

Hydrochloric Acid: Physiological Functions and Clinical Implications

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Abstract

Hydrochloric acid (HCl) secretion assists protein digestion by activating pepsinogen to pepsin, renders the stomach sterile against orally-ingested pathogens, prevents bacterial or fungal overgrowth of the small intestine, encourages the flow of bile and pancreatic enzymes, and facilitates the absorption of a variety of nutrients, including folic acid, ascorbic acid, beta-carotene, non-heme iron, and some forms of calcium, magnesium, and zinc. Numerous studies have shown acid secretion declines with advancing age and impaired HCl production and secretion is seen in a variety of clinical conditions. While the underlying etiological factors leading to impaired or complete lack of HCl secretion are not well understood, long term supplementation is safe and may be effective in certain patient populations and clinical conditions.

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Introduction

Historically, hydrochloric acid (HCl) was prescribed for many symptoms and clinical conditions and was listed as a therapeutic intervention in various pharmacopoeia. However, beginning in the late 1920s and early 1930s, its common use by the medical establishment began to decline. While the therapeutic efficacy of oral administration of HCl is still equivocal, largely due to a scarcity of outcome-focused clinical intervention studies, a substantial body of evidence indicates the necessity of proper gastric pH for optimal health. HCl secretion is required for protein digestion by activating pepsinogen to pepsin. It also renders the stomach sterile against orally-ingested pathogens, prevents bacterial or fungal overgrowth of the small intestine, encourages the flow of bile and pancreatic enzymes, and facilitates the absorption of a variety of nutrients.

Numerous studies have shown acid secretion declines with advancing age. The resultant rise in stomach pH can have a detrimental impact on nutrient absorption and may increase the risk of a variety of clinical conditions. It has been estimated 30% of U.S. men and women older than age 60 have atrophic gastritis, a condition in which little or no acid is secreted by the stomach,¹ and 40% of postmenopausal women have no basal gastric acid secretion.² Sharp et al tested 3,484 patients and found 27% to have achlorhydria. The greatest incidence (39.8%) occurred in females age 80-89. Among males, an increase in incidence was observed for each decade (except 50-59) until the age of 70. Over age 60, there was a significant increase in the incidence of achlorhydria in both males and females.³

Physiology of Digestion

Normally, the resting stomach contains appreciable amounts of free acid which substantially increases when the body is challenged to digest a meal. In general, free HCl is

present in an adequate concentration to maintain a pH between 1 and 2 in the stomach. The type of food eaten, nervous system integrity, micronutrient levels, structural alignment, the emotional mood of an individual, and other unidentified factors influence stomach acidity. In general, protein intake and a relaxed emotional state tend to increase stomach acidity.

Typically, digestion of food in the stomach is divided into two phases; however, both phases represent an integrated whole that overlap and mutually support each other. The cephalic phase of digestion refers to digestive activities dependent upon feedback and control occurring in the brain. The primary region of the brain regulating this stage of digestion is located in the medulla oblongata. In this phase, sight, odors or even the thought of food can stimulate the secretion of a small amount of gastric juice. Food placed into the mouth substantially increases gastric secretions, preparing the stomach to receive food. The second phase, the gastric phase of digestion, is regulated by stretch receptors which sense the arrival of food in the stomach and by chemoreceptors which sense the presence of dietary peptides. About 80% of gastric juice is secreted during the gastric phase.

The autonomic nervous system profusely innervates the digestive system with parasympathetic fibers (vagus nerve) which function largely to increase gastric secretion and motility, thus enhancing digestion. Sympathetic fibers, on the other hand, tend to indirectly reduce secretion and motility by constricting blood supply to digestive organs. Locally in the stomach, the submucosal plexus acts in a reflex manner to increase stomach acid secretion.

Gastric secretion is also responsive to hormonal control. The hormone, gastrin, is secreted by the stomach into the blood in response to ingestion of food, particularly dietary peptides. Gastrin secretion is also

enhanced by vagal stimulation and local activation of stretch receptors. Gastrin has a dual effect on digestion: it stimulates the parietal cells to secrete HCl and promotes contraction of the smooth muscles responsible for stomach motility. Gastrin is also believed to have an influence on maintaining the tone of the lower esophageal sphincter which prevents the reflux of stomach contents into the esophagus. Excessive amounts of gastrin have been associated with ulcer formation (Zollinger-Ellison syndrome).

Gastric secretions in the stomach consist of protective mucus, pepsinogen, and HCl. Pepsinogen, secreted by the stomach's chief cells (also called zymogen cells), is subsequently converted to pepsin in the stomach's lumen by HCl. Pepsin functions as a proteolytic enzyme, degrading food proteins into smaller peptides. It is most active at a pH of 1.8, and is completely inactive in a neutral or alkaline pH. Pepsin, once formed, also participates in an autocatalytic loop for the activation of pepsinogen.

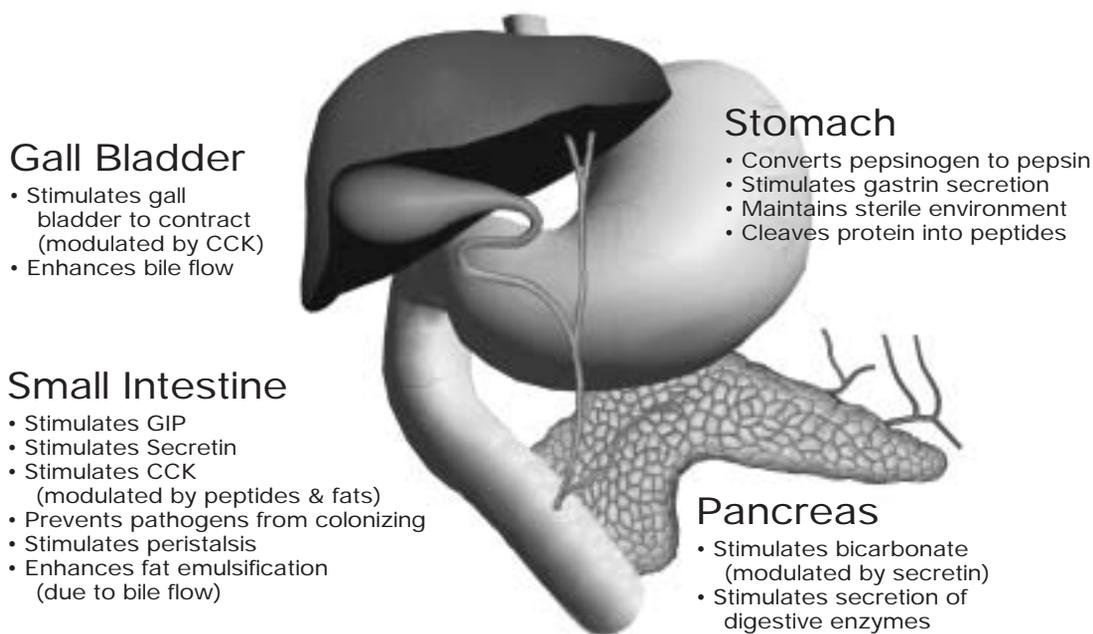
Figure 1. Hydration of CO₂



The parietal cells (also called oxyntic cells) secrete a concentrated solution of hydrochloric acid (H⁺ Cl⁻). Parietal cells contain the enzyme carbonic anhydrase, which promotes the hydration of carbon dioxide. (See Figure 1.) HCO₃⁻ is subsequently exchanged for a Cl⁻ ion which combines with a hydrogen ion to form HCl.

In the stomach, HCl's primary function is to maintain a sterile environment and to initiate the conversion of pepsinogen to pepsin. HCl dumping into the small intestine stimulates the release of gastric inhibitory

Figure 2. Direct & Indirect Physiological Functions of HCl



peptide (GIP), secretin and cholecystokinin (CCK) from the duodenal mucosa. GIP acts as a feedback mechanism to slow the emptying of acidic chyme into the duodenum. Secretin acts on the pancreas by augmenting the secretion of bicarbonate which assists in the neutralization of the acidic chyme. CCK release is stimulated by peptide fractions in the acidic chyme. Additionally, fatty acids are potent stimuli for the release of CCK. CCK stimulates the gallbladder to contract, releasing stored bile into the intestine. Bile assists in neutralizing acidic chyme and emulsifying fat, which facilitates its subsequent digestion by pancreatic lipase. CCK also stimulates the pancreas to release digestive enzymes.

Acidity in the duodenum varies in direct proportion to the acidity of chyme arriving from the stomach and the volume of alkaline fluids secreted in response. Even in the case of temporary achlorhydria, the duodenum may be rapidly invaded by microorganisms from the colon. High gastric pH is associated with gastric and intestinal bacterial colonization,⁴ and bacterial overgrowth occurs

in the proximal small intestine in all subjects when gastric pH is pharmacologically induced to greater than 4.0.⁵ In achlorhydria, colonic flora is found in the stomach in 25% of individuals. However, *Escherichia coli* is not found in the gastric contents of persons with achlorhydria for 1-2 weeks following a course of HCl administration.⁶ See Figure 2 for a summary of the physiological functions of HCl.

Indications

A variety of signs and symptoms can suggest decreased gastric secretions. For example Sharp et al found 80% of patients with achlorhydria had soreness, burning and dryness of the mouth, and low tolerance for dentures; 34% complained of indigestion and excessive gas; and 40% complained of malaise.³ Tables 1 and 2 list signs and symptoms associated with impaired HCl production. Several clinical conditions have been correlated with an increased incidence of impaired acid secretion. These conditions are listed in Table 3. While these symptoms, signs, and conditions

may help assess individuals for achlorhydria or hypochlorhydria, they are not always indicative of impaired gastric secretion.

HCl and Nutrient Interactions

Secretion of gastric acid and pepsin is a prerequisite for optimal digestion. Without adequate gastric secretions, macromolecules may be incompletely digested and may subsequently be absorbed into the systemic circulation. Acidic pH also impacts the absorption of selected micronutrients. It is probable gastric acidity impacts absorption directly, such as in making minerals more soluble, and indirectly by promoting the intestinal microflora needed for synthesis and absorption of selected micronutrients. Gastric acid may play an important role in mineral bioavailability by effecting the release of minerals from organic food matrices and by maintaining metal ions in solution.

Hydrochloric acid aids in the liberation of iron from food and facilitates its conversion to the ferrous form. In patients with iron deficiency anemia, optimal absorption of iron has been found to be related to maximal HCl output.²¹ While evidence indicates heme iron may be absorbed independent of acidity, non-heme iron appears to be dependent upon HCl secretion for absorption.²² In patients with histamine-fast achlorhydria, both ferric chloride and ferrous ascorbate were better absorbed when given with an acid solution. The acid solution did not alter absorption of hemoglobin iron.²¹

Zinc solubility is dependent on pH, with increasing solubility occurring as the pH becomes more acidic. The pharmacological impairment of stomach acidity with cimetidine has been shown to induce a reduction in zinc absorption.²³ Henderson et al also found that impairment of gastric acidity by famotidine diminished zinc absorption. They observed zinc from zinc acetate was absorbed more consistently at both low and high pH than zinc

oxide, and recommended against supplementation of zinc oxide in patients suspected of having impaired acid production.²⁴ Sandstrom et al observed a slight but not significant, decrease in absorption of zinc in individuals with achlorhydria compared with control subjects, and suggested achlorhydria does not effect zinc absorption. The 8 patients studied were diagnosed with atrophic gastritis and had no basal acid secretion and no acid secretion after pentagastrin stimulation. They found a 68 +/- 16% absorption of zinc from a zinc sulfate solution and a 33 +/- 10% absorption of zinc

Table 1. Symptoms associated with low gastric acidity

Bloating or distention after eating³
 Diarrhea or constipation^{3,7,8}
 Flatulence after eating^{3,8}
 Hair loss in women⁸
 Heartburn/epigastric distress^{3,8}
 Indigestion^{3,8}
 Pruritis ani⁸
 Low tolerance for dentures³
 Malaise³
 Multiple food allergies⁸
 Nausea or nausea after taking supplements^{3,8}
 Nocturnal encopresis⁷
 Prolonged sense of fullness after eating⁸
 Soreness, burning and dryness of the mouth³

from a test meal.²⁵ The information available seems to indicate zinc absorption in individuals with impaired acid secretion may be largely a function of the type of zinc supplemented.

Magnesium oxide is virtually insoluble in water and only 43% soluble in simulated peak acid secretion. Magnesium citrate has high solubility even in water (55%) and is substantially more soluble than magnesium oxide in all states of acid secretion. *In vivo* experiments in normal volunteers indicate

Table 2. Signs associated with low gastric acidity

Abnormal intestinal flora^{8,9}
Chronic candidiasis⁸
Chronic intestinal parasites^{8,9}
Dilated capillaries in the cheeks and nose (in non-alcoholics)⁸
Glossitis³
Increased excretion of urinary indican^{6,7}
Iron deficiency⁸
Post-adolescent acne⁸
Undigested food in the stool⁸
Weak, peeling and cracked fingernails⁸

a large dose of cimetidine does not completely eliminate gastric acidity, the results do not completely exclude an effect of gastric acidity on calcium absorption.²⁸ While still equivocal, it appears the form of calcium may have an impact on the amount absorbed in individuals with impaired gastric secretions. Until more evidence is available

magnesium citrate is significantly more bioavailable than magnesium oxide.²⁶ Although absorption studies of magnesium salts have not been conducted in achlorhydric or hypochlorhydric individuals, the citrate form, because of superior absorption in healthy individuals and because it is less pH-dependent for solubility, should be considered in individuals with diminished acid secretion.

Defective absorption of calcium has been thought to exist in patients with achlorhydria. Hunt et al found gastric secretion of acid is required for the absorption of calcium from carbonate; however, absorption of calcium from calcium monacitrate was consistent irrespective of pH.²⁰ Recker et al also found decreased absorption of calcium from the carbonate form in patients with achlorhydria. They found absorption of calcium as a pH-adjusted citrate salt was superior to carbonate in individuals with normal gastric acidity, and absorption of the citrate salt was not negatively affected by decreased gastric acidity. In fact, their findings indicate absorption of calcium as a citrate salt was significantly higher in individuals with achlorhydria than in subjects with normal gastric acidity.²⁷ In contrast to these studies suggesting the requirement of HCl for absorption of calcium from carbonate, Bo-Linn et al concluded stomach acid production was not important in absorption of calcium from carbonate in subjects given cimetidine to impair gastric acidity. However, since even

indicating calcium from carbonate is well absorbed in individuals with achlorhydria, calcium citrate or a similar calcium salt less pH dependent than carbonate should be utilized.

As far back as the 1940s, Allison believed HCl was essential for absorption of B-complex vitamins. While no research has been conducted to either prove or disprove his belief, his clinical experience with supplementation of HCl and B-complex vitamins merits investigation.¹⁷

Folic acid absorption in the small intestine has been shown to be influenced by gastric acidity. In patients with gastric atrophy, folic acid absorption is depressed (31 +/- 13%) compared with controls (53 +/- 13%). Supplementation with HCl in individuals with gastric atrophy increased absorption of folic acid to 56 +/- 14 %. No change in absorption was noted in controls when administered HCl.²⁹

Plasma vitamin C concentrations are significantly lower in individuals with hypochlorhydria when compared with individuals who have a pH less than or equal to 4 (p less than 0.005). Gastric secretory studies in five volunteers showed vitamin C concentrations increased significantly after intramuscular pentagastrin-stimulated HCl production.³⁰

A significant reduction in plasma response to beta-carotene supplementation has been demonstrated with pharmacologically-induced achlorhydria. When gastric acidity

Table 3. Clinical conditions associated with low gastric acidity

Addison's Disease ¹⁰	Gastric polyps ³
Alcoholism ¹¹	Gastritis ³
Anemia/Pernicious anemia ^{3,12}	Hepatitis ¹⁰
Arthritis/Rheumatoid arthritis ¹³	Hyperthyroidism/Graves disease ¹⁹
Asthma (of children) ¹⁴	Hypothyroidism ¹⁹
Celiac Disease ¹⁰	Lupus erythematosus ¹⁷
Carcinoma of the stomach ³	Myasthenia gravis ¹⁰
Chronic auto-immune disorders ¹⁰	Osteoporosis ²⁰
Depression ¹⁵	Psoriasis ^{16,17}
Dermatitis herpetiformis ¹⁶	Rosacea ¹⁷
Diabetes mellitus ⁷	Sjogren's disease ¹⁰
Diabetic neuropathies ⁷	Thyrotoxicosis ¹⁹
Eczema ^{16,17}	Ulcerative colitis ³
Flatulent dyspepsia ¹⁸	Urticaria ¹⁷
Gallbladder disease ¹⁸	Vitiligo ^{12,18}

was pharmacologically induced by omeprazole supplementation to a pH 4.6-7.4, the area under the blood response curve after a beta-carotene dose was reduced 50% compared with the response at a gastric pH of 1.0-1.5.⁵

Gastric secretions may be influenced by food constituents. Only a small amount of intra-intestinal amino acid concentration is required to produce gastric secretion. When five grams of lysine monohydrochloride (95% L-lysine) is added to a test meal consisting of toasted white bread, the amount of HCl in the gastric contents increases slightly, whereas a more uniform and quantitatively greater increase is observed with pepsin.³¹ Experiments on dogs also reveal intensified secretion of gastric juice, pepsin and hydrochloric acid from the effect of lysine-enriched bread.³² L-tryptophan administration has been shown to increase the amount of Cl- and H+ secreted in pigs.³³ In experimental animals, circumstantial evidence suggests a retinol deficiency may increase hydrochloric acid secretion.³⁴

In rats, a 5-week zinc-deficient diet significantly increased gastric secretory volume of both acid and pepsin.³⁵ In humans, administration of zinc sulfate has been observed to produce an anti-secretory effect

on gastric acid in duodenal ulcer patients. Gastric secretory testing showed zinc sulfate administered in doses of 60 ml/day (1% solution) for 10 days reduced basal acid secretion in duodenal ulcer patients by 57.7%. *In vitro*, zinc sulfate inhibits purified carbonic anhydrase activity in a dose-dependent manner. A similar dose-dependent inhibition was found with gastric mucosa carbonic anhydrase activity, with zinc sulfate reducing enzyme activity.³⁶

Experimental evidence suggests a component of a B-complex vitamin may be needed to maintain adequate HCl secretion. It has been demonstrated that a diet inadequate in the entire B-complex impaired gastric secretion in experimental animals. This effect was reversible upon administration of a diet supplemented with B vitamins. Brewer's yeast has been shown to restore normal gastric secretion in animals fed a B-vitamin deficient diet.¹¹

Cholinergic drugs stimulate production of acid and anticholinergic drugs inhibit it. HCl production is usually increased by caffeine, alcohol, histamine, and hypoglycemia. The production of pepsin is actively stimulated by any stimulant that increases HCl.³¹

Historically, beyond the direct administration of HCl, several other substances were

believed to stimulate either acid secretion or digestion. These include the use of diluted lemon or vinegar prior to meals; the use of herbal bitters, such as *Centaurium minus* or *erythraea* (common or red centaur), *Gentiana lutea*, (gentian), and *Zingiber officinale* (ginger); and the use of stimulants such as *Piper nigrum* (black pepper) and *Capsicum annum* (cayenne). While there is a little research on the majority of these substances, the bitter principles of the dried underground organs of *Gentiana lutea* have been shown to stimulate gustatory receptors in the taste buds, causing a reflex increase in the secretion of saliva and gastric juice.³⁷

Clinical Implications of Decreased HCl

Gallbladder: Gastric secretion is commonly decreased in conditions of gallbladder disease. Gatewood et al found of 192 patients with documented gallstones, 57 had gastric achlorhydria and 29 had diminished acid secretion.¹⁸ In a 1967 report, Capper et al observed 26/50 patients (52%) with cholelithiasis showed evidence of gastric hyposecretion with the augmented histamine test. They also noted flatulent dyspepsia and gastric hyposecretion often accompany cholelithiasis.¹⁸

Alcoholism: Joffe and Jolliffe investigated gastric acidity in 77 males and 28 female alcoholics. Inadequate secretion was found in 68% of the males and 71% of the females. The incidence of achlorhydria was 30% for males and 36% for females. While they were unable to demonstrate the incidence of achlorhydria varied with duration of alcohol consumption, alcoholics with symptoms of polyneuritis or pellagra had a higher incidence of achlorhydria than addicts without these complications.¹¹

Skin Diseases: The preponderance of information on gastric acid secretion, HCl supplementation and skin disease is based on the work of Allison published in 1945.¹⁷ In

Allison's experience, a normal balance of gastric acidity could be restored with administration of HCl during meals. In severe cases he recommended one capsule of HCl before, during, and after each meal. He was convinced, in severe hypochlorhydria or achlorhydria, B-complex vitamins were poorly absorbed. In patients known to be or suspected to be vitamin B-complex deficient, he invariably found achlorhydria or hypochlorhydria. In certain cases, he observed distinct psychic influences (primarily anxiety and nervousness) seemed to suppress acid secretion. In a series of 400 patients with skin disorders and suspected B vitamin deficiency resistant to local treatment, he investigated stomach acidity. Table 4 summarizes his findings.

He also observed the severity of acid deficiency was in direct proportion to both the duration and severity of the skin disease and digestive symptoms. Improvement in general health and skin condition was observed following treatment with HCl and B-complex (as brewer's yeast) in virtually all patients with impaired HCl production. Cases with very moderate HCl deficiency showed rapid improvement and early signs of intolerance to the acid, which he believed indicated a return to normal secretion.¹⁷ While Allison did not claim the ten dermatology conditions were always due to B vitamin and HCl deficiency, his observations and therapeutic results suggest gastric acidity should be investigated in individuals presenting with these conditions.

Ayers also commented on a correlation between eczema and psoriasis and reduced gastric secretions. He found 8 of 11 patients with eczema and 10 of 19 patients with psoriasis had functional hypoacidity. In one case, he reported a 52 year-old patient with a 46 year history of eczema had impaired HCl production. After supplementation with HCl, the patient's skin condition remained nearly normal during one year of follow-up.¹⁶ Francis reported that four patients with vitiligo and

achlorhydria experienced disappearance of vitiligo after starting HCl supplementation with each meal.³⁸

Osteoporosis and Arthritis: Experiments in rats indicate total gastrectomy or resection of the acid-producing part of the stomach (fundectomy) induces a marked and rapid reduction in bone wet weight, ash weight, and density.³⁹ Reduced gastric acidity may also cause alveolar bone resorption.⁴⁰

In 9 of 35 female patients (26%) with arthritis (average age 52), Hartung and Steinbrocker found achlorhydria. The incidence of achlorhydria in females of a similar age without arthritis is estimated to be 12-15.5%.⁴¹ De Witte et al observed in 53 patients with seropositive, definite or classical rheumatoid arthritis, only 50% had normal maximal acid output.¹³

D i a b e t e s :

Dotevall observed lower secretion of free and total HCl, both in the basal condition and after stimulation with histamine, in diabetics than in controls.⁴² Shay et al found the duodenal instillation of HCl prevented alimentary hyperglycemia after oral glucose load. They also reported individuals with anacidity had a greater tendency for disturbed carbohydrate metabolism. Abnormal glucose tolerance curves were found in 48% of the anacid group. Individuals in this group also showed a distinct tendency toward a greater deviation from a normal curve than individuals in the control group.⁴³ It has been reported diabetics with and without neuropathy have decreased acid secretion and delayed gastric emptying time, and the average incidence of achlorhydria in patients with diabetes mellitus is about 33%.⁴⁴ Rabinowitch reported in 50 diabetic patients

under age 40 and 50 patients age 40 and over, 18% of the younger group and 64% of the older group were achlorhydric. He also noted three diabetic patients with severe neuritis failed to improve with thiamin supplementation, but after receiving HCl supplementation, experienced marked improvement.⁷

Asthma: Gastric secretion of HCl following a standardized meal was studied in 200 asthmatic children (ages 6 months to 12 years) and compared with a control group of non-asthmatic children. Eighty percent of the asthmatic children had levels of HCl below normal while only 10% of controls had similar low levels. Hypochlorhydria was more prevalent in children under age seven with a trend toward normalization of HCl levels observed as the children approached puberty.

Table 4. Summary of the relationship between skin diseases and HCl¹⁷

Condition	# of Patients	achlorhydria	hypochlorhydria	normal
Acne Rosacea	30	40%	47%	13%
Alopecia	19	21%	74%	5%
Avitaminosis	37	30%	49%	21%
Eczema	106	25%	49%	26%
Lupus Erythematosus	9	22%	78%	0%
Psoriasis	9	56%	33%	11%
Seborrheic dermatitis	68	22%	65%	13%
Staph. Infection	12	8%	67%	25%
Urticaria	77	31%	54%	15%
Vitiligo	29	35%	55%	10%

The 160 asthmatic children with low HCl levels avoided known food allergens and were supplemented with HCl before or during meals. An immediate improvement in appetite, weight, and sleep was observed. Asthma attacks were shorter in duration and of lesser intensity.¹⁴ Wright has commented on hypochlorhydria and low pepsin production resulting in incomplete digestion of food and macromolecule absorption, increasing both the number and severity of food allergies, while simultaneously impairing micronutrient

nutrition. He also comments on the benefit of HCl administration as part of an integrated treatment protocol in childhood asthma.⁸

Hypo- or Hyperthyroid: A high incidence of achlorhydria, hypochlorhydria, and gastritis has been noted in patients with either hyper- or hypothyroidism. In 15 patients with thyroid disease, 10 with hyperthyroidism and 5 with hypothyroidism, basal secretion of acid was significantly reduced in all patients compared with controls, and 10 of 15 patients had no basal secretion of HCl. Twelve patients had significantly reduced secretion of acid following histamine challenge. In an earlier study of 15 patients diagnosed as hypothyroid, 8 were assessed as achlorhydric or hypochlorhydric.¹⁹ Williams and Blair reported a reduction in gastric secretion, with a 15.6% incidence of achlorhydria, in patients with hyperthyroidism.⁴⁵

Anemia: Achlorhydria is present in most patients with pernicious anemia. Because of the role of HCl in non-heme iron and folate absorption, HCl administration may also be efficacious in the treatment of other types of anemia. Glutamic HCl was dosed at 5 grains t.i.d. before meals to 25 diabetic patients with blood cell counts 4.2 million or less. Following treatment with glutamic HCl, the average red blood cell count had increased from 4.06 to 4.56 million. A subsequent combination of glutamic acid HCl with inorganic iron (ferrous carbonate 6 3/4 grains t.i.d.) increased RBC count to 4.85 million.⁷

Gastrointestinal Tract: A deficiency of stomach acid may result in increased susceptibility to, and severity of, enteric bacterial infections because of survival of ingested bacteria leading to intestinal overgrowth.⁹ H₂-receptor antagonists, by decreasing gastric acidity, frequently encourage *Candida* overgrowth, especially in women.⁴⁶ *Candida albicans* requires pH 7.4 for optimal growth but becomes completely inhibited at pH 4.5.⁴⁷ Twenty-seven achlorhydric patients were supplemented with betaine HCl and pepsin for

6 months. General improvement in physical condition and strength was noted in all subjects. Indigestion and excessive gas were relieved in all patients with this complaint. Signs of oral mucosal inflammation improved in 78% of the patients, and of 22 patients with a complaint of a chronic sore mouth, 5 had complete relief and 11 others noted improvement.³

Indican is derived from indole, a byproduct of putrefaction and aromatic amino acid breakdown. Increased levels of indican in urine are thought to be associated with bacterial overgrowth in the intestine.⁶ Elevated urinary indican has been observed frequently in individuals with achlorhydria.⁷ Test subjects with achlorhydria given HCl with meals showed no change with indican metabolism; however, when HCl was administered t.i.d. on a fasting stomach, indican metabolism improved markedly. No change in urinary indican levels was noted in patients with normal gastric secretion following HCl supplementation, irrespective of when administered. On discontinuing HCl supplementation, indican levels reverted to previous levels within 1-2 days.⁶

Dosage

Hydrochloric acid is available primarily as betaine HCl, although glutamic acid HCl is found in some formulas. The potency of a capsule or tablet preparation may vary from 5-10 grains with 1 grain equal to approximately 64.75 mg. Clinically, practitioners have reported administering dosages of betaine HCl from as little as 5 grains t.i.d. to levels as high as 60-80 grains t.i.d. While 5 grains of betaine HCl is not sufficient to allow the appearance of appreciable free acid in the stomach, Rabinowitch found this low amount often to be effective in relieving symptoms associated with achlorhydria.⁷ A standard protocol for HCl administration is provided as a patient handout.

Supplemental pepsin is obtained from the glandular layer of porcine stomach. Pepsin is standardized to digest not less than 3,000 and not more than 3,500 times its weight of coagulated egg albumin. Pepsin is administered to assist digestion and is typically given in conjunction with hydrochloric acid. It may also be combined with pancreatic enzymes. The usual dose is 500 mg of 1:3,000 potency pepsin. Although pepsin has a long history of medicinal use and is considered very safe, its actual therapeutic benefits are poorly documented.⁴⁸

If HCl is being administered to an individual with intestinal bacterial or fungal overgrowth due to lack of basal HCl production, it is recommended that one capsule of betaine HCl be supplemented between meals three times daily.

Conclusion

The normal sequence of digestion and absorption is dependent upon the anatomic and physiologic integrity of the upper gastrointestinal tract. When this system is disrupted, disorders of digestion and absorption can occur. Because of inadequate breakdown and assimilation, impaired gastric secretions are likely to result in nutritional deficiencies not withstanding adequate ingestion of nutrients. Secretion of gastric acid is required to destroy orally-ingested pathogens and to prevent their overgrowth in the stomach and small intestine. Additionally, the dumping of acidic chyme into the small intestine is necessary to stimulate the release of hormones, pancreatic enzymes, and bile. In order to have optimal absorption of several nutrients, including folic acid, ascorbic acid, beta-carotene, non-heme iron, and some forms of calcium, magnesium and zinc, adequate HCl production is required. It is quite probable the absorption of other nutrients are dependent on HCl secretion. HCl administration seems to be most indicated in aging people not

responding to nutrients which seem indicated, particularly B vitamins and minerals. Childhood asthma, alcoholism, chronic skin conditions, digestive disturbances, intestinal permeability, overgrowth by pathogenic bacteria or fungi, and evidence of parasites are conditions which may indicate impaired ability to secrete adequate HCl and which may benefit from supplementation. Diseases associated with the pancreas or gallbladder, since these organs indirectly require stomach HCl production to function optimally, may also benefit from HCl administration. While the etiologic factors leading to impaired or complete lack of HCl secretion are not well understood, long term supplementation is safe and warranted in certain populations and clinical conditions.

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