He discards all terms of signification in naming the secondary waves, such as "outflow remainder wave" or "tidal" wave, since the terms have not met with universal recognition, and simply names them, in their succession, first secondary wave, second secondary wave, and so on. The side pressure in the aorta was always obtained by inserting the glass tube of his manometer into the carotid.²² The animals were always narcotised by morphine.

The pressure movements varied during pulsation between 100 and 130 mm. Hg., so that the pulsatorial increase of pressure amounted to not quite one-third of the minimum pressure. He found the curve reached its first summit on an average in 0.025 seconds, and, in about the same time sank to a quarter or half the whole pressure movement to rise again to the height of the first summit. This second summit is the first secondary wave, and appears 0.05 second after the commencement of the primary wave. The curve sinks again from the second summit to a point either above or under half the height of the pulse, where a further upheaval occurs, which, however, does not attain to the same height as the foregoing. The second secondary wave occurs 0.12 second after the commencement of the curve.

Since the descent of the second secondary wave is more rapid than the rest of the descending side of the curve, we can distinguish yet a third secondary wave, which often comes out as a distinct rise. Finally, before the commencement of the next pulse a fourth secondary rising is noticeable, which is quite short and low, but clearly distinguishable.

A characteristic appearance of the pressure curve is furnished by different conditions of the vascular system. If the cervical sympathetic is irritated the second summit appears to raise itself some seconds after the commencement of irritation, more and more above the first, and so changes the appearance of the pulse curve that what was the first summit is only recognisable as a bend in the upward limb of the curve; by this means the limb becomes longer and ascends less rapidly, and the angle at the curve summit becomes more obtuse. When the depressor nerve is irritated there is a different change; the curve attains its summit very quickly, although the rise is higher than normal; thereupon follows just as sudden a sinking to beneath the level of the pulse height, so that the angle of the summit is very sharp; the first secondary wave now occurs from 0.05 to 0.01 second later than normal. The other secondary waves do not change their place in so constant a manner on irritation of the sympathetic and depressor.

These experiments clearly prove that changes of the lumen of the peripheric vessels cause certain changes in the pulse form. The estimation of blood pressure in the various forms of pulse scarcely allows a doubt but that the change of the tone of the vessels can only change the pulse form by means of the blood pressure. A change of pulse form is never to be seen without simultaneous rising or falling of the blood pressure. The tension of arterial blood alters with the tone of the vessels, through increase or diminution of the obstacles which oppose themselves to the blood stream. Thus in irritation of the sympathetic the minimum pressure is raised about 10 mm. Hg.; the pulsatorial excursion is in comparison somewhat smaller. In irritation of the depressor nerve the minimum decreases 10 to 50 mm. Hg., and the pulsatorial excursion becomes greater.

Hürthle found that the height to which the primary curve reached in a given time under a low pressure could amount to more than four times as much as the curve rises to under a high pressure. It has been already said that with high blood pressure the ascending limb rises more slowly. These time variations are suggestive, and enable us to form some idea as to the varied form of the primary wave, as seen in the accompanying diagram from Hürthle.

If A B C is the primary wave under normal blood pressure, then, on rise of the blood pressure, it is converted into the less excursive and more slowly rising form A' B' C' whilst under low pressure the rapidly rising and more excursive form A'' B'' C'' is produced. The descending limb of the pulse curve falls more quickly under low pressure than under high.

By accurate time measurements Hürthle found that the

first secondary wave occurred somewhat sooner than the primary under high pressure than under low. If the first

secondary wave occurs at the time point D under normal blood pressure, then the double pointed form of the pulse form A, B, C become anacrotic; on the other hand, if the wave appears under lower pressure, it occurs later, low in the descending limb of the pulse form A'' B'' C''. With regard to the nature of the first secondary wave, Hürthle says, "If one can apply the results of the experiments made by Fick and v. Kries on human beings to the pressure pulse of the rabbit, then the first secondary rising which follows the summit must be regarded as a positive wave, reflected from the periphery of the vascular system. The change which occurs in the time appearance of this wave with the alteration of blood pressure agrees with this assumption, for the velocity of propagation of the waves in the vascular system increases under increase of pressure, and *vice versa*." Hürthle moreover repeated his experiments on the cat, with a like result.

(To be continued.)

ABSTRACTS FROM THE INGLEBY LECTURES ON THE COMMON FORMS OF DYSPEPSIA IN WOMEN.

Delivered at Mason College, 1894.

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LECTURE I.

AFTER some preliminary observations, in the course of which he said that he would devote his attention to the consideration of the nature and treatment of the disorder present in the well-known type of dyspeptic woman which constituted two-thirds of the adult female patients in our hospital outpatient medical departments, and must form a considerable part of the daily work of every general practitioner, Dr. Saundby continued as follows:

A very great step has been made towards the clear understanding of stomach disorders in the method introduced by Küssmaul of examining the contents of the stomach during digestion, or a given time after the ingestion of food, by means of the stomach tube. By this and other means recently discovered, we can investigate the size of the stomach, the condition of its muscular wall, the quantity and quality of the gastric juice, and the presence in the stomach of abnormal substances which may injure its glandular mucous membrane, impair its function, and give rise to pain, flatulence, and other troubles.

If we wish to estimate the functional capabilities of an organ, we try to measure the work it does; and if it is a secreting gland, we obtain some of the secretion, and submit it to various chemical and other tests in order to determine its quantitative and qualitative characters. There is no better example of this method than the kidney, which has been the subject of close study for the last sixty years, so that we now possess a vast amount of theoretical and practical

²² A full description of his manometer will be found in *Pflüger's Arch.*, 1888, vol. xliii.

knowledge of its function and secretion, which is put to daily use by everyone of us. It would not seem unreasonable to expect that a similar line of study if directed to the stomach, its functions and secretions, would lead to equally definite and perhaps equally valuable results; and the only reasons that I know which have hindered this investigation have been the inherent conservatism of our professional methods, and an exaggerated notion of the difficulty of carrying out such examinations.

The problems we set ourselves to solve in a given case where there is dyspepsia—that is, where the function of digestion is slow and painful, attended with the evolution of gases abnormal in quantity or quality, or by signs of irritation of the mucous membrane of the stomach—are: Is the muscular wall active, and capable of discharging the stomach contents in due time? Are pepsin and free hydrochloric acid present in sufficient quantities, and does the gastric juice possess its normal power?

ATONY AND DILATATION.

The motor functions of the stomach may be determined by a series of observations upon the length of time after a meal of determined nature at which a distinct residuum of food could be extracted from the stomach by means of a tube or an emetic. But, although this has been done, a more convenient method has been devised, which depends upon two facts: (1) That the normal stomach contents are acid, but become alkaline after being mixed with bile in the duodenum; and (2) that salol (a compound of phenol and salicylic acid) breaks up in the presence of alkalis into its component parts, which are excreted in the urine, where their presence may be demonstrated in the usual manner by the perchloride of iron test. The dose is fifteen grains, given about one hour after food, by which time the stomach contents have become distinctly acid. According to the careful observations of Ewald, in from forty minutes to one hour and a-quarter the urine should give the characteristic reaction, while thirty hours later there should be none. If the reaction has not appeared in the time first mentioned, or has not disappeared at the end of the time stated, we are justified in inferring that the muscular wall of the stomach is defective in the performance of its function, and if there is no evidence of pyloric obstruction, this must be due to atony of the muscular wall. This condition of atony leads in time to more or less dilatation, which may be demonstrated in various ways. It may be possible to map out the stomach by palpation and percussion when distended by food or fluid, and in the latter case we may elicit a distinct succussion splash, but in less-marked cases this is not easy, and the thickness of the abdominal wall may interfere greatly with the precision of our manipulation. Some assistance may be derived by distending the stomach with a spray producer attached to a stomach tube introduced into its cavity. This method does not cause pain, and by the altered resonance on percussion gives very fair indications. The old plan of distending the stomach by administering an alkali and an acid separately is not to be recommended, as the carbonic acid gas evolved causes spasmodic closure of the cardiac opening, and great pain, and even fainting, may result from over-distension.

GASTRIC CONTENTS.

The examination of the gastric contents is rather more complex, but is based on even simpler principles. We give the patient—say at 8 A.M.—a breakfast consisting of 4 ounces of bread and a pint of weak tea without milk or sugar. Numerous observations made by Ewald and Boas have shown that for the first half or three-quarters of an hour lactic acid preponderates in the contents of the stomach; then distinct traces of hydrochloric acid begin to be present, together with lactic acid, and at the end of an hour the lactic acid disappears, and free hydrochloric acid alone is present. Therefore, one hour after the administration of the test breakfast the stomach tube, a soft long tube with a large fenestra, is introduced, and the contents of the stomach removed, an operation which is very easy, and is facilitated by voluntary compression of the abdominal muscles by the patient. In this way a few ounces of gastric contents are obtained, which consist of the remains of food mixed with the secretion of the gastric mucous membrane, with saliva, and possibly with a

little bile. This material is filtered, and its reaction tested with litmus paper. If alkaline or neutral, it indicates a very abnormal state of the gastric secretion, such as is only met with in a few cases of advanced organic disease. It is, as a rule, acid, and we have next to determine the nature of the acid present.

The test for lactic acid is made by adding to a 1 per cent. solution of carbolic acid a few drops of perchloride of iron, and diluting this, if necessary, with distilled water, until we possess a solution of a pale amethyst colour. On adding a little of the filtered stomach contents, the amethyst blue turns to a canary yellow if lactic acid be present. This reaction is given by lactates as well as by lactic acid, and by other substances, such as alcohol; but under the conditions of our experiment these fallacies are of no importance. If the solution turns pale yellow with a reddish or brown lustre, butyric acid is present; but this, too, is not important in the case of our test breakfast, though the fact is of value in the examination of vomited matter, for butyric acid is a product of fermentation in the stomach, and is a very powerful irritant of the gastric mucous membrane.

Whether or not lactic acid is present, we have to determine if there be any free hydrochloric acid in the gastric contents—that is, whether the secretion of hydrochloric acid by the peptic glands of the stomach has been sufficient to supply all the chemical affinities present in the food, and to leave a surplus, as should be the case in healthy digestion. There are many reagents which have been employed for this purpose, but I invariably make use of Günzburg's test, consisting of two parts of phloroglucin, one part of vanillin, and thirty of absolute alcohol. If I dip a splinter of wood in this solution, and then touch it with hydrochloric acid, it turns red. If we put a few drops of the solution into a capsule together with a few drops of filtered gastric contents, and warm them gently over a lamp so that the fluid is evaporated, the appearance of streaks and patches of bright red indicates the presence of free hydrochloric acid, and this test is so delicate that it will detect less than 0.05 part of free hydrochloric acid per mille.

Our next business is to determine whether the gastric juice contains a sufficiency of pepsin; and if we have proved that there is a due supply of hydrochloric acid, we must conclude that, if pepsin is not absent, peptone should be found in the filtrate. The test for peptone is what is called the biuret reaction, the rose red colour on adding peptone to Fehling's solution; but of late years it has been shown that the albumoses, which are a transition stage between albumen and peptone, give this reaction, so that we must first get rid of these by saturating the filtrate with ammonium sulphate, allowing it to stand twenty-four hours, filtering it, and perhaps saturating it again, warming it gently, and again filtering before we have a fluid in which we can with certainty affirm the presence of peptone.

Not very uncommonly it happens that the result of this proceeding in cases of dyspepsia is negative, yet we are not thereby enabled to conclude that pepsin is deficient, unless albumoses also are absent. It becomes necessary to carry our investigation a stage further by employing our filtered gastric contents as a digestive agent in an artificial digestive experiment. For this purpose we stamp out with a cork-borer a number of little discs of white of egg, and place an equal number of these—let us say six—in four test tubes. To the first of these we add only the filtered stomach contents; to the second, the gastric contents *plus* a few drops of 0.2 per cent. hydrochloric acid; to the third, gastric contents and pepsin; to the fourth, no gastric contents, but hydrochloric acid and pepsin. These are all placed in an oven and kept at the temperature of the stomach—100° F.—for three or four hours. At the end of that time they are examined separately for peptone. If the first shows peptone, the gastric juice was active but a little weak; if the second only or better than the first, that the hydrochloric acid was insufficient; if the third only or distinctly better, that pepsin was deficient; while the fourth is the control tube, by which we are enabled to assure ourselves that the hydrochloric and pepsin used, and the other conditions of the experiments, were good and efficient.

We have met with a good many cases of anachlorhydria or deficient secretion of hydrochloric acid, but only one in which pepsin was absent. Hydrochloric acid was as a rule absent in

cases of chronic gastritis and dilatation, but in only one where there was no evidence of such organic disease. It is known that the secretion of this acid may fail from nervous causes, and even without giving rise to dyspeptic symptoms, so long as the motor function of the stomach remains normal. The persistence of pepsin in sufficient quantity depends probably upon the small amount actually necessary, and agrees with the results obtained by Leube and others, but is strikingly in opposition to popular pathology, to judge by the large number of people who think themselves benefited by the use of preparations of pepsin.

This work necessarily takes up a good deal of time, and I do not suggest that every busy practitioner can investigate his cases so completely. In the majority of instances it is not necessary to do so; but I hope before very long it will be as easy to get assistance, where needed, in such investigations as it now is to have microscopical or chemical examinations made of urine, sputa, or morbid growths. All that would be required of the practitioner would be to order the test breakfast, attend to remove it at the proper time, and then seal up the fluid obtained in a bottle and send it for examination and report.

RESULTS.

A great number of experiments have demonstrated that atony of the stomach, involving delay in the transfer of the gastric contents into the intestine, is the most constant, the earliest, the most important factor in the production of the common dyspepsia of women. When the muscular wall of the stomach fails to act properly, it, in the first place, does not mix and churn the food so as to ensure all parts of the mass coming into proper contact with the gastric juice, so that the reduction to chyme is imperfectly performed.

It has been observed in experiments upon artificial digestion that the continuous peptonisation of albumen only occurs if the peptones already formed are removed, as they are formed, by dialysis, so that the accumulation of peptones in the stomach constitutes an obstacle to the further digestion of albuminous matter, and as little or no absorption takes place through the wall of the stomach, any delay in the discharge of gastric contents through the pylorus must be attended by this. It also gives rise to more or less stagnation of the blood and lymph currents, impairing the function of the glands, and leading to insufficient secretion of hydrochloric acid, as we have found in many of our in-patients. The defective movements of the muscular wall also lead to incomplete admixture of the gastric juices with the ingesta. By-and-bye the stomach becomes slightly dilated; a residue of food remains in the stomach until the next meal is taken, and this liquid organic matter, which contains normally many micro-organisms and is deficient in its natural antiseptic, hydrochloric acid, undergoes fermentative decompositions, giving rise to the formation of lactic and butyric acids, which cause irritation of the gastric mucous membrane, discomfort and pain, and are attended by the evolution of gases, which cause flatulence and often smell offensively. A stomach in such a condition is very prone to attacks of subacute gastritis.

This inflammatory reaction is slight at first, but it weakens the resistance of the membrane, renders it more prone to acute or subacute exacerbations, manifested by what are called bilious attacks, and, in course of time, determines atrophy of the glands of the stomach, with consequent diminution of the gastric juice, and as it extends through the mucous and submucous coats to the muscular layer further weakens this, leads to increased atony, and some degree of permanent dilatation. When this is established the vicious circle is completed and all the evils I have enumerated are intensified and the stomach troubles become very serious.

The initial evil—atony of the wall of the stomach—arises from many conditions, which are especially liable to occur in women:—from anæmia, from over-work, want of sleep, and anxiety, which are the lot of so many women in the course of the duty of bringing up a family; from superlactation, want of sufficient food, confinement to the house, and irregular meals; from acute diseases and want of proper rest, good food, and fresh air during convalescence. Does not the recital of these causes remind one too forcibly of the common lot of most of the women in our great towns, not only the wives of artisans or of the middle class of clerks, warehouse-

men, and shop assistants, but of the wives of many who are in fairly comfortable circumstances, but on whom sickness makes demands which they are not in a position to meet with all the resources of wealth?

Simple atony of the stomach due to debility, originating as has been described, constitutes the essential and sole lesion in the majority of women suffering from so-called dyspepsia. The symptoms commonly met with are pain between the shoulders, weight at the pit of the stomach coming on some time after taking food, and sometimes relieved temporarily by a meal. Flatulence and constipation are usually present, but the tongue is clean, and the secretions of the mouth are normal. The diagnosis may be made by the administration of a dose of salol, as already described, one hour after food, and the examination of the urine passed one hour and a-quarter after the dose of salol (gr. xv) has been taken.

A bilious attack is subacute gastric catarrh, but it is remarkable how little the subjective symptoms indicate the severe disturbance of the stomach; hence, I suppose, the general unwillingness to recognise the true nature of the lesion. This is very well illustrated by one of Beaumont's classical observations upon Alexis St. Martin.

St. Martin had been drinking ardent spirits pretty freely for eight or nine days. Two days later he had an uneasy sensation with tenderness at the pit of the stomach, some vertigo, and dimness and yellowness of vision on stooping down and rising again; he had a thin yellowish brown coat on his tongue, and his countenance was rather sallow, but he felt otherwise well, his appetite was good, and he slept as usual. Now mark the state of his stomach, as described from direct inspection. The inner surface of the stomach showed extensive erythematous livid patches, from the surface of which exuded small drops of grumous blood, large and numerous aphthous ulcers, the whole covered with thick mucus. The gastric fluids when extracted were mixed with a large proportion of thick ropy mucus and considerable muco-purulent matter, slightly tinged with blood, resembling the discharge from the bowels in some cases of chronic dysentery; yet with a stomach in this state he would have eaten anything!

It is easy to understand that once the patient has arrived at this stage it is only a question of time before the gastritis becomes chronic and the dilatation permanent and pronounced.

Where the nervous trouble produces marked hyperæsthesia, especially common in anæmic girls, the symptoms are pain immediately after food, epigastric in position, sharp in character, followed and relieved by vomiting; and these cases, as already mentioned, are very liable to be mistaken for ulcer of the stomach. It is noteworthy that the chemical and motor functions of the stomach do not necessarily suffer even in most severe cases of chlorotic anæmia—a fact which we have recently had several opportunities of demonstrating.

Closely allied to anæmic gastralgia is the condition which has been called anorexia nervosa, where there is entire loss of appetite for food, generally induced by the pain which has followed its ingestion. In this state the patient becomes extremely wasted, and may die from inanition. In most instances they recover rapidly when put to bed and properly fed; but they are apt to relapse, especially where there is a hysterical element in the case.

Ulcer of the stomach is undoubtedly a common occurrence in anæmic young women, but it has such a well-known pathological and clinical history that I need only allude to it here and elsewhere for the purposes of contrast and comparison. Undoubtedly until hæmatemesis has occurred it is impossible to differentiate ulcer from anæmic gastralgia, but this is no disadvantage, as both should be treated in precisely the same way.

GERMAN UNIVERSITIES.—The following figures show the number of students in the medical faculties of each of the universities of the German Empire during the present (1893-94) semester: Berlin, 1,279; Munich, 1,114; Leipzig, 788; Würzburg, 746; Griefswald, 380; Freiburg, 342; Erlangen, 338; Strassburg, 299; Breslau, 287; Kiel, 257; Bonn, 247; Tübingen, 235; Halle, 234; Königsberg, 230; Heidelberg, 218; Marburg, 216; Jena, 196; Göttingen, 192; Rostock, 120; and Giessen, 96. The total number of medical students in the twenty universities is therefore 7,714, being a decrease of 335 as compared with the corresponding period of 1892-93.

THE fourth Italian Congress of Chemists and Apothecaries will be held at Naples on September 2nd and five following days.