More posts: <http://survivingantidepressants.org/index.php?/topic/4361-tips-for-tapering-off-stomach-acid-blockers-or-ppis-esomeprazole-lansoprazole-omeprazole/>

PPIs interfere with acid secretion: you all know that. But acid is secreted in response to an enzyme, gastrin, in the blood (it's part of an overall biological feedback system - as I suspected). When the acid secretion is suppresed, gastrin levels in the blod increase -as the body tries to make more acid. Hypergastremia - itself a health risk!   
  
Wikipedia has an intersting article on gastrin. One of the effects is that high gastrin relaxes the lower oesophagal sphincter. So PPIs may actually cause more regflux - albeit non-acidic. Gastrin also induces gall-bladder emptying - which explains why bile reflux is a problem on PPIs.   
  
Oh - there's so much the doctors don't tell you. Probably because they do not know!

<http://www.barrettscampaign.org.uk/forum/forum_posts.asp?TID=560>

Hi http://forums.heartburn-help.com/images/smilies/smile.gif The type of poylps that PPIS cause are called fundic gland poylps . These are beneign in 99.9999 percent of cases . Lots of people the board have them and often they disapear themselves after a person stops PPIS . They are no big deal and really nothing to worry about. There is information about them in the PPI insert. They are considered a very harmless type of poylp . As LSAT said , noone here has every had a problem. Anything questionable is biopsied anyway.   
  
PPIS can cause atrophic gastritis which is a pre curser to stomach cancer , but you would have advance notice of that type of gastritis developing anyway. It usually happens over many years.   
  
As for low B12 caused by PPIS , thats mostly theroretical .. As of the end of 2005 , there was only one documented case in the world , and that man didn't have his B12 levels checked before starting PPIS . Ratobranco, i guess you posted the second case, with that 78 year old woman. ( they mention that there was only one previous case in the article you posted ) I don't know if there have been anymore cases since then . Low B12 is common anyway with older people . PPIS i imagine would exacerbate it. There have been no case reports of PPIS causing low B 12 in younger people in the entire world ...yet , although anything is possible, especially as people take them longer and longer ... but you're dealing with a needle in a haystack.   
  
You said Barrets is not such big problem since it can be eradicated using Halo 360. Just because Barretts appears to be gone , it doesn't mean there isn't a tiny cell floating around somewhere . It won't be known if ablation decreases the risk of cancer for many years. People have developed esophageal cancer 2-3 years later with other types of ablation therapies even though Barretts appeared to be gone . I'd use cautious optimism when it comes to ablation, it is an exciting advance though. Not all esophageal cancer risk factors are known yet either.   
  
Pshemek . Have you had an endoscopy ? What did it show ? Have you been ph tested ? Why do you assume you need PPIS for life ?

I just asked my gastroenterologist about B12 and he said that there are pancreatic enzymes that help absorb it, and they don't need stomach acid to be effective. I am going to do more research about it though, I know many others who do the sublingual supplement so I will likely ask to have my levels checked every so often, just to be sure. My doc did say that calcium absorption can be a big issue though, so he advised me to take a calcium supplement on top of dietary intake. Every appointment just seems to raise more questions..

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The PPIs are prodrugs. These prodrugs require gastric acid secretion to be converted to the active sulfenamide or sulfenic acid that blocks gastric acid secretion. All PPIs except tenatoprazole have short half-lives (about 1 hour) and all have good oral bioavailability. Most PPIs are metabolized by CYP2C19 and 3A4. Hepatic impairment and old age reduce clearance of the PPIs, as do mutations in CYP2C19.

Acid suppression studies comparing omeprazole, lansoprazole, rabeprazole, and pantoprazole show equivalent efficacy. Most studies using standard doses have not shown a significant difference between the four PPIs for the healing of reflux esophagitis or duodenal ulcer. Esomeprazole and tenatoprazole have stronger acid suppression, with a longer period of intragastric pH greater than 4.

<http://www.barrettscampaign.org.uk/forum/forum_posts.asp?TID=1319>

> A few years ago, I argued this point with two of the country's top  
> gastroenterologists who would not accept it as minerals are absorbed in  
> the duodenum not the stomach. They have since had to admit I was right.  
> If the minerals have not been sufficiently dissolved out of the food  
> before they reach the duodenum, the majority will pass through it into  
> the lower intestine too quickly.  
  
Minerals are converted to the chlorides by the hydrochloric acid in the stomach. For instance calcium catbonate is not soluble. HCl in the stomach converts it to calcium chloride - which is soluble - and carbon dioxide.  
  
Similarly magnesium and iron: the chlorides are soluble and easily assimilated.  
  
Other similarly soluble salts are formed with acetic acid (vinegar) and citric acid (lemon juice).  
  
If the minerals remain in insoluble form they cannot be assimilated.  
  
All this is very basic chemistry. It proves doctors are not taught chemistry!