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Gastric analysis: A review of methods of expressing acidity Gastric analysis : The results of a standardized basal and augmented histamine gastric analysis

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GASTRIC ANALYSIS. I. A REVIEW OF METHODS OF EXPRESSING ACIDITY. II. THE RESULTS OF A STANDARDIZED BASAL AND AUGMENTED HISTAMINE GASTRIC ANALYSIS.

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INTRODUCTION

Gastric analysis is the study of aspirated gastric contents under various specific conditions. The analysis of gastric contents is performed to study the normal function of the stomach and the effects of disease upon them. At first these methods were only a crude measure of gastric secretory function and were of questionable value in most cases because of the independent variables of gastric secretion, gastric emptying, and dilution of gastric secretion by the meal, saliva and biliary reflux. But with the introduction of the "basal" (interdigestive) and "maximal" (histamine) test of Kay,[26] gastric analysis has become a more quantitative method which is of primary importance: (1) in the diagnosis of achlorhydria, (2) in the study of chornic stomach pathology, (3) to serially follow patients with ulcer disease. (4) as an indication of the type of surgery or medical therapy needed, (5) in the elucidation of postgastrectomy dyspepisa, hemorrhage, and diagnosis of gastric or jajunal ulcer, and (6) in the diagnosis of the Zollinger-Ellison syndrome. The significance of the "basal" and "maximal" methods of gastric analysis in the above conditions would be greatly increased if there were agreement as to the method of carrying ou the test, the analysis of the specimens, the nomenclature used in expressing the results, and the separation of the results as to age, sex, medication, and the presence of other disease. With this standardization, it would be a simple matter to collect and compare the date obtained by different in-

vestigators and to integrate the results into a standard formulation for interpretation of gastric analysis. The following is a discussion of evolution of a simple quantitative method of expressing the results, and the presentation and discussion of a standard method of gastric analysis.

Acidity: In 1824 the concept of "free acid" and "total acid" were introduced by Prout.[46] Free acid is the acid equivalent of the amount of standard alkali necessary to titrate the sample to a salmon color, using the indicator dimethylamino-azobenzine (Töpfer's reagent). It is expressed in clinical units (which is the number of milliliters of 0.1 normal base needed to titrate 100 milliliters of gastric contents of the desired color). Total acid is expressed in the same units only the titration is done by using phenolphthalein as the indicator. Combined acid is a measure of the buffering capacity of the gastric contents and is expressed in clinical units as the difference between total and free acid. Prout did not indicate the derivation of the terms, but they were probably terms in common use by chemists during his time. In addition to introducing these terms to the field of gastric analysis, he also noted that the acid produced in the stomach was hydrochloric acid. This fact was confirmed by Szabo in 1877 and the term "degrees of acidity of stomach" was introduced in 1886.

These concepts for measuring and expressing acidity were generally accepted until 1909 when Sorensen[53] suggested the pH notation for

expressing the hydrogen-ion concentration. Following Sorensen's introduction of the concept of pH, there were attempts to use this notation, and to correlate pH with millinormality in the study of gastric analysis. Because the different investigators could not agree on the value of correlating these measurements, they did not gain wide acceptance.

Almost a century after Prout's definition of free acid and total acid, Michaelis[39] attempted to validate these concepts. He did this by comparing: (1) the curves obtained by titrating unknown concentrations of hydrochloric acid with 0.1 normal sodium hydroxide to the endpoints of Töpfer's reagent and phenolphthalein, and (2) those obtained with gastric juice following stimulation with both the Ewald-Boas test-meal and a bouillon test-meal. In interpreting his results, Michaelis thought that certain other fractions exists free in solution and a certain other fraction exists in combination with protein or other absorbing substances. He further stated that these fractions could be distinguished by the indicators dimethylamino-azobenzine (Töpfer's reagent) and phenolphthalein.

In attempting to prove this concept, Michaelis neglected to consider the buffering effects of the Ewald-Boas test-meal (fig.1). Also he used only the results of the Ewald-Boas test-meal and not the results of the bouillon test-meal in drawing his conclusions. The latter correlated very well with the curve of hydrochloric acid.

It was his contention that the point at which the titration curve of the gastric juice differed from that of a similar aliquot of 0.1 normal hydrochloric acid was the point (fig.1, point A) at which all of the free acid had been neutralized, and was equivalent to the amount of free acid present. In Figure two is shown the

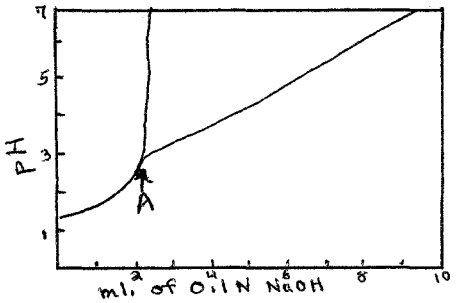

Figure 1. The titration curve of 3 ml. of gastric juice after an Ewald-Boas test-meal.

curves of seven one milliliter samples of gastric juice collected after histamine stimulation, compared with the titration curve of one milliliter of 0.1 normal hydrochloric acid. These results in-

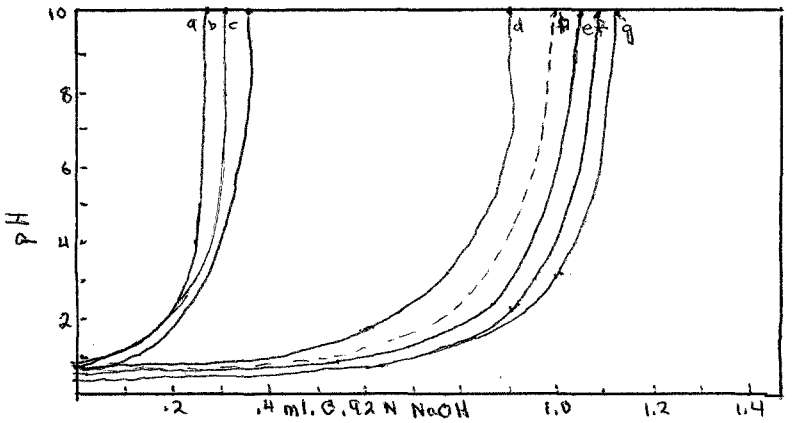

Figure 2. The titration curves of seven 1 ml. specimens of gastric juice collected after histamine stimulation compared with the titration curve of 1 ml. of 0.1 N HCl. (curve h).

dicate that the curves are very similar; and that all the acid is present as free hydrogen ions, when care is taken to avoid contaminating the gastric juice with buffering substances (food,and saliva). Considering the fact that each acid has its own respective titration curve and that the shapes of the curves in Figure two were all the same as that of hydrochloric acid, this would be additional evidence that the soul source of hydrogen-ions in gastric juice is hydrochloric acid.

Michael's proof of "free acid" and "total acid" was accepted and these terms continue to be widely used today in spite of reviews of his investigation revealing that his concepts were invalid--as illustrated in Figure two. In addition works by Shohl[51] and Hollander[19,21,22] show that free acid is related to free hydrogen-ion concentration but differs from the measurement of hydrogen-ion by titration when buffering substances are present (as in gastric juice). Also recognized was the fact that acids of the same normality, have different pH's, but they have the same titratable acidity. This can be explained by the fact that they have different dissociation curves--meaning that different amounts of the acid are in a ionized form in aqueous solution at different pH levels and the fact that strong acids(i.e. hydrochloric acid) are completely ionized in solution. These same worker's in the 1930's recommended the use of milliequivalents or milligrams of hydrochloric acid per liter as well as the use of pH in expressing

gastric acidity. Bockus[48] in 1950 reiterated the need for using the standard chemical units, milliequivalents per liter in expressing gastric acidity.

In recent literature on gastric analysis the units of milliequivalents per hour have been used for both basal and augmented histamine gastric analysis. This is an important advance in that it represents an attempt to quantitate the results of these tests, and increasing the acceptance of chemical units for expressing acid output. There is however still some confusion in the interpretation of the time used in calculating the "maximum acid output" (MAO) following the augmented histamine test. This term MAO was first defined by Marks[32,34] as the quantity of acid output between 15 minutes and 45 minutes after histamine stimulation, expressed as milliequivalents per hour, using phenolphthalein as an indicator. Other investigators have suggested the use of the whole hour, the first forty-five minutes, the first half hour, or the last half hour. The most appropriate definition for maximal acid output (MAO) would seem to be that suggested by Baron[2] that maximum acid output is the maximum acid output after "maximal" (histamine) stimulation for two consecutive fifteen minute samples expressed as milliequivalents per hour and that the endpoint be pH seven determined by an indicator or a pH meter.

Considering the above discussion, the availiability of pH meters, and micro-electrodes it is suprising that the phenolphtha-

lein endpoint, and in many cases Töpfer's reagent is still being used by informed workers in the field of gastroenterology.

The Concept of pH: The notation pH is a simple method of expressing minute concentrations of hydrogen-ions and is defined as the negative common logarithm of the hydrogen-ion concentration. In the scientific world it is customary to describe the acidity of a solution by saying: "The pH of the solution is three" instead of saying: "The hydrogen-ion concentration of the solution is 10^{-3}". It is universally accepted in the field of the physical sciences that acid means a pH less than seven, neutrality a pH of seven (i.e. the point at which there is an equal number of hydroxyl-ions and hydrogen-ions in solution) and alkalinity a pH greater than seven. The following pH values indicate important levels in gastric analysis:

pH 2---maximum activity of pepsin.
pH 3.5-end point of Töpfer's reagent.
pH 5---level above which peptic digestion does not occur.
pH 7---physico-chemical neutrality, and above which pepsinogen is stable.
pH 8.2-pH of mucus.
pH 8-9-end point of phenolphthalein.

Table I further illustrates the relationships of acidity and pH in gastric analysis and serves to illustrate the very small amounts being dealt with , as well as serving as a guide for interpretation of data.[22] Because these concepts are generally recognized and used by all of medicine except for the expression of acidity in gastric analysis and since further the end-points used in determin-

ing gastric acidity have no physiologic significance (as shown in the above discussion) it would seem most logical to use the notation pH when referring to the acidity of gastric juice.[6,19,21,22,31,53]

TABLE I

Arbitrary Divisions of Gastric Acidity[22]

mEq/L.	Degree of Gastric Acidity	pH	Normality
100.00		1.0	0.1
50.00	Hyper-acidity	1.3	0.05
15.00	Normal Acidity	1.8	0.015
1.00	Low Acidity	3.0	0.001
0.0001	Relative Achlorhydria	7.0	0.0000001
0.0000032	True Achlorhydria	8.5	0.0000000032

Electrolytes: The electrolytes in gastric secretion have been extensively studied with very similar results. It has been shown that the concentration of total chlorides in gastric juice under basal conditions parallel closely that of blood, but following histamine stimulation rise to an average of 140 milliequivalents per liter with the peak being 170 milliequivalents per liter. This rise whows a direct linear relationship between acid concentration and total chloride. A similar relationship is found following

Hisalog stimulation only to a lesser degree. Sodium has been shown to be almost inversely related to acid and chloride in a linear fashion. There are conflicting reports about the secrection of potassium but at present there does not seem to be any correlation with the changes in other electrolytes.[12,17,18,23,37,38,41,43] It is the opinion of most investigators that the most reproducible value would be that of total chloride following the augmented histamine test of Kay[26]. It is of additional importance in that if the value reported is below 125 milliequivalents it is thought that the gastric juice has been contaminated by other secretions.

Achlorhydria: In 1963 Rovelstad[49] reviewed the definitions of achlorhydria from its introduction by Michaelis as the absence of "free acid", to those recently proposed by Callender[8], and Card,[9] and it is evident that there is a great deal of confusion as to its meaning. From his discussion it would seem most desirable that the author describe the test and equipment used, as well as his definition of achlorhydria, at least until a standard universally acceptable definition is found. At present the most widely accepted definition of achlorhydria seems to be that of Card: "That state of gastric aecretion in which under the conditions of the test (Augmented histamine test of Kay) the pH of the secretion fails to fall below pH six following stimulation."[49] However, it would seem from the previous discussion of acidity and pH that the term "anacidity" as defined by Baron[2]: "A pH not less than seven --physico-chemical

neutrality--after the subcutaneous injection of four hundreths milligrams per kilogram body weight of histamine acid phosphate." would be preferred to the term achlorhydria.

One Hour Basal Gastric Analysis: The test to be discussed is a combination of a one hour basal and one hour augmented histamine test. The one hour basal (i.e. the juice secreted by the stomach in the absence of all intentional and avoidable stimulation)[49] gastric analysis consists of placing a nasogastric tube in the fundus of the stomach radiographically after a twelve hour overnight fast, removal and discarding the residium, and collection of secretions by continuous hand suction every 15 minutes for one hour, while having the patient lying on his left side and expectorating all saliva throughout the procedure. This test was developed as a result of the time, and patient discomfort involved in collecting nocturnal secretion. Its value was further enhanced by reports of its reproducibility in the same individual with considerable significance,[29,49,54,55] that the results of the one hour basal secretion test compare favorably with the results of the augmented histamine test in patients with duodenal ulcer. This increased secretion has been explained by a greater parietal cell mass.[4,7,24,54] Further correlation between the basal and augmented tests are those of the secretion being less in women than in men and that there is a decrease in acid output with increasing age which is most marked after the age of fifty.[3,7,30,,29]

Augmented Histamine Gastric Analysis: The second part of this procedure is based on the report by Kay in 1953 that increasing the dose of histamine acid phosphate above four hundreths milligram per kilogram body weight subcutaneously caused no further increase in gastric secretion or acid output.[26] This was further supported by Adam[1] in 1954. In performing the augmented histamine test, Kay used mypyramine maleate (Neoantergan) one hundred milligrams intramuscularly one half hour prior to giving the histamine. In reviewing the literature this antihistamine seems to be the one used most frequently. In a review article by Isaacson[25] it would seem that most antihistaminics have no effect on gastric secretion, although several have been reported to both increase and decrease secretion. In this same report is included a study of six patients with duodenal ulcer disease and abnormal gastric secretion, who were treated with chlorpheniramine maleate (Chlor-Trimeton) eight milligrams four times a day. These patients were followed weekly with repeat gastric analysis and after several weeks showed a return to a normal gastric secretory curve. Because of the time involved to bring about this change and because of the sedative effects of this drug it is this writers opinion that the use of chlorpheniramine maleate would not effect the gastric secretion during an augmented histamine test.

The use of this test is enhanced by the facts that it is reproducible in the same individual,[36,56] and that there is excellent

correlation between the maximal histamine response and the number of parietal cells in the stomach.[34,35,49]

Its primary usefullness is in the following areas. First, in the diagnosis of achlorhydria or anacidity. In 1955 Card[9] reported no cases of achlorhydria in patients other than those with pernicious anemia. The presence of achlorhydria does not however mean that the patient has pernicious anemia, but only indicates an absence of parietal cells capable of producing acid.[8,33] If achlorhydria is found in a patient with a gastric ulcer after repeated attempts to demonstrate acid secretion then the ulcer is malignant.[49] Secondly, the test is used serially to follow patients with ulcer disease. Thirdly, it is used as an indication of the type of surgery or medical therapy needed. Fourthly, it is useful in the elucidation of postgasterectomy dyspepsia, and hemorrhage. Absent or low acid output would virtually exclude a stomach or jejunal ulcer, while the higher the acid output these diagnosis become more likely.[31] Lastly, the ratio of the basal and maximal histamine secretions may help in the diagnosis of the Zollinger-Ellison syndrome.[35,48,49]

MATERIALS AND METHODS

The patients were selected from hospital patients at University Hospital, Douglas County Hospital, and Omaha Veterans Hospital. It was planned that at least five members of each sex and decade would

be included, but because of time and patient availability this was not possible. Patients with pregnancy, previous gastric resection, critically ill patients--especially those with cardiovascular disease, and those that might react adversely to the administration of histamine were excluded.

After a twelve hour overnight fast, a number sixteen Levin tube was introduced through the nose into the stomach, and the patient placed on the left side for the remainder of the procedure. To prevent contamination of the gastric juice the subjects were instructed to expectorate all saliva. The tube was taped in place where a maximum volume was withdrawn and this residium discarded. Following this, samples were collected every fifteen minutes for one hour by interrupted hand aspiration. After the second sample was collected, thirty milligrams of chlorpheniramine (Chlor-Trimeton) was given intramuscularly to reduce the systemic effects of the histamine which was given later. Following the drawing of the fourth sample four hundreths milligram per kilogram of histamine base was given subcutaneously, and four more samples collected at fifteen minute intervals.

The volume was measured and a three milliliter aliquot of each sample was checked for pH, and titrated with one tenth normal sodium hydroxide to a pH of seven electrometrically. The chloride content of each sample was obtained by using the Auto-Analyzer. The presence of bile and blood, as well as the systemic side effects of histamine were recorded.

The results were reported as follows. Basal: (1) Volume in milliliters per hour, (2) Acid output in milliequivalents per hour of hydrochloric acid, (3) Chlorides in milliequivalents per liter and milliequivalents per hour, (4) The average and individual pH. Histamine: (1) Maximum Volume as the greastest volume for a one-half hour period expressed as milliliters per hour, (2) Maximum Acid Output as the maximum acid output for a one-half hour period expressed as milliequivalents per hour. (3) Maxium Chloride as the chloride for a one-half hour period expressed as milliequivalents per hour. (4) The Maximum pH.

The arithmetic mean and the standard deviation for each sex and age group were calculated, as well as that for each sex.[11] Because of the small number of samples, the mixed population (patients with and without gastrointestinal symptoms and disease), and because the patients available tend to have chronic and multiple disease processes the results are not as significant. As a result the individual values of each case will be reported along with the diagnoses, and the results compared with those of recent investigators.[3,4,5,14,16,26,36]

RESULTS

Of forty-one patients (twenty-seven males and fourteen females) eighteen had aymptoms of gastrointestinal disease. Three male and three female patients had gastrointestinal bleeding--etiology unknown. Eight men and one woman had ulcer symptoms with negative

TABLE II
RESULTS OF STUDY
MEN

DISEASE	AGE	TEST	VOLUME	AVE. VOL.	pH	MAX. pH	AVG. pH	ACID OUTPUT	CHLORIDE mEq/L.	CHLORIDE mEq/Hr.	BILE	BLOOD
Chronic Duodenal	24	B	6.76		2.67				-	-	++	+
Ulcer symptoms			21.50		2.32				-	-	++	+
Mental Retardation			6.46		1.73				-	-	+	+
			2.85	8.17	1.45		2.04	1.34	-	-	+	+
		A	36.85		1.20				-	-	+	+
			89.65		1.15				-	-	+	+
			84.50		1.15				-	-	+	+
			87.64	355	1.06	1.06	1.10	39.8	-	-	+	+
Alcoholism	30	B	16.75		1.18				102	-	(-)	+
Pancreatitis			19.50		1.70				115	-	(-)	+
History of Peptic			22.55		1.50				97	-	(-)	+
Ulcer-neg. Radio-			10.25	69.1	1.54		1.48	4.20	80	6.97	(-)	+
graph.		A	41.50		1.21				68	-	(-)	+
			62.30		0.99				89	-	(-)	+
			61.15		1.11				107	-	(-)	+
			5.99	247	1.25	0.99	1.05	26.7	109	26.67	(-)	+
Asthma	30	B	0.10		0				-		+	+
Peptic Ulcer			0.10		0				-		+	+
			2.68		2.30				-		+	+
			2.30	5.18	2.32		2.66	0.06	-	-	+	+
		A	25.00		2.15				99		+	+
			40.35		1.64				105		+	+
			50.50		1.35				100		+	+
			70.60	242	1.36	1.35	1.35	17.7	99	24.22	+	+
Rheumatoid	32	B	1.32		2.50				-	-	(-)	+
Arthritis			4.00		2.50				-		(-)	+
			9.10		2.45				57	-	(-)	+
			4.25	187	2.52	.33	2.50	.33	69	1.18	(-)	+
		A	7.00		2.20				88	-	(-)	+

			29.50		1.30				87	-	(-)	+
			37.25		1.20				117	-	(-)	+
			55.55	186	1.22	1.20	1.21	7.45	100	20.03	(-)	+
Duodenal Ulcer	36	B	10		1.6				835	-	++	+
with internal			5		1.55				120.9	-	++	+
healing			4		1.55				133	-	++	+
X-ray Diagnosis			3	22	1.4		1.52	1.40	138	2.39	++	+
		A	18		1.4				159	-	+	+
			35		1.35				130	-	+	+
			50		1.20				133	-	+	+
			85	270	1.10	1.4	1.15	21.05	134	36.18	+	+
Chronic	39	B	1.48		6.40				108	-	++++	+
Bronchitis			1.56		6.50				117	-	++++	+
			2.60		7.01				116	-	++++	+
			7.66	13.3	4.01		5.78	0.02	102	1.43	++++	+
		A	37.5		1.08				114	-	+	+
			36.9		1.09				129	-	+	+
			72.75		1.00				135	-	+	+
			28.0	220.70	1.08	1.11	1.04	26.28	130	29.35	+	+
G.I. bleeding	42	B	26.25		2.54				120	-	(-)	+
Fatty Metamorph-			4.95		1.61				94	-	(-)	+
osis of Liver			48.0		2.10				99	-	(-)	+
Chronic Alcoholic			62.50	186.5	1.35		1.90	6.3	105	19.00	(-)	+
Hx. Chronic Ulcer		A	49.0		1.20				109	-	(-)	+
			58.0		1.12				85	-	(-)	+
			56.5		1.12				112	-	(-)	+
			49.6	229	1.10	1.10	1.10	21.0	89	22.67	(-)	+
Fibromyositis	42	B	15.5		1.15				101	-	+	+
upper back			7.8		1.25				95	-	+	+
Lipomata sub.Q			5.6		1.15				109	-	+	+
			12.1	40	1.15		1.17	3.2	105	4.18	+	+
		A	30		1.11				120	-	+	+
			45.5		1.08				114	-	+	+
			50		1.02				119	-	+	+
			76.5	253	0.99	0.99	1.00	32.3	116	29.85	+	+

Bronchitis	48	B	8.6		6.6				TLS	-	(-)	(-)
Steatorrhea			7.0		7.8				TLS	-	(-)	(-)
			6.5		7.5				TLS		(-)	(-)
			6.0	28.1	8.3		7.5	0.005	71	0.43	(-)	(-)
		A	15.0		8.50				79	-	(-)	(-)
			18.0		7.5				97	-	(-)	(-)
			16.5		2.30				96	-	(-)	(-)
			15.0	68	1.90	1.90	5.05	1.28	100	6.66	(-)	(-)
Small reducible	49	B	10		1.6				112	-	(-)	(-)
Hiatial hernia			7.5		1.6				114	-	(-)	(-)
and Poor diet			9.4		1.6				123	-	(-)	(-)
			4.6	14.75	1.7		1.62	1.48	122	3.70	(-)	(-)
		A	43.5		1.3				129	-	(-)	(-)
			48.6		1.15				131	-	(-)	(-)
			51		1.1				135	-	(-)	(-)
			45	199.2	1.1	1.1	1.10	21.3	133	28.49	(-)	(-)
Obesity	51	B	26.5		1.72				108	-	(-)	(-)
Sinusitis, frontal			10.9		1.85				97	-	(-)	(-)
No Ulcer			16.8		1.35				112	-	(-)	(-)
			18.7	72.9	1.21		1.52	3.3	109	7.84	(-)	(-)
		A	50		1.12				113	-	(-)	(-)
			85		1.00				115	-	(-)	(-)
			120		1.02				116	-	(-)	(-)
			225	740	0.99	0.99	1.00	88.92	121	87.32	(-)	(-)
Urinary tract	52	B	11.2		1.32				88	-	(-)	+
infection			10.1		1.30				95	-	(-)	+
			9.6		1.42				86	-	(-)	+
			11.0	41.9	1.45		1.37	2.44	90	3.77	(-)	+
		A	54		1.10				106	-	(-)	+
			100.0		1.10				117	-	(-)	+
			65.0		1.05				104	-	(-)	+
			112	424	0.98	0.98	1.01	51.2	101	53.25	(-)	+

Gastrointestinal	52	B	9.5		1.45				104	-	+	+
bleeding with			9.0		1.43				82	-	+	+
malena, history			12.5		1.40				104	-	(-)	+
of peptic ulcer			10.1	41.1	1.40		1.42	2.1	110	4.14	+	+
		A	30.5		1.25				88	-	(-)	+
			45		1.02				92	-	+	+
			87.5		1.00				116	-	+	+
			71	317	0.99	0.99	0.99	40.9	116	36.77	(-)	+
Malnutrition	53	B	0.2		0				0	-	-	-
Pul-emplysema			0.2		0				0	-	-	-
(mild)			0.1		0				0	-	-	-
Bronchitis			0.1	0.4	0		0	0	0	-	-	-
Diabetes adult		A	1.0		2.5				0	-	-	-
			2.0		2.10				51	-	-	-
			0		0				0	-	-	-
			3.50	8.0	3.55	2.10	2.82	.07	TLS	0.04	-	-
G. I. symptoms	54	B	5.6		5.50				114	-	+	(-)
gasoeusnem and			11.5		5.45				104	-	+	(-)
belches relieved			2.7		5.50				85	-	+	(-)
by antacids			1.5	21.3	5.35		5.45	0.04	106	-	+	(-)
possible peptic		A	15		3.70				130	-	(-)	(-)
ulcer			32		1.10				106	-	(-)	(-)
			45		0.92				123	-	(-)	(-)
			90	270	0.92	0.92	0.92	34.4	132	34.29	(-)	(-)
Leriche	59	B	11.54		5.00				101	-	++	(-)
syndrome			3.63		5.60				125	-	+	(-)
Folliculitis			3.10		6.65				118	-	+	(-)
			4.50	25.50	6.10		5.84	0.03	121	2.89	+	(-)
		A	28.45		1.56				118	-	(-)	(-)
			30.02		1.20				121	-	(-)	(-)
			38.95		1.10				134	-	(-)	(-)
			28.86	135.62	1.06	1.10	1.08	15.28	135	18.30	(-)	(-)

Adenocarcinoma	71	B	0.1		7.2				0	-	(-)	(-)
of stomach			0.1		7.20				0	-	(-)	(-)
BPH			0.2		7.25				0	-	(-)	(-)
Generalized			0.1	0.5	7.5		7.29	-	0	-	(-)	(-)
Arteriosclerosis		A	45.5		7.5				85	-	(-)	(-)
			46.5		7.5				64	-	(-)	(-)
			16		7.5				96	-	(-)	(-)
			14.5	184	7.5	7.2	7.5	-	102	18.22	(-)	(-)
Parkinsonism	72	B	1.5		3.4				89	-	+	(-)
Pulmonary			1.0		3.7				92	-	+	(-)
emphysema			1.0		3.4				104	-	+	(-)
			0.5	4.0	3.35		3.46	0.09	89	.28	+	(-)
		A	20		4.5				122	-	+	(-)
			15		2.2				85	-	+	(-)
			10		1.8				124	-	+	(-)
			40	120	1.85	1.8	1.82	3.1	112	14.16	+	(-)
BPH	74	B	9.0		1.82				95	-	(-)	(-)
History of			7.3		1.80				93	-	(-)	(-)
D.U. neg. X-ray			5.1		1.8				92	-	(-)	(-)
Pul. emphysema			1.0	22.4	1.65		1.77	0.9	80	2.08	(-)	(-)
		A	35		1.51				123	-	(-)	(-)
			87		1.28				121	-	(-)	(-)
			75		1.22				134	-	(-)	(-)
			93	360	1.20	1.8	1.21	31.7	100	42.12	(-)	(-)
	75	B	3.2		2.60				TL	-	(-)	(-)
			12.5		1.72				68	-	(-)	(-)
			8.6		1.50				85	-	(-)	(-)
			2.8	27.1	1.40		1.80	1.1	98	-	(-)	(-)
		A	50.0		1.12				105	-	(-)	(-)
			51.0		1.05				92	-	(-)	(-)
			47		1.02				102	-	(-)	(-)
			84	270	1.02	1.02	1.02	32.4	87	-	(-)	(-)

Thyroid	60	B	4.25		2.74				88	-	+	+
Pulmonary			4.7		3.1				87	-	+	+
emphysema			2		3.12				114	-	+	+
			2.5	13.45	3.3		3.06	0.16	120	1.31	+	+
		A	20.1		1.10				102	-	+	+
			26.5		1.32				132	-	+	+
			34.5		1.07				111	-	+	+
			26.6	122.2	1.06	1.06	1.06	13.52	110	13.44	+	+
ASHD with	66	B	16.25		1.4				122	-	(-)	+
Aur. Fib.			13.1		1.42				86	-	(-)	+
Hx. Chronic			5.6		1.5				73	-	(-)	+
D. U.			10.5	45.5	1.48		1.45	2.91	89	4.46	(-)	+
BPH		A	56.45		1.2				114	-	(-)	+
generalized			112.5		1.12				97	-	(-)	+
			127.95		1.12				120	-	(-)	+
			122.5	510.9	1.08	1.08	1.10	55.0	90	56.65	(-)	+
Gastric ulcer												
(low) X-ray	68	B	17.1		5.65				119	-	+++	(-)
			5.35		5.70				117	-	+++	(-)
			15.52		3.01				116	-	+++	(-)
			12.68	50.7	2.62		4.24	1.22	120	7.25	+++	(-)
		A	0		0				0	-	+++	(-)
			16.40		1.48				114	-	+++	(-)
			28.90		31.78				117	-	+++	(-)
			29.42	116.64	23.11	1.12	1.19	11.3	-	13.41	+++	(-)
Collagen	69	B	1.5		2.20				122	-	(-)	+
Disease			3.25		2.20				127	-	(-)	+
Rheumatoid			2.5		2.00				118	-	(-)	+
Arthritis			1.75	9.00	1.75		2.04	0.3	115	1.09	(-)	+
		A	51		1.20				117	-	(-)	+
			43		1.12				114	-	(-)	+
			62		1.12				113	-	(-)	+
			48	220	1.05	1.05	1.08-	17.6	112	24.64	(-)	+

Pul. emphyseme	76	B	6.2		1.7				55	-	(-)	(-)
RUQ pain etiol.			1.71		1.71				84	-	(-)	(-)
unknown			6.3		1.7				74	-	(-)	(-)
BPH			2.4	22	1.7		1.70	1.4	74	2.43	(-)	(-)
Gen. Art.		A	43.5		1.5				105	-	(-)	(-)
			25.6		1.45				107	-	(-)	(-)
			53		1.35				112	-	(-)	(-)
			74.5	255	1.20	1.5	1.27	21.6	106	27.80	(-)	(-)
CHF; ASHD;	77	B	5.5		1.80				TL	-	+	+
Anemia-etiol.			3.2		1.65				TL	-	+	+
Diabetic			2.1		1.50				TL	-	+	+
			1.5	12.3	1.52		1.61	.5	TL	-	+	+
		A	45		1.4				TL	-	+	+
			40		1.00				TL	-	+	+
			48		0.92				124	-	+	+
			88	372	0.92	0.92	0.92	39.0	118	44.64	+	+
Peptic Ulcer	78	B	2.2		1.7				82	-	(-)	(-)
			1.7		9.1				114	-	(-)	(-)
			1.65		11.7				105	-	(-)	(-)
			1.67	34.4	11.4		1.68	1.4	113	3.74	(-)	(-)
		A	1.31		76.5				107	-	(-)	(-)
			1.08		81				119	-	(-)	(-)
			1.00		59.5				128	-	(-)	(-)
			1.10	365	101.5		1.05	41.2	128	46.70	(-)	(-)

TABLE II
RESULTS OF STUDY
WOMEN

DISEASE	AGE	TEST	VOLUME	AVG. VOL.	pH	MAX. pH	AVG. pH	ACID OUTPUT	CHLORIDE mEq/L.	CHLORIDE mEq/Hr.	BILE	BLOOD
G. I. bleeding	26	B	4.5		1.5				120	-	(-)	Flecks in all samples
etiol. unknown			6.0		1.55				130	-	(-)	
2^{0} Fe definciency			7.5		1.5				89	-	(-)	
anemia			5.5	23.5	1.45		1.50	1.36	115	2.62	(-)	
		A	31		1.3				112	-	(-)	
			45		1.3				118	-	(-)	
			41		1.2				111	-	(-)	
			47	184	1.15	1.15	1.18	17.91	112	20.42	(-)	
Acute Gastritis	29	B	8.50		2.72				Lost	-	(-)	(-)
			15.0		1.25				Lost	-	(-)	(-)
			30		1.12				Lost	-	(-)	(-)
			21	55.6	1.10		1.55	4.8	Lost	-	(-)	(-)
		A	16.1		1.10				Lost	-	(-)	(-)
			33		1.02				Lost	-	(-)	(-)
			5.5		1.03				Lost	-	(-)	(-)
			50.5	167	1.00	1.00	1.01	11.06	Lost	-	(-)	(-)
Osteogenisis	38	B	14		1.55				113	-	(-)	(-)
imperferta			2		1.50				-	-	(-)	(-)
Athetous			10		1.40				124	-	(-)	(-)
			2	28	1.4		1.46	1.77	-	3.30	(-)	(-)
		A	2		1.45				-	-	(-)	(-)
			25		1.2				129	-	(-)	(-)
			34		1.2				131	-	(-)	(-)
			26		.8	.8	1.0	12.94	127	15.48	(-)	(-)
X-ray neg. for	39	B	20.5		3.92				121	-	++	(-)
Ulcer			15.25		3.98				109	-	++	(-)
Iron def. Anemia			20.5		3.82				92	-	++	(-)
			25.0	81.25	3.02		3.68	0.8	92	8.33	++	(-)
		A	25.5		1.38				110	-	++	(-)
			37.18		1.10				118	-	++	(-)
			44.6		1.10				104	-	++	(-)
			40.25	169.7	1.08	1.08	1.09	17.02	102	17.49	++	(-)

Pul. emphysema	47	B	11.0		2.50				101	-	+	(-)
Diabetes mellitus			2.75		3.00				95	-	+	(-)
Convulsive disorder			0.5		3.50				0	-	+	(-)
2° Hysteria			2.80	17.05	7.80		4.20	0.16	100	1.70	+	(-)
Psychoneurosis		A	3.65		4.50				111	-	(-)	(-)
conversion			17.5		1.50				120	-	(-)	(-)
			20.0		1.18				117	-	(-)	(-)
			33.5	107	1.15	1.15	1.16	9.36	117	18.25	+	(-)
Hypertension	49	B	6.10		1.54				115	-	+	(-)
Digitalis			6.5		1.58				104	-	+	(-)
Intoxication(mild)			6.85		1.56				108	-	+	(-)
			1.35	20.8	1.48		1.54	1.12	106	2.26	+	(-)
		A	7.45		1.32				107	-	+	(-)
			1.6		1.24				126	-	+	(-)
			1.2		1.20				106	-	+	(-)
			0	29.8	-	1.20	1.22	2.2	-	3.46	-	-
Obesity	51	B	3.4		6.2				TLS	-	++	+
Laennec			4.8		6.2				81	-	++	+
cirrohsis			5.6		6.5				78	-	++++	+
			2.8	16.60	2.3		5.24	.06	78	1.32	+++	+
		A	20		1.12				104	-	+	+
			35		1.02				114	-	+	+
			42		1.09				102	-	+	+
			46	176	1.00	1.00	1.04	21.4	118	19.36	+	+
Obesity	54	B	2.76		1.72				127	-	++	(-)
			0		0				0	-	++	(-)
			0.69		1.65				98	-	++	(-)
			2.06	5.51	1.63		1.67	0.026	110	.65	++	(-)
		A	5.8		1.53				120	-	++	(-)
			31.90		1.41				88	-	+++	(-)
			34.50		1.10				129	-	+++	(-)
			21.50	132.8	1.01	1.01	1.05	15.84	135	17.66	++	(-)

Obesity	56	B	1.65		4.50				63	-	(-)	(-)	
			21.2		3.00				TLS	-	(-)	(-)	
			18.0		3.10				60	-	(-)	(-)	
			1.40	42.25	2.9		3.37	.55	71	2.62	(-)	(-)	
		A	30.5		1.72				96	-	(-)	(-)	
			45		1.32				108	-	(-)	(-)	
			55		1.21				104	-	(-)	(-)	
			70.5	251.6	1.15	1.15	1.18	13.6	88	24.15	(-)	(-)	
Generalised Arteriosclerosis Obesity	59	B	2.5		3.75				73	-	+	(-)	
			7.5		1.85				90	-	+	(-)	
			4.5		1.80				95	-	+	(-)	
			4.0	18.5	2.00		2.70	0.61	84	0.73	+	(-)	
		A	47.5		1.40				123	-	+	(-)	
			63.2		1.30				132	-	+	(-)	
			49		1.30				134	-	+	(-)	
			25	224.4	1.20	1.20	1.25	20.2	131	29.62	+	(-)	
Bleeding from G. I. tract	62	B	12		Lost				0	-	(-)	+	
			4		Lost				0	-	(-)	+	
			1		Lost				0	-	(-)	+	
			3	-	Lost		-	-	0	-	(-)	+	
		A	85		1.00				0	-	(-)	+	
			85		1.01				129	-	(-)	+	
			80		1.05				114	-	(-)	+	
			70	340	.99	.99	1.02	39.8	125	40.46	(-)	+	
Digitalis intoxication Situational depression	69	B	3.55		2.10				92	-	+	(-)	
			2.60		2.68				88	-	+	(-)	
			2.35		3.20				79	-	+	(-)	
			3.76	11.26	3.06		2.76	.16	79	-	+	(-)	
		A	2.56		2.91				78	-	(-)	Flecks	
			16.58		1.41				111	-	(-)		
			16.50		1.19				115	-	(-)		
			19.56	72.1	1.10	1.10	1.14	6.8	106	-	(-)		

Pul. emphysema	75	B	1.10		2.80				71	-	(-)	(-)
Mild nutritional			.3		TL				-	-	(-)	(-)
deficiency			.2		TL				-	-	(-)	(-)
			.1	1.7	TL		2.08	0.016	-	.12	(-)	(-)
		A	.1		TL				-	-	(-)	(-)
			.6		1.42				-	-	(-)	(-)
			1.0		1.25				114	-	(-)	(-)
			.2	4.0	-	1.42	1.33	0.26	113	.46	(-)	(-)
Hypothyroidism	79	B	1.50		6.5				79	-	+++	+
History of nausea			2.7		6.6				84	-	+++	+
and vomiting			3.40		6.5				96	-	+++	+
			8.50	15.1	6.5		6.52	.026	107	1.59	+++	+
		A	4.25		1.37				111	-	++	+
			38		1.15				123	-	++	+
			88		1.15				114	-	++	+
			74.5	325	1.15	1.15	1.15	28.0	121	38.13	++	+

SECRECTION IN cc/hour

AGE Men	No.	BASAL Range	Mean	S.D.	AUGMENTED Range	Mean	S.D.
20-29	1	81.7	8.17	0	354.58	354.58	0
30-39	5	5.18-69.05	25.64	25.08	185.5-270.0	282.6	64.22
40-49	4	14.75-186.5	66.84	25.31	68-253	187.3	83.32
50-59	6	0-72.9	37.6	24.89	8-740	329.1	253.6
60-69	4	9-50.7	19.4	24.52	116.64-510.9	282.4	132.5
70-79	7	0.5-34.4	17.5	10.25	120-372	275	96.13
AVERAGE TOTAL	27	0-186.5	29.19	18.34	8-740	285.16	104.96
Women							
20-29	2	23.5-55.6	39.5	16.0	167-184	175.5	12.04
30-39	2	28-81.3	54.6	26.65	120-169.7	144.8	24.8
40-49	2	17.05-20.08	18.6	1.50	10.7-29.8	20.25	9.55
50-59	4	5-51-42.25	20.72	15.29	132.8-251.6	198.7	51.96
60-69	2	11.26	11.26	0	72.1-340	206	134
70-79	2	1.7-15.1	8.4	6.7	4.0-325	164.5	160.5
AVERAGE TOTAL	14	1.7-81.3	24.84	11.02	4.0-325	152.29	65.47

AVERAGE BASAL pH AND MAXIMAL AUGMENTED pH

AGE	No.	BASAL			AUGMENTED		
Men		Range	Mean	S.D.	Range	Mean	S.D.
20-29	1	2.04	2.04	0	1.10	1.10	0
30-39	5	1.48-5.78	2.79	1.76	1.04-1.35	1.16	.57
40-49	4	1.17-7.5	3.06	3.98	1.00-5.05	2.06	1.97
50-59	6	1.37-5.84	3.12	2.23	0.92-2.82	1.30	0.80
60-69	4	1.45-4.24	2.70	1.19	1.06-1.19	1.11	0.07
70-79	7	1.61-7.29	2.76	1.15	0.92-7.50	2.11	2.24
AVERAGE TOTAL	27	1.17-7.50	2.74	1.72	0.92-7.50	1.47	0.94
Women							
20-29	2	1.50-1.55	1.52	0.036	1.01-1.18	1.09	0.12
30-39	2	1.46-3.68	2.57	1.57	1.00-1.09	1.04	0.02
40-49	2	1.54-4.20	2.87	1.88	1.16-1.22	1.19	0.134
50-59	4	1.67-5.24	3.24	1.70	1.04-1.25	1.13	0.001
60-69	2	2.76	2.76	0	1.02-1.14	1.08	0.084
70-79	2	2.08-6.52	2.30	4.21	1.15-1.33	1.24	0.127
AVERAGE TOTAL	14	1.46-6.54	2.54	1.57	1.00-1.33	1.13	0.081

TITRATABLE ACIDITY TO pH 7 in Meq/hour

AGE	No.	BASAL			AUGMENTED		
Men		Range	Mean	S.D.	Range	Mean	S. D.
20-29	1	1.34	1.34	0	39.8	39.8	0
30-39	5	0.02-4.2	1.20	1.79	7.45-26.7	19.83	5.6
40-49	4	0.01-6.3	2.75	2.83	1.28-32.3	18.97	12.72
50-59	4	0-3.3	1.98	1.41	0.07-88.9	49.03	22.8
60-69	3	0.16-.3	1.12	1.40	13.5-55	28.7	22.8
70-79	6	0-1.4	0.67	1.28	0-39	25.9	13.75
AVERAGE TOTAL	23	0-6.3	1.51	1.45	0-88.9	30.37	13.67
Women							
20-29	2	1.36-4.8	3.08	2.40	11.06-17.9	14.48	4.81
30-39	2	.8-1.77	1.26	0.68	12.94-17.02	14.98	5.51
40-40	2	9.16-1.12	0.64	1.57	2.2-9.36	5.78	5.04
50-59	4	.03-.61	0.31	0.98	13.6-21.4	17.76	3.82
60-69	2	0-.16	0.08	0.036	6.8-39.8	23.3	23.32
70-79	2	.02-.03	0.02	0.00	.26-28.0	14.13	19.49
AVERAGE TOTAL	14	0-4.8	0.88	0.99	0.26-39.8	15.06-	10.66

CHLORIDES MEQ/hour

AGE	No.	BASAL			AUGMENTED		
Men		Range	Mean	S. D.	Range	Mean	S. D.
20-29	0	None	None	None	None	None	None
30-39	5	1.18-6.97	2.99	2.78	20.03-36.18	27.29	7.12
40-49	4	0.43-19.00	6.82	8.25	6.66-29.85	21.92	11.62
50-59	6	1.13-7.84	3.95	2.08	0.04-87.32	38.33	32.32
60-69	4	1.09-7.25	3.52	2.91	13.41-56.65	27.03	20.52
70-79	6	0.28-3.74	2.13	1.42	14.16-46.70	32.27	18.92
AVERAGE							
TOTAL	25	0.28-19.00	3.88	3.49	0.04-87.32	29.88	18.10
Women							
20-29	1	2.62	2.62	0	20.42	20.42	0
30-39	2	3.3-8.33	5.81	2.51	15.48-17.49	16.48	1.00
40-49	2	1.70-2.26	1.98	.28	3.46-12.52	7.99	4.53
50-59	4	0.65-2.62	1.33	0.92	17.66-29.62	22.69	17
60-69	2	1.05	1.05	0	7.93-40.46	24.19	16.27
70-79	2	0.12-1.59	0.85	0.74	0.46-38.13	19.29	18.83
AVERAGE							
TOTAL	2	0.12-832	2.25	1.08	0.46-40.46	18.51	11.52

TABLE VII

MEN

	BASAL				AUGMENTED			
	No.	Range	Average	S.D.	No.	Range	Mean	S.D.
Vol.	27	0-186.5	29.19	18.34	27	8-740	285.16	104.96
pH	27	1.17-7.5	2.74	1.72	27	0.92-7.50	1.47	0.94
Titratable Acidity	23	0-6.3	1.51	1.41	23	0-88.9	30.37	13.67
Chlorides mEq/hr.	25	0.28-19.00	3.88	3.49	25	.04-87.32	29.77	18.10

WOMEN

	BASAL				AUGMENTED			
	No.	Range	Average	S.D.	No.	Range	Mean	S.D.
Vol.	14	1.7-81.3	24.84	11.02	14	4-325	152.29	65.47
pH	14	1.46-6.54	2.54	1.57	14	1.0-1.33	1.13	0.08
Titratable Acidity	14	0-4.8	0.88	0.99	14	0.26-39.8	15.06	10.66
Chlorides mEq/hr.	13	0.12-8.33	2.25	1.08	13	0.46-40.46	18.51	11.52

This is a summary of tables III-VI illustrating the relationships between men and women for the total group irrespective of age.

radiographic evidence of ulcer disease. One male was found to have adenocarcinoma of the stomach and was subsequently operated upon successfully. Two males had radiographic evidence of ulcer--one duodenal and one gastric. The remainder of the patients had diseases not referable to the gastrointestinal tract, twenty-three of forty-one total.

In table II the results of each individual gastric analyis is tabulated. This was divided into male and female and the disease, age, test (basal or augmented histamine), volume, average and maximum volume, pH, average pH, Maximal pH, acid output, chloride, bile, and blood were recorded. Not included in this table were the comments about the patients reaction to the test and in particular their reaction to the histamine. All of the patients were somewhat apprehensive prior to passing the tube, but were much calmer to all appearances after the tube had been down a few minutes, and were resting quietly following the aspiration of the fasting sample. Following the administration of Chlor-Trimiton, many of the subjects went to sleep. During the histamine stimulation all of the patients were flushed and perspiring slightly, and in eleven patients there was profuse sweating, flushing, severe headache, and feelings of anxiety. It was also noted that if the subjects stood up immediately following the completion of of the test that they complained of feeling light-headed and had a marked decrease in blood pressure. This was not as significant

if they first sat up for a few minutes, prior to standing. Two subjects had a marked drop in blood pressure--fifty millimeters of mercury systolic. Both were difficult to evaluate because of a heavy emotional overlay. This effect could have been due to either the histamine or the chlorpheniramine maleate, since either in large doses could cause this effect. It is however most likely due to the histamine.

In table III the results of the volume of secretion for both the basal and augmented test are tabulated according to sex and age, as well as the number of patients in each age group, the mean and standard deviation of decade and for the men and women.

In table IV the average basal pH and maximal augmented pH are recorded by sex, by decades, and the numbers of patients in each group, the range, mean, and the standard deviation of each decade according to sex.

In table V the titratable acidity to pH seven in milliequivalents per hour are recorded in the same manner as the volume, and pH in tables III and IV.

In table VI the chlorides in milliequivalents per hour are reported in a similar manner.

In table VII the results of tables II through VI were tabulated according to sex, giving the volume, pH, titratable acidity, chloride, number of patients, range, mean, and standard deviation of each.

DISCUSSION

The results of this small group of patients supports the fact that women secret less gastric juice than men and have less acid output. For men the mean basal secretion was 29.19 (range 0-186.5; S.D. 18.34) and a mean histamine secretion of 285.16 (range 8-740; S.D. 104.96). While for women the mean basal secretion was 24.84 (range 1.7-81.3; S.D. 11.02) and a mean histamine secretion of 152.29 (range 4-325; S.D. 65.47). The titratable acidities showed a similar drop as shown by table VII.[3,5,7,15,25,26,28,33,36,50,54] In most of the studies relating age to gastric secretion there has been shown a decrease according to age which is more marked and more clearly demarcated after the age of fifty.[3,15,32,33,36,42] In this study there was no clear separation in any of the several components studied until after the age of sixty. This is most probably due to the fact that these were chronically ill hospital patients with poor nutrition, and to the small number of subjects in each decade.

There were no patients on medications whose effect were long enough to have any effect on the basal secretion. The patients did not receive their medications after the four P.M. dose the day before the test.

The evaluation of the chlorides was difficult because they were reported only by concentration in the literature. In this case both concentration (in milliequivalents per liter) and total chloride output (in milliequivalents per hour) were reported. The results of the basal values are very close to normal, which is

the same as that for blood. It is also observed from table II that there is arise in chloride, but not in proportion to the rise in acidity following histamine stimulation. This rise and the total peak of chlorides reached is not uniform nor as high as that observed by others.[12,17,43] The peak values in this study were forty milliequivalents per liter less than the top reported by other investigators. This indicate by the previouly discussed criteria that the gastric juice was contaminated, either by saliva or bile.

The following table will illustrate the maximum acid output (MAO) of this study compared with that of other studies. This

Authors	Males			Females		
	No.	Mean	Range	No.	Mean	Range
Kay (1953)	27	22.20	10.10-34.6			
Bruce,et,al.(1959)	14	22.40		18	14.60	0.10-31.30
Marks,et.al.(1959)	31	23.20	10.10-41.5	15	15.00	0.10-31.30
Card (1960)	29	22.65		28	17.20	6.20-34.60
Doteva11 (1961)	30	23.30	10.70-77.4	12	17.70	
Marks (1962)	94	25.10	10.70-77.0			
Baron (1963)	15	28.80		21	17.10	2.57-46.75
Bock (1963)		28.00	10.70-77.0		17.10	2.57-46.75
This Study	23	30.37	0-88.9	14	15.06	0.26-39.80

would indicate that the acid output in the males is higher than average and that that of the females is on the low end of the scale. However, this is not a valid comparison, in that the maximum values in each case were calculated by using different endpoints, and for different periods of time after stimulation. As a rough index of maximum acid output this study compares fairly well for overall values for both men and women, leaving out the relationship of age and acidity.

The acidity as measured by pH was in the normal range as per the limits listed in table I for the basal test, but not for the augmented histamine test. It is apparent from these data that additional material and studies of both normal and diseased states are necessary, before any definitive limits for normal can be defined for the augmented histamine test. In this study there was one achlorhydric patient as previously defined, who had adenocarcinoma of the stomach. The lowest pH was found in a patient without ulcer symptoms, or radiographic evidence of disease. All of the patients with gastrointestinal symptoms had a marked reduction in pH after stimulation and were among the lowest values found. This would be additional evidence of an ulcer diathesis in these patients.

CONCLUSION

The one hour basal and augmented histamine gastric analysis described, is a simple method of obtaining quantitative and reproducible results when the results are expressed in standard chemical units per hour.

It would also seem that the addition of pH and total chloride determinations to the list of other values routinely obtained would increase the reliability and usefulness of this test, and with the accumulation of more data they may prove to be of more importance.

The reporting of the results of further investigations using

this procedure as to sex, age, and disease would help to make the results of such procedures of more value.

The terms "free acid", "combined acid", and "total acid" should be abolished, and the pH and maximum acid output (MAO) be used for expressing acidity and acid output respectively.

The definition of achlorhydria given is of primary importance in the diagnosis of pernicious anemia, and in the detection of carcinoma of the stomach.

SUMMARY

In this study the following topics were presented and discussed. First, the methods of expressing electrolytes, acidity, and particularly the use of pH, and total chlorides. Second, the need for the development of a standard quantitative method of gastric analysis. Third, the definition of achlorhydria. Fourth, the relationship of age and acidity, Last the relationship of a standardized basal (interdigestive) and maximal (histamine) gastric analysis in normal and disease states were discussed, as well as the results of using this test in forty-one patients and their comparison with previously reported results for a similar test.

BIBLIOGRAPHY

1. Adam,H.M.,and others, Dose-response curves for effect of histamine on acid gastric secretion in man. Brit. Phar.,9:329,1954.

2. Baron,J.H., Editorial, Measurement and Nomenclature of Gastric Acid. Gastro. 45:118,1963.

3. Baron,J.H., Studies of basal and peak acid output with an augmented histamine test. Gut 4:136,1963.

4. Baron,J.H., An assessment of the augmented histamine test in the diagnoses of peptic ulcer. Gut 4:243,1963.

5. Baron,J.H., The relationship between basal and maximum acid output in normal subjects and patients with duodenal ulcer. Cl. Sc. 24:357,1963.

6. Bock,O.A.A., The concepts of "free acid" and "total acid" of the gastric juice. Lancet 2:1101,1962.

7. Bolo,P.O., Correlation of basal gastric secretion with peptic ulcer activity. Amer.J.Dig.Dis., 6:784,1961.

8. Callender,S.T.,and others, The augmented histamine test with special reference to achlorhydria. Gut 1:326,1960.

9. Card,W.I. and others, Observations on achlorhydria. J. Physiol. 130:18,1955.

10. Capper,W.M., The pH fields of the gastric mucosa. Lancet 2:1200,1962.

11. Cowden,D.J. and Croxton,F.E., APPLIED GENERAL STATISTICS, Prentiss-Hall, Inc.,1950.

12. Gray,J.S., and others, The relationships between total acid and neutral chlorides of gastric juice. Amer. J. Physiol. 132:504,1941.

13. Grossman,M.I., The pathologic physiology of peptic ulcer. Amer. J. Med. 29:748,1960.

14. Grossman,M.I. and Ventzke,L.E., Response of patients with duodenal ulcer to augmented histamine test as related to blood groups and to secretor status. Gastro. 42:292,1962.

15. Grossman,M.I. and others, Comparison of histalog and histamine as stimulants for maximal gastric secretion in human subjects and in dogs. Gastro. 44:620,1963.

16. Grossman,M.I. and others, Basal and Histalog-stimulated gastric secretion in control subjects and in patients with peptic ulcer or gastric cancer. Gastro. 45:14,1963.

17. Hirschowitz,B.I., Electrolytes in human gastric secretion; observations and a theory. Amer.J.Dig.Dis. 6:199,1961.

18. Hollander,F., Studies in gastric secretion. Statistical analysis of the neutral chloride-hydrochloric acid relation in gastric juice. J. Biol. Chem. 125:161,1931.

19. Hollander,F., An explanation of the various measure of acidity used in this investigation. J.Biol.Chem. 91:482,1931.

20. Hollander,F., Studies in gastric secretion. VI. A statistical analysis of the neutral chloride-hydrochloric acid relation in gastric juice. J.Biol.Chem. 97:585,1938.

21. Hollander,F., What constitutes effective neutralization of gastric contents. Amer.J.Dig.Dis. 6:127,1939.

22. Hollander,F., What is PH? An explanation of the various measures of acidity employed in gastroenterology. Gastro. 4:497, 1945.

23. Hollander,F. and Colcher,H., Evidence for the independence of K and HCl efflux into gastric secretion. Amer.J.Physiol. 1960.

24. Hunt,J.N. and Kay,A.W., The nature of gastric hypersecretion of acid in patients with duodenal ulcer. Brit.Med.J. 2:1444 1954.

25. Isaacson,J. and others, Some experiences with ChlorTrimeton in the treatment of duodenal ulcer. Med.Ann.District of Columbia. 20:63,1951.

26. Kay,A.W., Effect of large doses of histamine on gastric secretion of HCl; on augmented histamine test. Brit. Med. J. 2:77,1953.

27. Levin,E. and others, The variablity and periodicity of the nocturnal gastric secretion in normal individuals. Gastro. 10:939,1948.

28. Levin,E. and others, Nocturnal gastric secretion studies on normal subjects and on patients with duodenal ulcer, gastric ulcer and gastric carcinoma. Arch. Surg. 56:345,1948.

29. Levin,E. and others, A simple measure of gastric secretion in man, comparison of one hour basal secretion, histamine secretion and twelve hour noctural gastric secretion. Gastro. 19:88,1951.

30. Levin,E. and others, Variations in basal gastric secretions in man and the evaluation of gastric secretory stimulants. Gastro. 30:779,1956.

31. Marks,I.N., The augmented histamine test. Editorial. Gastro. 41:599,1961.

32. Marks,I.N. and others, The rationale of selective surgery in the treatment of duodenal ulcer. J.Roy.Coll.Surg.Edinburgh 4:85,1959.

33. Marks,I.N. and Shay,H., Augmented histamine test, Ewald test meal, and Diagnex test. Comparison of results. Amer.J. Dig.Dis. 5:1,1960.

34. Marks,I.N. and others, Maximal acid secretory response to histamine and its relationship to parietal cell mass in the dog. Amer.J.Physiol. 199:579,1960.

35. Marks,I.N. and Card,W.S., The relationship between the acid output of the stomach following "maximal" histamine stimulation and the parietal cell mass. Clin.Sci. 19:147,1960.

36. Marks,I.N. and others, The augmented histamine test. So. African Med. J. 36:807,1962.

37. Martin,L., The relationship of potassium to the electrolytes and to the proteins of the gastric juices of man. Gastro. 15:326,1950.

38. Martin,L., The electrolytes before and then changes at various periods after histamine stimulation. Ann.Int.Med. 6:91, 1933.

39. Michaelis,L., Some problems concerning the gasric juice. In the Harvey Lectures, p.59, 1926-27, from Bock,O.A.A. Lancet 2:1101,1962.

40. Milton,G.W. and others, Histamine secretory tests in cats; on evaluation of results based upon Hollander's two-component theory. Gastro. 44:642,1963.

41. McLean,H.W. and others, Variations in the acidity and total chloride contained the secretion from an isolated Pavlov pouch in the dog. J. Physiol. 65:77,1928.

42. Murray,F.A. and others, Gastric secretion in pregnancy. J. Obstet. and Gyn. Brit. Emp. 64:373,1957.

43. Neves,D.P. and Zaterka,S., The electrolytes in gastric secretion after histamine and histalog stimulation. Amer. J. Dig. Dis. 7:545,1962.

44. Piper,D.W., Gastric Secretion. Aust. Ann. Med. 12:171,1963.

45. Polland,W.S., Histamine test meals; on analysis of 983 consecutive tests. Arch. Intern. Med. 51:903,1933.

46. Prout,W. On the nature of acid and asline matters usually existing in the stomach of animals. Phil. Trans. Roy. Soc. 1 and 2, 45,1824.

47. Rider,J.A., Gastric Analysis; diagnostic and therapeutic uses. Western Med. 4:84,1963.

48. Roth,J.L.A. and Bockus,H., Why do a gastric analysis? I. Theoretical considerations. II. Uses of varous procedures. Gastro. 18:546,1951.

49. Rovelstad,R.A., Gastric analysis. Progress in Gastroenterology. Gastro. 45:90,1963.

50. Segal,H.L., Clinical measurement of gastric secretion significance and limitations. Ann. Int. Med. 53.1:445,1960.

51. Shohl,A.T., Determination of the acidity of gastric contents. I. Determination and significance of acidity. Bull. Johns Hopkins Hosp. 31:152,1920.

52. Shay,H. and others, Some fallacies in the clinical measurement of gastric acidity with special reference to the histamine test. Gastro. 15:110,1950.

53. Sörensen,S.P.L., quoted from Bock,O.A.A. The concepts of "free acid" and "total acid" of the gastric juice. Lancet 2:1101,1962.

54. Sun,D.C.H. and Shay,H., Basal gastric secretion in duodenal ulcer patients. Its conderation in evaluation of gastric secretory inhibitants or stimulants. J.Applied Physiol. 11:148, 1957.

55. Sun,D.C.H. and others., Relative effectiveness of anticholinergic drugs on basal gastric secretion. J.A.M.A. 158:713.

56. Sircus,W., The surgery of peptic ulcers. In peptic ulceration-a symposium for surgeons, edited by C. Wells and J. Kyle. from Gastro. 41:602,1961.