"Pharmaceutical Mythology: The Truth About Stomach Acid & Proton Pump Inhibitors"

- **Proton pump inhibitors cause osteoporosis.**
- **Proton pump inhibitors cause diseases associated with mineral depletion.**
- **Proton pump inhibitors increase the risk of intestinal infections.**
- **Proton pump inhibitors increase the likelihood of developing food allergies.**
- **Proton pump inhibitors cause deficiencies of vitamin B12 and vit. C.**
- **Proton pump inhibitors work no better than a placebo.**
- **Proton pump inhibitors cause stomach pain, gas, nausea, vomiting, and diarrhea.**
- **Eosinophilic Esophagitis is responsible for many symptoms misdiagnosed as GERD.**
- **Stomach ulcers are <u>not</u> caused by excess stomach acid.**

You have patients who are taking a proton pump inhibitor (or a histamine 2 blocker), which we should classify as a NUTRI-SPEC Red Flag drug --- drugs with such severe consequences that the damage to health <u>always</u> outweighs any symptomatic relief they offer.

<u>Proton pump inhibitors cause osteoporosis</u>:

Use of proton pump inhibitors for 7 or more years is associated with a significant increased risk of an osteoporosis-related fracture. There is an increased risk of hip fractures after 5 or more years of use.

Postmenopausal women have 3½ times the incidence of vertebral fractures if they use proton pump inhibitors long-term.

Targownik, et al. Use of proton pump inhibitors and risk of osteoporosis-related fractures. <u>CMAJ</u>, 2008.

Corley, et al. Proton pump inhibitors and histamine-2 receptor antagonists are associated with hip fractures among at-risk patients. <u>Gastroenterology</u>, 2010.

Ito, et al. Association of long-term proton pump inhibitor therapy with bone fractures and effects on absorption of calcium, vitamin B12, iron, and magnesium. <u>Curr Gastroenterol Rep, 2010</u>.

Roux, et al. Increase in vertebral fracture risk in postmenopausal women using omeprazole. <u>Calcif Tissue Int</u>, 2009.

Proton pump inhibitors cause diseases associated with mineral depletion:

The deficiencies of magnesium and potassium caused by proton pump inhibitors can cause severe life-threatening symptoms, including collapse from postanoxic encephalopathy, cardiac arrhythmia, and extreme electrocardiogram abnormalities.

There are reports of patients hospitalized for extreme muscular weakness of the arms and legs, vomiting, swallowing disorders, and hypoparathyroidism, all associated with low magnesium and calcium due to proton pump inhibitors.

Sundy, et al. Severe hypomagnesaemia in long-term users of proton pump inhibitors. Clin Endocrinol (Oxf), 2008.

Mackay, et al. Hypomagnesaemia due to proton pump inhibitor therapy: A clinical case series. QJM, 2010.

Ito, et al. Association of long-term proton pump inhibitor therapy with bone fractures and effects on absorption of calcium, vitamin B12, iron, and magnesium. Curr Gastroenterol Rep, 2010.

Hoorn, et al. A case series of proton pump inhibitor-induced hypomagnesaemia. Am J Kidney Dis, 2010.

Francois, et al. [Chronic use of proton-pump inhibitors associated with giardiasis: A rare cause of hypomagnesaemic hypoparathyroidism?] Ann Endocrinol (Paris), 2008.

Proton pump inhibitors increase the risk of intestinal infections:

The growth of bacteria, yeast, and parasites due to insufficient stomach acid is also reported with proton pump inhibitor use. Taking Proton pump inhibitors doubles the incidence of Clostridium difficile infection, and the incidence of other intestinal infections such as Salmonella and Campylobacter are increased by a factor of 2½.

Francois, et al. [Chronic use of proton-pump inhibitors associated with giardiasis: A rare cause of hypomagnesaemic hypoparathyroidism?] Ann Endocrinol (Paris), 2008.

Leonard, et al. Systemic review of the risk of enteric infection in patients taking acid suppression. Am J Gastroentrol, 2007.

Deshpande, et al. Association between proton pump inhibitor therapy and Clostridium difficile infection in a meta-analysis. Clin Gastroenterol Hepatol, 2011.

Proton pump inhibitors increase the likelihood of developing food allergies:

Undigested proteins due to use of proton pump inhibitors may persist in the GI tract, become absorbed, and thus stimulate an allergic reaction. Mouse models indicate that these drugs support the induction of Th2 immune responses that increase the occurrence of food allergies.

Impairment of gastric function by proton pump inhibitors is a documented risk factor for sensitization against oral proteins and against drugs.

Pali-Scholl, et al. Anti-acid medication as a risk factor for food allergy. Allergy, 2011.

Diesner, et al. Food allergy: Only a pediatric disease? Gerontology, 2011.

Proton pump inhibitors destroy the stomach's ability to produce vitamin B12, thus causing B12 deficiency:

The vitamin B12 deficiency caused by long-term usage of proton pump inhibitors is by a second undetermined mechanism in addition to the suppression of normal stomach acid.

----- Proton pump inhibitors also inhibit the bioavailability of vitamin C.

Laine, et al. Review article: Potential GI effects of long-term acid suppression with proton pump inhibitors. Aliment Pharmacol Ther, 2000.

Hirschowitz, et al. Vitamin B12 deficiency in hyper-secretors during long-term acid suppression with proton pump inhibitors. Aliment Pharmacol Ther, 2008.

Ito, et al. Association of long-term proton pump inhibitor therapy with bone fractures and effects on absorption of calcium, vitamin B12, iron, and magnesium. Curr Gastroenterol Rep, 2010.

Rozgony, et al. Vitamin B12 deficiency is linked with long-term use of proton pump inhibitors in institutionalized older adults. J Nutr Elder, 2010.

McCollke. Effective proton pump inhibitors on vitamins and iron. Am J Gastroenterol, 2009.

Proton pump inhibitors work no better than a placebo:

Cochrane Database of Systematic Reviews shows that both histamine-2 blockers and proton pump inhibitors are only slightly better than a

placebo at relieving non-ulcer upper GI symptoms. Histamine blockers had a relative risk reduction of 23% and proton pump inhibitors had a relative risk reduction of 13%. While these numbers are statistically significant, they may not be clinically significant. The Cochrane analysis concluded that while these results were significant, the benefits of these drugs is likely to be small, and patients would need to take them on a long-term basis, so the cost/benefit analysis may rule against these drugs. Furthermore, it was speculated that the slight clinically significant benefits may have been due to publication bias in the various studies done from 1988-2006.

In a study of patients with symptoms of heartburn, chest pain, and/or regurgitation despite taking proton pump inhibitors twice daily, their symptomatic episodes were evaluated both while on and off the proton pump inhibitor. It was found that proton pump inhibitors did reduce the number of episodes of upper GI symptoms, but the symptomatic episodes associated with low, not high, stomach acid were twice as high while taking proton pump inhibitors. These results illustrate that the symptomatic benefits of proton pump inhibitors only occasionally have something to do with decreasing stomach acid, and are almost entirely associated with accelerating the stomach emptying time.

Another study demonstrating that proton pump inhibitors achieve their symptomatic relief not by inhibiting acid, but by stimulating stomach emptying, shows that symptomatic improvement was achieved in patients on proton pump inhibitors in 36% of the cases, and only 6% of a placebo group --- but --- the vast majority of those who showed symptomatic improvement were those with a high body mass index. In other words, these would have been people whose symptoms were associated with increased mechanical pressure on the stomach due to the abdominal obesity. In these patients, the symptomatic relief would come not from inhibiting stomach acid but by emptying the stomach.

Moayyedi, et al. WITHDRAWN: Pharmacological interventions for non-ulcer dyspepsia. Cochrane Database of Systematic Reviews, 2001.

Fletcher, et al. BMI is superior to symptoms in predicting response to proton pump inhibitors. Gut, 2011.

Hemmink, et al. Esophageal pH-impedance monitoring in patients with therapy-resistant reflux symptoms: "On" or "off" proton pump inhibitor? Am J Gastroenterol, 2008.

Proton pump inhibitors cause stomach pain, gas, nausea, vomiting, and diarrhea:

Physician's Desk Reference

Eosinophilic Esophagitis is in many cases the true cause of symptoms attributed to GERD:

Gastroenterologists now are often uncertain whether to diagnose Eosinophilic Esophagitis or GERD. Some patients may show combined features of both GERD and EOE, which complicates the histologic analysis of these patients. In contrast to GERD, EOE typically involves longer lengths of the esophagus, affects the proximal equally or even more than the distal esophagus, and the pathological findings are often patchy in distribution. Since the inflammatory lesions in the esophagus look similar in the two conditions, it is now accepted (but absurdly irresponsible) practice among many gastroenterologists to continue routinely treating patients with proton pump inhibitors, and then when that therapy fails, to do a biopsy checking for EOE. Particularly when upper GI symptoms are present in young males, it is almost certain that the pathology is EOE, and not GERD.

Parfitt, et al. Eosinophilic Esophagitis in adults: Distinguishing features from GERD. Mod Pathol, 2006.

Odze R D. Pathology of Eosinophilic Esophagitis: What the clinician needs to know. Am J Gastroenterol, 2009.

Stomach ulcers are NOT caused by excess stomach acid, but rather by the bacterium Helicobacter pylori:

That H. pylori is the cause of peptic ulcer-associated gastritis was first determined in 1979, and was unequivocally proven by 1981. One of the original discoverers of the connection between H. pylori and stomach ulcers was so frustrated at his being ignored by the medical establishment that he actually inoculated himself with H. pylori by ingesting a culture of the bacteria, resulting in immediate gastritis, which he subsequently cured with the appropriate antibiotic plus bismuth salts. Gastritis and peptic ulcer have thus been conclusively shown to be infectious diseases, and have absolutely nothing to do with too much stomach acid. In fact, H. pylori thrives in a low acid environment, and even creates its own low acid environment as it damages the lining of the stomach.

Inhibition of acid secretion increases symptoms of gastritis in H. pylori infected subjects. H. pylori infection contributes to under-secretion of stomach acid. Data on gastric acid secretion in patients with esophagitis

suggests that acid secretion is normal or slightly diminished, and not elevated as is commonly believed. Proton pump inhibitors increase the tendency of H. pylori to cause atrophic gastritis. H. pylori actually impairs gastric secretion functions.

Pajares, et al. Helicobacter pylori: Its discovery and relevance for medicine. Rev Esp Enfrem Dig, 2006.

Calamj. Helicobacter pylori modulation of gastric acid. Yale J Biol Med, 1999.

Konturek, et al. Helicobacter pylori and impaired gastric secretory functions. J Physiol Pharmacol, 1997.

Doctors generally prescribe proton pump inhibitors (or histamine-2 blockers, or recommend over-the-counter antacids) for patients they believe have Gastro Esophageal Reflux Disease (GERD), or, who have heart burn, pain, pressure or other symptoms that are commonly blamed on "too much stomach acid," or "acid indigestion." Presumably, these symptoms result from the stomach producing too much hydrochloric acid, the acid essential for the first stage of efficient digestion of proteins, carbohydrates, and fats. Proton pump inhibitors have 2 effects --- they destroy the stomach's ability to produce hydrochloric acid, and they stimulate the stomach to empty its contents into the duodenum.

We will explain below why blocking the natural production of hydrochloric acid is always a bad idea, and often causes disastrous consequences. We will also explain that it is the drug-stimulated emptying of the stomach, not the decrease of stomach acid production, that gives the symptomatic relief when these drugs do give symptomatic relief. You will learn why blocking the secretory function of the stomach and stimulating stomach emptying (often at an inappropriate time) usually assures that the symptoms for which the drug is taken will return. In other words, even when the drug temporarily relieves symptoms, it makes the cause of the symptoms worse.

Truly, it is not excess stomach acid, but a deficiency of stomach acid (hypochlorhydria) that causes most upper GI symptoms. Sufficient stomach production of hydrochloric acid is essential:

- for efficient digestion of proteins, fats, and carbohydrates
- for efficient absorption of minerals and trace minerals
- for efficient absorption of vitamin B12 and vitamin C
- to kill ingested bacteria, viruses, yeast, fungi, and parasites
- to prevent food allergies
- to prevent stomach ulcers
- for efficient timing of stomach emptying

All these essential functions are crippled in patients given proton pump inhibitors by well-meaning but misinformed physicians.

The reason many patients feel they need proton pump inhibitors is due to their hypochlorhydria. Hypochlorhydria is extremely common. --- We call it a 50/50 condition. By that we mean that 50% of the people over 50 years old are at least somewhat deficient in hydrochloric acid. The only reason we do not make a bigger point of this with NUTRI-SPEC doctors is because many of those people that are low in hydrochloric acid are also low in either pancreatic bicarbonate production or bile production.

So, while they are low in acid, they are also low in alkali. Supplementing these people with hydrochloric acid (Proton Plus) can be easily overdone because while the Proton Plus is absolutely essential to improve stomach function, they do not have the pancreatic or liver capability of neutralizing that acid once it reaches the duodenum. --- But in those with at least close to normal pancreatic and biliary function, Proton Plus supplementation with meals is a tremendous benefit, not only in terms of digestive efficiency, but even more importantly in assisting the immune system in eliminating opportunistic and pathogenic bacteria as well as yeast/mold/fungi/and parasites.

Proton pump inhibitors inhibit the effective digestion of protein, and almost totally inhibit the absorption of minerals and trace minerals, particularly magnesium, calcium, potassium, copper, manganese, and iron. These drugs cause such extreme interference with mineral nutrient absorption they cause osteoporosis. The long-term effects of mineral depletion include increased hip fractures from osteoporosis in those who take these drugs over a long period.

There can actually be life-threatening low magnesium in those who take proton pump inhibitors daily for more than 3 months. Neurological symptoms, and neurovascular symptoms, and cardiac conditions are exacerbated by a magnesium deficiency. Even without these severe side effects, there are obvious disastrous nutritional and metabolic consequences of blocking the absorption of so many nutritionally essential minerals and trace minerals.

Another consideration as regards proton pump inhibitors is that many studies now show that they work little better than a placebo for upper GI and esophageal burning. Furthermore, one of the most common side effects of these drugs is <u>stomach pain</u>. Even more absurd than taking a drug that causes stomach pain for stomach pain, <u>and</u> is no more effective than a placebo, <u>and</u> causes mineral depletion and protein malabsorption, <u>and</u> will cause osteoporosis --- is that the condition for which these drugs are prescribed, (GERD), is not even the correct diagnosis in many patients.

The medical literature is quite clear now that many cases diagnosed as GERD are not GERD, but Eosinophilic Esophagitis --- one more pathology (like asthma and Eosinophilic Fungal Rhinosinusitis) associated with eosinophilic infiltration --- but in this instance, infiltration of the esophagus with resultant inflammation. No cause has been agreed upon for Eosinophilic Esophagitis. It is just loosely attributed to “allergies” with the standard treatment being food allergy testing and food elimination, along with a prescription for the immunosuppressant steroid drug, prednisone.

Gastroenterologists are in somewhat of a tizzy over Eosinophilic Esophagitis, since it turns out that so many who have been diagnosed with GERD in the last 25 years actually have eosinophilic esophagitis, and not reflux at all. But since openly recognizing that fact would eliminate the zillion dollar

pharmaceutical bonanza from selling proton pump inhibitors and related garbage, discussion of Eosinophilic Esophagitis has been suppressed.

Since eosinophilic infiltration is most common in patients who are Parasympathetic and/or Anaerobic, we would say it is almost a certainty that patients with those Metabolic Imbalances who also have upper GI burning, pressure, and pain, do indeed have Eosinophilic Esophagitis. There are many potential triggers to eosinophilic infiltration, but perhaps the most common is a reactivity to mold toxins --- mold spores and mold fragments in the living and working environment. But whatever the trigger, correction of the Anaerobic and Parasympathetic Metabolic Imbalances is essential.

Here are some essentials of gastric physiology:

Vagus nerve activity (Parasympathetic) excites stomach secretion of acid, and of pepsin, and of mucin, both directly by stimulation of the gastric glands, and indirectly through the gastrin hormone mechanism.

Control of the stomach emptying rate is largely under the influence of the hormone gastrin. Adequate secretion of gastrin from the stomach mucosa depends largely on the secretion of highly acidic (high hydrochloric acid content) gastric juice by the stomach. This very important highly acid stomach secretion is most effectively stimulated by the presence of meat in the stomach.

The secretion of large amounts of hydrochloric acid by the stomach is essential for many reasons. The hydrochloric acid helps breakdown proteins in the food, both directly, and by stimulating the stomach secretion of the enzyme pepsin that aids in protein digestion. The high hydrochloric acid environment is essential for the absorption of the minerals and trace minerals --- calcium, magnesium, potassium, manganese, copper, and iron.

The hydrochloric acid also regulates the rate of stomach emptying.

The hydrochloric acid also kills nasty critters that have entered the stomach --- including bacteria, viruses, parasites, yeast, mold, and fungi.

Another essential function of the hormone gastrin associated with adequate hydrochloric acid production in the stomach is its constrictor effect on the gastroesophageal sphincter. It thus prevents reflux of gastric contents into the esophagus.

----- In summary --- high hydrochloric acid production is essential for protein digestion, for mineral absorption, to kill pathological microbes in the GI tract, to ensure proper stomach emptying, and to prevent gastroesophageal reflux.

While timely stomach emptying depends largely on adequate production of hydrochloric acid in the stomach, there are also reflexes from the duodenum that inhibit stomach emptying --- thus assuring that the stomach does not empty before the first stage of digestion in the stomach is complete, and, assuring that the contents of the stomach are not emptied into the duodenum faster than the duodenum can accommodate the efficient digestion of proteins, fats, and minerals through the rest of the intestinal tract. Adequate acid from the stomach therefore not only stimulates the emptying of the stomach, but also inhibits the emptying of the stomach, as the duodenum senses the acidity of the chyme entering it from the stomach.

Normal, healthy gastric secretion is extremely acidic --- with a pH of less than 1 (0.8). That yields a normal overall pH of the stomach contents of between 1 and 2. Food entering the stomach generally has a pH near neutral (7.0). As the gastric secretions are stimulated and go to work on the food, the pH of the chyme gradually decreases to between 4 and 5, which is the ideal pH for the chyme to be slowly released into the duodenum. When the pH of the chyme entering the duodenum falls below 3.5-4.0, reflexes are activated to slow the stomach emptying until the duodenum can catch up.

So, excess acidity of the stomach (as in a Parasympathetic Imbalance, or patients who are taking certain drugs), will delay stomach emptying time. But stomach emptying time will also be delayed if the stomach produces inadequate hydrochloric acid such that the pH never gets down to 5. In either case --- hypochlorhydria or hyperchlorhydria, upper GI symptoms will occur --- and --- the symptoms of hyperchlorhydria and hypochlorhydria are virtually identical.

When chyme passes from the stomach to the duodenum with the ideal pH of 4-5, the pancreatic secretions and the bile from the liver/gall bladder that mix with the chyme in the duodenum increase the pH up to 6.0 to 7.0. This is the optimum pH in the small intestine for the digestion of proteins to amino acids, starches to sugars, and for the absorption of minerals and trace minerals.

Clearly, there is no such thing as damage from “excess stomach acid.” The stomach is well-equipped to handle a pH of 1 or less with no problem at all. The only people who can be damaged by too much acid are those who are taking aspirin or certain other non-steroidal anti-inflammatory drugs, or those on prednisone.

The Heidelberg test for stomach acid has revealed how common is insufficient hydrochloric acid. The test involves administering a challenge with baking soda, then monitoring the change in stomach pH over time.

In a patient with normal fasting stomach pH of 1.5, an alkaline challenge with sodium bicarbonate will immediately raise the stomach pH to 7.2, but keep it

there for only about 18 minutes. By 20-22 minutes, there is total re-acidification of the stomach in those who produce normal hydrochloric acid.

In some patients, a bicarbonate challenge will not yield a pH of 7.2, but a pH rise to only between 3.5 and 5. Such a failure to alkalize does not mean the patient has too much stomach acid, but rather that the fasting fluid volume in the stomach is higher for these people. So, with larger fasting juice volume, the normal quantity of bicarbonate used for the challenge is not enough to raise the pH all the way up to 7.2.

In patients with either achlorhydria or an extreme hypochlorhydria, the fasting stomach pH is not the normal 1.5, but is extremely high at a neutral reading of 7.0. In such patients the bicarbonate challenge actually raises the stomach pH slightly even higher for a few minutes, until the bicarbonate is passed from the stomach into the duodenum. In patients with such extreme deficiency of acid, food is actually dumped from the stomach into the duodenum very prematurely after a meal, particularly a large meal. The size of the meal stimulates the stomach to contract, but there is not enough acid in the chyme to activate the duodenal reflexes to inhibit stomach emptying, so the stomach just dumps very quickly, thus yielding completely ineffective first stage of digestion affecting both protein and mineral nutrition. Such patients often report passing completely undigested food in the stool.

These achlorhydric or extremely hypochlorhydric patients tend to suffer allergies, because of the insufficient digestion of protein. The partly digested protein particles are absorbed in the intestinal tract and pass into the bloodstream, thus eliciting the typical IgE/eosinophilic allergic reaction. Another possibility is a stimulation of an IgG immune reaction with delayed food reactivities.

In patients who are functionally but not extremely hypochlorhydric, the bicarbonate challenge of the fasting stomach will raise the pH from its typical state (somewhere between 1.5 and 3) up to a level of 7.0 to 8.0 --- but --- the pH will not fall back to normal in 22 minutes as it should, but may take several hours or longer.

In patients with a Parasympathetic Imbalance (particularly if accompanied by an excess of the stress hormone cortisol) there can actually be hyperchlorhydria. The excess stomach acid does not cause stomach ulcers. Patients with hyperchlorhydria will have a normal fasting stomach pH of between 1 and 2, and the bicarbonate challenge will raise the pH up to around 7, but the pH will drop back down to normal within 12-13 minutes.

The problem with too much stomach acid is not stomach ulcers, but the excess retention of food in the stomach for too long. Since the chyme released into the duodenum is excessively acid, the reflexes from the duodenum slow stomach

emptying. So, the excessively acid chyme sits in the stomach for a long time. Instead of the stomach content pH being the optimal 4.0 to 4.5, it will range from 1.5 to 2.8. This acid chyme can be retained in the stomach for 6 hours, or even as long as 24 hours.

These people experience extreme discomfort when it is time for their next meal since they are stuffing that meal on top of the previous meal that is still trapped in the stomach. These people can be ravenously hungry even though their stomach is still full. The solution to hyperchlorhydria is to correct the Parasympathetic Metabolic Imbalance. Also, Phos Drops taken before the meal can be helpful. The acidity of the Phos Drops inhibits the stomach's secretion of hydrochloric acid just enough that the pH of the chyme will reach the ideal 4.0 to 5.5 level so the stomach can gradually empty.

Heidelberg equipment is, as I understand it, reasonably accurate. However, I suspect it is rarely, if ever, necessary.

Your biggest concern is achlorhydria. But anyone with achlorhydria will have pernicious anemia, which will be evident on a CBC as an elevated mean corpuscular volume, and a follow-up vitamin B12 test will be low or low normal.

The MetaMetrix GI function profile can yield a pretty good idea of digestive function. If the triglycerides and so forth are elevated, then there is insufficient pancreatic function. If they are normal, then the presence of either putrefactive bacteria or an alkaline stool indicates a deficiency of hydrochloric acid.

Much easier than invasive and expensive laboratory testing for adequate stomach hydrochloric acid is the good old baking soda challenge. Quite simply, baking soda in water is taken on an empty stomach (after at least a 10-hour over-night fast), and the subject awaits an uncontrollable BURP. This test is probably as reliable as the Heidelberg test. When sodium bicarbonate reacts with hydrochloric acid in the stomach it produces water, sodium chloride, and carbon dioxide. --- The carbon dioxide is belched up. The simple chemical equation is:

$$NaHCO_3 + HCl \rightarrow NaCl + H_2O + CO_2$$

Why does this simple test work? Most people (including most doctors) do not realize that a healthy stomach, even when empty, is extremely acid. In fact, the normal stomach has a pH below 2, and ideally very close to 1 at all times when it is empty. So, adding baking soda to an empty stomach should yield a burp within a few minutes.

There are actually 2 variables in a stomach that contains no food. One is the hydrochloric acid content of the stomach fluid, and the second is the quantity of stomach fluid. Any healthy person has an empty stomach pH of between 1 and 2, but some have a very dry stomach, and some have more fluid secretion at rest. (Generally, those who have higher secretion are your Parasympathetic types.)

For the test, a person must be fasting for at least 10 hours. The patient drinks ¼-½ tsp. of baking soda in 4-6 oz. of cold water, then times how long until the big burp, or a series of small burps. People with normal acidity and normal quantity of stomach contents will belch within 2 or 3 minutes. Those (Parasympathetic types) with normal acidity but high fluid content will burp sooner. Those with low stomach acid will not burp for 4 or 5 minutes, and those with rather marked hypochlorhydria will never burp.

Additional notes:

- Those with low hydrochloric acid are those who are subject to H. pylori infections (and stomach ulcers), as well as yeast overgrowth of the GI tract. (--- Note that H. pylori is the cause of stomach ulcers, and H. pylori infection is both caused by and causes hypochlorhydria. Treating gastric/peptic ulcers with antacids is absolute insanity.)

- Esophageal reflux or GERD is never caused by excess stomach acid, but frequently involves low stomach acid. Most cases of GERD have either insufficient stomach acid, which slows stomach emptying time and allows the stomach contents to be forced up into the esophagus, or, have delayed stomach emptying for some other reason (such as the generalized autonomic failure that occurs as part of the aging process).

  Some individuals (the Parasympathetic types) have an overabundance of fluid secretion into the stomach accompanied by a cardiac sphincter (where the esophagus empties into the stomach) that is "stuck" partially open. Food or drink entering that stomach with an already high fluid pressure, forces the stomach contents up through the open sphincter ⇒ GERD. (The PPI drugs work, not because they decrease acid, but because they force the stomach to empty prematurely, thus relieving the pressure.)

  [Some cases of GERD have nothing to do with stomach function at all, but rather are misdiagnosed as GERD when they are actually Eosinophilic Esophagitis (--- a first cousin to Eosinophilic Bronchitis (asthma), and Eosinophilic Fungal Rhinosinusitis.)]

- The production of adequate hydrochloric acid in the stomach is dependent upon the formation of metabolically produced carbon dioxide. The carbon

dioxide is converted by carbonic anhydrase to form carbonic acid, which in turn dissociates into bicarbonate ions and hydrogen ions. The hydrogen is transported into the stomach, as are chloride ions, to form the stomach hydrochloric acid.

----- Insufficient carbon dioxide production for any reason (--- thyroid insufficiency, Respiratory Alkalosis, Ketogenic or Anaerobic Metabolic Imbalances, insufficient intestinal flora to produce CO_2 in the colon, etc.) will decrease stomach hydrochloric acid production --- yielding hypochlorhydria and the consequent inefficient digestion and likely upper GI symptoms.

- Note that histamine parallels Parasympathetic activity, so that antihistamine drugs inhibit gastric secretion, and thus cause inefficient digestion and upper GI symptoms. Of course, this is the mechanism by which histamine 2 blocker drugs give temporary relief of upper GI symptoms while actually perpetuating the cause of those symptoms. The antihistamine drugs decrease stomach acid and prematurely empty the stomach, thus relieving symptoms --- all the while they further decrease the essential production of hydrochloric acid, thus assuring that the symptoms will return as soon as the drug effect wears off. --- In other words, the more a person takes these drugs, the more the person feels the need for them. (--- Nice game for the drug companies to play.)

- Many GERD symptoms are associated with excess nitric oxide. Excess nitric oxide will cause inappropriate relaxation of the gastroesophageal sphincter (as will excess parasympathetic nerve activity). Nutrition supplements designed to inhibit nitric oxide biosynthesis include melatonin, along with tryptophan, vitamin B6, folic acid, vitamin B12, methionine, and betaine. Research shows this supplementation achieves symptomatic relief in 100% of the patients with GERD, compared to only 66% symptom improvement in those taking proton pump inhibitors. ----- Another effective combination of supplements includes melatonin 6 mg, 5-HTP 100 mg, methionine 500 mg, betaine 100 mg, taurine 50 mg, riboflavin 1.7 mg, vitamin B6 0.8 mg, folic acid 400 mcg, and calcium 50 mg. Reduction of melatonin to 3 mg results in the return of symptoms.

If you want a functional test for <u>gastric/peptic ulcers</u>, and often for duodenal ulcers, simply have the patient on an empty stomach ingest 1 <u>non</u>-enteric coated tablet of bromelain enzyme. If there is pain, then there is an ulcer. If there is no pain, then later that day or the next day on an empty stomach, swallow 2 tablets. If no reaction, then swallow 3 tablets some other time on an empty stomach, then as much as 4. If 4 tablets elicit no painful response, then go back to 1, then 2, then 3, then 4, completing 2 cycles of the proteolytic enzyme challenge. If there is never any pain, then there is no ulcer.

The mechanism behind bromelain as a diagnostic aid for upper GI ulcers is simple --- bromelain is a proteolytic enzyme. When there is an ulcer the mucosal tissue is exposed and unprotected --- essentially, it is raw meat. The bromelain will literally begin to "digest" the lesioned tissues on a small scale. (For your information, most meat tenderizers are either bromelain or papain --- and the way they work is by digesting the meat protein.)

- Note also that hydrochloric acid will not exacerbate the pain of a stomach ulcer. It will exacerbate the pain of reflux esophagitis only because the stomach contents that are being refluxed into the inflamed esophagus are acid, but <u>not</u> because the reflux is caused by <u>excess</u> hydrochloric acid.

Patients with upper GI symptoms

1. The first thing you need to do is familiarize yourself and make available to your patients the write-up on your NUTRI-SPEC website regarding proton pump inhibitors, GERD, H. pylori, peptic ulcers, and eosinophilic esophagitis.

2. By far the most common cause of the upper GI symptoms suffered by so many people is eating too often. The second most common cause is eating carbohydrates that ferment in the upper GI tract while held there awaiting completion of protein and fat digestion.

3. The "Burp Test" baking soda challenge is not infallible, but often yields clinically useful information.

4. Getting back to the eating too frequently --- A complete meal (as per the Nutri-Spec Fundamental Diet) takes at least 4 hours to clear the stomach in a perfectly healthy person younger than 28 years old. For those of us who are over 28, it takes longer than 4 hours --- at least 5 hours for the meal to even leave the stomach. So --- if anyone dumps anything into the stomach within 5 hours after the previous meal, it is a 100% certainty there is going to be at least some pressure and/or reflux.

5. If you are not doing NUTRI-SPEC testing on a patient, another approach is to do just the dermographics test.

   If you get a wide, red line, the patient with upper GI symptoms generally benefits from Phos Drops + seltzer water on an empty stomach before meals. If you do not get a red dermographics, the patient generally benefits from seltzer water before meals and Proton Plus after meals.

   If using Phos Drops, start with 10 Phos Drops + seltzer water on an empty stomach 2 times daily before meals. If symptoms improve but do not abate entirely, then increase in increments of 5 drops until maximum improvement is achieved.

   If using Proton Plus, have the patient drink the seltzer water before the meal, and take 1 Proton Plus 3 times daily after meals. (Due to the carbon dioxide in the seltzer water, Proton Plus is indeed to be taken after meals.) If symptoms improve but do not abate entirely, then increase the Proton Plus to 2 after each meal, or 3 for a particularly large meal.

   The amount of seltzer water to be used in conjunction with the Phos Drops or Proton Plus should be 6-10 oz., depending on thirst and hydration status. (Flavored seltzer water is less than ideal, but okay. Sweetened is a disaster.)

6. --- Remember --- the 5+ hours between meals is essential. Even a little munchy dumped into a full stomach will tend to cause pressure and bloating and perhaps burning.