A MODIFICATION OF THE SAHLI BUTYROMETRIC TEST MEAL

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The need of some improvement in our methods of determining the gastric functions is evident when we consider the limitations of the present methods. For instance, if after the administration of an ordinary test breakfast we aspirate an excessively large amount of contents, it is impossible to say whether the excess is due to abnormal secretion of gastric juice, to impaired motility, or to both. If, however, we should introduce with the test meal a certain amount of some substance which was not affected by gastric ferments, or absorbed by the stomach, something which would mix thoroughly with the gastric juice, then by estimating the amount left at the end of a given period we could determine how much of the test meal had passed on into the intestine; i. e., the gastric motility, and how much of the aspirated contents consisted of test meal, how much of the gastric juice.

Sahli[1] proposed the following meal, based on such considerations: An emulsion of fat is prepared by roasting 45 grams of flour, thoroughly mixed with 15 grams of butter; 350 c.c. of water are added and the whole boiled two minutes. This process makes rather an agreeable thick soup. Of this 300 c.c. are given the patient and 50 c.c. retained for a fat determination. After one hour the stomach contents are aspirated as completely as possible. As it is not possible to aspirate the total contents, the residue is calculated according to the method of Matthieu,[2] viz.: A definite amount of water—say 300 c.c.—is poured down the stomach-tube, thoroughly mixed with the remaining stomach contents, and the whole again aspirated. Then both diluted and undiluted contents are titrated and the residue calculated by the following equation: Let x = residue, a = acidity of undiluted contents, and b = acidity of residue diluted with 300 c.c. water. Then:

$$x = \frac{300 \times b}{a - b}$$

Adding this amount to the quantity originally aspirated, we have *TO,* or the total stomach contents at the time of aspiration.

1. Sahli: Klinische Untersuchungs Methoden, 1902, p. 412.
2. Matthieu: Arch. f. Verdauungskr., 1896, i, 348.

Two fat determinations are then made to ascertain how much fat the test meal contained and how much remained in the stomach at the time of aspiration. Sahli uses Gerber's acido-butyrometer, employed in dairies in Switzerland, in which the emulsion of fat is broken up by strong sulphuric acid and amyl alcohol, centrifugalized, and the fat per cent. read off in the necks of bottles empirically graduated. If we give a patient 9 grams of fat contained in 300 c.c. of soup and find only 3 grams left in the stomach after one hour, then this would correspond to 100 c.c. of soup, and if we aspirated 200 c.c. of stomach contents and 100 c.c. of it is test meal then the other 100 c.c. must be gastric juice. Algebraically expressed:

Let TO = total amount of stomach contents and F = per cent. of fat in original soup. Let f = per cent. of fat in aspirated contents. Let TM = test meal and GJ = gastric juice. Then TM or the amount of the test meal contained in aspirated contents $= \frac{f \times TO}{F}$ and of course GJ or the gastric juice $= TO—TM$.

We now know what part of the stomach contents is test meal and what part is gastric juice. The relation of these two to each other we call the secretory quotient or Q.

Furthermore, in an ordinary test meal what we titrate is the acidity, not of the undiluted gastric juice, but of gastric juice diluted by more or less of the fluid contained in the given test meal. If, however, we know the acidity of the mixture and how much of the mixture is gastric juice, it is easy to calculate that by the following equation:

Let a = acidity of the aspirated stomach contents (determined by usual titration) and A = calculated acidity of the undiluted gastric juice and TO = total amount in stomach at the time of aspiration and GJ = amount of gastric juice in the contents, then:

$$\frac{A = a \times TO}{GJ}$$

The motility in this method is represented by the relation of the amount of test meal which has left the stomach to the total amount given, or:

$$\frac{M = 300 - TM}{300}$$

As will be seen, the only extra work involved is the preparation of the test meal and two fat determinations, the calculations, of course, being very simple. Objections to the method soon appeared, however. It was asserted that the preparation of the meal was difficult, that the presence of such a large amount of fat in a meal inhibited gastric secretion, and, most essential, that the emulsion was not stable and did not mix evenly with the gastric juice. This last objection, if true, nullifies the whole procedure, which is based on the theory that the fat remains evenly diffused throughout the whole stomach contents. To take the instance given above, we supposed a soup containing 3 grams of fat to the 100 c.c. If in the stomach the fat should rise to the top and remain behind while the fluid passes through the pylorus, it will be seen that our only means of deciding how much of the original test meal is left in the stomach is gone.

After working some time with this meal it was found that these objections were justified, and a search was made for a simpler and more stable emulsion. After many fruitless attempts at artificial ones, a satisfactory substance was found in condensed milk. If we dilute condensed milk two or three times, add some gastric juice and keep the test-tube ever so slightly in motion, it will be found that the flocculi resulting from the coagulation are exceedingly fine, uniform in size and microscopically the fat globules almost as fine as in fresh milk and almost as evenly divided.

Of the different brands tested, that known as "Carnation" proved the most satisfactory and was used in the following work.

Table 1.—Results in Normal Individuals

Case Number.	Total Amount in Stomach=*TO.*	Ratio of Fat in Meal to Fat in Stomach Contents=*F:f.*	Calculated Amount of Test Meal in Aspirated Contents=*T. M.*	Calculated Amount of Gastric Juice in Aspirated Contents=*G. J.*	Secretory Quotient.	Motility.	Titrated Acidity.	Calculated Acidity of Pure Gastric Juice.	Diagnosis.	Remarks.
1	282	2.2:1.3	166	116	0.7	0.61	54	131	Normal.	
1	287	2.9:1.5	150	137	0.91	0.5	62	130	Normal.	
1	258	2.0:1.1	142	116	0.82	0.52	68	151	Normal.	
1	200	1.9:1.0	105	95	0.9	0.65	74	155	Normal.	
1	238	3.0:1.7	135	103	0.76	0.55	56	129	Normal.	
2	134	2.8:1.6	76	58	0.7	0.7	58	134	Neurasthenia.	Only 250 c.c. of meal were given.
3	85	2.0:1.0	42.5	42.5	1.0	0. 6	50	100	Neurasthenia.	

For making the fat determinations the Babcock method was employed, a small hand centrifuge being used, such as is sold for the determination of butter fat in milk and cream for dairy purposes. The following is the routine: 120 c.c. of condensed milk are diluted with water to make 350 c.c., 50 c.c. retained for a fat determination and 300 c.c. given the patient. In fifty minutes the contents are aspirated as completely as possible, 200 c.c. water poured down the tube, thoroughly mixed with the residue and again aspirated. A titration of the stomach contents, another of the diluted residue, a fat determination of the original meal, and one of the aspirated contents are all that are necessary for the final calculation. Blood, lactic acid, etc., are tested for as usual. The accompanying tables give results obtained by this method.

In normal individuals, then, the total amount in the stomach after fifty minutes varies considerably, viz.: from 85 c.c. to almost 300, depending, of course, on normal individual variations. Second, the relation between the amount of milk and the amount of gastric juice, which we call the secretory quotient, varies normally between 0.7 and 1 even in the same individual at different times, while between different individuals the variation is no greater. Third, the motility, or the relation between the amount of milk that has passed the pylorus to the amount given, varies between 0.5 and 0.86. If a less amount passes the pylorus we have hypomotility; if a greater amount, hypermotility. Fourth, the figures for titrated acidity are, of course, those known from the Ewald test breakfast and varied between 40 and 76, a variation from the mean of 31 per cent. The calculated acidity, however, was very much more constant, lying between 120 and 150 (occasionally a little lower), a variation of only 10 per cent. In other words, the acidity of the gastric juice as it is poured out by the glands is remarkably constant, much more so than the figures which we obtain from titrating a test meal would indicate. This fact, which was first demonstrated by Pawlow on dogs, was later verified by Bickel[3] in two patients with gastric fistulas. He found that the acidity was constantly between 110 and 140, only the first part secreted having a lower acidity, due to neutralization by the gastric mucus. Bickel also verified the results of Pawlow in regard to the quantity and found that it varied considerably according to the kind of food, condition of patient, etc. By the present method, of course, we do not estimate the total amount secreted during the whole digestive act, but simply the amount in the stomach at the time of aspiration. This, we have found, normally bears a constant relation to the amount of test meal in the stomach at that time, as indicated by the secretory quotient; or:

$$\frac{GJ}{TM} = 0.7 \text{ to } 1$$

3. Bickel: Deutsch. med. Wchnschr., 1906, xxxii, 323; 1907, xxxiii, 1201.

When the two do not bear this relation, the condition is pathologic; if the relation of gastric juice to test meal is greater than 1 we have hypersecretion; if less than 0.7, we have hyposecretion. Of course, if we remove the test meal earlier, we find a condition of hyposecretion; i. e., a lower proportion of gastric juice. For instance, removing test meal in thirty minutes, 321 c.c. remained in the stomach; of this quantity, only 85 c.c. were gastric juice, the remainder being test meal, a secretory quotient of 0.36. This was, of course, normal for a thirty-minute period, but would have been pathologic for a fifty-minute period. So much for the normal figures. There are theoretically six possible variations. There may be hypersecretion, or hyposecretion, hypermotility or hypomotility, hyperacidity or hypoacidity, or combinations of these.

The following table illustrates the findings in some typical cases of so-called hyperacidity:

TABLE 2.—HYPERSECRETION

Normal secretory quotient (*Q*) varies between 0.75 and 1. Hypersecretion is present when *Q* is more than 1.

Case Number.	Total Amount in Stomach=*T.O.*	Ratio of Fat in Meal to Fat in Stomach Contents=*F:f.*	Calculated Amount of Test Meal in Aspirated contents=*T.M.*	Calculated Amount of Gastric Juice in Aspirated Contents=*G. J.*	Secretory Quotient=*Q.*	Motility.	Titrated Acidity.	Calculated Acidity of Pure Gastric Juice.	Diagnosis.	Remarks.
4	156	3.0:1.3	68	88	1.3	0.77	72	128	Neurasthenia.	Test meal consisted of 125 c.c. each of condensed milk and water.
5	195	2.8:1.0	70	125	1.8	0.72	64	100	Neurasthenia.	Test meal consisted of 125 c.c. each of condensed milk and water.
6	474	2.8:9.0	152	322	2.1	0.39	82	121	Benign pyloric stenosis.	Test meal consisted of 125 c.c. each of condensed milk and water.
6	296	2.8:9.0	95	201	2.1	0.6	66	97	Benign pyloric stenosis.	Test meal consisted of 125 c c. each of condensed milk and water.
7	672	2.1:0.6	224	448	2.0	0.25	68	103	Benign pyloric stenosis.	Test meal consisted of 125 c.c. each of condensed milk and water.

Case 6 was a benign stenosis of the pylorus of ten years' duration. The stomach was thoroughly emptied before the test meal was given (and with some practice this can be done so that at the utmost only 25 to 30 c.c. are left). Fifty minutes later the amount removed was 496 c.c. Ordinarily we would have no certain means of knowing whether this excessive amount was due to impaired motility or hypersecretion of gastric juice, as either would produce this result. We find on analysis here, however, that, while the motility is somewhat impaired, yet the major portion of the excess is due to hypersecretion, there being 2:1 times the normal proportion present. In the first test the motility was markedly impaired, viz.: 0.39. In the second, after two weeks of gastric lavage, motility had improved somewhat, but did not remain permanently so till after a gastroenterostomy had been done. The titrated acidity in one estimate was 82, showing an apparent hyperacidity, but the calculated acidity of the pure gastric juice was 121, well within the normal limits. The functional diagnosis reads, then, hypersecretion and impaired motility, the characteristic findings of benign stenosis.

In Case 4 the total amount in the stomach was 156 c.c., but the relation of gastric juice to test meal in that was higher than normal, viz.: 1.3, instead of 1, a hypersecretion, in other words. The titrated acidity was 72, slightly above normal, but the calculated acidity was only 128. Therefore, the stomach was pouring out a larger amount of secretion than normal, but with normal acidity. Motility was normal.

Diagnosis: Simple hypersecretion with good motility, a neurosis, as the course showed.

To illustrate the value of this method in distinguishing between hypersecretion and impaired motility, we may compare Tables 2 and 3.

TABLE 3.—HYPOMOTILITY

Normal motility varies between 0.5 and 0.8. Hypomotility is present when less than 0.5 of meal passes pylorus.

Case Number.	Total Amount in Stomach=*TO*.	Ratio of Fat in Meal to Fat in Stomach Contents=*F*:*f*.	Calculated Amount of Test Meal in Aspirated Contents=*T. M.*	Calculated Amount of Gastric Juice in Aspirated Contents=*G. J.*	Secretory Quotient =*Q*.	*Motility.*	Titrated Acidity.	Calculated Acidity of Undiluted Gastric Juice.	Diagnosis.
27	436	2.9:1.4	210	2.26	1.08	0.16	58	112	Atony.
27	340	2.7:1.3	164	176	1.07	0.34	62	112	Atony.
27	370	2.7:1.3	150	160	1.07	0.4	60	116	Atony.
28	258	2.5:1.4	144	114	0.78	0.42	56	126	Stone in common duct.
28	386	2.6:1.1	160	226	1.4	0.36	60	146	Stone in common duct.
29	305	1.9:1.1	177	128	0.72	0.4	34	82	Atrophic cirrhosis of liver.
30	441	3.0:1.6	235	206	0.88	0.22	70	149	Benign pyloric obstruction.

The cases in Table 3 all show impaired motility of the first degree only; that is, the stomach emptied itself over night. In Case 27, for instance, all three tests gave large amounts of stomach contents—from 370 to 436 c.c., much beyond the normal. The same question arises as to whether this is due to impaired motility or hypersecretion. The results show, however, that the aspirated contents consisted of about equal parts of gastric juice and test meal; that is, the secretory quotient was about 1; and, therefore, that the increased contents must be due to impaired motility, and this the figures show, the motility varying from 0.16 to 0.4, considerably below normal. The same points are illustrated by Cases 28, 29 and 30, in all of which impaired motility without hypersecretion is found. As far as the reliability of this meal as a test of motor function is concerned, I may say that in all cases in which the Riegel motor meal showed impaired motility this method indicated it also, and in some cases of apparent slight impairment, as in simple atony, it proved positive where the Riegel meal was negative, so that it is, if anything, a more delicate indicator of motility.

Cases of impaired motility complicated by secretory changes are tabulated under the latter headings.

The third possible pathologic variation is hyperacidity, a name until recently used to indicate all of those conditions in which the titrated acidity was above a certain maximum, about 70. Rubow,[4] from theoretical considerations alone, concluded there was no such condition possible, all the cases of so-called hyperacidity being due simply to hypersecretion of juice of normal acidity. If the normal stomach contents one hour after a test meal consist of equal parts of test meal and a gastric juice of normal acidity (say 120), then the mixture would have an acidity of 60, and the titration would give us the latter or normal figures. If, however, the stomach contents contained one-quarter test meal and three-quarters gastric juice of the same acidity (120), then the mixture would have an acidity of 90 on titration, an apparent hyperacidity, due really, however, to a hypersecretion. Inasmuch as no case has ever been reported with an acidity above 150, or the upper limit of undiluted gastric juice, Rubow concludes that hyperacidity does not exist. As stated above, Bickel also found the gastric juice in the human being remarkably constant as to acidity. As shown in Table 2, at least some cases of hyperacidity are simple hypersecretion, the calculated acid as already stated being within normal limits. Since using the present modification of the Sahli meal I have been unable to demonstrate beyond doubt any case of true hyperacidity. For instance, in one case the first test meal resulted in a calculated acidity of 356. A second and third test meal gave figures of 92 and 100, respectively, so that the method

4. Rubow: Arch. f. Verdauungskr., 1907, xiii, 577.

was unquestionably at fault; i. e., the emulsion had possibly been destroyed so that the fat formed a layer at the top while the lower thinner fluid passed through the pylorus. While, therefore, I am certain that the diagnosis, "hyperacidity," ought to be made very much less frequently than at present, very much more evidence will have to be produced before the word is entirely dropped.

TABLE 4.—HYPOACIDITY

Acidity of undiluted gastric juice varies normally between 120 and 150 (calculated in terms n/10 NaOH).

Hypoacidity is present when acidity is less than 100.

Case Number.	Total Amount in Stomach=*TO.*	Ratio of Fat in Meal to Fat in Stomach Contents=*F:f.*	Calculated Amount of Test Meal in Aspirated contents=*T. M.*	Calculated Amount of Gastric Juice in Stomach Contents=*G. J.*	Secretory Quotient =*Q.*	Motility.	Titrated Acidity.	*Calculated Acidity of Undiluted Gastric Juice.*	Diagnosis.	Remarks.
16	166	2.7:1.4	86	80	0.95	0.7	26	54	Chronic gastritis.	Mucus++.
16	197	2.9:2.0	136	61	0.45	0.46	28	90	Chronic gastritis.	250 c.c. equal parts condensed milk and water given as test meal.
17	284	2.8:1.4	142	142	1.0	0.43	32	68	Chronic gastritis.	Mucus++.
18	97	2.5:1.4	54	43	0.8	0.78	20	45	Amyloidoses.	
19	55	2.8:1.5	29	26	0.9	0.88	20	41	Tb. ulcer of duodenum.	
20	123	?	64	59	0.92	0.78	7	15	Simple achylia gastrica.	
20	170	2.4:1.0	70	100	1.4	0.77	4	7	Simple achylia gastrica.	Removed in 30 minutes.
21	200	2.4:1.3	119	81	0.68	0.6	4	10	Simple achylia gastrica.	Removed in 30 minutes.
21	233	2.2:1.4	149	84	0.57	0.5	4	11	Simple achylia gastrica.	Removed in 30 minutes.
22	141	3.0:1.9	89	52	0 58	0.7	8	22	Simple achylia gastrica.	
22	100	2.6:1.8	69	31	0.45	0.77	8	26	Simple achylia gastrica.	
23	365	3.0:1.6	195	170	0.87	0.25	28	60	Carcinoma of pylorus.	
24	194	2.5:1.9	147	47	0.32	0 51	20	50	Carc noma of lesser curvature.	
25	70	2.2:1.6	51	19	0.37	0.8	4	15	Carcinoma.	
26	300	?	186	114	0.6	0.38	..	16	Carcinoma of fundus.	

The opposite variation, or "hypoacidity," is represented in Table 4. By uncomplicated hypoacidity we mean that, no matter what the titrated acidity is, on calculating the real acidity of the undiluted gastric juice, we find it below 100; i. e., the stomach is secreting a more watery fluid than normal.

This was found to be the case quite consistently in at least three conditions, viz.: chronic gastritis, achylia gastrica simplex and in some carcinomas. To take the most pronounced cases first, those of achylia gastrica, it was found that on titration the acidity varied between 4 and 8 (it was often necessary to aspirate in thirty minutes on account of the hypermotility), and that the amount of gastric juice secreted was generally normal, but the calculated acidity 26 or less. In other words, the gastric mucosa in some cases secretes almost pure water.

This seemed to indicate an error in the method, as both Martius[5] and Einhorn[6] define achylia as a condition in which there is entire absence of gastric secretion. A review of the literature, however, shows that the same condition has been found before not only in achylia, but in other conditions. It has been shown experimentally that the secretion of water, or hydrochloric acid, and of pepsin are three quite distinct functions of the gastric glands. Schiff[7] showed that in pathologic conditions there need be no parallelism between the variations of pepsin and hydrochloric acid; that by injecting pilocarpin the water could be increased, while acid diminished and pepsin remained constant. Schneyer[8] found that by stimulating the vagus of a fasting dog 3 to 15 c.c. of gastric juice flowed out, but without hydrochloric acid or pepsin, while if the animal had been fed within twenty-four hours both hydrochloric acid and pepsin were present. Roth and Strauss[9] showed that not only were hydrochloric acid and pepsin secretions distinct functions, but that water secretion was due to two distinct causes: (1) to osmotic phenomena in which the gastric mucosa acts as an animal membrane, and (2) to the specific activity of the gastric glands producing a "diluting" secretion often in defiance of the laws of osmotic pressure; and, furthermore, that in achylia gastrica, even when hydrochloric acid and pepsin secretions have ceased, this diluting fluid is still poured out. This is the condition indicated in Table 4. Hydrochloric acid was almost entirely absent, pepsin (not represented in the table) greatly diminished, while practically water alone was secreted. It will be noted that the quantity of water in most cases was practically normal; i. e., the secretory quotient was above 0.75.

In the case of chronic gastritis given here, the same condition was found, but to a less extent; i. e., the calculated acidity was higher (between 54 and 90), but still below normal. That this, too, is the usual condition in gastritis is shown by the work of Bickel[10] on dogs with

5. Martius: Achylia Gastrica. Ihre Ursache und Ihre Folgen. Reprint, 1897, p. 4.
6. Einhorn: Arch. f. Verdauungskr., 1896, i, 454.
7. Schiff: Arch. f. Verdauungskr., 1900, vi, 107.
8. Schneyer: Ztschr. f. klin. med., 1897, xxxii, 13.
9. Roth and Strauss: Ztschr. f. klin. Med., 1899, xxxvii, 144.
10. Bickel: Arch. f. klin. Med., 1906, lxxxix, 34.

Pawlow fistulas. Here, too, after irritation of the mucosa by local applications of alcohol and silver nitrate solutions, the amount of gastric secretion was found to be normal, but the hydrochloric acid content diminished.

TABLE 5.—HYPOSECRETION

Normal secretory quotient (*Q*) varies between 0.75 and 1. Hyposecretion is present when *Q* is less than 0.75.

Case Number.	Total Amount in Stomach=*TO.*	Ratio of Fat in Meal to Fat in Stomach Contents=*F:f.*	Calculated Amount of Test Meal in Aspirated Contents=*T. M.*	Calculated Amount of Gastric Juice in Aspirated Contents=*G. J.*	*Secretory Quotient* =*Q.*	Motility.	Titrated Acidity.	Calculated Acidity.	Diagnosis.	Remarks.
8	102	3.0:2.1	71	31	0.44	0.76	28	92	?	250 c.c. equal parts condensed milk and water given.
8	100	3.3:2.5	76	24	0.31	....	28	112	?	
9	139	3.4:2.6	107	32	0.3	0.65	34	140	Pernicious anemia.	
9	189	3.1:2.4	146	43	0.3	0.51	30	132	Pernicious anemia.	
10	150	3.2:2:8	131	19	0.14	0.56	20	158	Pernicious anemia.	
10	210	2.2:1.6	153	57	0.37	0 49	44	162	Pernicious anemia.	
11	132	2.1:1.6	100	32	0.32	0.67	24	99	Pernicious anemia.	
12	112	2.0:1.6	90	22	0.24	0.7	30	153	Connective tissue infiltration of walls (old ulcer).	
13	75	1.9:1.6	63	12	0.2	0.7	?	?	Carcinoma of fundus.	Large amount of lactic acid.
14	255	2.8:2.1	197	58	0.3	0.53	6	26	Pernicious anemia.	
15	228	2.1:1.6	173	55	0.31	0.42	38	157	Pernicious anemia.	Removed in 35 minutes.
15a	152	?	119	33	0.28	0.52	29	133	Carcinoma.	

A third condition in which the calculated acidity was subnormal was carcinoma. Here, however, the total amount of secretion was lowered; i. e., the secretory quotient was small. Now, according to Schiff,[7] the order in which the different functions are affected and disappear is: first, the hydrochloric acid secretion; second, the pepsin secretion; third, the diluting secretion. When only the first two are affected, as in achylia simplex, it is possible that there is only a functional disturbance of the mucosa, but when the diluting secretion is diminished we may be certain that a real atrophy of the mucosa is present and that the amount of the diminution measures the amount of atrophy. For example, in an instance of carcinoma of the pylorus (Case 23) the calculated acidity was 60 and the secretory quotient 0.87. In Case 24, a diffuse carcinoma of the lesser curvature (in which an exploratory laparotomy was done), the calculated acidity was 50 and the secretory quotient 0.32. Evidently the difference was due to the variations in the area involved. The motility in these cases follows the known rules; it is good in some and impaired in others, according to the situation.

In Table 5 is shown the series of cases in which the amount of water secreted was uniformly below normal, but the percentage of acid was normal, the so-called hyposecretion.

Taking the cases of pernicious anemia, we find that the amount of secretion was the factor most affected, viz.: a secretory quotient varying between 0.14 and 0.37, with normal acidity. This combination accounts for the low titrated acidity which we find in pernicious anemia; it forms a sharp contrast to the conditions in hypersecretion, given in Table 1.

Another cause for a low titrated acidity is hypoacidity, such as that which occurs in chronic gastritis and achylia, as seen in Table 4; these two conditions have, as we have seen, an entirely different significance functionally, though in both there is a low apparent acidity. If we accept the above-mentioned assertions of Schiff, that the last function of the mucosa to suffer is the secretion of water, the observation that it is here diminished in contradistinction to the condition observed in achylia, would suggest the conclusion that here the local process (atrophy) is more advanced than in the latter, or that the condition of the blood serum was such as to diminish the secretion. An inconsistency for which I can not account is the normal calculated acidity, which is contrary to what one might expect if one assumes that the acid secretion is the first to suffer. That the acid also is diminished in the more severe cases is shown by the figures in Case 14, an advanced case with marked cord changes. Here not only was the quantity of fluid diminished, but the quality impaired; i. e., a calculated acidity of 26. Here the atrophy was far advanced, as pathologic examination showed.

Patient 12 complained of pain, vomiting and emaciation. Motility and calculated acidity were normal, so that the low acidity was due to hyposecretion, the quotient being only 0.24. In the light of the above cases, the only justifiable diagnosis was atrophy, though one of probable carcinoma was made. Laparotomy disclosed simple connective-tissue infiltration of over two-thirds of the stomach from a healed ulcer, with atrophy of a considerable part of the mucosa involved, a condition just such as the clinical observations might have suggested.

Case 8 was an undiagnosed case, apparently not a carcinoma, more probably an arteriosclerotic atrophy, to judge from the hyposecretion.

The above are some of the more interesting considerations suggested by a study of the results obtained by this method. That they have a solid basis is shown by their experimental verification.

The method does, however, fail occasionally, probably for the reason that the emulsion is destroyed. Failure is particularly liable to occur under the following circumstances:

1. In cases in which there is low acidity and hypermotility. In such cases there will be so little fluid left in the stomach in fifty minutes that no accurate fat determination can be made. When such a condition is suspected, it is best to aspirate in thirty minutes.

2. In cases of gastritis in which there are very large amounts of mucus which destroys the emulsion and by rising to the surface of the Babcock bottles obscures the readings.

3. In cases of greatly impaired motility owing to the difficulty of completely emptying the stomach preliminary to lavage.

Even where it fails it still, however, offers as much information as the ordinary Ewald test breakfast.

SUMMARY

1. The method here proposed for the study of the gastric functions is a modification of the Sahli test meal, consisting of one-third condensed milk and two-thirds water, the object being to obtain a stable emulsion of fat. With a very little extra work we can calculate the amount of gastric juice in the stomach at the time of aspiration, and so determine its real acidity as well as the gastric motility, etc.

2. We have, then, definite standards for comparison which enable us to make more accurate functional diagnoses than are at present possible otherwise.

3. Among the advantages of this meal over the Ewald and Riegel meals is the consideration that the conditions of most of the gastric functions may be determined by a single aspiration. Its advantages over the Sahli meal are its constancy of composition, the ease with which it may be prepared and administered, its strong secretory stimulating powers, and the stability of the emulsion.

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