

## CORRELATION BETWEEN CHLORIDES OF BLOOD AND ACIDITY OF THE GASTRIC JUICE

by

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The presence of hydrochloric acid in the gastric juice and the high concentration that it attains in it is a remarkable fact that has excited much interest. A survey of literature reveals that the origin of hydrochloric acid in the stomach and the mechanism involved in it is still obscure. In a previous communication (4), a definite correlation between the amount of blood bicarbonate and the intensity of gastric acidity was observed—a persistently raised level of blood bicarbonate in hyperchlorhydria, a subnormal level in case of hypochlorhydria, and a normal level in isochlorhydria. It was also suggested that a raised blood bicarbonate level seem to be an etiological agent of some significance in development or persistence of hyperchlorhydria.

In the present paper an attempt has been made to determine the parent source of Cl<sup>-</sup> ions of the acid and to find out if there is any relation between chlorides of the blood and the acidity in the stomach. It was thought that a satisfactory correlation between chlorides of blood and intensity of hydrochloric acid secretion may throw some light on the mechanism of hydrochloric acid secretion by the stomach which may help to control the excessive acidity in the clinical disorders.

### MATERIAL AND METHOD

The cases were collected from the medical wards and the out-patients department. The patients came from the middle or lower income group of population. In all, 31 cases were investigated and they were equally distributed as regards hypochlorhydria, hyperchlorhydria and isochlorhydria. Some of the isochlorhydria cases were normal subjects without any clinical symptoms. The fractional test of gastric juice and estimation of chlorides in blood were carried out in each case.

Fractional test was done in the usual way (1). Alcohol meal was used as the stimulus in most cases. Histamine (0.5 mg) was given subcutaneously as a stronger stimulus in some cases to find out if the outpouring of acid into the stomach had any effect on blood chlorides. Free and total hydrochloric acid was estimated by titration with N/10 NaOH using Topfer's reagent and Phenolphthalein as indicators. Tests for blood, starch, bile and mucus were also done.

The estimation of chlorides in blood was done by Whitehorn's method (8). In all cases, blood was taken out immediately after the last sample of gastric contents was withdrawn. In some cases, however, blood was also taken just before the Ryle's tube was introduced.

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## OBSERVATION AND RESULT

The highest free Hcl level in India, in isochlorhydria has been said to be about 65 ml. N/10 Hcl and figures above that level have been taken to indicate hyperchlorhydria with the oatmeal gruel as the test meal (7, 2). But in the present work the highest free acid level of isochlorhydria has been taken as 55 ml. N/10 Hcl because most of the persons with gastric acid levels above 55 ml. N/10 Hcl in this series suffered from hunger pain and other signs and symptoms usually associated with hyperacidity. The lowest free Hcl level in isochlorhydria has been taken as 20 ml. N/10 Hcl. Thus cases have been divided in three groups: subjects showing a level of free Hcl ranging between 20 and 55 ml. N/10 Hcl were grouped as isochlorhydria, subjects above the level of 55 ml. N/10 Hcl were taken to be hyperchlorhydric and those below 20 ml. N/10 Hcl were classed as hypochlorhydric. Figures for blood chlorides mentioned by Philip B. Hawk (1954) have been taken as normal averages. The figures represent the weighted averages of the observation of several investigators. Chlorides as NaCl:—450-500 mg/100 ml. of blood.

The result obtained in cases of isochlorhydria are shown in Table I.

TABLE I

*Blood chlorides and gastric juice acidity in cases of Isochlorhydria*

Serial No.	Case No.	Age & Sex.	F. T. M. Max. free Hcl. ml. N/10 Hcl. per 100 ml. of gastric juice	Blood chlorides mg/100 ml. (corrected)
1.	3	18M	53.0	478.3
2.	4	60M	32.5	462.2
3.	5	28M	52.0	587.0
4.	11	25M	52.0	486.5
5.	12	30M	31.0	454.2
6.	15	40F	42.0	458.2
7.	17	22M	54.0	482.5
8.	22	20M	29.5	378.0
9.	25	20F	37.0	416.0

In the cases of isochlorhydria, it is seen that chlorides in the blood were normal in 6 cases out of 9. Two cases (No. 22 & 25) had, however, subnormal and one case (No. 5) more than the normal.

The results obtained in hyperchlorhydria are given in Table II.

TABLE II  
*Blood chlorides and gastric juice acidity in cases of hyperchlorhydria*

Serial No.	Case No.	Age & Sex.	F. T. M. Max. free Hcl. ml. N/10 Hcl. per 100 ml. of gastric juice	Blood chlorides mg/100 ml. (corrected)
1.	1	17M	103.5	534.5
2.	7	30M	65.0	402.0
3.	8	37M	82.0	683.5
4.	10	28F	70.5	434.3
5.	16	35M	112.0	382.0
6.	19	25M	121.0	408.0
7.	21	32M	59.0	373.7
8.	23	26M	116.5	361.7
9.	26	20M	76.0	392.0
10.	27A	48M	99.0	398.0
11.	29	32M	60.0	424.0
12.	30A	22M	99.0	402.0

In the cases of hyperchlorhydria, it is evident that blood chlorides were decreased in 10 cases out of 12 and in 2 cases (cases No. 1 and 8) they were more than normal.

The result obtained in hypochlorhydria are given in Table III.

TABLE III  
*Blood chlorides and gastric juice acidity in cases of hypochlorhydria*

Serial No.	Case No.	Age & Sex.	F. T. M. Max. free Hcl. ml. N/10 Hcl. per 100 ml. of gastric juice	Blood chlorides mg/100 ml. (corrected)
1.	2	22F	20.0	438.2
2.	6	50M	0.0	442.2
3.	13	15F	14.0	402.0
4.	14	50F	18.0	430.1
5.	20	35F	14.0	371.7
6.	28	30F	20.0	428.1

In cases of hypochlorhydria, it is seen that the blood chloride level is below normal in all cases.

The results obtained in the cases of histamine stimulation are recorded in Table IV.

TABLE IV  
*Gastric secretion under histamine stimulus and its effect on the blood chlorides*

Serial No.	Case No.	Age & Sex.	F. T. M. Max. free Hcl. ml. N/10 Hcl. per 100 ml. of gastric juice	Blood chlorides mg/100 ml. (corrected)		Difference
				Before	After	
1.	9	50M	76.0	426.1	414.0	12.1
2.	18	28M	86.0	402.0	359.7	42.3
3.	24	25M	117.0	402.0	432.1	30.1
4.	27	48M	131.0	380.0	357.7	22.3
5.	30	22M	142.0	388.0	366.0	22.0
6.	31	29M	108.0	412.0	382.0	30.0

A marked decrease ranging from 12.1 to 42.3 mg. in the blood chloride level was observed in 5 cases out of 6. However, it showed an increase of 30.1 mg. in one case (case No. 24).

Table V shows the blood chloride level under alcohol stimulus.

TABLE V  
*Gastric secretion under alcohol stimulus and its effect on the blood chlorides*

Serial No.	Case No.	Age & Sex.	F. T. M. Max. free Hcl. ml. N/10 Hcl. per 100 ml. of gastric juice	Blood Chlorides mg/100 ml. (corrected)		Difference
				Before	After	
1.	19	25M	121.0	446.5	408.0	38.5
2.	21	32M	59.0	418.0	373.7	44.3
3.	23	26M	116.5	400.0	361.7	38.3
4.	26	20M	76.0	410.0	392.0	18.0
5.	27A	48M	99.0	406.0	398.0	8.0
6.	29	32M	60.0	452.5	424.0	28.5
7.	30A	22M	99.0	412.0	402.0	10.0
8.	20	35F	14.0	402.0	371.7	30.3
9.	28	30F	20.0	432.1	428.1	4.0
10.	22	20M	29.5	398.0	378.0	20.0
11.	25	20F	37.0	446.5	416.0	30.5

The secretion of gastric juice under alcohol stimulus also shows a definite depletion in the blood chloride level ranging from 4 to 44.3 mg.

#### DISCUSSION

The relation of chloride to the acidity of the stomach has been a subject of fragmentary study. Kahn has shown many years ago that the ultimate source of chloride is the sodium chloride of the blood. Animals fed for some weeks upon meat deprived of its chloride by prolonged boiling in distilled water secreted an acid free gastric juice. It has been known for long time that the ultimate source of the constituents of the hydrochloric acid can not be other than the blood itself. Leaving the question of the mechanism of formation and transport of  $H^+$  ions across the canalicular wall and focussing the attention only on the chlorides, one can not but be led to the conclusion that the amount of chloride secreted as hydrochloric acid must be coming from the blood for two reasons. First the parietal cells are not known to accumulate chloride except during activity (3) and secondly chloride is an inorganic element ion which can not be synthesized otherwise in the body and has to be supplied from outside as a dietary essential. If therefore the body needs a supply of chloride from extraneous sources, the absorbed chloride has to travel via the blood stream to reach the site of requirement. These considerations justify the statement that the chlorides of the blood are the potential sources for the secretion of Hcl in the gastric juice.

In the present series of experiments, it was observed that in majority of the cases of isochlorhydria, the chloride level was within the normal limits (Table I). 6 out of 9 cases, result ranged from 454.2—482.5 mg/100 ml blood. However, in two cases it was below normal 378 and 416 mg/100 ml blood and in one case it was above normal 587.0 mg/100 ml. blood.

In the case of hyperchlorhydria (Table II) and hypochlorhydria (Table III) generally a low blood chloride level (361.7 to 442.2 mg/100 ml blood) was observed with two exceptions in the case of hyperchlorhydria in which it was above normal-viz. 534.5 and 683.5 mg/100 ml blood.

From the consideration of the above, it seems that the acidity of the gastric juice depended on the chloride content of the blood. This finding was further confirmed by estimating the blood chlorides before and after the injection of histamine (Table IV) and also before and after the alcohol test meal (Table V). It was observed that the depletion of the chloride content of the blood was of such a magnitude (12.1 to 42.3 mg. in the former and 4 to 44.3 mg. in the latter) that it could not possibly have been to any other cause than the secretion of Hcl in the stomach.

It has been reported by Lahiri (6) that during convalescence of patient from chloric diarrhoea and allied condition, where there is profound depletion of the chloride, the patient shows signs of hypochlorhydria or even complete achlorhydria. These findings are indirectly in conformity with the results obtained in the present study, but as the cases selected by Lahiri were in convalescent stage after having undergone severe ionic and endocrinologic upset, much importance can not be attached to the finding of low acidity. This may be due to causes other than hypochloremia. In this connection it is worthwhile to mention that many of the cases which succumb to Cholera are those

who do not possess the normal gastric acid-barrier and therefore it is not unlikely that even prior to the onset of the disease some of the patients might have been hypochlorhydric or achlorhydric.

However, the one exception in which the low chloride level was increased by 30.1 mg (Table IV) could not be accounted for unless further work is done with a larger number of cases.

From the present work, it may be suggested that the chloride of the blood serve as the source for the formation of hydrochloric acid. The association of low acidity with low blood chlorides and also of hyperacidity with low chlorides could be explained by assuming that in the former case the source of chloride was already depleted leading to deficient acid secretion where as in the latter case the blood chloride were depleted considerably because of the secretion of a large amount of acid.

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