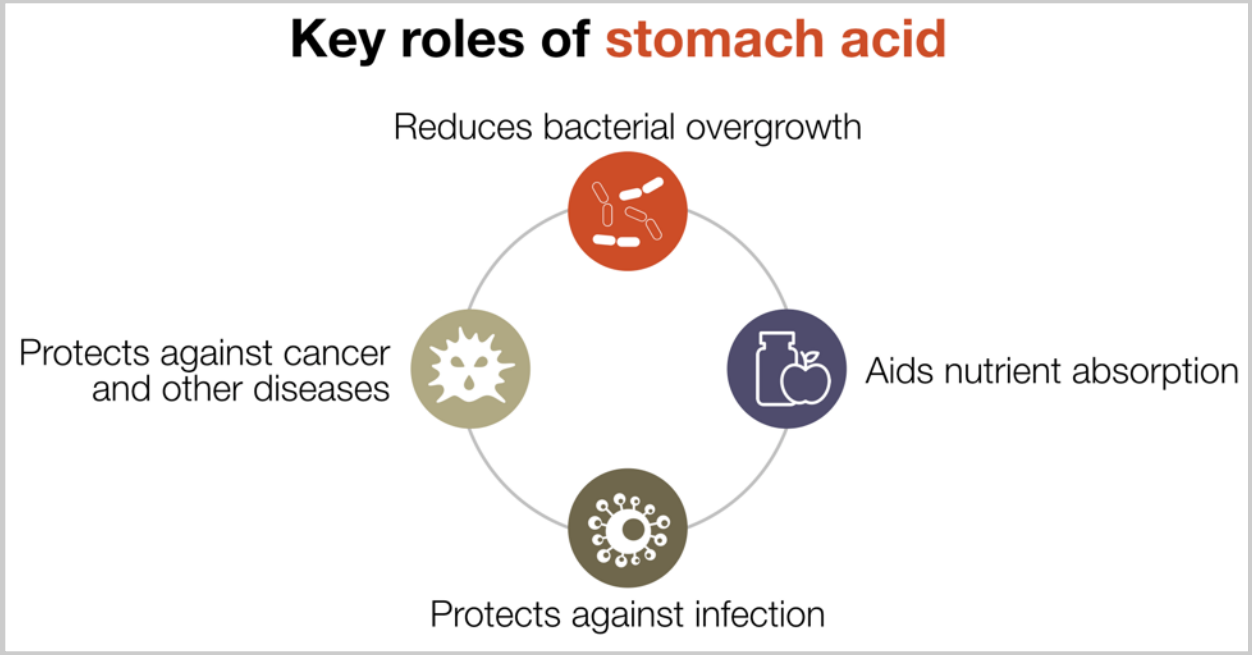
Gut Treatment Protocols: GERD, IBD & IBS, Part 2

Many patients will come to you with GERD and heartburn, and they'll be on acid-suppressing drugs, primarily PPIs, and they will often have been on these drugs for years and even decades, in some cases. One of the biggest ironies in medicine is that these drugs may help relieve the symptoms of GERD, but they not only don't address the cause, they likely even make it worse. They can suppress stomach acid production to nearly zero, and as we've talked about, low stomach acid is probably one of the main contributors to bacterial overgrowth, which in turn is a major contributor to GERD and heartburn. So getting patients off acid-suppressing drugs and supporting stomach acid, bile, and enzyme production is one of the most important steps you can take in treating GERD, and again, check out the comments on that article that I just mentioned, where you'll see hundreds of testimonials from people who were able to get off their PPIs, even if they've been on them for more than a decade, with the approach that we're talking about here.



There are a lot of other reasons for patients to stop taking PPIs. In the most general sense, stomach acid plays a vital role. It's not just there to give us heartburn. The consequences of low stomach acid are directly related to its important functions, so in addition to protecting us from bacterial overgrowth, it also helps with nutrient absorption, protects against infection, and protects against cancer and other diseases. I've written in detail about these risks in the GERD e-book; we're going to put a link to it in the supplemental materials, so again I'm not going to go into detail

here. I'm just going to review and you can refer to that e-book for the research studies and all the nitty-gritty.

Cold turkey?

Address **underlying pathologies first,** then titrate

The longer they've been on, the longer it will take to come off

Must work with **prescribing physician**

But how and when should a patient stop taking PPIs? In some cases, the patient will be able to stop cold turkey if they switched to a Paleo, very-low-carb diet and started taking HCl and enzymes and had really great results. In other cases, you have to address the underlying pathologies like SIBO, H. pylori, nutrient deficiency first, and then transition the patient off of the PPIs afterwards. If patient has been on the drugs for a long time, you may need to titrate off the acid blockers slowly and gradually because their endogenous production of stomach acid has been so suppressed for such a long time. As a general rule of thumb, the longer they've been on the medication, the longer it will take for them to come off of it, and you have to evaluate based on patient needs and response to treatment.

Of course, this goes without saying, if it's not within your scope of practice to prescribe medication, they must work with their prescribing physician as they make these changes. But you can educate your patients on the risks and benefits of PPIs and what alternatives are, and that's really your role as a clinician if you're not able to prescribe. And of course, it's your role as a prescribing physician as well, and many patients just don't get enough education about the medications they're taking and informed consent, so this is helpful no matter where you fall on the spectrum.

Next consideration is reducing consumption of carbohydrates that can feed bacteria in the small intestine. Now as we discussed, a low-FODMAP diet can be particularly helpful in this situation, but it may not be a good long-term approach because FODMAPs also feed bacteria in the colon. But in the short term, while you're addressing the GERD and while you're helping the patient to stop PPIs, a low-FODMAP diet can be really useful. We will typically start with a lower-carb Paleo diet as a foundation, and then we might add the low-FODMAP recommendations if they're not getting the success that we would like them to.

High FODMAP foods

Excess fructose	Lactose	Fructans	Galactans	Polyols
Fruit: apple, mango, nashi, pear, tinned fruit in natural juice, watermelon	**Milk:** milk from cows, goats or sheep, custard, ice cream, yoghurt	**Vegetables:** asparagus, beetroot, broccoli, brussels sprouts, cabbage, eggplant, fennel, garlic, leek, okra, onion (all), shallots, spring onion	**Legumes:** baked beans, chickpeas, kidney beans, lentils	**Fruit:** apple, apricot, avocado, blackberry, cherry, lychee, nashi, nectarine, peach, pear, plum, prune, watermelon
Sweeteners: fructose, high fructose corn syrup	**Cheeses:** soft unripened cheeses eg. cottage, cream, mascarpone, ricotta	**Cereals:** wheat and rye in large amounts eg. bread, crackers, cookies, couscous, pasta		**Vegetables:** cauliflower, green capsicum (bell pepper), mushroom, sweet corn
Large total fructose dose: concentrated fruit sources, large serves of fruit, dried fruit, fruit juice		**Fruit:** custard apple, persimmon, watermelon		**Sweeteners:** sorbitol (420), mannitol (421), isomalt (953), maltitol (965), xylitol (967)
Honey: corn syrup, fruisana		**Miscellaneous:** chicory, dandelion, inulin		

FODMAP stands for "fermentable oligosaccharides, disaccharides, monosaccharides, and polyols," and these are all carbohydrates that are poorly absorbed, and they can linger in the gut and become food for bacteria there. I've put some examples of FODMAPs listed here on the slide. The low-FODMAP diet has been studied extensively for system relief for IBS, and it provides that in over 70 percent of cases, which makes it one of the single most effective treatments for IBS. Strangely, I haven't yet seen a single study investigating the efficacy of the low-FODMAP diet in GERD, probably because the conventional approach doesn't really recognize low stomach acid or bacterial overgrowth as significant causes or contributors to GERD, but in my clinical experience, I've found that the low-FODMAP approach can be very effective for GERD.

Foods with moderate or high fermentation potential (FP)

Vegetables	Fruits	Starches	Other
Green peas	Banana	Green plantain	Milk
Jerusalem artichoke	Blueberry	Taro root	Fruit juice
	Apple	Basmati rice	Beer
	Cherries	Sweet potato	
	Payaya	Yam	
	Passionfruit	Yuca *(cassava)*	
	Persimmon		

Another option is a low fermentation potential diet. Fermentation potential's a concept originally presented by Norm Robillard in his book ***Fast Tract Digestion Heartburn***, and it's a measure of how likely carbohydrates are to be fermented by intestinal microflora. Carbohydrates that are rapidly absorbed high up in the small intestine are given a low fermentation potential, or FP, and those that take longer to break down and thus may be fermented by gut microbes are given a higher FP.

So white rice, interestingly enough, depending on the type of white rice, can have either a low fermentation potential, so jasmine white rice, a very, very high glycemic index, actually higher than glucose itself, has a low fermentation potential, but basmati white rice, because it has a lower glycemic index, has a higher fermentation potential. So, foods are assigned a numerical value using a mathematical formula for calculating fermentation potential, and then they're categorized as low, medium, or high in terms of their propensity to feed intestinal microflora. So I've listed some examples of foods with high fermentation potential on the slide, and they're basically a mirror image of some of the fermentable carbohydrates we discussed that are beneficial for long-term gut health. If you want to learn more about this approach, you can get some charts and more information on his website, which we'll put a link to in the resources section. I can say we don't typically use this, we rely more on a basic Paleo approach plus low-FODMAP, but in some cases patients have found it to be useful.

Low fermentation potential	Low FODMAP
How quickly a carbohydrate is absorbed according to formula including glycemic index	Excludes certain **carbohydrates that are poorly absorbed**

The concept behind the low fermentation potential on the low-FODMAP approaches is similar; the idea is to reduce the levels of microbial overgrowth in the small intestine, whether bacteria or fungi or parasites, by depriving them of their food source. However, they employ a different strategy for achieving that goal. Low fermentation potential is determined primarily by how quickly a carbohydrate is absorbed as measured by its glycemic index, whereas the low-FODMAP diet excludes specific types of carbohydrates that are poorly absorbed. Notice that foods with low fermentation potential are those that are rapidly absorbed into the bloodstream like jasmine and short-grain white rice and white potatoes, and some of those foods, of course, for patients who have severe blood sugar issues, may not be beneficial, so you have to consider that when you're choosing which approach to use, and let's talk a little bit more about that.

I recommend starting in general with the low-FODMAP approach and seeing how that goes. It's less restrictive and I think it's healthier for beneficial bacteria in the colon and overall, over the long term. If symptoms don't improve significantly, you could consider adding low fermentation potential on top of low-FODMAP, but that will be an incredibly restrictive diet, and it's also highly effective in starving bacteria not only in the small intestine, but in other parts of the gut, so I think it should only be used therapeutically for the short term, and remember, neither of these approaches should be used at all when a patient's on an antimicrobial protocol. Gut bugs need to be happy and well fed to be killed. Remember the saying, "Gotta feed 'em to kill 'em." So you might use these approaches in patients who haven't yet been tested or treated for SIBO or H. pylori or the other underlying causes, or can't be tested for some reason, or you might use them in cases of recurrence after treatment.

Step two is to replace stomach acid enzymes and nutrients that aid digestion. These include hydrochloric acid enzymes and bile. So let's start with HCl. This is what the stomach should normally produce, and in its supplemental form, it's best to take as betaine hydrochloric acid, in a form that contains either pepsin or acid-stable protease, which are proteolytic enzymes. If the stomach is not producing enough acid, it's likely it's not producing enough protein-digesting enzymes.

HCL Protocol

Step 1	Step 2	Step 3	Step 4	Step 5
Take **one capsule of HCL w/pepsin** in early part of meal	If no side effects after 2-3 days, **increase to two capsules**	**Continue increasing every 2-3 days** by one capsule, until you experience mild burning sensation in epigastric area	**Reduce dosage** to previous number of capsules you were taking before you experienced burning sensation	**Reduce dosage over time** as underlying mechanisms are addressed

Ideally, we'd be able to easily test for stomach acid levels to determine the optimal dose of HCl, but in practice that's difficult because it requires special equipment and a test called the Heidelberg capsule test, which is not commonly offered, even among gastroenterologists, so in practice most clinicians resort to an empirical method known as the HCl challenge. With the HCl challenge, you start with one 650-milligram capsule of hydrochloric acid with pepsin in the early part of each meal. If there are no problems after two or three days, you increase the dose to two capsules at the beginning of meals. Then, after another two days, you could increase to three capsules, and you would increase the dose gradually in this step-wise fashion until the patient feels a mild burning sensation. At that point, you would reduce the dosage to the previous number of capsules they were taking before they experienced that burning and stay at that dose. If the patient doesn't have any burning sensation by six or seven capsules, I'd say further improvement from increasing beyond that is rare, so we would generally advise the patient to stop increasing at that point. In some cases, even one capsule will be too much and could cause an exacerbation of burning, and you need to use a different kind of product with maybe 200 milligrams of HCl in it, in order to do the HCl challenge with that patient so that they can be more incremental with the dosing. Over time, patients will often find that they can reduce the dose of HCl as the underlying mechanisms leading to GERD or heartburn are addressed.

Important thing to be aware of is that HCl should never be taken and this HCl challenge should never be performed by anyone who's using any kind of anti-inflammatory medication like corticosteroid, prednisone, aspirin, ibuprofen, or other NSAIDs. These drugs can damage the GI lining, and HCl might aggravate that damage, increasing the risk of gastric bleeding or ulcer, and by the same token, HCl should not be used by anyone who currently has an ulcer.