing quantities of water; at first ¼ of a liter is taken, and the location of the greater curvature determined; then, in short intervals, ¼ of a liter is taken at three successive periods, and after each ¼ of a liter increment the further descent of the greater curvature is determined by palpation and percussion. A healthy stomach will not reach the line of the umbilicus under these conditions, while an atonic stomach may have transgressed far beyond it. Auscultation elicits sounds only when the stomach contains liquids or shortly after they are ingested. It is best to use the binaural stethoscope in these cases, as then both

hands are free to palpate and move the stomach to obtain the percussion sound. Boas holds that we have no reliable method to test the gastric elasticity and tonicity (*loc. cit.*, p. 76). We consider that our method of recording the gastric peristalsis on the kymograph, as described in the first part of this book, is also an excellent method for investigating the gastric tonicity, for, as our stomach-shaped intragastric bag on being distended gradually fills out the lumen of the stomach exactly, the indications of pressure which are obtained on the kymograph are reliable representations of the tonicity. Moreover, we have experimented with an electrodiaphane contained within our stomach bag, so that when the bag was distended in a dark room the gradual descent of the greater curvature could very plainly be seen. By reference to the description of the apparatus on pages 76 to 78, it will become evident that we can easily determine the amount of air or water with which the bag is distended within the stomach; so with this method, which in a modified form was also used independently, after our first publication by Professor Moritz, of Munich, for studying the gastric motility, we may determine also the elasticity and tonicity of the stomach.

Percussion.—In percussion of the stomach, we must attempt to define its four limits, viz.: the upper, lower, right, and left limit. The lower limit may, on percussion, be confounded with the transverse colon if the latter still be in its normal position. The way out of this difficulty is to fill the transverse colon with water, which gives a dull percussion note through the abdominal wall, while the stomach may be distended with air or gas, giving a clear tympanitic sound. When both the stomach and the colon are filled with gas or with solid material simultaneously, it is almost impossible to distinguish between the two by percussion. It is best to evacuate the colon and fill the stomach with water, or vice versâ to evacuate the stomach and fill the colon with water. In our clinic we use the rubber stomach-shaped intragastric bag methodically, and when it is distended there is no difficulty at all to percuss and palpate the stomach. The determination of the upper border of the stomach is, in our experience, no easy matter, since there are no very striking differences in the percussion note of the lower edge of the left lung and the highest portion of the gastric fundus which is normally covered over anteriorly by the lung in inspiration. The upper border may be best determined by filling the stomach with water

and then percussing over the left lung along the parasternal line from above downward. Pacanowski (*Deutsch. Arch. f. klin. Medizin*, Bd. XL, S. 342) gives the following determinations of the upper limit of the stomach. In the left parasternal line it is at the lower edge of the fifth rib or in the fifth intercostal space. In the left mamillary line the limit is in the fifth intercostal space extending to the sixth rib, or into the seventh rib. In the anterior left axillary line the upper limit is at the lower edge of the seventh or eighth rib, rarely under the eighth rib. The determination of the left and right gastric limits seems most hyperthetical to us and not of diagnostic value, because here we may confound the percussion notes of organs superimposed upon the stomach.

In our experience, a clear conception of the size and location of a normal stomach can only be obtained when it is distended by gas, air, or water. Naturally, this can not be done if there is any suspicion of recent ulcer, cancer, or perigastritis. We have already spoken of the value of the electrodiaphane in ascertaining the size and location of the stomach, and, notwithstanding numerous objections, consider the method practical. According to Kuttner and Jacobsohn (*Berlin klin. Wochenschr.*, 1893, No. 39), a prolapsus of the stomach may be distinguished from a dilated stomach by the fact that the dilated organ changes its position with inspiration and expiration because it is in juxtaposition to the diaphragm, whereas a gastroptosis does not change its position during respiration. C. A. Meltzing (*Arch. f. Verdauungskrankheiten*, Bd. II, S. 436) has confirmed the observations of Schwartz that gastrodiaphany has an undoubted diagnostic value. It is important to emphasize this fact, since the method has been criticized by Renvers, Kuttner, and Jacobsohn, who have asserted that intestinal loops filled with gas, when adjacent to the stomach, will also become translucent or luminous when the light is introduced into the stomach, and Langerhans (*Wiener med. Blätter*, 1895, No. 45) denies that electric transillumination of the stomach has any diagnostic value whatever. We believe that the method is not only practical, but very useful, and that the objections of its adversaries have been squarely met and disproved by the report of Meltzing (see pp. 102 to 104).

After all, the most convenient method of determining gastric atony, and that which is available for every practitioner, is by means of the double test-meal used in our clinics (see pp. 111 and 112).

According to Boas' suggestion, a full meal is preferably given in the evening, when a healthy stomach will show no demonstrable signs of food particles the next morning. It should not be forgotten, in this connection, that even healthy stomachs may contain mucus, gastric juice, and bile in the morning before food is taken. The chemical analysis of the stomach contents in gastric atony yields no results useful for diagnosis, because the state of the secretion varies considerably according to the degree of the mechanical insufficiency. In the primary stages of gastric atony superacidity is, as a rule, present; at other times the secretions may be normal, and, in the latter stage, we may have subacidity or even achylia. The drawn stomach contents of atony, on settling in a glass vessel, do not show the three characteristic layers of solid, liquid, and froth which are usually found in the drawn stomach contents of dilatation. We have never observed processes of fermentation in simple atony. The secretion of pepsin and rennin is generally found to be proportionate to the secretion of HCl. Thirst is normal, and the amount of urine passed is not reduced. Disturbances in nutrition may gradually develop if the diet is inappropriate or if the patient refuse to eat sufficiently for fear of causing gastric distress.

The course of gastric atony is a chronic one, and the symptoms are subject to many deviations. Stiller and Boas hold that gastric atony comparatively rarely develops into dilatation. Notwithstanding this, the disease generally produces considerable systemic weakness. A variety of nervous disturbances accompany the malady.

Prognosis.—In recent cases, the prognosis is favorable, provided that they are systematically treated and the fundamental causatives of the disease can be removed; but in pronounced atony, and that of long standing, complete recovery is rare.

Diagnosis.—Gastric atony and myasthenia may be confounded with chronic gastritis, nervous dyspepsia, dilatation, and megalogastria. In chronic gastritis the stomach is not enlarged, as a rule, since the motility is good; excess of mucus is common in gastritis and rare in atony; as atony may predispose to gastritis, the two affections may sometimes exist side by side. Nervous dyspepsia is characterized by a great deviation and uncertainty in the symptoms; even the motility may be at times seemingly much affected, but at others, if the case be strictly watched, the motility will be found to be perfect. In nervous dyspepsia there are painful points in

the district supplied by the great abdominal sympathetic plexuses —the celiac, solar, and hypogastric. The painful spots are rare in simple atony. Nervous dyspepsia and atony may exist simultaneously, and one may cause the other; in such cases it will be difficult to determine which is the primary disease. The differential diagnosis between dilatation and simple atony should present no difficulties when modern methods of determining the size and capacity and motility of the stomach are used (pp. 97 to 104); neither should there be any difficulty in distinguishing atony from megalogastria, since the latter is not a disease, but simply a condition of big stomach, which performs its functions normally.

Treatment.—The most important part of the management of gastric atony is prophylaxis, which includes the avoidances of all known causes of the affection. Defective teeth, irregular mode of life, hasty eating, and abnormal burdening of the stomach with food and drink, constipation, as well as the frequent abuse of purgatives. Even where the distinct cause of the malady is not known, one will do best to prevent the full development of myasthenia by rational dietetic and hygienic treatment before functional disturbances become manifest. We have already remarked that atony may be inherited. Whenever this is noticed, such persons should be particularly guarded and careful in the selection of their diet. There are a number of constitutional diseases which, in our experience, undoubtedly predispose to this state. These are tuberculosis, syphilis, anemia, chlorosis, and cholelithiasis. It is present also after exhaustive hemorrhages, and is particularly ominous when the condition occurs after hemorrhages from gastric ulcer.

Typhoid fever has, in our experience, frequently been followed by gastric atony; the same is true of infectious diseases generally, particularly scarlet fever, malaria, diphtheria, and influenza. We have also noticed gastric atony follow a number of operations for abdominal tumors, and particularly ovarian neoplasms. It is very probable that the relaxation of the gastric walls is here largely due to mechanical causes, similar to that which occurs after very frequent and rapidly consecutive pregnancies. In all of these instances the abdominal walls do not regain their tonicity. We have described this condition fully in the section on Gastroptosis. Prophylaxis consists in appropriate hygienic living, much sleep (at least nine hours in the twenty-four), and strengthening of the abdominal mus-

cles—the latter is one of the most important elements, not only in prophylaxis, but also in the treatment of atony. The training of the abdominal muscles should be carried out according to rules laid down in Sandow's text-book on physical culture. The treatment proper of a fully-developed atony must have regard for the fundamental cause. For instance, in syphilis specific treatment will be the only proper course to pursue; in anemia we must have recourse to preparations of iron which have no direct deleterious effect upon the mucosa. Among these preparations we prefer the iron albuminates and peptonates, also ferratin and extract of bone-marrow. With pronounced enteroptosis, particularly in women, abdominal gymnastics can not be effectively carried out, on account of the great exhaustion of the patient.

In some cases of this type palliative results may be obtained by abdominal massage, faradization of the abdominal muscles, baths, and, last but not least, a well-fitting abdominal bandage. In a few cases it will not be possible to trace any cause whatever, though even in these it is well to carefully study the alimentary tract itself before giving up the hope of determining the etiology. The most important factors of direct treatment are: (1) diet, (2) hydriatic, (3) electric procedures, (4) massage, and (5) medicines. The principle underlying the diet in gastric atony is that of frequent and very small meals, which, although quite nutritious and digestible, must not be voluminous. The diet should, as a rule, consists of fats, carbohydrates, and proteids, mixed. If there is an excess of HCl, there is no objection to increasing the proteid food, but in doing so it is well to watch the ratio of the ethereal to the combined sulphates, and the indican in the urine. If the ratio of the preformed to the combined sulphates is very high, and there is an excess of indigo in the urine, it is, in our experience, worth the trial of adding more fats and artificially prepared amylaceous foods, such as dextrinized flours, etc., for it has been found that the general symptoms, as well as the aforementioned indications in the urine (in rare instances, in which proteid diet does not agree in hyperacidity), will improve if the proteids are cut down and the other food substances increased. Together with this diet in hyperacidity, the use of alkalies, magnesia usta, sodium bicarbonate, etc., and of ptyalin, or of malt or taka diastase, is unavoidable. The diet which we recommend for gastric atony is the following:

8 A. M.—250 gm. of bouillon, oat meal, or 100 gm. of milk and 100 gm. of tea or coffee.

10 A. M.—One soft-boiled egg, or 70 gm. of finely-scraped tenderloin, either raw or broiled, and 20 gm. of toast.

12 M.—A broiled sweetbread, or 150 gm. of broiled oysters, or little neck clams, or 100 gm. of finely-scraped beef slightly fried in butter; 200 gm. of potato purée, and if hyperacidity is present, we give half a wine-glassful of some reliable malt extract.

3 P. M.—200 gm. of Mosquera's beef chocolate (P., D. & Co.)

6.30 P. M.—60 gm. of scraped raw ham, or the same amount of fried perch or carp; 50 gm. of toast, and 30 gm. of butter.

10 P. M.—100 gm. of some approved light Moselle wine.

In the section on Dietetics we have given other diet lists suitable for this affection (pp. 226 and 228). A number of competent authors consider the treatment of atony and a chronic dilatation together in the same chapter. Personally, we draw a very sharp distinction between these two affections and their treatment. The therapy of dilatation is considered in the chapter devoted to this subject.

The total quantity of liquids should be limited to 1½ liters a day. This includes the soups, coffee, tea, milk, alcoholic beverages, and water. Alcohol, except in the quantities suggested in the diet list, should not be allowed. Purgatives and narcotics are forbidden. If the thirst is intense, which, however, is rarely the case, water may be introduced by enema. When milk is well digested, and no idiosyncrasy exists against it, so-called milk cures may have a beneficial effect. There is no doubt that an exclusive milk diet insures rest and is very sparing upon the stomach, but it is a two-edged sword. We have seen cases in which the atony undeniably became aggravated by the milk diet. The diet must vary also with the amount of HCl secreted. With increased secretion of HCl the meats may be permitted to prevail. All meats should be run through the meat chopper. Eggs in all forms are permissible in this state. When the secretion of HCl is diminished, we permit the use of spinach, carrots, beans, cauliflower, and asparagus. All vegetables should be cooked and given in the form of purées; among these are the potato, rice, sago, pea, and bean purée. The use of beer should be forbidden or very greatly limited; we do not, as a rule, forbid small doses of good wine. Where good wine can not be obtained, it is safest not to prescribe wine of a doubtful quality, but to order dilute whisky or brandy. Constipation should be met with proper

diet, and medicines should not be used unless they are positively unavoidable. We have already spoken of the diet best suited for constipation (p. 236). In very stubborn and protracted cases glycerin suppositories and water injections will be more effective than medicines given by the mouth. (See E. A. Ewald, "Ueber d. habituelle Obstipation u. ihre Behandlung," 1897.) An advantage is gained by going to stool at a definite hour. There are cases, however, in which a natural stool that occurs every two or even every three days spontaneously is much better, and will do more toward gradual recovery from the evil of constipation than a stool produced artificially every day. Where the patient insists on medicine, and it is really unavoidable, cascara sagrada is most favored by the author. (The syrup cascara, "active," Clinton Pharm. Company, and the elixir of cascara sagrada, P., D. & Co., or S. & D., can be safely recommended.) Podophyllin in the form of pills is a proper medication. The following formula is the one which we favor :

℞.	Podophyllin, ...	0.26	grs. iv
	Ext. belladonnæ, or ext. hyoscyami, ...	0.1	gr. ij
	Strychnin sulphate, ...	0.2	gr. ⅓
	Glycyrrhizæ, q. s.		
	F. pil. No. xii.		M.

SIG.—One pill before supper and one at bedtime. Dose increased to two pills the next day if necessary.

The compound extract of rhubarb is also an effective combination ; but calomel, aloes, colocynth, jalap, and scammony, and the very concentrated purgative waters, such as the Hunyadi Janos, the Rubinat-Condal, and Veronica, must be strictly avoided. Boas, in contrast to other authors, states that lavage is not only unnecessary, but harmful in simple atony, because stagnation does not occur in this disease and there is therefore no necessity for washing out the stomach. We use lavage, however, not to combat any presumable stagnation, but as a sort of intragastric hydropathic massage. For this purpose we use an intragastric douche with hot and cold water alternating. The electrical treatment with which Einhorn has achieved remarkable results is undeniably a valuable means of therapeusis in this affection. It may be applied externally with large felt-covered plates applied to the abdomen directly, or by the intragastric electrode. We usually apply the current fifteen minutes and repeat it daily for three weeks. Massage should be

applied, not only to the stomach, but to the entire abdomen. The method of application (concerning abdominal massage see Hoffa, "Technik d. Massage," Stuttgart, Enke, 1893, also Penzoldt and Stintzing, "Handbuch," Bd. IV, S. 34–39) is described on pages 290–299.

Treatment by Medicines.—Perhaps the only drug which one may depend on for improving the gastric tonicity is strychnin. When atony is accompanied with suppression of gastric juice and inacidity, we can practically associate it with HCl and gentian in the following manner:

℞.	Strychnin sulphate, ...	0.02	gr. ⅓	
	Dil. hydrochloric acid, ...	15.6	f℥iv	
	Elixir of gentian, ... q. s.	180.0	f℥vj.	M.

SIG.—One-half of an ounce in two ounces of water, after meals, through a glass tube, three times a day.

Where there is excess of HCl it is well to combine the strychnin in the following manner:

℞.	Strychnin sulphate, ...	0.02	gr. ⅓	
	Bismuth salicylate, ...	7.5	℥ij	
	Sodium bicarbonate, ...	11.25	℥iij	
	Magnes. ustæ, ...	4.0	℥j	
	Peppermint water enough to make	180.0	℥vj.	M.

SIG.—A tablespoonful in a wineglassful of water after each meal t. i. d.

Creosote has been recommended by Klemperer (*Berlin. klin. Wochenschr.*, 1889, No. 11) and A. Pick (Forl. üb. Magen- u. Darmkrankh., 1895), but Fleischer has found that the motility is still more reduced under creosote. Ergotin, which is recommended by Leube, is, in our opinion, a doubtful remedy for this purpose. Ichthyol has been claimed by Pick to benefit atony, particularly when it is associated with fermentative processes in the bowel. In severe cases of gastric atony with recurrent gastric distress, we have had very gratifying results by rectal feeding for from six to eight days, and total exclusion of food from the stomach; that is, we treated the atony as we would treat a severe gastric ulcer. During the period of rectal feeding the patient must remain in bed.

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CHAPTER X.

SENSORY NEUROSES.

HYPERESTHESIA.

Hyperesthesia depends upon a morbid increase in the irritability of the sensory nerves of the stomach. It is probably a neurosis of the vagus, and a mild form of gastralgia. Clinically, the two forms of gastric sensibility, viz., gastralgia and hyperesthesia, are differentiated by the following facts: The unpleasant sensation of pressure, fullness and pain in the epigastrium with eructations, nausea, and vomiting, occur in hyperesthesia only after the ingestion of food; that is, there must be a digestive stimulation of the mucosa. The distress occurs only after meals, very rarely with an empty stomach; but in gastralgia the pains and other distress occurs with equal intensity in the full as well as in the empty stomach; digestive irritation is not necessary to cause gastralgia. Hyperesthesia lasts several days, weeks, or even months, with uniform or gradually increasing intensity, and during this time dyspeptic symptoms occur daily after every meal; in gastralgia, however, the pains last only during the attacks, generally for a few hours only. In the intervals between the attacks the excitability of the nerves is so completely arrested that even strong irritation—like the overloading of the stomach with food—does not cause a return of the pain. The various acts constituting normal digestion, the movements of the gastric wall and of the contents of the digestive tract are phenomena of which a healthy person is not conscious, but they may be perceived by patients with increased sensitiveness of the gastric nerve endings. As a result of this nervous state, sensations reach consciousness from these localities which in the normal being would not pass the threshold of consciousness. The disturbance of the nerves need not necessarily be in the end distributions of the stomach; they may be in the nerve itself or in the central organ. Most frequently the seat may be in the peripheral nervous end organs in the stomach; these are the cases that have been caused by improper mode of life and various insults to

the mucosa. In other rare cases the gastric hyperesthesia may be a perception due to increased excitability of the nervous centers. In order to intelligently appreciate the sufferings of patients with increased sensibility it is necessary to bear in mind that the increased irritability brings about the perception of transactions into the digestive tract, which in themselves are not pathological, and if present to the same degree in a healthy individual would not be perceived. The natural process of digestion and absorption in such patients is a train of uninterrupted distressing sensations. The patients themselves generally misinterpret their condition or exaggerate it, and as a consequence of the various impressions that they perceive, assume that they suffer from severe organic disease. They often become hypochondriacal. Hyperesthesia may be an independent, idiopathic or secondary, symptomatic neurosis.

Causation.—The primary idiopathic form occurs very frequently as an accompaniment to chlorosis and anemia, particularly with women and young girls. Also after repeated overloading of the stomach with indigestible food. Long-continued use of very salty or acid or spiced foods, and the ingestion of very hot or very cold drinks after long fasting, and in debilitated states following excesses (in venere et Baccho). It has been observed to occur also after chloroform narcosis. Secondary hyperesthesia occurs with hyperacidity and supersecretion in hysterical patients, also in neurasthenia and tabes. Gastralgia may follow hyperesthesia, and there are cases in which both neuroses may be present simultaneously.

Symptomatology.—Patients with this neurosis frequently feel the pulsations of the abdominal aorta, and complain of beating and pulsating in the stomach. Then, again, they have a feeling of heat or cold, or a gnawing, burning sensation, and an impression of restlessness through the entire stomach. The ingestion of food, no matter of what consistency, causes a sensation of discomfort, fullness, nausea, and even vomiting. These sensations may increase to a typical gastralgia, and are felt only during the first period of digestion, or they may last as long as food is contained in the stomach. Some patients complain for a while even after food has left the stomach. The pains are absent in the morning, when the stomach is entirely empty. If the hyperesthesia depend upon hyperchylia, the pains do not become pronounced until the second period of gastric digestion, when the acidity of the gastric chyme reaches its highest degree. In some cases of hyperesthesia it may

happen that the distress is temporarily relieved by the ingestion of albuminous food or the taking of alkalies. The burning, sticking, and beating in the stomach may be accompanied by bulimia. These gastric symptoms are generally accompanied by other nervous phenomena which are probably symptoms of the fundamental etiological disease; thus we meet with migraines, cephalalgia, and neuralgias in other parts of the body, etc., etc. The emesis which occurs in hyperesthesia induces the patients to restrict their diet more and more, whereby the general nutrition and bodily resistance become very much reduced. Concerning the appetite and the foods which are best digested, the patients show the most manifold contrasts. Some of them feel more distress after liquids than after solids. The appetite does not seem much affected; some patients have an intense feeling of hunger. There are no very constant anomalies of motility or secretion. The bowels are generally constipated.

Prognosis.—The prognosis is favorable, as the hyperesthesia ceases when the detrimental and irritating conditions which excite the sensibility of the stomach can be kept away, and when the fundamental disease can be removed.

Diagnosis.—The affection may be confounded with gastralgia, and with the painful symptoms of organic gastric diseases. From gastralgia it can be distinguished by the fact that the symptoms occur daily for a long time, regularly after each meal, and that they are absent when the stomach is empty. Gastralgia occurs only spasmodically, rarely lasts longer than several hours, and is of equal severity in an empty as in a full stomach. The intervals between the attacks are perfectly free from gastric distress. Concerning the differential diagnosis between hyperesthesia and the distress of diseases of the stomach connected with anatomical alterations, we refer to the differential points stated in the diagnosis of gastralgia. We might emphasize here that, in the organic diseases, the pains are entirely absent when the stomach is empty. Atrophic gastritis forms an exception to this rule. The intensity of the pains is influenced by the quality of the food, which is not the case in hyperesthesia, and that organic diseases are mostly associated with tolerably constant disturbances of secretion and motility.

Treatment.—The treatment will be directed in the first place to the correction of the underlying fundamental disease. Wherever

this is not possible, or wherever an idiopathic form of hyperesthesia is present, all irritants which can exert detrimental influence upon the stomach must be excluded. All bodily and mental exertion must be avoided. In severe cases, the Weir Mitchell rest-cure, together with a Leube ulcer cure, has, in our experience, been very efficacious. Hot moist applications to the stomach are very soothing. In a very pronounced case of gastric hyperesthesia in a colleague, which returned regularly whenever he was under great mental strain, the symptoms disappeared entirely after a sojourn at the seashore for one month. Galvanization is a capital method of treating this affection. The intragastric method may be used, but when the patient is not accustomed to the swallowing of the electrode we have obtained good results from the external application of the large abdominal plates. Excessive use of tea, coffee, tobacco, and alcohol must be avoided, as these things have been known to keep up a hyperesthesia. Rosenheim has suggested the following treatment (*Berlin. klin. Wochenschr.*, 1890) internally:

℞. Argenti nitras, ... 0.2 grs. iij.
Aquæ menthæ pip, ... 100.0 ℥iij. M.

Sig.—Two teaspoonfuls in a wineglassful of water, on an empty stomach, in the morning, and a half hour before each meal.

The patient must be kept in bed, and warm cataplasms applied to the epigastrium. The diet consists of milk taken by tablespoonful doses, later on soft eggs, and scraped beef and dipped toast. When the stomach becomes more resistant, the patient can return to solid food. To remove the cause, Rosenheim advises treatment of the general underlying affection, bodily and mental rest, and hydrotherapeutic measures. Severe hyperesthesia is sometimes relieved by bromid of strontium and codein. We have also obtained very good results from spraying the stomach with a solution of morphin, cocain, and menthol. In doing this, a spray must be used by which we can tell the exact amount of cocain and morphin which reaches the stomach with the spraying liquid. It is well not to put more into the spray than we wish to put into the stomach, otherwise the patient may absorb too much cocain and morphin.

Gastric idiosyncrasies are those peculiar forms of hyperesthesia in which neuropathic and sometimes perfectly healthy persons have morbid sensations only after ingesting certain foods. These

sensations consist of headache, light fever, skin erythema, and urticaria. The author has observed persons who developed urticaria after eating crabs, potatoes, cheese, or strawberries. One of our patients regularly develops an acute acne whenever she eats cheese. Another patient regularly develops fever whenever he partakes of crabs. Although a heightened irritability of the sensory nerves may be instrumental in the development of these idiosyncrasies, it is very plausible that auto-intoxication plays a very important rôle in them. It is probable that in individuals who develop urticaria after eating certain foods, there must be micro-organisms that develop toxins from these foods, which, in turn, act in the manner indicated. Pick states that his cases suffered also from constipation, which naturally favors the putrefaction of the ingesta. Acting upon the theory of auto-intoxication caused by intestinal putrefaction, Pick very strongly recommends the internal use of creosote. (See "Albu. Auto-intoxicationen des Intestinaltractus," part on The Skin, p. 88); also p. 396 of this volume.

GASTRALGIA (Cardialgia, Gastrodynia).

Gastralgia, or neuralgia of the stomach, occurs in periodical and spasmodical attacks of severe gastric pain, alternating with intervals of freedom from pain. Pains of greater or less intensity occur with all gastric diseases, particularly with ulcer, carcinoma, gastritis atrophicans, and toxic gastritis. These pains are a consequence of the anatomical alterations which these organic diseases effect in the gastric wall, brought about most probably by exposure, distortion, and compression or inflammation of the sensory gastric nerves. Such pains have been described in the chapter on various Organic Diseases of the Stomach. Gastralgic pain results from functional, not from structural disturbances of the sensory nerves. Gastralgia is characterized by the irregular intervals in which it occurs. and its independence of the quality and quantity of the ingesta, The attacks come on either suddenly, or there may be such premonitory symptoms as feeling of pressure and fullness in the stomach, eructation, nausea, vomiting, headache, and salivation. The pains have a gnawing, boring, burning, tearing, or cramp-like character. They are felt principally in the epigastric region. In some cases the pain radiates to the hypochondriac regions, the entire abdomen and back, and may be accompanied by unmistakable signs of collapse and the feeling of impending dissolution. The

pains occur as well after food that is easily digestible as after indigestible food.

In some hysterical patients the so-called "clavus hystericus," a sharply localized pain, as if a nail were driven into a part, is well described by the sufferer. There is also, in some of these cases, a sudden and transient sensation as if a tremendous ball were rising in the throat (globus hystericus). Nausea and vomiting, as well as bulimia and an urgent desire to urinate, are occasional symptoms. The paroxysms may last a few minutes or several hours, and extend through the entire night; they may begin at any hour of the day or night. The intervals of relief may amount to days, weeks, or months. We have observed a number of cases of malarial gastralgia in which the attacks occurred at regular intervals, and could be distinctly associated with an evolution of the characteristic malarial parasite in the blood. These malarial gastralgias are not infrequent in fishermen, and even sportsmen who sojourn for weeks along the shores of the Chesapeake Bay in Maryland. In a wealthy patient of this city, the gastralgic attacks persisted, notwithstanding the most careful treatment, until the patient could be persuaded to give up his ducking sport on the Chesapeake Bay. The attacks occur generally without any demonstrable cause. As a rule, only one attack occurs in the day, but there may be as many as four in one day. The end of the attack may culminate in very profuse vomiting, which brings a great relief, the pains ceasing thereafter as rapidly and suddenly as they came on. Gastralgia may be a primary idiopathic and independent disease or a secondary reflex neurosis.

Causation.—The gastralgia is frequently a result of motor or secretory neuroses,—of gastrospasm, pylorospasm, and cardiospasm, hyperacidity, and supersecretion. The root of the vagus nerve may be irritated by functional and anatomical diseases of the medulla and adjacent portions of the central nervous system. Boas (*loc. cit.* II, S. 214) enumerates the following causes of gastralgia: (1) Those that attack the stomach itself and its immediate surrounding. (2) Central causes. (3) Infections and intoxications. (4) Reflex causes emanating from other organs. (5) Neurasthenia and hysteria. (*a*) The causes that emanate from the stomach and its immediate surroundings are gastric ulcer, gastric carcinoma, gastritis acida and atrophicans, various forms of perigastritis, and peritonitic adhesion with the pancreas, liver, gall-bladder, spleen, and

transverse colon, and other portions of the intestines. Furthermore, hypersecretion and gastroxynsis, tumors of neighboring organs and pancreatic cysts. (*b*) Of the central causes he mentions the attacks occurring with tabes (" crises gastriques "). In myelitis and brain tumors, gastralgic pains have been observed. (*c*) Infections and intoxicants may cause gastralgia. Of the first, a prominent cause of infection in our latitude is malaria, either in its outspoken form or in its masked and latent type. Of intoxicants, nicotin poisoning and the auto-intoxication associated with uric acid and gout are well-known causative factors. (*d*) Among the reflex causes emanating from other organs, diseases of the genito-urinary organs occupy the first place in both sexes. Prominent among these are displacements of the uterus, inflammations of the ovaries and tubes, and uterine and ovarian neoplasms. (Panecki, " Retroflexio uteri und Magenneurosis," *Therapeut. Monatshefte*, 1892, S. 79.) Independent organic gastric diseases that occur simultaneously must be carefully differentiated from the typical gastralgia. Gastralgias may be associated with genito-urinary diseases in the male. (Peyer, "Ueber Magenaffectionen b. männlichen Genitalleiden," *Volkmann's Samml. klin. Vortr.*, No. 356.) The gastralgias which occur as a consequence of enteroptosis have been fully considered in the chapter on Gastroptosis. (*e*) Stomach neuralgia which occurs in hysterical and neurasthenic persons without any apparent cause, and those which occur in anemic patients, should prompt a very careful examination before we decide that there is no real organic trouble at the foundation of the gastralgia. Occasionally we may find that gastralgias occur with small median herniæ of the linea alba. Whenever motor insufficiency exists with these herniæ, we presume that the omentum is fixed in the hernial sac. Such cases have been recently reported by Rosenheim (*Berlin. klin. Wochenschr.*, 1897, No. 11). Horner (" Ueber Cardialgia, Verursacht d. præ-peritoneale Lipome," *Prag. med. Wochenschr.*, 1892, S. 310) reports a case of severe gastralgia caused by preperitoneal lipomata. These herniæ of the linea alba can be treated successfully only in an operative or orthopedic method (Bandages).

F. Bardenhauer (" Ueber den epigastrischen medianen Bauchbruch," in *Gesammelte Beiträge a. d. Gebiete d. Chir. u. Medizin*, etc., Wiesbaden, 1893, S. 35), Vulpius (*Beitr. z. klin. Chirurg.*, Bd. VII, H. 1), and Roth (*Archiv f. klin. Chirurg.*, Bd., XLII, H. 1, S. 1) consider this subject from the surgical side. It is possible that in

some cases of gastralgia, in which we can not find other diseased conditions which may have caused the affections, secondary anatomical changes in the stomach may exist. Among these are erosions without hemorrhage, follicular inflammation, adhesions with neighboring organs, and cicatrices.* These conditions can not be excluded with certainty, because they may not cause symptoms for a long time. The author advises every caution in the diagnosis of idiopathic gastralgia, as many a case that is diagnosed as a genuine form of gastralgia of this character is found after a very thorough examination to be a result of some anatomical change, or a motor or secretory neurosis of the stomach, or of a disease of some other organ. Idiopathic gastralgia should only be diagnosed when symptoms and indications of other diseases can not be discovered after an exhaustive anamnesis, and repeated thorough examination by means of the most modern methods, and instituted during the intervals between the attacks when the patient is free from suffering. The author has rarely made the diagnosis of idiopathic gastralgia.

Idiopathic gastralgia may occur in connection with chlorosis, anemia, chronic nicotin poisoning, nephritis, incipient tuberculosis, and convalescence from continued fevers, and also as a result of alcoholic and sexual excesses. The gastralgias which occur with arthritis, malaria, and chronic rheumatism are particularly interesting from an etiological point of view. We have repeatedly observed that gastralgic attacks in gout may take the place of an expected acute attack of the joints. The association of malaria with gastralgia can be established beyond a doubt by the blood examination for the malarial parasite, and this kind of gastralgia can be cured by the administration of quinin and sometimes of arsenic, and ceases entirely if the patient removes to an environment that is free from malaria. The occurrence of gastralgia during gout has been explained by some by assuming that the deposits of uric acid and uric acid salts actually occur in the walls of the stomach and thereby irritate the endings of the sensory nerves. This theory explains that gastralgia may occur vicariously in place of expected attacks of gout.

* A negro suffering from the most intense gastralgia with hyperacidity was operated on at the Maryland General Hospital, by Dr. John D. Blake, upon the author's advice. The stomach was bound to the liver, diaphragm, and transverse colon by numerous adhesions, those going to the liver being inseparable.

Secondary Gastralgia.—Cases of this type have been reported which were very severe and obstinate during life, and in which tumors were found at the autopsy drawing upon or compressing the fibers of the vagus and sympathetic. It has been observed, also, in Basedow's disease, but is more frequently the result of direct or indirect irritation of the roots of the vagus nerve in consequence of organic or functional diseases of the spinal cord or brain. We have already referred to the frequent attacks of gastralgia occurring in tabes, which have recently been explained by a sclerotic degeneration of the nucleus and of the main stem of the vagus (Kahler, Oppenheim, Demange, Dejerine). The gastric crisis, which we have described elsewhere, demands a greater interest because it may occur in tabes as an initial symptom when the other characteristic signs, such as absence of the tendon reflexes, rigidity of the pupils, and Romberg's symptom, are not yet present, and in some cases the typical ataxia has not been known to occur for from six months to a year after critical gastralgias of this kind. Erb has established a very probable causal relation between syphilis and tabes, and the hope has been expressed that these early gastric crises should stimulate exhaustive clinical examinations of the patients with a view to combatting the disease by mercury and iodids at a time when the spinal changes are not far progressed. Leyden has described gastralgia with subacute myelitis, and Oser with myelitis due to compression.

Symptomatology.—The symptoms of the attacks are generally quite characteristic, and the course so typical that they can not be misinterpreted. Prodromal symptoms, such as depressed spirits, headache, salivation, nausea, pressure, and fullness in the stomach may occur, but, as a rule, are not observed and have no diagnostic value. Generally, the cases begin very suddenly with severe gastric pains, which are sometimes so intense as to baffle description. Strong pressure upon the stomach sometimes relieves the pain,—in fact, the patients are often found doubled-up in bed, pressing both hands upon the epigastrium. If the pain has been caused by hyperchylia, it is relieved by alkalies or albuminous food. The bowels are constipated and the urine suppressed. The forehead is covered with large drops of cold perspiration, the pulse is small, occasionally irregular and accelerated. In rare cases it has been reported to have been much retarded. Great prostration and muscular cramps, and even general convulsions, have been known to follow. At the

end of the attack the patients usually indulge in repeated yawning, eructation, and sometimes vomiting, and in hysterical patients a copious dilute urine is sometimes voided.

Diagnosis.—As idiopathic gastralgia can rarely be logically diagnosed, it will be more correct to consider gastralgia as a symptom, not as a disease *per se;* although the fundamental disease causing it, may remain obscure or be missed entirely in the beginning of the disease, it may become pronounced eventually. Gastralgias may have to be differentiated from the pain of ulcer, acute and chronic gastritis, toxic gastritis, carcinoma, from rheumatism of the abdominal muscles, myalgia, intercostal neuralgia, nephrolithiasis, cholelithiasis, and intestinal colic. The differential diagnosis from ulcer of the stomach has been stated in the chapter on Ulcer. The ulcer pain is sharply circumscribed in the epigastrium and in the dorsal regions. It is directly dependent upon the quantity and quality of the food. Pains from gastric ulcer are relieved by rest in bed, and made worse by movement. This pain does not occur in paroxysms—it is usually a lasting discomfort. There may be atypical cases of ulcer in which the diagnosis becomes much involved. Boas (*loc. cit.*, S. 38) emphasizes the diagnostic value of the painful point situated at the left of the spinal column between the tenth and twelfth thoracic vertebræ.

Von Leube advises, when other symptoms are missing, to treat the disease as if it were ulcer, and Boas recommends the internal administration of nitrate of silver for three or four weeks. The good result of both of these treatments would speak for gastric ulcer. The acute and chronic gastritis are rarely so painful as to be confounded with gastralgia. The pains of chronic atrophic gastritis occur only at a time when complete atrophy of the mucosa has supervened; and, inasmuch as the secretion in gastralgia is never suppressed or lost, this factor will constitute an important diagnostic feature, since HCl is, in a great majority of cases, absent in gastritis. From toxic gastritis the diagnosis is made by help of the clinical history; from carcinoma, by means of ascertaining the state of the motility and secretion, which is, as a rule, lost in carcinoma, and normal in gastralgia. The pains of carcinoma as well as of ulcer increase on pressure; in gastralgia they diminish on pressure, and in carcinoma we have anemia and cachexia as prominent signs. It has been said that the galvanic current with the anode on the epigastrium and the cathode on the spinal column

relieves the pain. These signs are not reliable, and as there is nothing typical about gastralgic attacks which should distinguish them from painful paroxysms issuing from other abdominal organs, we may say that, up to the present time, no pathognomonic sign or symptom of gastralgia exists. There are attacks of rheumatism and myalgia of the abdominal muscles which seem to become focused in the upper part of the abdomen, so that they may be confounded with gastralgic pains. Myalgic pains may occur from severe exertion of the abdominal musculature. These pains are increased by pressing or pinching the sore muscles; they are not accompanied by any gastralgic symptoms whatever, are very much improved by rest, and, if they are rheumatic, by salol and salicylate of soda. Intercostal neuralgias can be distinguished by the excessive and permanent sensitiveness to pressure which the affected nerves exhibit all along their course from the spinal column to the sternum. Cholelithiasis, or the pains of an incarcerated or passing gall-stone, frequently irradiate so prominently to the epigastric region that they are more marked there than over the liver, but whenever the stone obstructs the ductus choledochus temporarily the gall-bladder may be palpable by its dilatation, and icterus and clay-colored stools are evident signs; but in those patients in which the stone is impacted in the cystic duct and does not completely obstruct it, or rapidly passes through it, a differential diagnosis is difficult, because the symptoms before mentioned are absent. But even in these cases great sensibility of the liver to pressure, anteriorly and posteriorly, is usually present. The liver is usually enlarged, and there is a painful point in cholelithiasis at the twelfth dorsal vertebra, a few centimeters to the right of the spinal column. A careful search for gall-stone particles must be made in the passages. Gall-stones, as a rule, cause vomiting, while gastralgia does so rarely. The differential diagnosis between hepatalgia and gastralgia presents great difficulties. In nephritic colic, the dyspeptic symptoms may be exactly like those of gastralgia. The diagnosis between the two affections can be made with certainty by careful urinary examination, for fragments of calculi and traces of blood, or by catheterization of the ureters in the intervals. In intestinal colic, the pains may be located in the upper part of the abdomen. They are mostly due to excessive gaseous distention of the intestinal loops, and are associated with constipation, and cease after the copious discharge of gas.

Treatment.—In the treatment of gastralgia, the fundamental cause must, if possible, be discovered and removed. In malarial districts the treatment by quinin and tonics is the most effective, if the causal relation can be established. Chlorosis and anemia should be treated by albuminate or peptonate of iron, ferratin, bonemarrow, arsenic, and highly nutritious diet. In some cases there is no better remedy than the tincture of the chlorid of iron. If the patient is an inveterate smoker, he must be cautioned to cease his habit. Enteroptosis, gout, and rheumatism must have suitable therapeutic attention. Disturbances of the genito-urinary organs, particularly of the female sexual organs, will command the attention of the specialist. Wherever we can find no cause for gastralgia, the only thing that can be done is to treat it symptomatically. The most effective agent in our experience for this purpose has been the galvanic current. Large felt-covered copper plates are dipped in water as hot as the patient can stand it, the anode placed on the epigastrium and the cathode on the spinal column extending from the cervical region downward between the scapulæ. For this purpose we use very strong currents, not less than 25 milliamperes. Oser ("Die Neurosen d. Magens," etc., Vienna and Leipzig, 1885) claims to have observed cessation of the pains after application of the faradic current. Where the pains are not too intense, the internal administration of phosphate of codein, ½ of a grain every three hours, chloral hydrate, 15 grs. every two hours, Dover's powder, tincture or extract of hyoscyamus, extract of belladonna, and camphorated tincture of opium are available remedies. Compound spirits of ether and the ethereal tincture of valerian, 20 drops every two hours, are useful when collapse is associated with the pain. Exalgin, antipyrin, and antifebrin have been recommended by Penzoldt. If the collapse is marked, wine, whisky, ether, and ammonia should be given until it has passed over. In pains of great intensity, the sovereign remedy is a hypodermic injection of ⅓ of a grain of morphin sulphate, together with $\frac{1}{100}$ of a grain of atropin sulphate inserted directly into the epigastric region. Boas recommends suppositories of extract of opium and extract of belladonna. All of these agents are useful for the immediate treatment of a paroxysm; they probably have no curative effect on the underlying etiological trouble. The irritability of the mucosa can be effectively reduced and gastralgic attacks sometimes altogether prevented from recurring by intra-

gastric irrigation, with lukewarm carbonated water (Malbranc, Kussmaul), or by treating the mucosa according to Fleiner's method—with suspensions of bismuth subnitrate. We have seen excellent results from irrigations containing bismuth subnitrate (℥ij), bismuth subgallate, ℥ss in one pint of camphor water. The outflowing camphor water must be measured so as to ascertain that not over ℥j remains in the stomach. Although the pains of gastralgia are not influenced directly by the character of the food, the diet should be very bland and unirritating, and should not be taken in large quantities.

Gastralgokenosis.—Under this name Boas describes a painful emptiness of the stomach which occurs one to two hours after meals, and may be so severe as to embarrass the respiration of the patient. The paroxysms last but one-quarter to one-half an hour, and are not connected with bulimia. These attacks are said to be relieved by the ingestion of milk, bread, etc. One of the cases of Boas developed into an attack every time he drank wine or champagne, or ate cake. We have never seen a case that corresponds to Boas' description of this malady, and would suggest that it is probably a gastric hyperesthesia associated with hyperperistalsis and a strong secretion of HCl, particularly as the cases reported by Boas show that the reactions for HCl were quite strong.

ANOMALIES OF THE SENSATIONS OF HUNGER AND APPETITE.

BULIMIA, OR HYPEROREXIA.

Morbid increase of the sensation of hunger may occur as an independent idiopathic neurosis, as a result of abnormal irritability of the center controlling the sensation of hunger, or as a symptom of organic diseases. An intelligent insight into the pathogenesis of bulimia is only possible with a knowledge of the origin of the sensation of hunger. A modern physiological theory suggests that the hunger center in the medulla oblongata is stimulated normally by the blood as soon as it has become impoverished in nutritive substances, and that the sensation of hunger ceases when the blood is saturated with nutritive substances. Stiller and others assert that the sensation of hunger results from excitation of specific hunger nerves in the stomach, and that from here the sensation is conducted centripetally to the hunger center, and

that, therefore, the normal sensation, as a rule, is brought to consciousness indirectly. Neither of these theories is supported by satisfactory clinical and experimental evidence. The appetite ceases when the stomach is filled with food, but that does not imply that the nutritive materials are already absorbed into the blood. This may require from three to four hours. In many gastric diseases the feeling of hunger is indirectly affected by the local disease, either increased or diminished. There are also general (metabolic) diseases which directly or indirectly increase or diminish the sensation of hunger. In some persons, even in the normal condition, vehement emotional excitations may cause a loss of hunger and appetite, although the blood is undoubtedly impoverished in nutritive substances, so that we have clinical evidence sufficient to demonstrate both a local, gastric, and a remote or central nervous excitation of hunger. According to one hypothesis, hunger results every time the stomach becomes entirely empty, and Leo ("Ueber Bulimia," *Deutsche med. Wochenschr.*, 1889, Nr. 29 und 30) has asserted, in a most comprehensive report on this affection, that the abnormally rapid evacuation of the stomach is the cause of bulimia. This would naturally include that bulimia is very frequent with pyloric insufficiency, in which, as we know, the ingesta at once enter the intestine from the stomach. Bulimia should also then be frequent in cases where a gastro-enterostomy has been executed for benign stenosis of the pylorus. Ewald and Fleischer have reported cases of bulimia in which there was no hypermotility. The combination of bulimia with hypermotility may possibly be explained by the fact that intense excitation of the hunger center may extend to neighboring centers in the medulla, and involve even the vagus center, which responds by affecting a more rapid evacuation of the gastric contents into the intestines. Some of the accompanying symptoms of bulimia (tinnitus, and roaring in the ears, palpitation of the heart, and fainting) are attributed by R. Ewald (the physiologist) to secondary irritation of nervous centers lying in close proximity to the hunger center. The affection expresses itself by violent sensations of hunger coming on suddenly, even shortly after the completion of a full meal, and if the desire for food is not immediately gratified, the patients exhibit signs of fright, weakness, headache, pallor of the face, palpitation of the heart, roaring noises in the ears, and gastric distress. The attacks may sometimes occur periodically,

but, as a rule, occur irregularly. In the intervals between the attacks, hunger and appetite are normal. According to Boas, bulimia may alternate with anorexia.

Causation.—Bulimia may be an idiopathic, central neurosis connected with abnormal irritability of the hunger center, or a symptomatic affection which Leo (*loc. cit.*) has observed in exophthalmic goiter, with gastric ulcer and hyperacidity, chronic gastritis, tapeworm, diarrhea, and menorrhagia. It has been observed even with carcinoma and dilatation. Fleischer states, without reserve, that the hyperexcitability of the hunger center is not caused by sudden and excessive impoverishment of the blood in nutritive substances, because the attacks may occur immediately after an abundantly nutritious meal which has brought about a feeling of satiety, and because, in other cases, the morbid sensation may be relieved by a mouthful of bread or a swallow of beer or wine. The fact that the sensations of hunger and thirst are normal in the intervals between the attacks, or even at times entirely absent, argues against the assumption that bulimia is always caused from a condition of the blood acting upon the central nervous system. The following are morbid conditions in which bulimia has been observed to occur: Cerebral tumors, epilepsy, psychoses, hysteria and neurasthenia, focal diseases of the brain, cerebral concussion, Basedow's disease, Addison's disease, tuberculosis, syphilis (according to Fournier, *Gazette Hebdom.*, 1871, Nos. 1–3, it occurs between the third and sixth month of this disease), diabetes mellitus, uterine disease, chronic gastritis, ulcer, dilatation, carcinoma, enteritis, and intestinal parasites. Bulimia has also been observed during the puerperium. Some authors classify the ravenous appetite following exhaustive continued fevers, as well as that following abundant loss of blood, with bulimia. This, in our opinion, is not a justifiable classification, because the increase of hunger in these cases can be explained in a simple and natural way without assuming a hypothetical excitability of the hunger center. In diabetes mellitus we may assume the existence of an abnormal irritability of the hunger center because these patients are not satisfied even shortly after large meals. It has been supposed that the glucose circulating in the blood is the agent that causes this irritation of the hunger center. In diabetic patients in which the sugar in the blood and urine has been reduced by a diet limited exclusively to fat and albuminous food, the torturing feelings of

hunger disappear, to return again if the mellituria is allowed to increase on other diet. According to Pettencofer and Voit, the metabolism of diabetic patients is much increased; which, of course, means a more rapid consumption of the nutritive elements of the blood. The impoverishment of the blood is further augmented by the fact that the sugar which is formed from the amylaceous substances of the food is only partially or not at all utilized in the economy. The diagnosis of bulimia should only be made in diabetes if the violent sensations of hunger continue notwithstanding very rich and very abundant meals, or if it recurs very soon after such meals, by which the blood must have been charged with nutritive substances for a longer time. Ewald and Boas have observed that the attacks grow less after bodily exercise in the open air. According to Rosenthal, the affection is more frequent in women than in men, and occurs most often between the twentieth and fortieth year.

Symptomatology.—The main and most characteristic symptom is the impulsive sensation of hunger, which by any and every means commands the ingestion of food. The pallor, weakness, and terror, with attacks of fainting and roaring in the ears, we have already described. This sensation comes on generally within one to two hours after meals, or it may occur within ten minutes after meals. We have known several old gentlemen who were for a long time aroused in the middle of the night by this torturing sensation of hunger. Some patients complain of gnawing and boring pain if the hunger is not gratified. Very small quantities of milk, beer, or wine, or only a few mouthfuls of cracker or bread, will cause the entire train of symptoms to disappear. Peyer (*Correspondenzbl. Schweitzer Aerzte*, 1888, Nr. 20) reports a case of a paroxysm of bulimia occurring in a female patient when she was away from home visiting a friend. The weakness ensuing is described as being so great that she could not return home. Peyer asserts that in three-quarters of an hour she consumed three pints of milk, 23 eggs, and two pints of strong wine before the bulimia and pain in the stomach ceased. The patient then fell asleep, and returned home perfectly well on awakening. Potton reports the case of a young, hysterical girl who was obliged to take 11 or 12 meals a day, and even eat during the night; she is claimed to have ingested between 10 and 12 kilograms per diem, and was finally cured by

gradually-increasing doses of morphin. This was a case of so-called continued bulimia.

Diagnosis.—Wherever the abnormal sensation of hunger occurs shortly after abundant food has been taken, the diagnosis is not difficult; at other times it may be confounded with polyphagia and acoria. In polyphagia, the desire for eating is very much increased, but it does not occur until some time after the meals, and occurs gradually, not developing the intense hunger suddenly. So, polyphagia is simple increase of the normal sensation of appetite, such as we find in diabetes mellitus. It is impossible to strictly separate polyphagia from bulimia—both occur under similar conditions and as primary or secondary neuroses. Bouveret ("Traité des Maladies de l'Estomac," Paris, 1893, page 654) refers to a case in which a patient seventeen years old could devour 100 pounds of meat in twenty-four hours, and Rosenthal reports an instance of a woman, aged twenty-eight years, who ate at one meal a whole roast of goose and a large portion of bread. There is a so-called continued form of bulimia which alternates with acoria, or the absence of the feeling of gratification or satisfaction after meals. If it can be found that the feeling of hunger is very great, and even continues or returns after abundant meals, then we are dealing with bulimia; but if the sensation of hunger is normal or reduced, and ceases after larger meals, but without causing the feeling of satiety, we are dealing with acoria. The continued form of bulimia has hitherto been found only with diabetes mellitus and hysteria. We have had two cases in hospital practice which illustrate that bulimia and polyphagia may be developed by practice. Both cases occurred in negroes who had, as a result of a number of wagers, eaten large quantities of food. One colored man was a waiter at a hotel at Cape May, N. J., and used to exhibit himself in eating a huge watermelon together with eight pies. The other negro, who was a patient at Baltimore, gradually developed his polyphagia from participating in rival encounters with other individuals of his race to see who could eat the most oysters. It is claimed that this man could eat three quarts of oysters, with a large amount of crackers and beer. Both negroes found later that the habit had developed into a disease, the tremendous appetite developing very often within a half hour after the big meals of bread, fish, and egg had been taken. One of them has been cured by dram doses of bromid of ammo-

nia four times daily. The diagnosis of an affection of this character can not be stated in such exact terms as that of an organic disease, as individual opinions of specialists as to what really constitutes bulimia will probably vary greatly. (The *treatment* will be considered together with that of acoria.)

ACORIA.

This word is derived from α and χορέννυμι,—I become satiated.

Absence of the normal feeling of satiation, even after very abundant meals, without increase of hunger or appetite.

Acoria is not identical with bulimia or polyphagia, for in both of these there is a very strong feeling of hunger, while in acoria we may have absence of appetite. Even in polyphagic gluttons the feeling of satiation will eventually supervene, but not in acoria. The disease is generally secondary to neurasthenia, hysteria, and certain psychoses. It is occasionally met with in sexual neurasthenics. The feeling of satiation is no positive sensation; it occurs when hunger and appetite have been appeased, and is therefore a negative sensation. Hunger and appetite cease normally when the hunger center passes from a sensation of excitation to that of rest. The amount of food required to accomplish this varies greatly in different persons, and even in the same person at different times. One hypothesis has attempted to explain acoria on the basis of overexcitation of the hunger center. If this were the case, we would find, periodically at least, an increased sensation of hunger after large meals which is never observed in acoria, for hunger and appetite are normal, or even subnormal, in acoria. Some patients even state that after meals hunger ceases, but they have no feeling of satiation; in fact, no impression whatever from the stomach informing them that they have eaten enough. It is well known that many people are not satisfied to introduce food until the appetite has been appeased, but they continue long enough to perceive a feeling of pressure and slight fullness in the stomach, which is a result of a moderate distention of the gastric walls by ingesta. While moderate eaters perceive this sensation as uncomfortable and indicating supersatiation, gormandizers gradually become accustomed to this feeling of pressure and fullness, sometimes from early childhood, so that eventually they do not believe themselves satiated before this distention occurs, and this

fullness and distention is finally confounded with the normal sensation of satiety.

The next step in the development of this nervous anomaly is that the feeling of pressure and fullness may mimic a temporary normal feeling of satiation, while at the same time the excitation of the hunger center continues. The sensation of hunger, when it is not very strong, may be in some cases removed by filling the stomach with perfectly indigestible material, such as leaves and sawdust. In the voyage of the *Jeanette* (Journal of Lieutenant de Long commanding the expedition, 1883), the crew of the surviving members subsisted upon scraps of deer skin, which, from its bulk in the stomach, seemed to afford relief from hunger. After everything was exhausted they lived upon an infusion made from arctic willow, containing really no nourishment, and ate two old boots. As the feeling of satiation is absent after copious filling of the stomach with food, and as it can not be disguised by an abnormal feeling of hunger, because hunger is normal or subnormal in acoria, another explanation that has been offered for this nervous affliction is that it is due to loss of sensibility, or anesthesia, of the gastric sensory nerves. This seems to be a very probable explanation, since we have personally had at least one experience that would suggest a local gastric anesthesia as in explanation of acoria. The case we have in mind is that of a young lady whose stomach we had sprayed with a three per cent. solution of cocain and menthol. She returned on the same day, stating that, although she had taken a long bicycle ride after the spraying, and returned home feeling quite hungry, she had the impression that the food she ate never reached the stomach. She had no feeling in her stomach that the meal effected satiation. At first we overlooked the causal relation between the spraying with menthol and cocain for this temporary acoria, and our attention was attracted to it after the same symptoms were complained of each time the menthol and cocain were used. These agents had been employed for the relief of gastralgic pains resulting from erosions. The case ultimately recovered by treating it with suspensions of subnitrate of bismuth. It is conceivable that anesthesia of the stomach nerves may occur from repeated overdistention, as occurs in bulimia, polyphagia, diabetes mellitus, and dilatation of the stomach.

Symptomatology.—As the only symptom is the absence of satiation, the clinical picture is not very manifold. The complaints

of the patients are limited to the statement that large meals cause no sensation of having had enough to eat, and that they do not know when to cease eating; that they have to measure out their food previous to beginning to eat, in order to know when they have sufficient. Some of these patients try to compel a feeling of satiation by the ingestion of enormous quantities of food and drink. This has been reported as a cause of gastritis, atony, and dilatation. The prognosis varies according to the fundamental disease. The diagnosis is made from the single important symptom and the exclusion of bulimia and polyphagia. Sometimes we find transitions from acoria to bulimia, which Boas explains by a reactive hyperesthesia following an anesthesia of the gastric nerves. Acoria is distinguished from polyphagia by the increased desire for food, which is marked in the latter, very likely as a result of increased oxydation, while the diagnosis from bulimia hinges upon the ravenous desire for food in the latter; in both the feeling of satiation will eventually supervene.

Treatment.—The treatment of bulimia when it is a secondary disease must have regard for removal of the primary cause, such as intestinal parasites, genito-urinary diseases, hyperacidity, or ulcer, and any existing neurasthenia, hysteria, or psychosis. The bromids are very valuable remedies to reduce the irritability of the hunger center; they should be given in the form of bromid of ammonia, or strontium, 30 gr. of either four times a day, preferably in peppermint water. The following formula will be found useful in bulimia:

℞. Tinct. opii camph., ... 8i.o f℥iij
Tinct. belladonnæ, ... 1.o gtt. xl
Elix. simplic., ... q. s. 180.o f℥vj. M.

Sig.—One-half of a fluidounce three times a day.

Arsenic in form of Fowler's solution, beginning with three to five drops, and gradually increasing the dose to 10 to 15 drops, is highly recommended by Boas. Rosenthal recommends subcutaneous injections of extract of opium, and has seen good results in bulimia from cocain hydrochlorate. Morphin is a remedy that has been followed by good results in this affection. An attempt should be made with the use of electricity. In one of the colored patients to whom we referred as champion gluttons, and who had subsequently developed bulimia, the symptoms improved very much

under the intragastric use of the constant current. The treatment of acoria should be mainly that of neurasthenia; climatic changes and electrical hydropathic cures are most effective. Intragastric douches, with alternating warm and cold water, have been recommended. It is very important that these patients should be watched by healthy friends during their eating; thorough mastication and insalivation should be insisted upon. Strychnin and massage of the stomach suggest themselves as rational means of treatment.

NERVOUS ANOREXIA.

By anorexia is meant an entire absence of appetite and loss of the sensation of hunger. The superlative degree of this sensation is expressed in the disgust and repugnance toward all food. There are probably no pathological conditions, neither of the stomach nor of any other organ of the body, in which anorexia is not occasionally met with. In most anatomical diseases of the stomach anorexia is a regular accompaniment. The separation of appetite and hunger is not as clear as one might presume; the two are not necessarily synonymous, nor does one include the other. Penzoldt defines hunger as the warning or admonition, and appetite as the pleasure of eating (Bibliothek der ges. medizin. Wissenschaften, herausgegeben von Drasche: article on "Anorexia"). There may even be appetite when there can not possibly be hunger. We have already spoken of the various forms of anorexia that may accompany the organic and functional diseases of the stomach. By nervous anorexia we mean loss of appetite, and even repugnance to food, that may extend over weeks and months, with a perfectly intact digestive apparatus; this affection is found principally in women, and is based upon neurasthenia, hysteria, anemia, chlorosis, and certain neuroses of the stomach. It is found in those addicted to the excessive use of alcohol and tobacco, and as a symptom of the morphin habit. It is, therefore, not a disease peculiar to itself, not a typical morbid entity, but rather a sequence. Whether or not nervous anorexia may be an independent disease of central origin, a neurosis connected with a reduced irritability of the hunger center, has, up to the present time, not been satisfactorily investigated. The course and the prognosis depends upon the degree of the repugnance for food. Among the insane and very neurasthenic patients fatal cases have been reported.

Symptomatology.—The patients who, from loss of appetite or distress, can not take food, grow anemic and weak, look very emaciated, have a slow, feeble pulse, cold hands and feet, and may even give the impression of tuberculous patients. Rosenthal ("Magenneurosen," etc., Vienna and Leipzig, 1886), Gull (*The Lancet*, 1868), and Charcot ("Oeuvres Complètes," tome III, p. 240) have reported fatal cases of nervous anorexia. Insomnia is a frequent symptom of this affection. Very slight anatomical changes in the stomach may cause anorexia ; it is, therefore, almost impossible to make the diagnosis of secondary or primary anorexia with precision.

Diagnosis.—There is no difficulty about the diagnosis of anorexia, but it is not always easy to discover the real cause of it. We will find under the consideration of enteroptosis that almost any abdominal organ when dislocated may produce this symptom. Organic affections of the stomach must be excluded before we can make the diagnosis of nervous anorexia. Very frequently chronic gastritis, incipient tuberculosis, and carcinoma begin with this symptom before any other signs or symptoms are manifest.

Treatment.—The primary object of the treatment must be to improve the general nervous condition, to correct any existing fundamental disease, to act upon the psychic sphere by pursuasion, suggestion, and firm but kind argument, and, finally, to combat the anorexia itself directly. Any existing neurasthenia and hysteria should be treated by methods that have been spoken of repeatedly for these affections. Dujardin-Béaumetz ("Traitement des Maladies de l'Estomac," 1891, p. 326) speaks very highly of arsenic in the treatment of nervous anorexia. In anemia, mild preparations of iron (the peptonates, albuminates, ferratin) are almost indispensable. Dyspeptic patients, as a rule, do not tolerate the ordinary preparations of iron,—the chlorid, sulphate, phosphate, and iodid of iron ; possibly the least irritating of these to sensitive stomachs is the chlorid. In order to improve the general nutrition, the Weir Mitchell rest-cure,—which consists in isolating the patient from his family and placing him under the supervision of a trained nurse and experienced physician, and feeding him so abundantly that gradually a gain of weight is accomplished,—together with the use of baths, massage, and electricity, has, in many cases in our experience, produced happy results when other means have failed. Where there is an absolute repugnance for food, or where the patient is insane, artificial com-

pulsory alimentation by gavage should not be postponed too long. We have considered this fully on page 182. In the beginning of the trouble the bitter tonics are available to stimulate the appetite. The basic orexin, five to ten grains, three times a day, in a cup of hot bouillon, produces very excellent results in these cases of nervous loss of appetite.

Boas speaks very highly of the cinchona bark. It may be prescribed in the following formula:

℞.	Tinct. cinchonæ comp., ...	40.0	f℥ jss
	Acid. sulphuric. dil., ...	10.0	f℥ ij
	Syr. zingiber., q. s., ...	240.0	f℥ vj. M.

Sig.—One-half of a fluidounce in two ounces of water, through a glass tube, three times a day.

In some cases in which the anorexia was due to a feeling of pressure and discomfort after eating, Rosenthal reports good results from 10 to 15 grs. of bromid of sodium given before meals. Boas cautions against the use of mineral waters in the treatment of this neurosis. One most approved combination for anorexia is given on page 450; it contains dilute HCl, because we have found, in a very large number of cases of intense nervous anorexia, that the gastric secretion is very much reduced or entirely lost.

CHAPTER XI.

NEUROSES OF SECRETION.

HYPERCHYLIA (Hyper- or Superacidity, Hyperchlorhydria).

Most digestive pathologists whose clinical and laboratory experience renders them competent to judge, consider hyperacidity and hypersecretion of the gastric juice to be neuroses of the secretory function. They are regarded as functional disturbances of the nerves of the stomach, which may occur as individual diseases or as part of other neurotic conditions. This view no doubt is correct in a large number of the cases. It includes the opinion that in this disease no characteristic changes in the structure of the gastric mucous membrane are demonstrable. Judging from the results of Hayem, Cohnheim and Einhorn, and the author, which we have already quoted (pp. 130 to 135), it is beyond a doubt that in more than one-half the cases of hyperacidity examined, proliferation of the glandular elements is present.

We have not only examined fragments of mucosa that were accidentally found in the wash-water, but have had opportunities of autopsies on cases of pronounced and prolonged hyperacidity that died of intercurrent diseases. In serial sections of these stomachs it was found that the prevailing state of the mucosa was that of glandular proliferation, with increase in the number of oxyntic cells. Such stomachs do not show the same conditions throughout. On making serial sections of large pieces of the secretory portion, one occasionally meets with areas in which the glandular structure is apparently normal. At very rare intervals one can even find sections showing partial glandular atrophy. This is so rare as to be insignificant. Even in normal stomachs one sometimes finds indications of atrophy in serial sections, and we consider that these changes are very limited, and, perhaps, may be considered as processes of reconstruction and transition, where accidentally injured or exhausted glands break down in minute foci and are replaced gradually by newly-formed gland-cells. The prevailing condition,

then, in hyperacidity, according to our opinion, is proliferation of the glandular elements.

A large number of microscopical investigations will be necessary to confirm this opinion. We have thus far examined the entire stomach of four cases that gave the clinical picture of hyperacidity before death. In all four of these cases the proliferation of the glandular elements was uniformly present.

Sir William Roberts ("Digestion and Diet," p. 240) holds that the acid in what he calls acid dyspepsia (which seems to us an objectionable term, since it does not define which of the diseases that are connected with excess of acidity he refers to) is not unmixed HCl, but that lactic, butyric, tartaric, and malic acids are present. These are probably derived from salts of the organic acids present in articles of food which are decomposed by the HCl of the gastric juice. There may, of course, be a hyperacidity due to organic acids, which may present all the symptoms of hyperchlorhydria; at the same time there may be no HCl secreted at all. What we refer to clinically as hyperacidity, however, is an excessive formation of hydrochloric acid from the gastric glands. Concerning the nature and origin of this acid we have nothing but theories.

It has been suggested that the hyperchlorhydria is due to an excess of chlorids in the organism, from which it liberates itself by excretion into an organ where the freeing of the system from chlorids could at the same time become of utility as a digestive secretion in the form of HCl. The author has made a number of experiments by feeding carnivorous animals with food from which the chlorids had been removed so far as was possible. The acidity of the gastric juice of the dog will become very much reduced if the chlorids are withdrawn from the food. This, however, is not proof of the fact that the reduction of chlorids is the cause of the diminished secretion of HCl, because foods containing much chlorids are a healthy stimulant to the normal secretion of HCl, and food deprived of chlorids can not exert this stimulation upon the mucosa. Personally, the author considers it very probable that hyperacidity is frequently an *adaptive* process; that is to say, the glandular layer gradually develops greater secretory powers, because more secretion of HCl is required by the nature of the ingested food. We have been told by physicians practising in Japan, that hyperacidity, as well as gastric ulcer, are practically unknown in that country, which may be partially explained by the

exclusive carbohydrate diet upon which the middle and lower classes of that nation exist.

It is a well-known fact that the gastric juice of carnivora is much stronger in HCl than that of the herbivora. It may not be so well known that the gastric juice of a carnivorous animal can be made to contain a less amount of HCl by being fed upon a carbohydrate diet for a long time. Two dogs of the same litter (*a*), fed exclusively on milk, potatoes, and rye bread, and (*b*) fed exclusively on beef, mutton, pork, fish, and water: At the end of one year dog (*a*), fed upon carbohydrates, had a gastric juice, one hour after a roll and a half pint of water, containing 3 per 1000 of HCl; dog (*b*), who was fed upon a meat diet, had a gastric juice containing 6.540 per 1000 HCl. These two dogs were raised in two entirely different families. Dog (*a*) was raised by a gentleman living in a country district where meat was not easily obtained and milk was very abundant; dog (*b*) was raised in the city, and lived upon the refuse meats from the table. Unless conducted in this manner, and watched by competent observers for a long time,—at least one year,—the experiment is of no practical utility. It is conceivable that we do not as yet know all of the constituents of the gastric juice; clinically, it has been very frequently observed that the secretions of the intestines may contain traces of products of metabolism and other toxins when the function of the kidney is suppressed or lost. The gastric juice of epileptics may contain toxic substances. Augustini, who recently investigated this subject, found that the gastric juice of an epileptic, when injected into the abdomen of a rabbit, proved fatal, with general toxic symptoms and clonic convulsions. This was especially true when the gastric juice was obtained immediately before or after an attack. Normal gastric juice was found to produce no such evil effects. Augustini concludes from these experiments that systematic lavage and disinfection of the stomach and intestines are indicated in all cases of epilepsy. What we wish to emphasize in this introduction to the consideration of hyperacidity is that hyperchlorhydria, although frequently a neurosis, is, in our opinion, very often a process of adaptation of the mucosa.

Acidity of the Urine and Gastric Contents in the Healthy and the Dyspeptic.—Mathieu and Tréheux (*Arch. Gén. de Méd.*, November, 1895) have made researches on this subject. They examined the urines hourly during the afternoon, after the midday meal, carrying out their investigations on 12 persons, after 84 differ-

ent meals, thus making over 400 estimates of the degree of acidity of the gastric contents and urine. The individuals examined were the subjects of hyperchlorhydria, with and without symptoms of gastric dilatation, carcinoma, etc. The authors conclude that: (1) There is a relation between the acidity of the gastric contents and the urine. (2) The greater the production of acid, whether by secretion or fermentation, the greater the amount of acid excretion in the urine. (3) Normally, the acidity of the urine falls during the first three to five hours after eating, thereafter it increases. (4) Most often there is an almost absolute parallelism between the two curves of gastric and urinary acidity, but this is destroyed after a repast by the presence of polyuria. (5) If the acid is withdrawn by any means from the stomach, the amount in the urine falls also, and the latter may even become alkaline. (6) The average quantity eliminated by the urine hourly is greater in hypochlorhydria (subacidity) than in hyperchlorhydria (hyperacidity). (7) Milk increases the acid in the urine, owing to its giving rise to lactic acid in the stomach. (8) It is not possible, at any rate at present, to trace the curves of urinary acidity so as to bear indirectly on the question of the chemical variety of the dyspepsia. (9) Milk must be excluded from test-meals when these curves are to be studied. (10) Patients should be subjected to a constant regimen for some time before the investigations.

Nature and Concept.—As the name implies, the factor with which we are most particularly concerned in this neurosis is the HCl. With superacidity, a gastric juice unusually rich in HCl and pepsin is secreted in very large quantities during digestion, as a result of the stimulation of the foods. On this account free HCl may be proved in the stomach after test-meals much earlier than under normal circumstances, and the acidity of the digestive mixture is further increased as digestion proceeds. Superacidity may be an independent disease confined to the stomach alone, or a partial symptom of hysteria, neurasthenia, and melancholia. It may also be noticed as a reflex neurosis with renal calculus and hepatic colic, and as the companion of organic changes of the stomach (ulcus ventriculi, gastritis acida).

Historical.—Even if superacidity, like supersecretion, has been demonstrated with certainty only in the last twelve years, through the researches of Reichmann, Jaworski, van den Velden, Riegel, the latter's pupils, and others, and the aspect of the disease has been

precisely defined by them, nevertheless, as Ewald justly emphasizes, it would be an error to believe that we have to do with an entirely new discovery, since both these anomalies of secretion, as also their nervous origin, were known to older physicians in the beginning and middle of this century,—men celebrated in England, France, and Germany (Trousseau, Todd, Budd, Copland, Pemberton, Hübner, and others). By some of these, the most important symptoms were also correctly stated. It is not intended that the merit of the previously-mentioned investigators of these neuroses of secretion shall be in any way diminished by this older historical reminiscence, for as the older physicians, owing to the lack of exact methods, could not recognize those anomalies of secretion with certainty, their results were soon forgotten; Reichmann was the first who, by a thorough examination of the contents of the stomach, with the help of newer and constantly-improving methods, furnished certain proof of the existence of secretory disorders which had previously been only suspected, while it was Ewald especially who emphasized particularly the nervous origin of supersecretion and superacidity, so that soon they were generally recognized as neuroses. The observations of Reichmann were soon after confirmed by von Noorden, Honigmann, Riegel, Jaworski, Saly, and others, and to-day there is a consensus of opinion concerning the nature and consequences of both neuroses. Jaworski designates both neuroses as very frequent disorders, since he could prove them in almost two-thirds of his patients who had diseases of the stomach, while Riegel also observed them very frequently in Hessia—although not quite so frequently as Jaworski—Ewald, with whom the author can agree, states that they, especially supersecretion, occurred only in a fraction of his patients with diseases of the stomach, so that supersecretion should be called rare, rather than frequent.

Etiology.—The fundamental causes of superacidity are still unknown. The little that has up to date become known concerning the etiology of the disease is confined to the knowledge of a few predisposing factors. Very excitable people, predisposed to nervous disorders, more frequently become affected with superacidity than those of calm temperament, who do not lose their equanimity so easily, although it is not found entirely wanting in the latter. Jaworski found hyperacidity very frequently in the excitable Jewish population of Galicia, pre-eminently disposed

to nervous disorders. With hysterical patients superacidity was noticed by Jolly, and in melancholic subjects by von Noorden, and it is also a frequent companion to neurasthenia. The disease is more common in men than in women. The educated, and particularly the learned, classes furnish the main body of patients suffering from hyperchylia, though it is not infrequent in the laboring

FIG. 40.—HYPERTROPHY AND PROLIFERATION OF GLANDULAR ELEMENTS.
From a case of persistent hyperacidity found in the eye of the tube.

classes. Local causes seem to play an important rôle in the etiology of hyperchylia, and this is vouched for by the frequent occurrence of the disease in Galicia and Hessia (Riegel), while it is much rarer in other districts. It would be a valuable contribution by various gastro-enterologists of the United States if they collected and reported the frequency of hyperacidity and other gastric diseases occurring in their localities, so as to throw light on the influences

of race, climate, geographical distribution, etc. It is our opinion that a diet rich in fish, meats, and proteids in general predispose to hyperacidity. That the frequent occurrence of superacidity with cholelithiasis and nephrolithiasis is a causal relation and not a mere accidental coincidence is shown by the fact that the stomach complaints dependent on superacidity generally disappear quickly after the passage of the calculi into the intestines and bladder respectively. The relation between superacidity and peptic ulcer has been sufficiently dwelt upon in the discussion of the pathogenesis of the latter; whether superacidity is a cause or result of the ulcer, it has, up to date, been impossible to decide with certainty. In the first half of this work the author has laid down reasons why hyperchylia may in some instances be sufficiently explained by the proliferation of glandular elements observed in fragments of mucosa found in the wash-water in one-half to two-thirds of the cases of hyperacidity examined (p. 135). Whether this condition is the cause or the result of the neurasthenia is difficult to determine, though we observed it when no neurasthenia could be detected.

Symptomatology.—*Disturbances of Sensibility.*—The subjective complaints consist chiefly in contracting, boring, burning, or gnawing pains in the entire region of the stomach, which generally radiate forward or toward the back. As they are the consequences of a strong irritation of the mucous membrane of the stomach by its superacid contents, they are generally only noticed during digestion, appearing some time after eating, and generally increasing perceptibly with the progress of digestion. They are much influenced by the quantity and composition of the food; with meats they are much less than with an amylaceous diet. After the introduction of food very rich in albumin they appear later than with a diet poor in albuminates, mainly because in the former case the appearance of free HCl in the contents of the stomach is postponed. At the height of digestion the symptoms are generally most severe. Temporary, strong, cramp-like pains in the region of the pylorus are generally the result of a spasm of the muscular sphincter of the pylorus. By alkalies, as also by the renewed taking of milk, eggs, or meat, the painful sensations are generally soon alleviated or temporarily done away with, so long as the acid is held in combination by the alkalies or albuminates. If a strong generation and collection of gases occur, the region of the stomach is swollen, and in some degree sensitive to touch.

If the escape of the gases upward or downward is temporarily prevented by simultaneous cramp of the cardia and pylorus, the complaints are considerably increased, owing to the strong expansion of the walls of the stomach. Other gastric symptoms, such as nausea, belching, and vomiting of very acid masses accompany hyperchylia very frequently. Belching causes slight, passing relief,while copious vomiting brings greater and more lasting relief. If small quantities of the very acid contents of the stomach are brought up through the eructations, and if the mucous membrane of the esophagus is subjected to caustic action by the latter, heartburn develops, which may increase to a severe contracting pain under the breastbone, which extends to the pharynx (pyrosis hydrochlorica, Sticker). The same complaints, naturally, may also appear after the vomiting of the contents of a very acid stomach. The manifold subjective symptoms previously stated are sometimes also observed with neurasthenic patients in whom the acidity of the gastric juice is normal. An abnormal sensibility of the gastric nerves to hydrochloric acid must be supposed in these cases (Talma). The appetite is generally undisturbed with superchylia. Thirst and appetite are often much increased.

The Influence of Hyperchylia upon the Transformation of the Foods in the Stomach.—As is well known, with increasing acidity of the gastric juice (at least, up to a certain limit) its digestive power for albuminous foods is increased, and peptonization proceeds more quickly and freely the sooner free hydrochloric acid is present in the contents of the stomach. Both conditions are given with patients suffering from hyperacidity. The acidity of the gastric juice is much greater than that of the normal secretion, which amounts to 0.15 to 0.2 per cent. It varies between 0.3 and 0.6 per cent. in hyperchylia; in severe cases between 0.4 and 0.6 per cent., so that in these 80 to 120 c.c. of a decinormal solution of caustic soda are necessary to neutralize 10 c.c. of the gastric contents drawn at the height of the digestion of a test-meal. Free HCl may be shown in the contents of the stomach in ten minutes after a test-breakfast, instead of in an hour, and in one hour, instead of in three to four hours, after the full test-dinner; therefore, the peptonization of albumin proceeds in a very prompt and free manner. On the other hand, gastric amylolysis is much disturbed, as the effectiveness of the ptyalin is interrupted very early by the appearance of free HCl. On this account one finds, in three

to four hours after an experimental meal, no undigested muscular fibers and albuminous particles in the contents of the stomach; but many unchanged amylaceous particles may still be found in the residue on the filter. (This is best studied with the double test-meal as recommended on p. 111.)

If the acidity of the gastric juice exceeds a certain maximum (0.6 to 0.7 per cent. and over), even the digestion of the albumin is in some way retarded (Schwann). But up to the present time such great quantities of free HCl have not been met with in the contents of the stomachs of patients suffering with hyperchylia.

A copious formation and collection of gases may occur in this neurosis of secretion.

The Effect on the Motor Function.—With very severe irritation of the mucous membrane of the stomach by the overacid contents, abnormally severe contractions of the musculature are produced reflexly, by which a quicker flow of blood to and from the gastric walls is effected, a more intimate contact of the ingesta with the mucous membrane of stomach hastens the secretion, resorption, the digestion of the albuminates, and also the passing over of the chyme into the intestines. Therefore one should expect a quickened emptying of the stomach in patients with hyperacidity; but in many cases exactly the opposite is noted; namely, a retarded passage of the contents of the stomach into the intestines. It is generally caused by a stubborn, often-recurring cramp of the pylorus, brought about by the strong irritation of the mucous membrane of the pylorus by HCl, which even the most extreme contractions of the rest of the musculature can not overcome. These overexertions of the latter may lead to fatigue and to atony, and the appearance of this state is furthered by the fact that the coats of the stomach are burdened and distended by the stagnating ingesta more than under normal circumstances. Since, however, the musculature can rest and recuperate with an empty stomach, the atony does not very frequently pass over into a chronic state of dilatation of the stomach. The formation of the dilatation is, however, to be feared when the cramp of the pylorus brings about a hypertrophy of its ring muscles, and with it a stenosis of its lumen. The disturbances of circulation in the coats of the stomach, caused by the cramp of the pylorus, in the presence of a very acid and potent gastric juice may favor the formation of peptic ulcer. The troublesome thirst so

often complained of by patients was formerly explained by a reduction of the power of resorption of the mucous membrane, caused by the strong irritation of the same (HCl) and spasm of the smaller vessels. The quick cessation of thirst after copious drafts of water has been explained by the dilution of the acid chyme and a decrease of the irritation of the mucous membrane.

That superacidity may cause disturbance of resorption is not to be denied. Since it is known, however, that the greater part of the water introduced is absorbed only in the intestine, and that the resorption of water in the normal stomach is only very slight, it might be more correct to trace back the thirst with superacidity to the cramp of the pylorus and the longer retention of the water in the stomach. After copious drinking of water, with dilution of the contents of the stomach, the irritation of the mucous membrane of the pylorus decreases, the cramp is lessened, and the water, passing into the small intestine, is then quickly reabsorbed in the latter. That, indeed, a cramp of the pylorus with hyperacidity often causes retention of the contents of the stomach may be proven by the introduction of the tube before and after taking the water—especially when it contains alkalies (Saratoga Vichy). Before the taking of water, the contents of the stomach still show abundance of food, while some time after the stomach is generally found to be entirely empty.

Urine.—After copious vomiting, by which a part of the HCl is permanently removed from the organism, the urine has an alkaline or neutral reaction (Sticker, Gluzinski, Jaworski, and Hübner); but this may be observed during the digestion in the stomach without vomiting. Since the phosphates more easily separate out of the alkaline urine, it may happen that the urine is turbid when emptied out of the bladder, a circumstance sometimes unnecessarily alarming the patient. Or, after standing a while, the urine may show a very plentiful sediment. The chlorids are sometimes decreased.

The state of nutrition generally remains good, especially at first. If considerable disturbance of the motility appears, the nutrition becomes impaired.

The Digestion of the Foods in the Intestine.—If the contents of the stomach enter the intestine in an overacid condition, it requires much more than the usual time to attain the alkaline reaction in the contents of the intestines with the help of the mixed alkaline

intestinal juices. An alkaline medium is indispensable for the digestion of fats and carbohydrates.

In the period immediately succeeding the passage of the chyme into the small intestine, so long as there is still free HCl present a small part of the undigested albumin is peptonized by the pepsin which has passed over along with it, while, on the other hand, the transformation of the carbohydrates and fats is arrested completely in this time.

Consequently, the digestion and assimilation of the foods in the intestine are much retarded in severe cases of hyperacidity, and the evacuation of the bowels is correspondingly delayed. The very acid chyme occasionally produces an abnormally intense peristalsis of the intestine, so that in exceptional cases the evacuation of the intestines may be diarrheic in character.

Prognosis.—This is favorable if the disease is of recent origin; older cases, however, are often stubborn. The ever-recurring pains and disturbances of digestion may, in the course of time, wear out the patient. With symptomatic superacidity the prognosis must be made in accordance with that of the primary complaints. If these—*e. g.*, hysteria, neurasthenia, melancholia, peptic ulcer, or cholelithiasis and nephrolithiasis—are successfully relieved, the superacidity will quickly disappear. In the author's experience, permanent relief may follow persistent treatment, even in chronic cases.

Diagnosis.—In this disease our double test-meal (see p. 111) is a special diagnostic aid. If a strong reaction for HCl is shown in the contents of the stomach after ten to twenty minutes succeeding the test-breakfast, or in one to one and one-half hours after the test-dinner, and if the macroscopic and microscopic examinations in the residue on the filter still show abundant remnants of carbohydrates, with complete absence of muscular fibers and particles of albumin, then the diagnosis of hyperacidity is established, even though the other previously-mentioned symptoms may be absent. In order to prove or disprove a co-existent supersecretion, we recommend to let the patient take the test-meal at night, and the next morning introduce the tube before breakfast. If it contains large quantities (over 100 c.c.) of a potent secretion, then supersecretion exists side by side with superacidity; and if the stomach be empty, or if only a small quantity of gastric juice be found in it, then supersecretion is not present. The result is still more certain if a few hours after the last evening meal the stomach is thoroughly washed

out and the next morning the tube is introduced into the jejune stomach. Schreiber asserts that supersecretion is found only in atonic stomachs.

The differential diagnosis between the ulcer and hyperchylia is of practical importance on account of the therapeutics to be followed. With both diseases, the manifold subjective complaints appear usually after eating: during digestion, with peptic ulcer patients, generally very soon after eating; with those suffering from hyperchylia, later on at the height of digestion, though the onset varies considerably according to the quantity and character of the foods.

With ulcer, the pains are generally confined to one region, namely, the epigastrium (circumscribed pain caused by pressure), and they very often radiate forward, laterally, toward the loins and shoulder-blades (dorsal pain-points), while in the case of superacidity a diffused sensation of pain exists in the whole region of the stomach and the radiating pains are wanting, and also the dorsal pain-points, which, in the case of ulcer, will be present in about one-third of the cases.

In the case of ulcer the pains are generally strongest when food in itself difficult of digestion has been introduced in large quantities, and it is irrelevant in this case if the food consisted mainly of carbohydrates or meats; in the case of hyperchylia the pains are generally less severe after abundant meals, even if the latter consisted of foods in themselves difficult of digestion, *provided only* that they are rich in albumin; after smaller meals with little albumin the pains are not relieved.

If the symptoms observed in the patient do not suffice to establish a positive differential diagnosis between ulcer and hyperacidity, it is safer to treat the patient for ulcer,—this may be the case when it is impossible to pass the tube on account of suspected ulcer. If the pains become less or cease entirely soon after beginning this treatment, it is probably a case of ulcer, while if they remain the same or increase it is a case of hyperacidity. If the hyperacidity is removed by this treatment at the same time as the ulcer, this fact suggests that the hyperchylia was not the cause, but the result, of ulcer. On the other hand, if, after curing the ulcer, the hyperchylia continues, we may presume either an accidental coincidence of the two, or else the hyperacidity has caused the ulcer. In the latter case, the breaking out of the ulcer or

the formation of new ulcers is to be feared in the future should the hyperacidity continue.

Therapeutics.—(*a*) *Diet.*—The selection of proper diet for these cases is one of the most important duties of the general practitioner and specialist. There are two systems of dietetic treatment for hyperchylia. One favors the use of amylaceous diet and the restriction of proteids, because the latter are powerful stimulants to HCl secretion. The advocates of the other argue that, since the manifold complaints with hyperchylia are a result of the irritation of the nerves of the stomach by free HCl, therefore, in the fixing of a rational diet such foods must be chosen which combine with the greatest quantity of HCl; that is, in the first place, meats; in the second, vegetables especially rich in albumin. All the stimulants which might further increase the irritation of the nerves of the stomach must be avoided, viz.:—pungent spices, tapioca, pepper, also mustard, horse-radish, ginger, organic acids, such as lactic, acetic, citric, and tartaric acids, fatty acids (rancid fat); cooking salt must, so far as possible, be kept out of the stomach; drinks rich in alcohol are just as injurious,—strong beer, heavy wines, but especially whisky and cognac. Food and drink must not be taken too cold (not under eight to ten degrees, 10° R. to 12.5° C.) and not too hot (not over 45° R. or 56° C.). After foods in themselves difficult of digestion, the complaints are not stronger if they contain much albumin than after easily digestible ones, nevertheless the latter are to be preferred, and careful preparation is requisite in order to avoid a mechanical irritation of the nerves of secretion. A diet consisting only of carbohydrates is, in our experience, not injurious. We have exhaustively stated the advisability of amylaceous diet in hyperacidity on pages 187 to 189. Pure fats are allowed in the same quantities as in the case of healthy people. As sugar does no harm in the case of most patients, such baked foods (cakes) are to be permitted in which, by a long process of baking or roasting, a part of the starch has been dextrinized; that is, well-toasted bread, cakes, breadcrust soaked in milk, and also certain dextrinized flours (Horlick's food, Kufeke's flour, and others). On account of frequent disturbance of the motility large quantities of water, by which the muscularis of the stomach is unusually distended, must not be introduced at one time; it is much better to give small quantities frequently, by which at the same time the acid contents of the

stomach are diluted. Burning thirst is best quenched by frequent imbibing of small quantities of alkaline mineral waters (Saratoga Vichy, Geyser, Capon Springs, Apollinaris water, Seltzer, Giesshübler, Fachinger water), which are rich in carbonic acid, as the latter is not only soothing, but also aids resorption. If this does not suffice, repeated injections of water into the rectum must be ordered.

Since the percentage of albumin of the various foods, and hence also their valence toward HCl, vary remarkably, a table of all these foods is given on page 239, showing how much hydrochloric acid they may hold in combination until the appearance of a weak but distinct reaction with the Baeyer-Gunzburg reagent.

In the two columns, those foods occupy the first places which combine with the least amount of HCl; those which combine with the greatest amount of hydrochloric acid (meat, poultry, game, fish, vegetables, etc.) stand at the end of the column.

From this survey it appears that for patients with hyperchylia, veal, beef, mutton, and raw ham (which hold in combination two to three times as much HCl as the same quantities of sweetbread, liver pudding, and calf's brain) are to be recommended. For the same reason the Leube-Rosenthal meat solution, which in itself is easily digestible, can be recommended. Also cooked ham and pork are suitable meats. Of the other foods, Swiss cheese, Roquefort, pea sausage, brick cheese, and of the various kinds of bread, especially pumpernickel and rye bread are available; wheat bread is not so suitable, as 700 gm. of it are necessary to combine with the same quantity of HCl which is held in combination by 300 gm. of pumpernickel or 100 gm. of veal. Beer is not suitable. Milk is to be recommended, however, both on account of its digestibility and composition. If, on account of muscular disturbances, it is desired to avoid large quantities of liquids, condensed milk should be advised; 600 gm. milk, condensed to one-fourth of its volume, combines with as much HCl as 100 gm. veal. Cocoa is also to be recommended.

If gastric distress and pain appear after supper before bedtime, the patient should drink a cup of lukewarm milk, bouillon with egg, and meat solution, or eat raw ham scraped fine, or one egg, which also combines with a great deal of HCl (see p. 319).

This large ingestion of proteid and albuminous food, especially advocated by Fleischer for hyperacidity, does not lead to

permanent relief, in the author's experience. According to carefully conducted experiments and analyses, he is of the opinion that a proteid diet may keep up a hyperacidity because it is a stronger stimulation to HCl secretion (see p. 187).

(*b*) *Medicinal Treatment.*—We refer to pages 322 to 326 in explanation of the use of alkalies in hyperacidity. Magnesia is preferable to the carbonates because it can not form chlorids, which may irritate the mucosa. As may be seen from the table (p. 239), the albuminates of the foods may take up considerable quantities of HCl and after their transformation into peptone hydrochlorate they aid the nutrition of the body, which is not the case with the chlorids. The digestive products of albumin, hemialbuminose, and peptone, combine with more HCl in the formation of their hydrochlorate compounds than albumin itself (peptone almost twice the amount), and the peptonizing, as well as the decomposition, of the peptone hydrochlorate in the blood requires some time, so that the HCl combined with it does not readily become free again.

Some of the alkalies may be taken in the form of alkaline mineral water—Saratoga Vichy, St. Louis Spring (Mich.), Apollinaris, Biliner Water, Fachinger, Salters, Vichy Water. Magnesia usta, bicarbonate of soda, sodium biborate (Jaworski, L. Wolff), are best prescribed as indicated on page 325. If there is an inclination to the formation of gases, the bicarbonate of soda is to be replaced by magnesia usta in order not to increase the amount of gas by the CO_2 which is set free from the former. Magnesia usta has also the advantage of forming a chlorid which has a mild aperient effect. According to Jaworski's experiments, the continued use of large quantities of Carlsbad salt and the thermal waters of Carlsbad reduce the secretion of the acids of the stomach; this might explain the beneficial influence of a protracted stay at Carlsbad (see p. 323). In our country we are assured that the Bedford and the Saratoga Carlsbad mineral waters have an equally beneficial effect. If the pains in the region of the stomach continue in spite of the remedies discussed thus far, narcotics are prescribed, especially extract of belladonna (0.03 gm. daily), and atropin sulph (0.0005 to 0.001 or $\frac{1}{100}$ of a gr.), given with advantage, together with magnesia usta or ammonio-magnesium phosphate—substances which not only have the effect of reducing pain, but also inhibit the secretion of the glands; codein phosphate (0.03 gr. ¼ to ⅓ daily, is a

reliable drug for this purpose). Cocain muriate is not suitable on account of the fact that its effect passes away rapidly, but bromid of sodium and bromid of ammonium (2.5 to 4.0 in twenty-four hours), when taken for some time, often do good service. Strontium bromid is even better tolerated than the sodium or ammonium salt.

On the other hand, the use of morphin muriate is to be as limited as possible. According to Hitzig and Alt, morphin muriate is, to a great extent, excreted in the gastric juice and also with the saliva, and therefore reaches the stomach again after absorption. Small quantities of morphin, however, excite the nerves more than they calm them.

If the patients complain of severe pains even on an empty stomach (without being able to prove supersecretion or hyperesthesia), then lavage of the stomach, irrigation of the mucous membrane, and internal douches, which were first recommended by Malbranc, will give more permanent relief. (See pp. 286 and 287, Lavage and Douching.)

With very stubborn cramp-like pains in the region of the pylorus (pylorospasm) there is nothing to be done but to remove the strongly acid contents of the stomach with the tube and to wash out the stomach, first with lukewarm water, then with bicarbonate of sodium, and to leave a small part of the latter in the organ.

The electrical treatment has been used successfully by Einhorn for this purpose, and he especially favors the internal galvanization of the stomach. Since the anode has a calming effect upon the irritated nerves (Heidenhan), it is perhaps best to introduce the anode with the intragastric electrode into the stomach filled with moderate quantities of lukewarm water, and to apply the cathode to the sternum, epigastrium, or spine.

Constipation is to be fought with rhubarb preparations (pulv. rad. rhei, 40.0; natr. sulph., 20.0), Carlsbad Sprudel salts, by injections, massage of the intestines, and glycerin suppositories. Since superacidity is frequently a neurosis, we must, in general, influence the nervous system favorably by a sojourn in the country, in the mountains, at the seashore, by cold rubbings, gymnastics, and abstention from severe mental labor. A treatment recommended by Biedert and Langermann (*loc. cit.*) has been found quite serviceable by the author. The stomach is first washed out by a solution of sodium bicarbonate. When the water returns clean we

pour in a one per cent. suspension of magnesia usta; when this has run out it is followed by a one-half per cent. solution of tannin. In the place of the latter, particularly when it is not well tolerated, we often use a suspension of bismuth subnitrate; when the pains are severe, the author prefers the latter together with bismuth subgallate.

PERIODIC ATYPICAL FLOW OF GASTRIC JUICE, GASTROXYNSIS (*Rossbach*), GASTROXIE (*Lépine*). GASTROSUCCORRHEA PERIODICA (*Reichmann*).

Gastroxynsis, or periodic flow of gastric juice, is an atypical secretion of the peptic glands, atypical because it does not occur after a normal digestive stimulation, but rather when the stomach is empty. The attacks are associated with intense gastric distress, severe spasmodic pain, and vomiting of considerable quantities of very acid gastric juice. This peculiar neurosis is found only among the educated classes, and particularly among those individuals who are subjected to unremitting mental exertion. In exceptional cases persons belonging to the laboring classes are attacked by it. The malady occurs in attacks which last from one to three days, returning in some instances every week, and in others at intervals of months. The attacks are more frequent when the mental exertion is severest, and become rare as soon as pauses of mental rest intervene. During vacation of these brain-workers, or sojourn at the seashore or in the mountains, the attacks disappear entirely, to return again when the sufferer applies himself to his profession. The pains which are most probably caused by irritation of the mucosa, by the intensely acid secretion, are generally preceded by nausea, eructation, and pyrosis. Eventually the emesis of large quantities of acid liquids supervenes, and, as a rule, terminates the attack. The sufferers generally recuperate quickly. Jürgensen and Ewald have reported cases of typical migraine, that were also associated with superacidity. Rossbach (*Deutsch. Archiv f. klin. Med.*, Bd. XXXV, 1885) and Rosenthal (*loc. cit.*) have suggested hypotheses attempting to explain the pathogenesis of periodic flow of gastric juice; their theories are not supported by experimental evidence, and have not at all cleared up the subject.

Etiology.—Among the incidental causes, we meet with excessive and exhausting mental exertion, intense emotional excitement,

anger, nicotin poisoning, and occasionally dietetic errors. The so-called periodic flow of gastric juice, as first described by Reichmann (*Berlin klin. Wochenschr.*, 1882, Nr. 40), and the gastroxynsis of Rossbach, are, in our opinion, simply phases of the same neurosis, not different diseases.

Symptomatology.—The attacks occur very acutely, more frequently on an empty stomach, and with a feeling of pressure in the head increasing to intense headache, pain in and over the eyes, distress, pressure, and fullness in the stomach, increasing to gastralgia. Eructation, pyrosis, and nausea usher in abundant vomiting of highly acid mucous masses, the acidity of which is equal to 0.2 to 0.5 per cent. of HCl. Repeated vomiting will bring up mucus and bile. When the vomit occurs while the stomach still contains ingesta, this will show the same chemical reactions as are found in hyperacidity. The drinking of water relieves the gastric distress by diluting the acid, but generally increases the vomiting. In our experience the attacks occur, as a rule, in the middle of the night, or in the early hours of the morning. The patient has a very pale appearance and the extremities are frequently cold. A few hours after the first vomiting of gastric juice the attack may be repeated, and again an equally large quantity of gastric secretion containing no food particles whatever may be vomited up. Occasionally the gastric pains are the only symptom, and headache follows later on; in fact, the symptoms might be differentiated into gastric and cerebral symptoms—at times the former prevail, and at others the latter. The very acid masses in the stomach very likely cause a reflex spasmodic pylorospasm. Eructation, insufficiency of the cardia, and pneumatosis are frequent accompaniments. Periodic atypical flow of gastric juice may be an independent neurosis of secretion, or reflexly caused by diseases of the central nervous system. The gastric crises occurring in tabes have been classed with periodic secretion by some authors, but according to von Noorden (Charité Annalen, 1890), Bouveret (*loc. cit.*, p. 680), and Boas (*Deutsch. med. Wochenschr.*, 1889, Nr. 42) the liquids vomited in gastric crises are not always acid, and frequently may be found alkaline. They are not associated with the very severe phenomena of highly-increased acidity of gastric juice; namely, the strong pyrosis, and the feeling of a corrosive substance in the stomach. Bouveret has expressed his doubt concerning the existence of a central form of periodic gastrosuccorrhea.

Diagnosis.—Gastroxynsis may be confounded with the migraine associated with gastric symptoms, with intermittent forms of severe hyperchylia, and with the gastric crises. The diagnosis can be made by chemical analysis of the vomited matter. The attacks usually occur in the midst of good health, and the severe thirst, loss of appetite, cephalalgia, and great prostration are characteristic symptoms. Rossbach found an acidity of four per thousand (HCl) in one of his cases, and Boas found that there was a hyperacidity even in the intervals between the attacks, and that the amount of gastric juice during the attacks was not much increased as compared to that found in the intervals.

Example I.—Miss M. G., age twenty-four, of neuropathic extraction, has frequently had attacks of vomiting and gastric pain during childhood. For about six years she has suffered with intense pyrosis, which was relieved by bicarbonate of sodium tablets. Sometimes the heartburn ceased after the ingestion of food. She is a music teacher, and frequently spends eight to ten hours a day teaching pupils and giving singing lessons. The appetite is at all times very good, bowels slightly constipated. The acidity after our double test-meal taken in the interval between the attacks is equal to 0.3 per cent. HCl. About once a week she has distressing attacks of gastralgia, associated with severe headache and vomiting of very acid liquid masses. The attacks occur generally between two and three o'clock in the morning, when she has spent a day at hard work teaching pupils. The patient awakes suddenly with a feeling extending from her stomach to her throat, which is described as a twisting of the gullet. Severe cephalalgia, giddiness, nausea, and vomiting follow. Sometimes she does not vomit, but the attack is passed off by rapidly drinking a half-pint of water with a teaspoonful of bicarbonate of soda. Physical examination, entirely negative. Urine, the indican is increased. Uric acid, ratio high. No splashing sound in the stomach prior to ingestion of food or drink. Acidity of filtrate of vomited matter, which apparently was free from bile, was equal to 2.8 per thousand (HCl), or 0.28 per cent. The fact that the acidity was less during the attack than during the intervals, suggested that the HCl had been neutralized through bile, duodenal secretions, or saliva, but the careful examination for these constituents was negative. During a summer vacation in which the patient undertook a trip to Europe, she vomited daily from seasickness, but in three months, while she was in Germany, she did

not have one attack. On returning, the acidity, after a similar test-meal as before stated, was equal to 1.5 per thousand (HCl). General condition much improved.

Example II.—This case is that of a colleague, a friend of the author, who has described his case with great accuracy on repeated occasions. The attacks usually occur at night, associated with headache and gastric distress, and culminate in the vomiting of large masses of highly acid material. The doctor is an indefatigable brain-worker, allows himself very little, if any, recreation, and rarely leaves the house. His general nutrition is good, and he has found that his attacks are rapidly relieved by the taking of ordinary cane sugar (Ewald).

Periodical vomiting, when associated with hyperacidity, must be carefully distinguished from gastroxynsis.

Treatment.—This includes, in the first place, the avoidance of stimulants and narcotics; alcohol and tobacco, as well as strong coffee. It is most essential that the patients should avoid mental overwork. They should, in fact, refrain from brain-work altogether, and allow themselves three to four months a year to enjoy recreation in the mountains or at the seashore. Physical exercise should be indulged in moderately but systematically. The bicycle is an excellent remedy for periodic flow of gastric juice; and also horse-back riding, swimming, rowing, fencing, gymnastic exercises, and out-door games. During the attack itself, the effects of the excess of acid should be counterbalanced by copious drafts of suspensions of calcined magnesia, ammonio-magnesium phosphate, or bicarbonate of sodium. Where the vomiting has occurred at short intervals, one should not hesitate to pass the stomach-tube, wash out the stomach with sodium bicarbonate, and afterward treat the mucosa with suspensions of bismuth subgallate (℥ij to Oj). A mustard plaster should be placed over the epigastrium. If this can not be conveniently had, a hot-water bag will act similarly. The bromid of strontium and bromid of ammonium, in doses of 30 grs. three times a day, have an undeniable effect upon the frequency of the attacks. The diet should be carefully adjusted to the digestive capacity of the stomach. We usually recommend Penzoldt's diet order, which is given among the diet lists. In the intervals between the attacks the patients should undergo treatment as outlined for hyperacidity.

CHRONIC CONTINUOUS FLOW OF GASTRIC JUICE (CHRONIC HYPER- OR SUPERSECRETION (*Riegel*), GASTROSUCCORRHEA CHRONICA (*Reichmann*).

We have our doubts whether such a condition of permanent irritation of the gastric secretory nerves and uninterrupted secretion exists as a primary disorder. Chronic gastrosuccorrhea, which Reichmann claimed to have observed and first described in 1882 (*Berlin. klin. Wochenschr.*, 1882, Nr. 40; 1884, Nr. 48; 1887, Nr. 12) as a disease peculiar to itself, is stated by him to be a disorder characterized by the chronic uninterrupted secretion of gastric juice at all times, even where there is no food in the stomach. In a fasting condition in the morning, Reichmann and others claim that gastric juice could be drawn from the stomach in these cases. As we have seen in the description of the organic gastric diseases, particularly in severe reflex neuroses, in dilatation, and gastric ulcer, continued flow of gastric juice is a frequent symptom, associating itself with alteration and loss of substance in the mucosa. Occurring as a secondary affection, it may still be able to effect severe damage to the gastric walls. The diagnosis of this hypothetical disease hinges upon the presence of gastric juice containing HCl and ferments in the jejune or fasting stomach. This question has been very carefully investigated by Schreiber (*Deutsch. Archiv f. klin. Medizin*, Bd. LIII, S. 90). He found, in Königsberg, that in over 70 per cent. of his patients a digestive secretion was contained in the fasting stomach.

We have very carefully investigated this subject from quite a large clinic in Baltimore, and can confirm the results of Professor Schreiber. Physiologically speaking, an absolutely clean and empty stomach should, in the morning, contain no gastric juice, as the glandular apparatus is normally in a resting state, but practically the human stomach is very rarely, or perhaps never, in this condition. It contains at all times epithelial detritus, dust, bacteria, secretions from the mouth, larynx, and pharynx, particularly saliva, which at different intervals are swallowed consciously or unconsciously. Now these albuminous, mucous masses, which are generally weakly alkaline, and which collect particularly during the night, incite the specific gastric glands to secrete their physiological product just as any other weakly albuminous food would do. This is a kind of pseudo or frustrate digestion, because, so far as nutrition is concerned, this slow digestion going on constantly is of

no value. (See pp. 138, 139, and 140.) In dilatation this slight pseudo digestion, which is present perhaps at all times, is augmented and multiplied by the permanent presence of actual food masses. The frustrate digestion becomes a real one. In all dilatations with retention of food we have a permanent, real digestion and an augmented permanent secretion corresponding to it.

This so-called continued hypersecretion leads to the digestion and assimilation of proteids and albuminous bodies of the food, while the normal digestion of carbohydrates is impeded. The phenomena that are claimed to be typical of chronic continued hypersecretion are unavoidable consequences of dilatation. The cardinal point of distinction,—namely, that digestive secretions are contained in the stomach on the morning following a very effective washing out executed the evening before, is certainly not peculiar to chronic hypersecretion, but occurs also with dilatation. Schreiber has called attention to the fact that it is exceedingly difficult—even impossible—to completely evacuate and clean out a dilated stomach. During gastrotomies food substances have been found in the stomach, notwithstanding very energetic efforts to free it of all remnants beforehand. When a patient with a dilated stomach is to be tested for the gastric contents prior to taking food in the morning, it is expedient not to be satisfied with the simple expression method of Ewald where this is negative, but to place the patient in a horizontal position, and, while he makes efforts at straining as if he were bearing down for stool, the operator must compress the stomach near the fundus, which, in these cases, is sometimes found below the umbilicus.

In the reports of a number of advocates of chronic hypersecretion as a primary disease *per se*, one frequently finds that the authors state that small quantities of food remnants were found in the fasting stomach. The argument generally follows that such small quantities of food could not be the cause of the large quantity of gastric juice secreted, contending that the latter must have been secreted spontaneously. At the same time, the illogical position of these writers is demonstrated by their assertion that the momentary contact of a soft stomach-tube with the mucosa is the cause of the secretion of 80 to 100 c.c. of gastric juice. This assertion, it should not be forgotten, is made by a number of those who have found food remnants in the stomachs of these cases. That is to say, the effort is made to ignore the physiological

stimulus of food, which is contained in the stomach for hours, and emphasize the rather insignificant momentary stimulation caused by the introduction of a tube. As Schreiber correctly points out, the freedom of the stomach contents from food particles is very often only an apparent, not a real, one. It is caused by imperfections in our methods of investigation, and when one closely considers the facts of chronic hypersecretion, as they are described by the adherents of Reichmann, they seem to be identical with those of dilatation. We have been able to exclude the hypothetical factor of the stomach-tube in causing a secretion of gastric juice in a normal fasting stomach. In a number of our students to whom we administered a hypodermic injection of apomorphin before they had eaten anything in the morning, we demonstrated the presence of HCl in the vomited matter. There is, therefore, to some degree a physiological normal continued secretion of gastric juice, as Schreiber correctly asserts (*Deutsch. medizin. Wochenschr.*, 1894, Nr. 18–21), the stomach secretes gastric juice normally and independently of the ingestion of food. Ewald and Boas cite a case which has been quoted by Riegel (*Deutsch. med. Wochenschr.*, 1893, Nr. 31, 32) in opposition to the views of Schreiber. This female patient had a peculiar gastric neurosis founded on a hysterical basis. For six years the patient vomited everything that was ingested; fluids were vomited immediately and solid food after two to four hours. When she had taken 100 c.c. of water on an empty stomach in the morning, she vomited it very soon thereafter, and Ewald and Boas could not find free HCl in it, and therefore concluded that the fasting stomach secretes no gastric juice under normal conditions. Aside from the fact that this woman may have had a spasm of the esophagus or cardia preventing the small amount of water from ever reaching the stomach, it is not fair to decide a physiological question from results obtained from a chronic neurotic patient; for, as we know, in this class of individuals the greatest variation in the state of the gastric secretion exists. The results obtained on human beings with gastric fistulæ (see W. Beaumont on his Canadian hunter, Alexis St. Martin–Kretschy–Richet) are inadmissible to the solution of this physiological question, because they are made on individuals under pathological conditions.

The existence of HCl in normal stomachs may be demonstrated by giving healthy individuals long pieces of thin thread,

which with some practice they can learn to swallow on an empty stomach; this silk or thread is so thin that it does not irritate the gastric wall to any degree, at least not so much as a stomach-tube; the thread is then rapidly withdrawn and pressed between pieces of Congo paper, when it can be seen that the Congo paper turns dark blue. We have also introduced the thread already stained with Congo red, and obtained the blue discoloration from fasting normal stomachs. Undoubtedly there are great individual variations in the genuine as well as in the frustrate digestion, caused by the secretive power of the glands and the character of the food. Morbid conditions influence both of these types of digestion, quantitatively and qualitatively. Conditions which incite the glands will increase the HCl, and conditions which weaken the mucosa, such as gastritis, diminish the HCl during real digestion after meals, as well as during the frustrate digestion occurring during the night and on a fasting stomach. These variations are also frequently found in dilatation. If the gastrectasia occurred on the basis of an ulcer, the true secretion, as well as the permanent secretion of a frustrate character, will be increased, and reversely, when carcinoma or chronic gastritis is present together with dilatation, both kinds of secretion will be diminished, or they may not contain HCl at all. In diagnosing a dilated stomach it is important to bear in mind that a stomach may be very much enlarged and still its greater curvature may not have descended to any considerable extent. Frequently the very cause which has brought about the dilatation,—for instance, perigastritis, or adhesions about the stomach,—make a descent of the greater curvature impossible, simply because it can not descend, being bound down into this position in the upper part of the abdomen by inflammatory adhesions. Therefore it is possible that a stomach may be dilated and yet give no splashing sound about the neighborhood of the umbilicus, nor need it be much displaced from its normal position. The stomach may, in fact, enlarge in an upward and backward or lateral direction when its descent is made impossible by adhesions. The presence or absence of food particles in contents drawn from the fasting stomach can not always be recognized by the naked eye. What looks like a turbid liquid free from ingesta to the naked eye, will often show undigested rice, bread, and other carbohydrates under the microscope. We have personally seen cases which according to the description of Reichmann and Riegel would have to be classed as

typical chronic hypersecretion, in which the stomach was apparently in its normal place, and no organic gastric disease could be determined with the most exact methods of examination. In three of these cases we could extract from 100 to 150 c.c. of gastric juice from the fasting stomach, apparently containing no food contents but frequently containing traces of bile. The acidity of this secretion when filtered was equal to 80° HCl by decinormal solution of sodium hydroxid and either Congo or dimethyl-amido-benzol.

The total acidity was 110 in one of the cases—that of a young bank clerk twenty-four years of age. He also suffered from constipation, pyrosis, increasing to pain, and vomiting which came on very soon after meals. The examination of the contents one hour after our complex test-meal gave the following results: Total acidity, 108; free HCl, 84; biuret reaction, positive; patellar and pupillary reflexes, normal. For eight consecutive days free HCl and gastric ferments could be detected in the contents from the fasting stomach. As his trouble was persistent and he was determined to get well, he consented to a course of exclusive rectal feeding. He was nourished for ten days by the rectum, and at the same time his stomach was washed out with solutions of magnesia usta every day. Under these conditions, when no food was ingested per os, the gastrosuccorrhea rapidly diminished, and disappeared entirely on the fourth day, so that not even the swallowed masses of mucus and saliva could sufficiently stimulate the mucosa to produce a secretion of HCl. This has occurred in three of our cases where the amounts of gastric juice found on an empty stomach exceeded 100 c.c. A second one of these cases was that of a young girl with chronic flow of gastric juice, who was operated on upon our suggestion by Dr. R. W. Johnson at the Maryland General Hospital. After the abdomen was opened and the stomach incised, no anatomical cause for the persistent vomiting and gastralgia could be detected. On replacing the stomach, however, and inserting the finger into the pylorus, a rather sharp bend in the duodenum was evident to the author. Undoubtedly this kink became more manifest when food was ingested, the stomach thereby dragging upon this acute angle in the duodenum. It was one of those cases of motor insufficiency which Broadbent has described (*British Medical Jour.*, vol. II, 1893, pp. 1193 and 1268) due to kinking of the duodenum by an abnormally short duodeno-

hepatic ligament. During the operation the pylorus was also enlarged by sewing together the oblique incision which had been made (it is true only for explorative reasons), but was in closing up sutured in such a way as to resemble a pyloroplastic operation. The patient made a perfect recovery, and there was no more gastrosuccorrhea, vomiting, or gastralgia.

She remained in the hospital for two months after the operation, and was not supplied with specially prepared diet, but lived upon the regular hospital fare without gastric distress, and was discharged in good condition. Einhorn (*loc. cit.*, p. 313) agrees with Reichmann as to the existence of a pathological continuous gastrosuccorrhea, although he restricts this name to cases not presenting organic lesions of the stomach. Whenever the latter exists (lesions) he looks upon the accompanying gastrosuccorrhea as a consequence of the main trouble, but not as a cause of the organic lesion. It is the exclusion of these organic troubles, particularly of enlargements of the stomach and motor insufficiency, in which the greater curvature has not descended, which presents so much difficulty. In all the cases of chronic continued hypersecretion that we have examined with regard to this question, we were enabled to discover some organic lesion, most frequently an atony, pyloric stenosis, or dilatation from some cause. After a careful investigation of a large clinical material we can not, from our own experience, affirm the existence of chronic gastrosuccorrhea as an idiopathic neurosis *sui generis*. We hold that chronic hypersecretion is not a spontaneous, idiopathic neurosis, but a secondary symptomatic phenomenon. We base our conclusion upon the following facts: (1) That gastric juice is contained normally in the fasting stomach; the secretion of the peptic glands being set up by the presence of mucus, saliva, dust, bacteria, epithelial detritus, etc. (2) That apparently clear gastric juice obtained from a fasting stomach may show presence of food particles microscopically. (3) That it is not possible to exclude dilatation nor ulcer in all of these cases, particularly when the dilatation is not marked by the descent of the greater curvature. (4) The liquid obtained from undoubted dilatations of the stomach may contain absolutely no food particles, thus simulating the condition for chronic gastrosuccorrhea. (5) Gastric contents obtained from dilatation of the stomach do not always show the presence of products of imperfect starch digestion (erythrodextrin). This is particularly the case when we

meet with dilatation accompanied by hyperacidity, but with a fair motility or where the peristalsis is only periodically lost. In any case of hyperacidity it is possible that the products of starch digestion may be absent when very little carbohydrate food has been ingested. This may, of course, also happen with gastrosuccorrhea. (6) In cases of typical, so-called chronic, continued supersecretion, the symptoms cease entirely and the stomach contains no gastric juice in the morning after the patient has been fed by the rectum for four to eight days. (7) Diseases presenting the classical picture of Reichmann's disease have been known to disappear entirely after a gastro-enterostomy or a pyloroplastic operation was performed.

Symptomatology.—This is essentially the same as in motor insufficiency with hyperacidity. (See chapter on this subject.)

The periodic flow of gastric juice, which we have already described (p. 727), is either a functional neurosis or a reflex affection (tabes dorsalis), or connected with an affection of the sympathetic. It is identical with the gastroxynsis of Rossbach. Possibly, also, the periodic vomiting of Leyden belongs to this group of neuroses. The chronic gastrosuccorrhea *is a symptom*, and has not the claim to be considered a morbid entity like the periodic or spasmodic gastrosuccorrhea. Chronic flow of gastric juice may be a complication of ulcer and motor insufficiency. From a pathological standpoint, it is well established that gastritis may accompany ulcer as well as dilatation (Rokitansky, Lebert, Orth, Cruveilhier). The gastritis which accompanies these diseases, and which shows hyperacidity, has been called by Korczynski and Jaworski (*Deutsch. Archiv f. klin. Med.*, Bd. XLVII, p. 578) "catarrhus acidus," and by Hayem (*Gazette Hebdom.*, 1892, Nos. 33 and 34) it has been designated as "gastrite hyperpeptique." These expressions signify the same complexity of symptoms as those first described by Reichmann under the name of "gastrosuccorrhea." The "gastritis acida" of Boas is quite a different thing (p. 434), a characteristic form of chronic gastritis.

Diagnosis.—As Boas says, the main question to decide is not whether we are dealing with chronic gastrosuccorrhea, which is not very difficult to find out, but to determine which disease it is a consequence of. The most frequent causes are ulcer and mechanical insufficiency. For a fuller explication of these subjects and their consequences, we must refer to the chapters in

which they are considered. It is much to be regretted that a number of authors still insist upon the identity of chronic gastrosuccorrhea, and regard the symptom or complication in the light of a disease *sui generis*.

Treatment.—If ulcer or dilatation can be demonstrated to exist, the treatment must be directed to these fundamental causes (see treatment of ulcer and motor insufficiency). In the absence of any definite etiological factor, the treatment is that described under hyperchylia. Under all conditions massage of the stomach and intestines, intragastric application of the galvanic and faradic currents, and washing out of the stomach are very essential adjuncts to treatment. Where there is much secretion of gastric juice, even on an empty stomach, the use of a stomach-tube can not be consistently neglected. It is the only way to remove the excess of secretion directly; then, again, the best treatment of a hyperchylia is that which is supplied directly to the mucosa in the form of irrigations with calcined magnesia, sodium bicarbonate, tannin (½ per cent. solution), and suspensions of bismuth subnitrate. The methodical use of alkalies affords great relief to the pyrosis and eructation, and facilitates carbohydrate digestion. The alkalies which we recommend most strongly are the magnesia usta and the ammoniophosphate of magnesium. Einhorn speaks very favorably of spraying the stomach with a solution of nitrate of silver, 1 : 1000. Reichmann administers the nitrate of silver in solution or in gelatin capsules.

The diet will vary with the underlying causative disease. It will be either that for ulcer or dilatation, or that for hyperchylia. Where the stomach is extremely sensitive, the diet orders of Penzoldt may be safely tried, because they are very sparing and make little demands upon the capacity of the stomach.

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11. Debove et Rémond, "Les Maladies de l'Éstomac."

12. Johnson und Behm (*Zeitschr. f. klin Med.*, Bd. XXII, S. 478), report 106 cases of supersecretion, including all cases where slight amounts of gastric juice were found, and give complete literature.

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23. Lyon, G., "L'Analyse du suc Gastrique," Paris, 1890.

24. Text-books of Leube, Riegel, Boas, Ewald, Debove et Rémond, Bouveret, Penzoldt, Fleiner, S. Martin, A. Pick, Mathieu, Einhorn.

SUBACIDITY (HYPOCHLORHYDRIA OR HYPOCHYLIA).

Subacidity, as a neurosis, is a disease in which, even during the height of digestion, gastric juice is secreted in which the HCl, and with it the pepsin and rennin, are present in smaller amounts than normal. We will not consider under this head those secretory anomalies in which the secretion of gastric juice is absent entirely. These states will be considered under Achylia Gastrica or Inacidity. In subacidity HCl is still secreted, but in such small amounts that it enters into combination with albuminous foods

entirely, and we can detect it only as combined HCl. Cases in which free HCl can be detected by Congo paper after our double test-meal do not logically belong in this class of subacidity, because the presence of free HCl means that more HCl is present than can enter into combination with the food; there is an excess of acid beyond that required for digestion. Technically, we may, therefore, define hypochylia as a secretory neurosis in which free HCl is absent at the test-meal, but combined HCl and the ferments are still present. Nervous hypochylia is in reality but a phase of nervous dyspepsia (or neurasthenia gastrica). But, as it is desirable to represent all secretory neuroses seriatim, we have here abstracted the symptoms of nervous depression of gastric secretion. It was formerly believed that subacidity was always connected with some organic gastric disease (carcinoma, gastritis), or occurred in the train of infectious diseases, or, as a result, with anemia and leukemia. Subacidity, however, may exist on a purely nervous substratum, in hysteria and neurasthenia and in psychoses. It then occurs, independently of anatomical changes in the stomach, as a functional disturbance of the secretory nerves, the irritability of which has been reduced. Functional disturbances of this character may be limited to the secretory nerves and not involve the remaining nervous apparatus of the stomach. The amount of HCl secreted is not sufficient to saturate the albumin present in the proteid food; in other words, an HCl deficit exists. It is probable that the secretory nerves become exhausted sooner than the motor nerves, and that, therefore, subacidity may be an expression of exhaustion or weakness in the secretory apparatus. In this way we have repeatedly observed prolonged subacidity followed by pronounced hyperacidity.

Etiology.—Nervous subacidity or hypochylia is a secondary phenomenon occurring with neurasthenia, hysteria, tabes, and the psychoses.

Symptomatology.—When the motor function of the stomach is good, symptoms may be absent entirely, but the slightest insufficiency of the motor power is rapidly followed by decomposition in the gastric contents, caused by bacteria, for the amount of HCl secreted is not sufficient to inhibit or prevent the action of microorganisms. As a consequence of this, organic acids are formed, and gaseous formations create gastric discomfort, and, at times, intestinal distention. There is nothing characteristic in the symp-

tomatology of subacidity. The result of the depressed state of the secretion and the general symptomatology are the same as those in achylia gastrica, and will be described under that heading. It is natural that amylolysis should proceed more rapidly in the absence of free HCl, since nothing can disturb the activity of the ptyalin in that case. On the other hand, the digestion of meats, eggs, etc., is most unsatisfactory. As HCl is one of the principal normal stimulants to peristalsis, the disease is frequently accompanied by constipation, which, in turn, produces increasing putrefaction of the intestinal contents, which is more pronounced because the disinfecting action of the HCl is missing.

Differential Diagnosis.—Carcinoma and chronic gastritis might be confounded in the incipient stages with nervous subacidity. (For the differential diagnosis from carcinoma and gastritis, we refer to the chapters on these diseases.) But when the enzymes, pepsin and rennin, can be demonstrated in the gastric contents, or even if only the proenzymes, pepsinogen and rennin-zymogen can be demonstrated, one can not, as a rule, exclude chronic gastritis and carcinoma. A patient and prolonged study of nervous subacidity will not fail to demonstrate that great variations exist in the amount of hydrochloric acid that is secreted. Occasionally it may be found that a transition to a normal acidity, or even to hyperchylia, has taken place. Lactic acid is a very rare occurrence in nervous subacidity; its presence and the Oppler-Boas bacillus would speak for carcinoma.

Treatment.—In most cases it will be sufficient to supply an amount of dilute HCl which is commensurate with the deficit. In rare instances it will be impossible to administer sufficient HCl to give the reaction for free HCl, because it is not well tolerated in this quantity. In that case we advise adding the HCl to beef juice, either Wyeth's, Valentine's, or the Mosquera beef jelly. This makes a sauce which can be poured over the finely-divided meat foods that are to be eaten. The meat-dissolving power of the acid is not destroyed by this method of preparing it, although the acid is mostly in a combined state. Abnormal fermentations and decompositions are rare, and therefore the stomach-tube can, as a rule, be dispensed with. The bitter tonics—quassia, cinchona, calumbo, gentian—and the basic orexin (in five-grain doses three times a day) very often increase the appetite and favor a secretion of HCl. Strychnin and the intragastric use of the faradic current

we warmly recommend for this purpose. When the motility is good, those mineral waters which are rich in sodium chlorid are worth a trial. (See section on Mineral Waters.)

The Diet.—It is an interesting fact that patients with subacidity instinctively avoid a meat diet, and are large carbohydrate eaters. It is well, however, not to let them persist in the exclusive use of carbohydrates, but to train up the digestive capacity of the stomach to a more abundant digestion of proteids. All meats should be given in a finely-divided state; before the meal, it is well to stimulate the appetite and secretion by giving a few sardelles or the roe of potted herring, or, what is more palatable and easier to procure a sandwich spread with Russian caviar. Surf baths, cold sponge baths at home, proper movements of the bowels, and at least eight to nine hours of sleep, are indispensable agents in the management of this secretory defect.

CHAPTER XII.

ACHYLIA GASTRICA.

Synonyms.—Absence of the Secretion of Gastric Juice, Nervous Inacidity, Atrophy of the Stomach, Anadenia Ventriculi, Phthisis Ventriculi, Achlorhydria.

Nature and Concept.—The term *achylia gastrica* means, literally, without gastric chyle, and was first proposed by Einhorn (*New York Medical Record*, June 11, 1892) to designate a class of diseases in which no gastric juice is secreted.

The affection is found to exist in two varieties: First, the primary, idiopathic, possibly inherited, achylia; secondly, the acquired or secondary achylia. The primary idiopathic or symptomatic achylia is characterized by the fact that absence of secretion is evident before any marked anatomical changes have occurred in the mucosa which could explain the loss of function. It is, therefore, as a rule, not regarded as a result acquired from a real disease, but as an individual peculiarity, possibly an inherited functional debility. There are undoubtedly persons in whom gastric secretion may be absent for years, or permanently wanting; yet who, apparently, may enjoy robust health. The majority of these individuals, however, have suffered from frequent dyspeptic complaints, which are partly of a purely nervous character. In these cases severe anemic and cachectic conditions are usually absent, and while the general nutrition may occasionally be found disturbed, it is easily remedied with proper dietetic treatment.

The last-named type of cases demonstrates that the function of the stomach may be permanently lost so far as its digestive power is concerned, yet with no apparent effects upon the general constitution. A very convincing argument for the compensatory digestive power of the intestine! Lubarsch ("Achylia Gastrica," etc., von Martius u. Lubarsch, 1897, p. 74) raises the question whether gastric digestion may not be entirely dispensed with, or whether it is not superfluous, which of course implies that the secretion of HCl may possibly be an unnecessary function. There

can be no doubt, however, that deficiency of gastric secretion is a disease. Individuals affected with symptomatic achylia are very much more sensitive in general, and more susceptible to gastric diseases, than their fellow-men equipped with normal stomachs. The idea that gastric digestion is superfluous and dispensable impresses us as being a reactive opinion induced by the other extreme view formerly held, according to which the stomach was the most important of all digestive organs. Gastric secretion is by no means a useless function. Lubarsch says: "Those who have lost it, have one weapon less in the struggle for existence," and clinical experience teaches that persons who have no secretion of gastric juice are much more liable to diseases of the stomach. When such are attacked by intestinal diseases, the prognosis actually becomes serious.

Achylia may exist upon a nervous basis, it may be congenital, or acquired, in consequence of some organic gastric disease.

The results of the examination of the gastric contents, in simple, uncomplicated achylia, are quite characteristic: The fasting stomach, examined in the morning before any food has been taken, is empty. We have never been able to obtain more than 20 to 30 c.c. of neutral, slightly mucoid liquid; remnants of ingesta of the previous day are never observed. One hour after the Ewald test-breakfast, the contents of the stomach have the same appearance as they have in the mouth before they are swallowed. This appearance is claimed by Einhorn and others to be quite characteristic. Contents drawn in this manner are generally slightly acid. Blue litmus paper is very slightly reddened. The total acidity varied in our cases from two to eight. This degree of acidity can be found in the test-meal before it is eaten. Whenever the acidity of the drawn stomach contents does not exceed that of the meal before it is swallowed, it may be safely assumed that free and combined HCl are absent; in other words, no HCl has been secreted. Whenever the total acidity is equal to four, it is due to acid that has been introduced in the food; with a total acidity no higher than four, one hour after a test-breakfast, it is, therefore, unnecessary to make further complicated analyses for the detection of HCl. The gastric contents, when filtered and mixed with HCl sufficient to produce the reaction with Congo paper, can not digest discs of egg-albumen.

Milk taken by achylic patients can be drawn out twenty to thirty

minutes afterward perfectly unchanged, or, rather, uncoagulated. The secretion of pepsin and rennin is, therefore, absent. By proper tests it can also be found that pepsinogen and renninzymogen are also wanting. Lubarsch and Martius assert that the isolated loss of HCl, without loss of secretion of pepsin and rennin, does not exist; and for these cases of loss of gastric secretion (not the HCl simply, but all the constituents of gastric juice), the terms anacidity, inacidity, and achlorhydria are not as expressive and logical as the designation "achylia gastrica." In the progressive destruction of the mucosa accompanying carcinoma and gastritis there are stages in which HCl is totally wanting, and yet, by proper methods, secretion of ferments, or of the proenzymes, can be detected. All other cases of loss of secretion not due to carcinoma or atrophic gastritis may logically be classed as achylia. A further pronounced sign of achylia is the abnormally small quantity of gastric contents found one hour after the test-breakfast. Biedert ("Diätetik u. Kochbuch," etc., 1895), who suffers from this affection himself, found that his stomach was very rapidly emptied, so that he had to draw the contents within forty-five minutes if he wished to obtain any at all.

Julius Miller (*loc. cit.*, *Archiv f. Verdauungskrank.*, Bd. 1, p. 233) found that strong solutions of sodium chlorid are very much diluted when they are brought into the human stomach. It is further known that strong solutions of common salt, when brought into the stomach, arrest HCl secretion. The tendency to dilute solutions that are put into the stomach is so persistent that it continues even after the concentration of these solutions has inhibited the HCl. It is not known whether this dilution of the stomach contents is caused by absorption of solid substances or by the secretion of water into the stomach itself. In achylia gastrica, however, the stomach differs very much in this respect from the normal organ, since it has then lost its power of diluting the gastric contents.

The fact that concentrated solutions of sodium chlorid inhibit the secretion of HCl has been made available in the treatment of hyperacidity. From these facts it is very probable that, in achylia, we are dealing not only with loss of the characteristic secretion, the gastric juice with its HCl and ferments, but also that there seems to be no secretion of any kind issuing from the mucosa. The diluting secretion of the stomach is, under normal conditions, not exclusively made up of the normal gastric juice,

and we are here confronted with a physiological function of a very complicated character, concerning which very little of a positive nature is known. There is a general consensus of opinion, which we can confirm on the basis of large clinical material, that, in achylia, there is an exceptionally great vulnerability of the mucosa. It is a frequent experience with achylic patients to find that particles of the mucosa showing slight hemorrhages are unintentionally scraped or torn off during the drawing of test-meals.

Lubarsch (*loc. cit.*), Einhorn (*loc. cit.*), Biedert (*loc. cit.*), Cohnheim (*Archiv f. Verdauungskrank.*, Bd. 1, p. 274), Jaworski (*Münch. med. Wochenschr.*, 1887, Nr. 7 und 8), have observed this phenomenon, and the first-mentioned author asserts that the vulnerability of the mucosa in achylia is as great as in carcinoma. We have devised a stomach-tube with a lower extremity of somewhat harder rubber than the remainder of the tube. It is beveled off like a chisel, and its edge is rather sharp. By means of this tube we effect the chiseling off of small portions of the gastric mucosa, which are afterward washed out for diagnostic purposes. In achylia it is almost impossible to avoid the scraping off of portions of the superficial mucous membrane, no matter what shaped tube is used, and if it is desired to avoid scraping the mucosa at all it is safer to use a tube which is entirely closed at its lower end and has but one velvet eye-opening at the side (Tiemann & Co., New York). This is an entirely different procedure from the accidental sucking off of rather large pieces of mucosa by the stomach-tube. Scraping off of minute particles is a harmless procedure, but the tearing of larger pieces by suction may be followed by extensive hemorrhages.

Total loss of gastric secretion, even when a consequence of a fully-developed atrophy of the mucosa (anadenia), can not cause anemia or cachexia *per se*. Those cases in which anemia has been observed in connection with achylia were most probably complicated by a mechanical insufficiency of the stomach, or by other diseases; thus, in some cases, syphilis or tuberculosis, and extension of the atrophic process to the mucosa of the intestine, have complicated the gastric derangement.

The credit of having first pointed out the association of gastric atrophy with anemia is usually attributed to S. Fenwick (lecture on "Atrophy of the Stomach," *The Lancet*, July 7, 1877; also "On Atrophy of the Stomach and Certain Nervous Affections of the

Digestive Organs," London, 1880, J. and A. Churchill). Both Einhorn (*loc. cit.*, p. 321) and Martius (*loc. cit.*, p. 16) assert that Fenwick's report is the pioneer observation on this subject. As a matter of fact, it was our countryman, Austin Flint, who first called attention to the relation between anemia and atrophy of the gastric gland (Austin Flint, *The American Medical Times*, 1860). He expressed the opinion that some cases of profound anemia are dependent upon atrophy of the glands of the stomach. (The further contributions of Flint to this subject are to be found in the *New York Med. Jour.* for March, 1871, and in his "Principles and Practice of Medicine," p. 477, Philadelphia, 1881.) The priority of Flint's publications have been emphasized by Professor William H. Welch ("A System of Medicine by American Authors," vol. II, p. 616). Since Flint's publications, cases have been reported by Quincke, Brabazon, Nothnagel, Rosenheim, and G. Meyer. The purely American contributions to this subject are very valuable. They have been made by Henry and Osler ("Atrophy of the Stomach, with Clinical Features of Progressive Pernicious Anemia," *Amer. Jour. Med. Sciences*, April, 1887); F. P. Kinnicutt ("Atrophy of the Gastric Tubules; Its Relation to Pernicious Anemia," *Amer. Jour. Med. Sciences*, vol. XCIV, p. 419, 1887); Allen Jones ("Gastric Anacidity," *New York Med. Jour.*, p. 573, May, 1893); D. D. Stewart (*Amer. Jour. Med. Sciences*, Nov., 1895); Einhorn (*Med. Record*, June 11, 1892); also in Boas' *Archives of Digestive Diseases*, volume I, p. 158. Although the anemia which supervenes in these cases of achylia can not be directly ascribed to the gastric atrophy, and too much importance was attributed by Flint and others to the state of the gastric mucosa, the reports of this author are, nevertheless, very valuable, because the secondary states, which we have mentioned as really causing the anemia and cachexia, are most probably brought about by, and owe their origin to, the primary degenerative changes in the gastric mucosa which Flint and Osler have described.

In the first part of this work (pp. 129 to 135) we have reported examinations of fragments of mucosa derived from 12 cases of anacidity or subacidity; of these, ten were cases of typical achylia gastrica. In these 12 cases, proliferation of glands, with marked round-cell infiltration, was found once. The fragment was, apparently normal in two cases, and of the ten cases of typical achylia, granular gastritis and atrophy of the mucosa could be established

in nine. In making the diagnosis of simple achylia gastrica, we excluded all those cases of permanent loss of secretion evidently due to carcinoma or pronounced chronic atrophic gastritis. In fact, before making these detailed examinations, we supposed it was possible that this form of achylia existed simply as a neurosis, because all of the ten cases which we described occurred in neuropathic patients. We had also inclined to Einhorn's view, that some forms of achylia might be of purely nervous origin. We have since then examined a number of new cases in addition to those reported, making in all 14. In none was the mucosa found perfectly normal. It seems improbable that a permanent cessation of a normal function could be caused by a neurasthenic condition. This explanation of achylia would be justifiable only in case we could demonstrate that in this affection the gastric mucosa was perfectly normal. To our knowledge, there is no case of well-authenticated achylia on record in which a cure or an improvement in the neurasthenia was reported to have cured or improved the secretory defect. The question has also suggested itself, whether achylia gastrica could not be the cause of the neurasthenia. Nor on this point are there any authenticated observations. Experience has taught, however, that neurasthenic disturbances disappear in these cases with an improvement of the general condition, while the achylia continues, which would not be the case were the latter the cause of the neurasthenia. The loss of function in this disease is not a relative or transient one, but it is absolute and permanent. Biedert (*loc. cit.*, p. 173) gives it as his opinion that the persistent loss of HCl and ferments gives the impression of a lasting defect, not of a variable increasing or decreasing inhibition. The absence of the gastric secretion is the same, whether the patients are very much run down and emaciated and subject to much suffering or whether they are in a state of good health. The supposition of Biedert, that there may be a great many who possess this defect and are unaware of it, has been verified by a number of observations among the students at our clinic. During our studies on the question, whether the normal healthy stomach contained digestive juice in the fasting condition, we discovered an athletic, robust student who had no HCl whatever in his stomach, whether fasting or otherwise. The total acidity, taken after test-meals on six different occasions, varied between one and four ; as these analyses were made shortly before the examinations

for the degree of M.D., we did not inform the candidate of the physiological defect in his stomach, fearing that it might cause him some mental annoyance, and for all that we know he may still be unaware of his achylia and continue in vigorous health. Our results concerning the condition of the gastric mucous membrane are in accordance with those of Cohnheim (*loc. cit.*), Einhorn (*N. Y. Med. Record*, June 27, 1896), Hayem (*Allgem. Wiener med. Zeitung*, 1894, Nr. 2–17), and have recently been supported by Martius and Lubarsch ("Achylia Gastrica," pp. 112 to 170). The results of the very exact investigations of these last authors make it probable that a more or less pronounced granular gastritis exists in the majority of cases of achylia.

The anatomical changes, however, are not, in all cases, sufficiently advanced to explain the permanent loss of function. Excepting the germicidal power of HCl in the gastric secretion, there is no indication at present for determining whether glandular gastritis is the cause or result of achylia. It is intelligible that a weak gastric parenchyma should be less resistant to exterior detrimental influences—such as bacterial invasion—than a robust gastric tissue.

It is evident from what has been said in the etiology of the various diseases of the stomach that the organ is exposed to many external aggressions of a thermic, chemical, mechanical, and bacterial nature, and it is a matter of astonishment what intense maltreatment a healthy stomach will endure without reacting pathologically. It is, therefore, conceivable that the anatomical loss of the glandular apparatus will render those individuals afflicted with primary simple achylia more susceptible to bacterial invasion. Most observers agree that the increased vulnerability of the mucosa goes hand in hand with the loss of secretion; this lessens the power of resistance, and eventually induces a state of chronic granular gastritis, effected by causes which a healthy stomach would resist without any change.

Symptoms.—The disturbances of function may long remain latent. Persons with achylia may have no subjective or objective disturbances of any kind; but sooner or later dyspeptic complaints arise. The subjective sensations are not characteristic, but are essentially those of nervous dyspepsia, accompanied by eructation, fullness, and pressure after eating, gradually leading to attacks of severe

gastralgia. The symptomatology, as based upon the complaints of the patient, is most accentuated in neurasthenics. In persons with a perfectly sound nervous system achylia may exist, and the individual may be unaware of it; this is proven by the case of the medical student reported above. Oppler (*Deutsche med. Wochenschr.*, 1896, Nr. 32, S. 511) has reported a number of cases which make it probable that loss of gastric secretion predisposes to diarrhea and intestinal catarrhs, which are not benefited until the achylia is discovered, when rational treatment effects improvement. The personal description which Professor Biedert (*loc. cit.*) gives of his own case is a weighty argument pointing to the fact that achylic patients are very much predisposed to diarrhea. Among the achylic patients which we have studied (14 in all), we observed attacks of diarrhea in five. So far as we could determine, the colon and the duodenum were in a normal condition. We also studied the state of the duodenum by our method of duodenal intubation, showing the pancreatic and hepatic secretions to be normal. This makes it probable that these diarrheas are possibly not due to an extension of the anatomical changes in the stomach to the intestine, but to fermentative processes, developed in the absence of HCl secretion. These diarrheas confirm Bunge's view of the antiseptic effect of the HCl secretion.

Martius' conclusions (*loc. cit.*, p. 101) are the following: Achylia gastrica is due to two conditions: (1) A primary secretory debility of the stomach, constituting simple achylia gastrica; (2) atrophy of the gastric mucosa (anadenia), which is secondary achylia gastrica. The primary achylia gastrica is either congenital or developed on the basis of a very early predisposition. It is associated with inherited debility of the nervous system, and prevails among so-called neuropathic patients.

Primary secretory debility of the stomach is an individual peculiarity, which may remain latent for years, and without demonstrable detriment to the general organism. This is particularly the case when the motor function is well preserved, and the motor, secretory, and resorptive functions of the intestine continue normal.

The mucosa, which is devoid of secretion, exhibits a diminished vital resistance to all external detrimental influences. This explains the fact that anatomical alterations of varying intensity are rarely

absent in simple achylia gastrica. The structural changes bear no proportionate relation to the absolute gravity of the loss of function.

It is, therefore, probable that there are forms of so-called atrophy of the gastric mucosa (the primary non-carcinomatous anadenia) which develop preferably on the basis of congenital secretory weakness of the stomach.

Accordingly, there are gradual transitions, clinically and anatomically, from congenital simple achylia with but immaterial alterations of the mucosa to achylia with chronic granular gastritis eventuating in complete atrophy of the secretory mucosa.

The grave results for the total organism (progressive anemia, malnutrition, etc.) which have been ascribed to the latter type do not in reality develop until the mucous membrane of the intestine is extensively involved by the atrophy.

Pathological Histology.—The investigations made by the authors quoted in the literature at the end of this chapter show in general a marked increase in the interstitial connective tissue. The surface epithelium contains many goblet cells. The vestibules to the glandular alveoli are very tortuous, and so dilated that they resemble minute cysts, filled with homogeneous, slightly glandular masses, that stain with acid anilin. The epithelial cells lining the vestibules present marked variations in structure and staining qualities. Those most prominent are: (1) Ordinary long cylindrical epithelial cells. (2) Somewhat shorter cylindrical cells with dark protoplasm and dark-staining nucleus, without an upper end. (3) Goblet cells. (4) Cells as in type 2, but with a very dark protoplasm (Stöhr cells). (5) Cells with a marked fuchsinophilic granulation. In some vestibules, only cells answering to the description of type 2 are found, and in them an abundance of mitotic figures. In other vestibules we find goblet cells in addition to these. There are very few vestibules which contain normal surface epithelium.

Among the other characteristics that were found in freshly hardened stomachs of achylic patients are: (1) Immigration and permeation of leukocytes. (2) The occurrence of mitoses in the surface epithelium and in that lining the vestibular alveoli. (3) Occurrence of acidophilic leukocytes. (4) Frequency of goblet cells. (5) Occurrence of so-called Stöhr's and Nussbaum's cells. (6) Occurrence of hyaline spheres.

Referring to No. 1 of the above observations, it should be stated

that Sachs ("Zur Kenntniss d. Magendrüsen b. krankhaften Zuständen," Breslau, 1886) has found an abundance of lymph-cells migrating through the surface epithelium and glandular substance. The pyloric region seems to be more invaded than any other part of the stomach. Stintzing considers the immigration of leukocytes in the normal stomach a very rare occurrence. Permeation of the gastric mucosa with leukocytes at the height of digestion is a normal occurrence, and has been frequently observed in animals. The difference in the achylic stomach, with regard to the permeation of leukocytes, is simply one of degree. Lubarsch found that the glandular lumina were actually packed full with leukocytes, and that they were acidophilic; this property has not been found in the leukocytes of the normal stomach. It is more than probable that the invasion of the mucosa with acidophilic leukocytes to such a degree as Lubarsch describes is pathological.

Concerning No. 2, the presence of mitoses in the epithelia of the normal stomach is denied by Sachs (*loc. cit.*) and Oppel ("Lehrbuch der vergleich. mikroskop. Anatomie d. Wirbelthiere," Bd. 1: Magen), and the occurrence of karyokinetic figures in the chief and border cells is extremely rare. For a closer study of the character and significance of the mitotic processes we refer to the literature given in Dock's article under the diagnosis of gastric carcinoma (p. 527). The hyaline spheres which Lubarsch describes are composed of cell granules that have become confluent and enlarged, but are still contained within the body of the original cell. These hyaline spheres are considered pathognomonic for atrophic processes in the gastric mucosa.

The histological changes found by various authors in achylia, and which we have been enabled to confirm in cases which we had opportunity to examine at autopsies shortly after death, indicate the proliferation of the interstitial connective tissue, the occurrence of acidophilic migrating cells; and, in addition, the disappearance of the specific glandular elements and cell proliferation, emanating from the vestibules of the glands; also transformation of the gastric mucosa into intestinal mucosa. The process eventuates in complete atrophy of the mucosa. Einhorn has reported a case of achylia in which a bit of gastric mucosa was found in the wash-water, which under the microscope appeared normal. We obtained normal mucosa from two cases of achylia, when strips were cut from achylic stomachs running from

the esophagus along the greater curvature to the duodenum; on serial sections made at intervals of one inch apart, small districts of microscopically normal mucosa were found, particularly near the cardia, while most other portions of the stomach showed distinct atrophic changes, with profuse immigration of leukocytes, and proliferation of the interstitial connective tissues. A small bit of mucosa accidentally found in the wash-water does not indicate the state of the entire stomach. When such normal fragments are found in achylia, it is still probable that portions of the stomach may be diseased. We do not, therefore, consider the evidence satisfactory that achylia may exist with a perfectly normal gastric histology.

In the great majority of cases of achylia, a progressive atrophic gastritis may be found to exist. There may be periods in the history of achylia when this condition exists without any apparent alteration in the gastric mucous membrane, and the fact that most patients do not consult the physician until the process has developed to a very advanced state, may explain the observation that the occurrence of achylia with perfectly normal stomachs is thus far supported by very few reliable microscopic examinations of gastric tissue fragments. All achylic patients give a history of years of gastric disturbances when they first present themselves for treatment, the anamnesis thus making it probable that the gastric changes must have progressed very far. In the case of the healthy medical student in whom we found achylia on six different examinations, we did not succeed in obtaining a piece of the gastric mucosa. In these cases frequent examinations for fragments of mucosa are necessary to decide the relation between the histological alteration and the clinical history. These examinations should be made at frequent and regular intervals, and in case of autopsies on achylic patients the stomach should be previously preserved by pouring in alcohol or Zenker's fluid within a half hour after death, so as to prevent autodigestion. What relation exists between the atrophic process of the intestines and that of the stomach is unknown. It may be a direct continuation of the progressive gastritis, since it is very probable that the same detrimental agencies that cause the disease of the stomach give rise also to the intestinal atrophy. One might assume also that excessive demands are made upon the digestive power of the intestines in the absence of the preparatory digestive function of the stomach. Again, it is probable that bacterial fermentations occur to a much greater degree when the

disinfecting power of the HCl is lost. In two autopsies on subjects who had shown the symptoms of achylia, the author observed that the coeliac axis, and all branches arising from it, were of unusually small size. The gastric arteries were smaller than those of normal stomachs. The intestinal and mesenteric arteries were also smaller in diameter than normal. The dimensions of the hepatic and splenic arteries were smaller. The arteries of the heart appeared normal in size.

Etiology.—Aside from the probability that achylia may be either congenital or developed upon a neuropathic basis, not much is known of the causation of the progressive atrophic gastritis. It has been supposed that bacterial infection is an etiological factor in bringing about this state of the mucosa. We may conceive the bacterial invasion to have occurred in a similar manner to that pictured under the head of ulcus carcinomatosum. In one of our drawings the presence of bacilli is represented beneath the floor of the ulcer, some of them located in the muscularis. (Plate VIII and Fig. 38, p. 497.) It is not known whether this bacterial invasion is a cause or a result of these processes. Syphilis and tuberculosis may be predisposing factors.

Example of Clinical History.—Mr. L. W., thirty-two years old, reporter on a daily newspaper. Up to 1894 he was physically well and in good health, although he admits to have frequently abused his stomach by overeating and overdrinking. His mother is a highly neurasthenic woman who imagines she is afflicted with all sorts of ailments; father high-strung and arbitrary. His duties necessitate that he should be awake during the night and sleep during the day. As a consequence of this, he is compelled to take his meals at very irregular hours. He frequently does not obtain sufficient sleep, being awakened by noises in the street (trolley cars, etc.) and about the house in which he lives. He usually gets to bed about five o'clock in the morning, and, if his nerves are quiet, sleeps until eleven or twelve; then, arising, he takes his breakfast. His main meal is taken between five and six o'clock in the afternoon. At seven o'clock he must report for duty as night clerk or reporter of the Associated Press. The work he has to perform is frequently of an exciting and enervating character. During the summer of 1894, while it was very hot, he had indulged in very cold beer during the night while following up some sporting occasion, and since then has suffered from dyspepsia, nausea, eructation, etc. Sometimes, after a meal, he will be attacked with palpitation of the heart and a feeling of giddiness, which has recently been associated with sensations of precordial fear. His appetite in the summer of 1895 was very poor; the food was described as weighing down his stomach like a lump of lead. Heart palpitation so strong that he can not sleep because of the noise his heart makes. In one month of the summer of 1895 he lost 11 pounds.

Analysis of Test-meal.—The first test-meal, which was the one used by us and described on page 111, had disappeared from the stomach entirely fifty-five minutes after it had been eaten. The second test-meal was drawn fifty minutes after it had been taken; the amount was about three ounces, and it consisted of chewed particles of wheat bread entirely unchanged. Total acidity = 5; free HCl, negative; combined HCl, negative; lactic acid, trace; propeptone and peptone, absent. On test by milk digestion, rennin and renninzymogen, absent; pepsinogen, absent; very slight quantity of mucus; very slight amount of filtrate gained by pressing the drawn ingesta through a sieve. Test of motor function, by the Hemmeter method, shows a rather increased peristalsis.

Absorption (Penzoldt and Faber's method) is abnormally delayed. Examination of a fragment of mucosa shows irregular dilatations of the peptic gland-ducts; there is some increase of the interglandular connective tissue, which is infiltrated with tremendous numbers of leukocytes, which have also pervaded the epithelial cells of the vestibules. Here and there the entire lumen of the gland-duct is packed full, and apparently pushed apart with an enormous invasion of lymphoid cells. The characteristic spindle-shaped connective-tissue cells are present, but unless carefully sought for they escape detection, on account of a copious, round-cell infiltration, and of the invasion of leukocytes, to which reference has been made. Many eosinphilic cells present. In the epithelia, numerous chromosomes and cells containing more than one nucleus. The neuclei in these cells are in various stages of indirect division, showing atypical figures.

Anatomical diagnosis, chronic granular gastritis. This patient improved very much after a vacation of six weeks in the summer, which brought him the necessary sleep at night and rest. Remedies to restore HCl secretion have been tried persistently for one year and six months without effect. He repeatedly suffered in the hot season of the year from attacks of diarrhea, which were easily controlled by administering HCl and subnitrate of bismuth, together with a proper diet. The patient has relapses whenever he indulges in overwork, with loss of sleep. Since 1895, 23 test-meal analyses have been made, not one showing a trace of HCl or ferments.

Treatment.—We are in the habit of prescribing dilute HCl for all these cases whenever the acid agrees well. As considerable HCl is needed to effect any appreciable digestive action and to exert a disinfecting influence, we give 20 drops of the officinal dilute HCl every half hour after meals until 60 drops have been taken. The acid must be largely diluted and taken through a glass tube. Our experience, which is based upon a large number of cases of this sort, has convinced us that the acid is not only well tolerated, but is almost indispensable to the patient. Although it may be argued that achylic patients sometimes get along without any treatment whatever, simply because they exhibit no symptoms, nevertheless when they do apply for treatment they generally

present a complexity of symptoms, which are much benefited by carefully selected but nutritious diet, sometimes rest in bed, strychnin, and HCl. When the appetite is absolutely lost, it may be restored by washing out the stomach with bitter tonics, such as gentian and quassia. In neurasthenic patients, strychnin sulphate improves not only the local gastric symptoms, but also the symptoms of the general neurasthenia. A number of gastric patients of this kind refuse to eat, because they fear that distress will be caused by the food. In such cases it may become necessary to place the patient in a well-managed institution for the dietetic treatment of digestive diseases. They must gradually be convinced that food that is ingested with appetite can do no harm. When the motility is interfered with and symptoms of dilatation are manifest, gastric lavage is indispensable. Concerning the use of pepsin and pancreatin see pp. 331, 332.

Diet.—The achylic patient is an individual who has an internal infirmity, due either to a congenital defect, or to an acquired irregularity in the gastric structure. Whatever may be the condition and the cause, we are dealing with individuals who are essentially weak and debilitated. We have found it expedient not to be too exacting with diet orders. In fact, we make it a rule never to give a standing diet order to an achylic patient without carefully inquiring as to the food which he knows from experience agrees best with him. The stomach is a protective and selective organ, preparing the food for the intestines. By its selective property, when the motility is in a normal condition, it permits only the semisolid and liquid masses to pass first, while the more consistent masses are retained to be further softened and disintegrated. We have shown, in the preceding pages, that the stomach of the achylic patient has lost the power to dilute its contents by a secretion from its walls. This is one of the main reasons why we permit the ingestion of liquids during meals, and of largely diluted HCl after meals. For the same reason all foods should be well chewed, or preferably finely divided during the process of cooking, for the main object of all treatment must be to preserve the peristalsis, and to insure a healthy state of the mucosa. Therefore, the food should generally be taken in form of gruels, pastes, or in any semisolid, easily swallowed state. The meat should be very soft,—scraped or run through the meat-chopper. Fish, sweetbread, calf's brain, and soft-boiled eggs are, as a rule, of such soft

consistency that they need no further preparation. Our experience is that the more food is ingested and well tolerated, the better for the patient in these cases. We will give no outline here of detailed diet list, but refer the reader to the diet order for anacidity and Penzoldt graded diet order given in the chapter on Dietetics. In many cases, physical and mental rest, hygienic surroundings, and a nourishing diet will be all that is needed for insuring comparative well being of the patients. Where the motor power becomes defective, the treatment will be that outlined in the chapter on Motor Insufficiency.

A remedy little known—but a very valuable adjuvant in treatment for lack of dietetic ferment—is the juice of fresh pineapple. This has decided proteolytic power, and, besides, is a pleasant, easily-procured remedy.

There is no treatment that is universally applicable to all cases of achylia. The ability of the practitioner in discerning the special indications for each individual case is put to the test severely in the therapeutic management of this disease. Sometimes the treatment will be that of chronic gastritis, sometimes that of nervous dyspepsia. Excessive strictness in dietetic regulation has, in the author's opinion, occasionally developed gastric "hypochondriacs." It is more advisable to train up, or, as Broadbent says, "level up," the gastric digestion to a higher plane. We make it a rule to show these gastrophobic patients the contents of their stomachs at a proper time after full meals, to convince them that they can digest thoroughly, for, as a rule, every vestige of food will have passed out of the organ within one and one-half hours.

The literature on achylia gastrica will be found compiled in the article by Martius and Lubarsch, published by Franz Deuticke, Leipzig, 1897.

CHAPTER XIII.

NERVOUS DYSPEPSIA (Leube).—NEURASTHENIA GASTRICA (Ewald).

The original definition which Leube gave of this affection characterized it as a neurosis of sensibility, without any well-defined and constant objective disturbances of digestion, but exhibiting a large variety of subjective symptoms connected with the digestive act and occurring independently of any demonstrable changes in the stomach.

In his first paper, Leube ("Ueber nervöse Dyspepsie," *Deutsch. Arch. f. klin. Med.*, Bd. XXIII, 1879) emphasized that the gastric digestion may be perfectly normal so far as the chemistry and motility are concerned, and that he has used the term "dyspepsia," or difficult digestion, because this act is accompanied by manifold complaints that are traceable to an abnormal excitability of the sensory gastric nerves. Since then Leube has expanded the conception of nervous dyspepsia to the effect that it includes anomalies of secretion and motility.

R. Geigel and Abend (pupils of Leube) later on demonstrated that the secretion of HCl may be extremely variable in nervous dyspepsia, and that we may have a normal acidity, or euchlorhydria, subacidity, anacidity or achylia, or hyperacidity. Accordingly, the important symptoms of the trouble, viz., the annoying gastric distress, can not be traced to fermentations of the gastric contents with sub- or inacidity, or to the products of this decomposition, nor to irritation of the gastric nerves in superacidity. It is natural that the definitions and conceptions of various authors concerning a disease that is so vague and indefinite in its clinical history and pathology, should differ greatly. Leube distinguishes more recently between two kinds of nervous dyspepsias: (*a*) Nervous dyspeptic symptoms in which the nervous channels are sympathetically involved by anatomical changes in the stomach, and altered chemistry of digestion caused by these changes. (*b*) Nervous dyspepsia with an apparently normal

anatomical state of the organ. Boas distinguishes a third form of nervous dyspepsia, which originates reflexly from other organs; for instance, the kidneys, uterus, ovaries, male genito-urinary apparatus, and intestine. Constitutional diseases, such as tuberculosis, syphilis, diabetes mellitus, anemia, uric acid diathesis, may form the basis of this complexity of symptoms. The disease may occur in an idiopathic form, independently of any demonstrable gastric changes, or in a secondary form consequent upon neurasthenia, hysteria, and the other pathological states referred to. Whatever the underlying basis or etiology of the disease, the ultimate symptoms can be described to a functional sensory neurosis and overexcitability of the gastric nerves, which may become so acute that they react in a pathological manner upon the influence of normal digestive stimulation.

Pathology.—Jürgens has discovered total degeneration of the plexus of Meissner and Auerbach in 41 cases of nervous dyspepsia. In one of the cases in which the sensory disturbances were predominant, and the intestinal functions involved also, this author found a distinct degeneration of the muscularis of the stomach and intestine. Further exact pathological and histological investigation will very probably restrict the number of cases at present classed under nervous dyspepsia. The conceptions of various authors concerning the nature of nervous dyspepsia vary considerably. Leube, as is well known, states that nervous dyspepsia is of central origin, while Stiller applies the name to all digestive disturbances that are transmitted to the stomach through the central or the sympathetic nervous system. Stiller attributes greater importance than Leube to disturbances of the secretory function, which he could demonstrate in a majority of his cases. We interpret the disease as a mixed neurosis, in which the motor secretory and sensory nerve apparatus are affected contemporaneously or alternately; an anatomical substratum is present in one-half of the cases, but it is not of a constant type.

Etiology.—Neurasthenia gastrica has been observed after intense emotional excitement, exhaustive mental work, alcoholic and sexual excesses, after abuse of tobacco, and associated with pulmonary phthisis, nephritis, and malaria. The sensibility of the normal gastric mucosa is very slight, and the digestive irritation causes no distinct sensation in the normal individual, but when a healthy person transgresses the customary amount of food, unpleas-

ant sensations of pressure, distention, fullness, eructation, and nausea will ensue, indicating that the organ has been overloaded. These sensations cease when a part of the chyme has passed out into the intestine.

Narcotic substances, such as very strong coffee, tea, or tobacco may relieve or remove these symptoms, showing that they are of a purely nervous character. If the excitability of the sensory nerves is for any reason increased, then the normal digestive irritation brings about such gastric difficulty. With an intensely excitable nervous apparatus in the stomach, such symptoms as we have described may come on even when the organ is empty. It is characteristic of nervous dyspepsia that gastric distress is perceived only after meals, and is absent when the stomach is empty. The nervous dyspepsia associated with malaria should induce the physician to examine the blood of the patient for the malarial parasite. The symptoms in these cases generally abate under the influence of quinin.

As a secondary neurosis, neurasthenia gastrica is generally the result of general neurasthenia or hysteria. Grave anatomical alterations of the brain and spinal cord, which frequently bring on other gastric neuroses, are, so far as we know, not reported to have any causal relation to nervous dyspepsia.

Reflexly, the disease may result from irritation arising from the genito-urinary organs in both sexes, from menstrual and puerperal disturbances. The dyspepsia during the puerperal period has been attributed to traction or compression of the sympathetic. In a portion of the cases it is impossible to attribute any cause. It is a disease which prevails among the male sex.

Symptomatology.—The clinical picture of neurasthenia gastrica is extremely variable. It is, therefore, impossible to give a well-defined typical representation of the disease that can be applicable to the majority of the cases. We will, therefore, simply designate the most important and frequent symptoms. It is characteristic of nervous dyspepsia that the gastric distress is directly dependent upon the ingestion of food—that it occurs only after meals, and not on an empty stomach.

Furthermore, it is characteristic that the quality and the quantity of the food and dietetic errors exert no influence upon dyspepsia. At times the most indigestible food causes no difficulty whatever, and at other times the most digestible food brings on distress.

The sensations of the patient are very much under the influence of the emotional state. The dyspeptic symptoms are: Unpleasant sensations, pressure, fullness, distention of the stomach, occurring shortly after meals. After the patients have slept well, they are in a cheerful state of mind in the morning, but immediately after breakfast they are tormented by manifold sensations in the stomach. The suffering is most severe when the neurasthenia gastrica is accompanied by hyperacidity. In this case it increases during the second period of digestion as the acidity becomes greater. Such types are relieved by the administration of alkalies, which, however, are useless with achylia. The epigastric region is not very sensitive, nor are there any characteristic pain-points, so far as we could determine.

Leven ("Estomac et Carvau," Paris, 1884) attributes great importance to the appearance of these so-called painful spots, which are supposed to be due to an irritation of the solar plexus. Burkhart ("Pathol. der Neurasthenia Gastrica," Bonn, 1882), Fleischer, Ewald, Bouveret, and Richter do not attribute much importance to this symptom. In some cases very peculiar sensations are described by these patients. Some have a crawling feeling in the stomach, as if some live animal were moving about in it. Some have a sensation of tickling, others describe it as a beating, burning, or sticking sensation. A most unusual sensation is that described as a restless, wavy, or undulating motion. Persistent eructation is a very frequent and annoying symptom. The eructations occur in an explosive manner, and without any regard for the surroundings. If there is hyperacidity, these eructations are accompanied by severe pyrosis. Emesis is rare, but when it does occur the character and consistency of the vomit depends upon the composition of the gastric juice. With normal acidity or hyperacidity it has a very sour taste and is void of proteid food when the acid is present in excess. With subacidity or inacidity, it contains much undigested meat and eggs and but little carbohydrate food. Although inacidity may be present, the gastric contents do not decompose because there is no stagnation. The appetite is variable. There may be a normal appetite, bulimia, or anorexia. As a rule, thirst is increased. The behavior of the sensory gastric nerves is capricious. When the patient is in a cheerful, pleasant humor, or occupied with a congenial, interesting piece of work, he will digest articles of diet which will cause very

great distress when he is emotionally depressed. Excessive mental or bodily work, cares and worries concerning the vocation, disappointments in business enterprises, grief, etc., all cause a condition of excitability in which digestion is much impaired.

Secretory Function.—In neurasthenia gastrica there may be a normal secretion, hyperacidity, or inacidity. When inacidity exists the ferments can still be demonstrated in the gastric contents. This important fact will serve to distinguish this type of nervous dyspepsia from typical achylia gastrica.

Motor Function.—The peristalsis of the stomach is undisturbed in neurasthenia gastrica.

Intestinal Disturbances.—The most constant symptom is obstinate constipation. Very frequently there are rumbling noises in the intestines and extensive flatulence.

Nervous Symptoms.—These consist of pain and pressure in the head, giddiness, tinnitus aurium, flashes before the eyes, rapid pulse, exhaustion, cool extremities, attacks of fainting, palpitations of the heart, dyspnea. It is very probable that all these symptoms are connected with the deranged intestinal digestion, and that they are due to the absorption of toxic products formed during the putrefaction of food in the intestines. C. A. Herter and E. E. Smith (*N. Y. Med. Jour.*, June 22 and 29, July 6, 13, and 20, 1895) have published clinical histories and detailed analyses showing the relations of psychic disturbances, melancholia, etc., to the toxicity of the urine. There is no doubt that the production of these symptoms, particularly frontal headache, beating in the head, congestions, pulsations of the large arteries, globus hystericus, melancholia, and insomnia, is in some way related to excessive intestinal putrefaction. When the nervous dyspepsia is comparatively recent, the symptoms are limited to the gastro-intestinal tract, but when the disease is of long standing, the nervous symptoms may submerge the digestive, and it may be difficult to decide whether the latter or the former constitute the primary derangement.

Prognosis.—Inasmuch as the course of the disease is a chronic one, it may, in severe cases, by continued and progressive loss of strength and emaciation, prove fatal. In those cases in which a cure has been effected relapses may occur, so that the prognosis should be guarded. Sometimes, when the fundamental disease is remediable, for instance, in genito-urinary disturbances, malaria, etc., or when the cause, such as sexual excesses, abuse of alcohol

and tobacco, bodily and mental overexertion, can be removed, the resulting nervous dyspepsia may be permanently cured.

Diagnosis.—As a general rule, it will be found that the nervous dyspepsia is connected with some organic disease of one of the digestive organs. We refer to the various anomalies of position of the intra-abdominal organs that are described in the chapter on Gastroptosis and Enteroptosis. Frequently, dislocated kidneys, small tumors, herniæ of the median linea alba, morbid changes in the male or female sexual organs, and organic diseases of the stomach and intestines, will be found to exist. There will be no connection between the quality and quantity of the food and the digestive difficulties, but sleep, emotional state, and psychic condition will be influential factors. The complaints of the patient are frequently described in exaggerated language. One of our patients, who is the owner of a brickyard, describes his feelings "as similar to the rolling of a ton of bricks in his belly"; another compares his sensation to being "stabbed with a red-hot knife"; still another describes her abdomen as being "distended to bursting, like a balloon," or, at other times, as feeling compressed as though it were in a vise. At other times these same patients, without recognizable reason, will make no complaints at all, will be very happy and cheerful, and digest well. These variations in the functional powers of digestion are very characteristic of nervous dyspepsia. Leube has called our attention to the emptiness of the stomach seven hours after a rather heavy test-meal.

Heterochylia (from ἕτερος, meaning "other" or "different," and χυλός, meaning "juice" or "secretion").—This term is suggested by the author to denote a rapidly-alternating state of secretion, occurring chiefly in nervous dyspepsia. In making a large number of analyses in these cases, Dr. E. L. Whitney and the author have observed within one week that euchlorhydria, hyperchlorhydria, and inacidity will be found after the same test-meals. In the discussion of a paper read before the American Medical Association, in Philadelphia, June 4, 1897, a colleague, Dr. E., of Brooklyn, stated that in his own case he had observed hyperacidity and inacidity on the same day, after he had taken the identical meals and drawn a sample within one hour after they had been ingested. Dr. E., who is an able chemist, made quantitative analyses of his gastric juice on many occasions, and, so far as he can tell, these variations in the secretion are

independent of his emotional state. In a number of the cases examined by Dr. Whitney and the author they found hyperacidity with rapid digestion of proteids, and defective carbohydrate digestion with symptoms of pyrosis and eructation that were relieved by alkalies. Two days afterward, the author examined the same cases, to find that two of them showed no reaction with Congo paper and a pronounced HCl deficit. There was no pyrosis nor eructation, but the patients complained of a sense of fullness and weight in the stomach, together with anorexia, which symptoms were relieved by two doses of dilute HCl (30 drops per dose). At the end of this week we had occasion to examine the same cases again, and found the acidity normal. For want of a better expression we have designated these rapidly alternating states of secretion by the name *heterochylia*. When the acid is in excess, the proteids are absent from the test-meals, and rice and bread almost undigested. When the acid is absent, one may frequently find a defective proteid digestion but a rapid carbohydrate digestion. We have no explanation to offer for these cases beyond those which are purely hypothetical, but it is conceivable that a closer histological study of the finer ramifications of the gastric nerves, such as has been carried out with such admirable regard for detail by Henry J. Berkley in other organs of the body, may throw some light upon this puzzling phenomenon. For instance, we may sooner or later be instructed that the oxyntic or border cells receive a different nervous supply from the chief or central cells, or that both chief and border cells are supplied by nerves of widely differing character, one set exciting the function, the other inhibiting it; *i. e.*, anabolic and catabolic secretory fibers; but all this is premature and problematical.

Differential Diagnosis.—Nervous dyspepsia with inacidity may be confounded with chronic gastritis or carcinoma, and nervous dyspepsia with hyperacidity may be confounded with ulcer, while still other forms may bear a striking resemblance to atony or myasthenia. For the separation of chronic gastritis from nervous dyspepsia, the following facts are of importance: Chronic gastritis is accompanied more frequently by vomiting; the stomach contains large quantities of mucus and a few blood streaks; we may have also stagnation of the contents. The course of chronic gastritis is more uniform and typical, and the dyspeptic symptoms are directly influenced by the quality and quantity of the ingesta. In carci-

noma, the distress is present at all times, even on an empty stomach, vomiting is frequent, and the ferments are absent when the HCl secretion is lost. In nervous dyspepsia the ferments are still present, though HCl may be absent. When symptoms of stenosis have occurred, there can be no difficulty about the diagnosis. The differentiation of nervous dyspepsia from ulcer becomes difficult only in those cases in which there has been no hematemesis. The constant dependence of gastric pain upon the food, the sharply circumscribed pain-points in the epigastric region and in the back, are unmistakable criteria. We have spoken more fully of the differential diagnosis in the sections on the various gastric diseases with which nervous dyspepsia may be confounded. It must not be overlooked, however, that nervous dyspepsia may be associated with some form of organic gastric disease.

Treatment.—The fundamental causes of disease should be hunted up, and, if possible, removed. The prospects of doing this are favorable if the cause can be found in the existence of intestinal parasites, floating kidney, malaria, and certain remediable diseases of the genito-urinary organs. In those forms of nervous dyspepsia which depend upon an undue excitation of the nervous system, due to sexual excesses, abuse of alcohol and nicotin, or excessive mental and bodily exertion, improvement can not be hoped for unless these states are remedied. Patients must be impressed with the fact that drugs and other treatment will not improve them if they persist in their bad habits. Particularly American business men, who, with admirable energy but with little regard for their own health, persist in executing work which is too severe for their mental and physical constitution, must be taught that the prime factor in successful treatment is rest, *rest*, REST! This class of cases will, in the long run, prove to be very grateful patients if this truth is emphasized, and false expectations concerning the efficacy of drugs, and washing out the stomach, etc., corrected at the beginning of the treatment. Better results can be obtained in all of these cases by a change of environment, with absolute psychic and physical quiet, removal from the cares and worries of business and household, than by the most detailed and complicated treatment.

In connection with this we must emphasize the value of rational psychic treatment of nervous dyspepsia. The physician must, in a dignified manner, attempt to merit the absolute confidence of his

patient. For this purpose we consider it important that he should show a warm, sincere interest in the suffering of his patients, even if, after a repeated and thorough examination, he should become convinced that the patient's complaints are unreal and exaggerated. It is a great comfort to these neurasthenics to listen patiently and sympathetically to their complaints, and not to ridicule or criticize them. The sufferings of the patient, psychically considered, are equally intense, whether they be real or imaginative.

Gymnastics.—The author has frequently observed marked improvement after a course of mild gymnastic training under an experienced training-master. The bicycle, moderately used, is a better means of promoting appetite and regular evacuations than drugs. In a similar way, horseback riding, rowing, fencing, etc., are to be recommended.

Climatic Treatment.—A sojourn in the mountains or at the seashore is a great help, inasmuch as it not only removes the patient from surroundings which keep up his disease, but at the same time insures rest, quiet, and, above all things, invigorating fresh air. In seeking a resort, fashionable places and those thronged with society should be avoided. The greater part of the day should be spent in the open air—if possible, in taking extensive walks. This will favor good sleep during the night. If there be persistent hyperacidity with constipation, the patient will be benefited by a sojourn at Bedford Springs, Pa.

Massage.—There is no doubt that massage improves the nutrition of the muscles and nerves, and favors a vigorous circulation, metabolism, and regular evacuation. Massage should not be permitted from inexperienced quacks. Nine-tenths of the persons claiming to be masseurs at the present time are charlatans. To be effective, the massage must be studied by the physician who has the case in hand, and though he can not execute it himself he should, at least, supervise it.

Hydrotherapy.—Cold sponge baths, taken in the morning immediately after arising, have a bracing effect. A good method is to wrap the entire body of the patient in a sheet dipped in cold water, and while the patient himself kneads and beats the parts of his body that are accessible to him in front, another person must perform the massage of his back; after this the patient is thoroughly rubbed with a coarse Turkish towel. These cold rubs should not last longer than three minutes, after which the patient

must dress and take a walk of about one mile. The favorable effects of hydrotherapeutic methods do not become manifest until they have been applied for two to three weeks. They are then followed by improvement in the appetite and sleep. When the insomnia is persistent, we are very fond of prescribing a warm salt bath, at the temperature of the body, containing four per cent. of sodium chlorid and two per cent. of sodium carbonate. The patient is placed in this bath about half an hour before bedtime, and remains in it for about twenty minutes.

Irrigations and Douches of the Gastric Mucosa.—These are used to reduce the hyperesthesia of the gastric nerves, and for this purpose carbonated waters are preferable to still waters. The gastric tube should be used which contains numerous small lateral openings instead of a few large terminal openings. If a carbonated water can not be conveniently obtained, it can be prepared by adding citric acid or lemon juice to a one per cent. solution of sodium bicarbonate. The amount poured into the stomach should not exceed 20 ounces at a time. (See p. 287.)

Electricity.—Galvanization of the abdomen and the spinal region and general faradization are applicable in these cases. The faradic current should be applied to every muscle in the body with large, broad, felt electrodes. A good method consists in placing the feet of the patient upon a large plate electrode (cathode), while the other pole is placed on the various muscle groups of the body. It is well to allow the large electrode to remain on the epigastric region for about five minutes, while the remaining one is passed up and down over the spinal column. The intensity of the current and the duration and localization of the treatment must be varied according to the individuality of the case. According to Erb, Beard, and Rockwell, this treatment improves the appetite and sleep, reduces the psychic irritability, and creates a more favorable disposition to bodily exercise. Personally, we may, without defining the exact benefits derived from electric treatment, pronounce it to be an indispensable adjunct to the treatment of neurasthenia gastrica. Perhaps it influences the nutrition of the nervous centers, or perhaps it is nothing but systematic massage. At all events, it effects an improvement in the sufferings of this class of patients.

The Diet.—In this disease, more than in any other, the physician must see that the articles of food possess considerable variety and

are well cooked and appetizing. The behavior of the digestive functions are so grotesque that it is impossible and useless to suggest stereotyped diet lists. Experience is the best guide, and the common phrase "the proof of the pudding is the eating of it," is certainly applicable in these cases. It is very beneficial to the patient if he can take and well digest large quantities of milk; aside from its high nutritive value, milk acts upon the gastric mucosa like a soothing liquid ointment, and is a dietetic intestinal antiseptic. Sometimes when the patient is prejudiced against it, it is possible to mix it with the food surreptitiously, and our diet lists and "dietetic kitchen" give many formulæ for foods containing milk. In the selection of the remaining foods, the taste, likes, and dislikes of the patient should be consulted so far as is consistent with rational dietetics. Articles of luxury, such as good fruit, grapes, pears, figs, dates, and, if anacidity exists, fresh pineapples, should not be forbidden. If constipation is obstinate, the diet should contain a large amount of these foods, and particularly apples. Concerning the use of alcoholic beverages no definite rule can be given. On the whole, we believe that wines and beer should be avoided, unless they are needed for stimulation and to improve the appetite. Large colon enemata with pure olive oil (300 c.c. at a time) are sometimes curative in the nervous constipation, particularly the membranous colitis present in these patients. Perhaps the most effective treatment, on the whole, is that designated as the Weir Mitchell rest-cure, a combination of hydrotherapeutic, electrical, and dietetic treatment, with gymnastics, rest, massage, and as much sleep as possible. Where the state of the nutrition has been much reduced, the so-called "Mastkur," a system of fattening by highly nutritious diet and passive exercise, is, in our experience, very effective in bringing about a reduction of the symptoms and improvement in the digestive functions. This "Mastkur" is not applicable to all classes of patients; those of an irritable and restless temperament and those who have organic gastric diseases will become aggravated rather than improved by it.

Drugs.—Those that have been employed in neurasthenia gastrica are the tonics, sedatives, and hypnotics. In anacidity, the basic orexin, five grs. three times a day, has been very much lauded by Penzoldt. The fluid extract of condurango, one teaspoonful three times a day, and the bitter tonics, calumbo, gentian, quassia, in doses of one dram three times a day, are sometimes of value, though

personally we have seen no marked results follow their administration. The remedy we have most faith in is the sulphate of strychnin, $\frac{1}{30}$ of a grain three times a day. When malaria is associated with the nervous dyspepsia, quinin is the remedy "par excellence." Boas and Einhorn speak very favorably of the use of bromids; both of them employ mixtures of the ammonium and sodium bromids. While the remedies may have a temporary value, and are indispensable for producing sleep and diminishing the excessive irritability of the nervous system, they must not be used continuously. We have assured ourselves, by quantitative analyses of the toxic products of the urine, similar to the studies of Herter and Smith (*loc. cit.*), that the toxicity of the urine is increased in nervous dyspepsia as soon as the total quantity of bromids that is administered exceeds six gm. in twenty-four hours. Maximowitsch recommends the following in neurasthenia gastrica on a basis of anemia:

℞. Ferri bromati,
Chinin bihydrobromic, ... āā 4.0
Ext. et pulv. rad. rhei. q. s. u. f. pil. No. CXX. M.
SIG.—D. S. two pills three times daily.

The use of mineral spring waters is of doubtful efficacy. Where an improvement is noticed at the mineral springs, it is probably due to the hygienic surroundings, the removal from care and worry and responsibility, and the discontinuance of the detrimental habits encouraged at the home of the patient. For further consideration of the effect of mineral waters we refer to the chapter on this subject (pp. 300 to 315). If the insomnia is persistent, chloral may be unavoidable. It should, in these cases, be given by rectal enema and not by the stomach. Fifteen grains in two ounces of starch water are usually sufficient to secure rest. An effective combination consists of eight grs. of chloral hydrate and eight grs. of sulphonal. Opium and belladonna are best excluded from the treatment. Sulphonal and trional are available remedies for the insomnia, but, like the chloral, they have a deleterious influence upon the stomach, and should be preferably given per rectum. But the treatment producing the most lasting results is that which tones up and invigorates the neuromuscular apparatus and increases the will power.

AUTHOR'S SYNOPSIS OF SCHEME FOR EXAMINING STOMACH PATIENTS AT THE MARYLAND GENERAL HOSPITAL.

Medical No ... *Name.....* *Address.....* *Age...* *Color.....*

Sex..... *Social Condition.....* *Diagnosis.....* *Date.....*

HEREDITARY FACTS OF IMPORTANCE.

PREVIOUS HISTORY.—Severe constitutional diseases? First appearance of symptoms, and cause? Did they appear suddenly? Intensely? Or gradually? Continuous? Or remittent? What intervals? Occupation? Habits? Alcoholism? Tobacco? Cold? Change of climate? Mental strain? Trauma? Malaria? Did it begin with or without a chill? Fever? Yellow fever? Constipation? Diarrhea? Dysentery? Typhoid fever?

PRESENT HISTORY.—Diseases of other organs? Dyspeptic symptoms? Pressure? Local- and subjective complaints? Fullness? Pain? Distention? Restlessness? Sounds in the digestive tract? Bowel movements? Nausea? Eructation? Vomiting? Hematemesis? Appetite? Taste? Thirst?

LOCAL SUBJECTIVE SYMPTOMS.—Any difficulty or pain on deglutition? If so, its regularity? Irregularity? Intensity? Duration? Effect of food on pain? Do they occur in every position of body? Or only in certain positions? Time of onset after meals? Pain at night? On an empty stomach? Improved by eating? Exaggerated by eating? Is pain diffuse? Or circumscribed? Deglutition sounds?

ERUCTATION.—Duration? Occurring on full or empty stomach? Is gas tasteless? Odorless? Acid? Decomposed? After what foods? Presence of pyrosis, or heartburn?

NAUSEA AND VOMITING.—Occurs on full or empty stomach? Frequency? Taste of vomit? Appearance of matter? Food particles? Proteids? Starches? Mucus? Bile? Pus? Blood? Food eaten several days before? Does emesis relieve symptoms?

APPETITE AND THIRST.—Accustomed diet (let the patient state in detail what is eaten during the entire day)? Mode of life? Anorexia? Bulimia? Aversion to meat? Thirst?

BOWELS.—Constipation? Diarrhea? Undigested particles of food? Mucus? Pus? Blood and source?

RESULTS OF BLOOD EXAMINATION.

GENERAL NUTRITION.—Emaciation? Loss of weight in pounds? In what time?

PHYSICAL EXAMINATION.

INSPECTION.—Change in form of abdomen? Tumor? Gastric or intestinal peristalsis?

PALPATION.—Time of examination? Temperature? Outline of stomach? Upper border? Lower border? Presence of tumor? Movement of tumor? Was stomach full or empty? Pain on pressure? Diffuse or circumscribed? Succussion sound?

PERCUSSION.—Limits of the stomach?

DISTENTION WITH AIR OR GAS.—Limits of stomach? Results with intragastric bag? Does tumor move with distention? Made more or less distinct?

ELECTRODIAPHANY.—Limits of stomach? Tumor?

EXAMINATION OF TEST-MEALS.

Double test-meal of the Maryland General Hospital (see p. 111) at say 9 A. M.
Ewald test-meal at say 2 P. M. Contents drawn at say 3 P. M. Date.....

MICROSCOPICAL EXAMINATION.—Quantity? Color? Odor? Food particles? Froth or gas? Pus? Mucus? Bile? Blood? Fragments of tissue? Bacteria? Oppler-Boas bacilli? Sarcinæ?

CHEMICAL EXAMINATION.—Reaction? Free acid? Free HCl? Lactic acid? Amount free HCl? Combined HCl? Amount acid salts and organic acids? Total acidity? Erythrodextrin? Deficit of HCl?

PEPSIN.

Albumin digested in pure filtrate in....minutes. Albumin digested in acidified filtrate in.... minutes. Albumin digested in HCl and pepsin filtrate in....minutes.

RENNET.

Milk coagulated by rennin in....minutes. Milk coagulated by rennin-zymogen in....minutes. Rennin-zymogen active in dilution 1.

CONTENTS.—After meal previous evening at 8 P. M.

CONTENTS.—After lavage previous evening at 8 P. M.

TIME OF SALOL REACTION ...minutes.

TIME OF IODID OF POTASSIUM RESORPTION TEST....minutes.

URINE.—Amount? Urea? Reaction? Indican? Preformed sulphates? Albumin? Tube casts? Ethereal sulphates? Ratio? Sugar? Specific gravity?

TREATMENT.

Diet? Medicines? Electricity? Massage? Hydrotherapy? Lavage? Mineral spring water? Gymnastics? Orthopedic treatment? Results?

LIST OF AUTHORS.

The *List of Authors* and *Table of Contents* were compiled by Mr. James W. Kissling (*Candid. Med.*). The author herewith expresses his thanks for the intelligent way in which this work has been executed.

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Compiled by the Author's pupil, Mr. James W. Kissling (*Candid. Med.*).

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