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VIRTUAL LAF CONFERENCE

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SUBJECT: LAF, GERD & Diaphragm Displacement

Several Bulletin Board threads have dealt with the connection between GERD (gastroesophageal reflux disease, heartburn) and afib, while other more recent ones have considered the possibility that a displaced diaphragm may somehow be involved.

GERD is associated both with an ANS (autonomic nervous system) dysfunction and an excessive generation of oxygen-based free radicals (oxidative stress). The most recent LAF survey revealed that 34% of the 100 respondents suffered from GERD. This is not significantly different from the prevalence in the general population. However, the majority of GERD-suffering afibbers felt that there was a connection between a flare-up of GERD and the initiation of a paroxysmal afib episode or a worsening of symptoms in the case of permanent afibbers.

Belching and indigestion in general and perhaps a displaced diaphragm could put physical pressure on the heart muscle and possibly initiate PACs that could turn into an afib episode.

I believe it may be worthwhile to explore these connections in depth once more to see where it leads us. Certainly, I agree with all the posters that emphasize the importance of stress (past and present) in the etiology of afib. I am convinced it is a major factor in my own case. Perhaps there is a connection between GERD, diaphragm abnormalities, indigestion and stress. Some afibbers have found, at least temporary, relief from afib episodes by taking Nexium or other GERD medications – are there other approaches out there that have been found useful?

So I would like to lead off this new Conference Room subject by reposting Mike F's excellent contribution. I do feel that this subject ought to be covered in the Conference Room rather than on the Bulletin Board so that our deliberations will be preserved for "posterity" in the Conference Proceedings.

Hans

Hi folks,

Further to my suggesting it in a recent thread, Hans asked me to post a new topic to the CR regarding the possible/likely (for many) between AF and GERD/esophageal problems/diaphragm displacement.

Problem is, I'm not sure how to approach this, or even whether I should given it's been well discussed before by others more learned than I.... Furthermore, my views and opinions on AF and its likely cause/s seem to evolve day by day the more I learn (largely here on this forum) and deliberate.

A recent thread encouraged everyone to put forward their own concise theory of why in their opinion AF has struck them. It is interesting to note that whilst a wide range of theories are proffered, many of them fall into broad categories including chronic stress (physiological and/or psychological), digestive maladies, MSG and/or other man-made neurotoxins and pollutants, hormonal imbalances, electrolytic imbalances, and genetic factors. I tended to go in broad terms with James D.'s 'channelopathy' take on things. It's looking more and more likely that the primary cause of AF is a fault - either temporary, occasional/random, cyclical, or more persistent - with ion channel operation in the cell membranes of cardiac cells.

As I already commented in my own response to the aforementioned thread, this channelopathy can be totally hereditary (in those individuals whose extended families include many other AFrs), largely lifestyle-influenced/precipitated (neurotoxins, pollutants, other chronic physiological and/or psychological stressors), or truly temporary e.g. 'holiday heart syndrome'. It's relatively easy to acknowledge the existence of a group of hereditary AFrs, as it is similarly easy to understand how a bout of severe binge-drinking can put some otherwise (but still with a very slight degree of predisposition - NOT so easy!) 'normal' folks into AF. The real problem is tackling that broad group in the middle which largely excludes both aforementioned groups and comprises the majority of us here on this forum i.e. those of us who for some reason/s all of a sudden in our middle age develop AF.....although we should remember at this point that everyone of us must have some SLIGHT degree of genetic predisposition to AF (since some adults can binge drink, worry/stress, eat poorly etc etc etc and NEVER get AF - although they'll likely get something else instead in due course.....).

WHY DO WE DEVELOP AF????? OK, so we're back to the old question - why me. Accepting that channelopathy is the cause of AF, then we can refine our question to 'why have my ion channels (predominantly K and Ca?) become faulty'???? This is where things get complicated since soooo many factors can throw our systems out of sync. - especially where such factors are multiple, exacerbate/potentiate each other, and are chronic.

The reason I personally keep returning to digestion is founded upon the old adage..... we are what we eat.... or, more correctly, we are what we ABSORB. There is little doubt that many of us here - including myself - have had, and continue to have, problems with GERD, heartburn, gas/bloating etc. OK, so many adults have these problems who never get AF. But not as BAD and/or OFTEN as many AFrs.... or should I say, not as CHRONICALLY as many AFrs. Is this enough to cause AF, or is something else required?

This brings me on to chronic psychological stress - either externally imposed or - perhaps more significantly - self-imposed in the form of anxiety (also possibly resulting from previous and long-standing exposures to external stressors). I know that I have always as an adult been a VERY anxious individual. Is it this anxiety which has resulted in my digestive maladies? Or is it this anxiety PLUS my digestive maladies which have resulted in my AF? Chicken and egg..... AGAIN! Moving onto esophageal/diaphragm displacement, what is it that pulls these muscles out of place? TENSION/CONTRACTION. Could this not also be down to chronic psychological (e.g. bad diet/improper nutrition) and/or accumulative psychological stress?

Each and every one of us is unique. Nonetheless, each one of us here has a slightly above 'normal' genetic predisposition towards developing AF. In essence, my equation for AF for the majority of us here would be:

Slight predisposition towards some appropriate form of channelopathy PLUS physiological stressors (such as improper nutrition and/or compromised digestion) AND/OR chronic psychological stress in any form EQUALS imbalances in the ANS/hormones/electrolytes which will activate the channelopathy thus resulting in AF.

It should be noted that the types of imbalances in the above equation might be transient and without pattern for some and highly cyclical for others.

In my view, all each of us can do is to strive to - one-by-one - reduce the numerous physiological and/or psychological stressors in our lives. This takes a LOT of TIME and EFFORT. Just look at Erling and Fran and Jackie’s (and others) example. But it's good to know it CAN be done. Whatever the upshot of our healthy endeavours, they can only serve to slow the typical progression of AF (as well as to make us healthier and more disease-resistant in general) until such time as near 100%-effective drugs and surgical procedures become available).

OK folks, that's one hour's worth of thinking and 'tapping' over with... and all because Hans asked me to kick off a new

topic. Truth is, as I've said, I think we've already covered that area before, and such a topic is only part of a much bigger and intricately woven picture which we're still collectively trying to unravel. Well that's it from me: I'm off out to jump in my car, put the roof down, and have a ride along some country lanes and forget about AF for a while!!

Mike F.

"We are what we eat.... or, more correctly, we are what we ABSORB" might be paraphrased "we are what we think, feel and ABSORB consciously and unconsciously.

There is no reason why ion channel malfunctions, like all other intricate, complex, and interrelated systems of the body, are not caused by back loads and bombardments of tension and stress.

It is easier and perhaps more satisfying for us to focus on the empirical, materialistic functions than on the ephemeral, unseen factors.

Carol A.

Carol A - Most definitely true, but moreover, in addition to repressed emotions and stress that have cellular memory, is the physical factor of damaged cellular lipid layers which do not allow the ion channels to function properly or at all. This is physical destruction from the wrong type of fats and not enough of the Omega 3 essential fatty acids which allow the lipid layer of the cells to remain unclogged and flexible.

Just the factor of damaged magnesium receptors, alone, could be enough to disrupt the flow of essential minerals or electrolytes in and out of the cell.

Both the damage from stress be it physical, emotional, environmental and damage to the physical structure of critical cellular components is at the very core of any ailment - not just AF.

Because the unseen is difficult to comprehend, this makes it all the more critical to be aware that "everything happens at the cellular level."

Jackie

Mike F.... regarding your examination of chicken/egg digestion/absorption/stress/illness cycle....

One doesn't have to look very far to find support for the fact that unless we break down food so that it can be assimilated into the body (where the nutrients from food support all of the cellular functions of the body), it makes perfect sense that the body will eventually break down or not function.

It goes beyond absorption - and to that of assimilation or the actual using of the nutrients required. If they aren't there, or can't get in side the cell for utilization, all the supplements or good food in the world will not correct the good condition.

So....first, the right foods, then proper digestion, then proper absorption through the small intestine which is healthy and not leaking or clogged with candida, and into the blood stream where it is actually assimilated into cells.... each facet has to function well for optimal utilization.

All the while, the body works on this process, additionally, it has to sort out and dispose of the toxins that come in to the body with food or other sources such as drugs, alcohol, environmental toxins coming through the skin, water, etc. It's a big job.

But the basic step is 1) chewing well 2) breaking down food with either a good supply of one's own gastric juices or

aided by digestive enzymes. and 3) sending it off to the small intestine for absorption after it is treated by the gall bladder and pancreas and placed in a usable form for the body.

The GERD and the gas and the hernia or diaphragm are just results of improperly or inadequately performed initial steps...malfunctions either mechanical or chemical, compounded by a physical distortion which impacts both the diaphragm and the physical location of the vagus nerve and its comfort.

Then to go on and compound the problem, if we eat improperly and as a response, stimulate adrenaline because of either toxic overload of alcohol, too much sugar and other intakes not suited to our body's tolerance, we set off the other cascades of events that prime us for not only AF but other reactions.

Everything happens at the cellular level. Always has; always will.

Good post, Mike. Creating awareness is always important.

Jackie

I found this excerpt from the book "Why Stomach Acid is Good For You", to be of interest.

Certain medications can weaken the LES. These include bronchodilators used to treat asthma, NSAIDS, certain types of blood pressure lowering drugs (e.g. calcium channel blockers, beta blockers), the antianxiety drug Valium, the narcotic analgesic Demerol, and nitroglycerine used to treat coronary artery disease. These drugs all function in one way or another to relax muscles, including those that surround the airways and blood vessels, but also those that comprise much of the GI tract.

Some medications can directly irritate the gastrointestinal lining, leading to heartburn, esophageal and peptic ulcers, and leaky gut. These drugs include aspirin, NSAIDS (e.g. ibuprofen, naproxen, and many others), the antibiotic tetracycline, the cardiac arrhythmic drug quinidine, potassium chloride tablets, and iron salts. GI irritation is a major side effect of some of these drugs, causing great injury and severely limiting their use.

Foods that irritate or weaken the esophageal lining are: fats, chocolate, coffee, mints (esp. peppermint and spearmint), sugar, onions, some alcoholic beverages, citrus fruits and tomato based foods, spicy foods, carbonated beverages.

Richard

Hans - I totally agree with the connection between the diaphragm displacement and afib..... hence my previous posts on the success I had with correcting the displacement.

Also - in addition to belching and indigestion, remember, the symptom that led me to the investigation of the DD or HH (hiatal hernia) possibility was the advent of hiccoughs and then almost immediately, afib.

Two things were factors - 1) definite displacement and 2) hiccoughs are a symptom of magnesium deficiency.

If readers new to this post have not read the report I reference in the hiatal Hernia article by Steve Rochlitz, PhD entitled,

"A Missing Link To Chronic Illness, Allergies and Longevity?: Vagus Nerve Imbalance/Hiatal Hernia Syndrome," by Steve Rochiltz, PhD,

then, now would be a good time to go to this post again and note either the excerpts or the full article listed in the reference area.

Acid Reflux, GERD, Hiatal Hernia and Vagus Nerve Imbalance
Author: Jackie (---.73.204.229.Dial1.Chicago1.Level3.net)

Date: 03-18-04 12:24

I think this topic is very important since I had the greatest success in eliminating the postural triggers of AF once my diaphragm was adjusted.

Important information here and it deserves to be examined in detail.

Thanks Hans and Mike.

Jackie

This is an excellent topic for discussion and I have been pondering my present feeling on it ever since it appeared on the BB in a post by Dean on 4/7/04. Having read all the excellent posts on that thread I would like to add my own here in the CR.

Why do some of us get LAF while others do not? We mustn't forget the fact that AF can be induced in all dogs, via either fast pacing (adrenergic) or injection of a cholinergic (vagal). This would suggest that everyone has the potential for LAF, given the right environment.

It is clear that there is no one "answer" to the LAF puzzle. However, I believe that it can be distilled a bit. I've divided the below discussion into direct effects on substrate (the heart) and effects on electrolytes and pH.

SUBSTRATE

There has been much talk about heredity and LAF. This has been primarily in connection with a channelopathy. This is certainly understandable given the relatively recent announcement by a team of Chinese investigators that familial AF was due to a potassium channelopathy. However, I think we are possibly overlooking a more plausible explanation on this.

Like our fingerprints, the three dimensional map of intraatrial nerve fibers (both SNS and PNS) are distinct for each individual. Our "individuality" is more than biochemical. It is structural as well.

I lifted much of the following from the ANS section (p. 24) of a Swedish book on AF, available at http://www.ub.rug.nl/eldoc/dis/medicine/a.e.tuinenburg/

"Both sympathetic and parasympathetic (vagal) nerve fibers supply the atria. The right hand part of the ANS subserves the right atrium and SA node, whereas the left hand part subserves the left atrium and AV node. Right-left differences in atrial autonomic innervation are therefore inrinsic. Furthermore, the distribution of vagal innervation of the atria is not uniform (Alessi 1958)." I'm sure sympathetic atrial innervation is similarly nonuniform or nonhomogeneous. Both sympathetic and vagal stimulation cause shortening of the refractory period and the nonhomogeneity of nerve fibers causes dispersion of this refractoriness.

This might explain much of the dysautonomia in LAF. One could make an argument that ion channels, while critical in explaining remodeling due to AF, might not be essential in explaining its development.

To further analyze this possibility, it might be instructive to look more closely at some kinds of episodes. Many LAFers state that some episodes are triggered by a short period of intense anaerobic activity, e.g., lifting something, sprinting at the end of a workout, etc. Quoting from the same page of the above source:
"Moreover, sympathetic and vagal activity do not exclude one another; both may be active at the same time. In fact, vagal effects are more pronounced in the presence of high sympathetic tone and vice versa ("accentuated antagonism") (Levy 1971). When a premature atrial beat hits the atria at a point in time when autonomic tone has created favorable conditions for atrial arrhythmia to occur (in particular shortened refractoriness), both initiation and perpetuation of AF are facilitated. In addition to the above, vagal stimulation produces a marked variability in atrial refractoriness, which also facilitates AF (Liu 1997)."

I believe that GERD, hiatal hernia, and dietary glutamate all impact LAF through the vagus nerve. Like swallowing or lying down, they stimulate vagal afferents (sensory nerve fibers) that cause crossover stimulation of the SA node. I've posted many times on how I think this happens. For example, it has been reported that AF has been triggered by gastroscopy, a procedure in which a fiberoptic tube is introduced into the stomach to visualize its inner lining. During the procedure air is manually pumped in to separate the mucosal folds for better viewing. Perhaps this gastric distention is what triggers the AF in gastroscopy.

IMHO PVI works because it decreases vagal tone. Efferent vagal nerve fibers (motor nerves) enter the heart through the pulmonary veins and are transected by the procedure.

ELECTROLYTES AND pH

K and Mg are at the root of the problem. PH is only a player insofar as it impacts these two cations. The primary determinant of blood K is aldosterone and cortisol. Cortisol, which can bind to the same mineralocorticoid receptors, is at 1000 fold higher concentrations than aldosterone and should not be overlooked in the genesis of hypokalemia.

Aldosterone regulates blood K via three separate and distinct mechanisms, 1) RAAS (renin angiotensin aldosterone system), 2) K/Na ratio via direct effect of K on the adrenal cortex (zona glomerulosa), 3) ACTH (adrenocorticotropic hormone), which is probably the least significant of the three. This latter however, is very significant in stimulating the production of cortisol and would result in low renin levels.

Conditions that cause urinary K and Mg wasting include stress (low renin), dehydration (high renin), hypoglycemia, alkalosis (hyperventilation and mountain climbing) and thyroid imbalance.

Stress is most noteworthy because 1) there are many avenues for it to cause loss of these two electrolytes, including increased production of glucocorticoids (cortisol) and mineralocorticoids (aldosterone), hyperventilation (causing alkalosis), catecholamine production, etc., and 2) of its direct effects of the latter on the heart (see above). Aldosterone and cortisol also directly affect the heart, but I personally think this is more responsible for heart disease and not LAF. Please read Mildred Seelig's recent book *The Magnesium Factor* on this.

Hypoglycemia causes not only release of catecholamines but also ACTH. Both of these result in hypokalemia. Hyperthyroidism causes LAF primarily via hypokalemia. The mechanism for hypothyroidism is less clear to me.

And, of course, IMHO besides stress, the main player resulting in insufficient K and Mg is dietary shortfall. It takes many years for K and Mg to drop to levels sufficient to cause LAF and it will take years to sufficiently replenish stores. The late Dr. Philippe Coumel described his seminal case of VMAF in 1986. I can think of no other plausible explanation for this rather recent appearance of this entity.

According to an article that Hans underscored in the May 2004 AFIB Report under AFIB Vulnerability, atrial refractory period increases with age. This means that vagal tone decreases with age. Therefore, if your AF episodes are increasing as you age, then vagal tone is not the culprit. Remodeling and possibly increased PACs are the likely culprits.

In summary, the mix of conditions creating substrate susceptibility for adrenergic and vagally mediate LAF is a sliding scale. Just the right mix of refractoriness, automaticity and dispersion for the given "structural individuality". The diurnal differences between adrenergic and vagally mediated LAF support this interpretation. However, for all LAFers insufficient K and Mg is required to potentiate the episode. For example, I could sometimes terminate a VMAF episode triggered in the afternoon (by holding my breath) but could never do this in the PM. The HR was always higher for the former episodes, which I tended to associate with hypoglycemia and dehydration.

So there is my long-winded response to the survey.

PC

With very rare exceptions, each of my afib episodes has been accompanied by or caused by or caused gastronomical distress. Although right now I am in about a 6 or 7 week hiatus from afib, almost all of my episodes had a gastronomical element. Frequently, I would awake in the middle of the night with a burning sensation in my upper chest/lower throat area that I eventually identified as heart burn or something similar. I would be in afib. Typically, I would take an extra rhythmol and a pepcid and the symptoms would abate.

I went through a course of prilosac, or whatever, otc, at my general practitioner doctor's suggestion and it seemed to really help the heart burn/indigestion issues. At the same time, I started taking potassium and increased my intake of elemental magnesium. The combination, or some part of it, has resulted in almost 2 months of no afib, fewer flutters and less heart burn.

Don't know what comes first, the chicken or the egg, heartburn or afib, but there is a connection.

John

John - both potassium and magnesium, in addition to other things, reduce acidity and raise alkalinity. Hence, heartburn will diminish.

Jackie

John,

Heartburn is like stress in the sense that it not only has a direct impact on dysautonomia (increased vagal tone for GERD and increased HR, PACs, etc., with stress) but it also potentiates this substrate effect by causing hypokalemia (alkaline tide). This latter occurs during the secretion of H and Cl into the stomach lumen concomitant with K and HCO3 into the blood (and from there into the urine).

This increase in vagal tone is compounded by the natural increase in night time vagal tone. And, of course, lying down is certainly going to exacerbate the reflux.

PC

PC,

In a post above you mention the following about gastroscopy:

"During the procedure air is manually pumped in to separate the mucosal folds for better viewing. Perhaps this gastric distention is what triggers the AF in gastroscopy."

This has jogged my memory about a GP visit I had. About 2 years ago my GP mentioned on 2 occasions that I was what is termed an "air gulper". That is, I regularly swallow mouthfuls of air. At the time I didn't take any notice of my GP.....thinking he was away with the fairies....

But upon reading your post this now seems to be of much greater significance.

Do you know anything about this "air gulping"?

Dean

Dean,

Sorry, but I've never heard that phrase before. All I know is that distention of the GI tract results in stimulation of the vagus nerve and subsequent peristalsis to move the GI contents along their way. For some reason when this occurs in the upper GI tract there seems to be crossover stimulation of additional vagal motor nerves, i.e., those to the SA node.

PC

Dean - air gulping is commonly found in people who eat fast - hastily - gulping and swallowing both food and drink fast. It results in (obviously) air in the stomach. It's mentioned in any of the books written about digestive problems.

Jackie

Richard,
I just about lived on those foods you mentioned...not now though. Have cut them all out except for onions, alcohol, tomato based and some citrus.

As for the medications I just about lived on antibiotic tetracycline, especially during puberty. Was always coming down with tonsillitis (tonsils not removed) and was taking it for acne as well.....I often wonder about antibiotics.

No wonder I have a LES and GERD problems.

Dean

Dean,

I've always had problems with eating onions, unless they're cooked, and spicy foods were bad, as well. Since doing the Paleo diet, I have no problems, but don't eat sugars, dairy, wheat, etc.... You might find the following interesting, esp. about the air bubble.

To All,

Here's another excerpt found at KOMO TV:

Symptoms from an uncomplicated paraesophageal hernia usually develop in adult life and may consist of a sense of pressure in the lower chest after eating and occasionally palpitations, due to cardiac arrhythmias. All of these are pressure phenomena, caused by the enlargement of the herniated gastric pouch when food displaces the fundic (stomach's) air bubble. Since complications are frequent in paraesophageal hiatal hernias, even in the absence of symptoms, operative repair is indicated in most cases. The usual method is to return the herniated stomach to the abdomen and affix it with sutures to the posterior rectus sheath (anterior gastroplexy). The enlarged hiatus is closed snugly around the gastroesophageal junction with interrupted sutures. It is unnecessary to excise (cut out) the hernia sac. The results of surgical management are generally excellent. Most patients (80 percent) with clinically significant reflux have a sliding hiatal hernia. In these patients, the cardioesophageal junction and the fundus of the stomach are displaced upward into the posterior mediastinum, exposing the lower esophageal sphincter to intrathoracic pressure. At least half of all sliding hiatal hernias are asymptomatic and require no treatment.

For the rest, here's the link:
http://ww3.komotv.com/global/story.asp?s=1230509

Richard

And here's a bit more of a question posed by someone:

From: "Frederick J. Lieberman" <goatfarm@servtech.com>

Subject: Re: Arrhythmias: Magnesium has no effect- alternatives?

Does anyone have any information on the treatment of arrhythmia aside from the use of drug preparations such as beta blockers etc. ie. alternative medicine?

Hiatal hernia and chemical exposure are two major causes of arrhythmias. It is far easier to correct the cause than treat it for the rest of your life.

Fred

http://www.uni-marburg.de/herzzentrum/ismnt/heartdisease/arrhythmiashiatalhernia.html

The above was from the University of Marburg. Does anyone know where this is? I went to the main site, by backing up on the link, but couldn't read the language.

Richard

Dean,

You and me both mate! That list of food Richard provided was virtually ALL I ate for many years. And like you, I was plagued with tonsillitis throughout my childhood and adolescence - it finally backed off when I was in my early 20s. I was scheduled for a tonsillectomy when I was in my late teens but I bottled it. Jeez did I take a lot of tetramycin over the years.

Mike F.

Marburg is just north of Frankfurt Germany. :-)

Ella

For the record, I wanted to put this link, from previous BB posting about HH and arrhythmias, here, for ease of finding.

http://www.yourhealthbase.com/forum/read.php?f=3&i=8949&t=8949

Richard

Richard,

I have just gone for a long walk and to buy my lunch and take it back to work to eat. While I was walking back I could feel gas build up in my stomach, right up high, under the breast bone, and a tightness and pressure around the diaphragm. Had a couple of pac's and forced myself to burp to relieve the pressure.....no more pac's.

I come back to the office and log onto the conference room to find a post by you about HH that describes exactly how I felt 10 mins ago. Unbelievable!

Also Jackie answers my question about "air gulping". The answer perfectly describes my eating and drinking habits. I always eat and drink quickly, just like everything else I do. I am a naturally "quick moving person", always have been. Will have to think about lifestyle changes on this one.

I am keen to hear how Marshal (MJM) goes with his hernia operation. This operation and his reports should prove very interesting for us GERD/LAF sufferers.

Dean

Dean,

That's strange that you experienced that, and then came back to read this post. Synchronicity.

I too am anxiously waiting to see how Marshall does. I'm very excited for him. What a difference this could all make in our search and recovery. There has to be something to this. I go on Medhelp.com or org, and people are always asking the docs if GERD could be related to AF, and the answer is always NO. How can they say definitely not, when there are studies out there proving otherwise. Blows me away.

Take care of those gas bubbles,
Richard

These discussions are very interesting, I do have some conclusions on why the LAF is triggered. I believe the intake of alcohol is definitely a trigger, but possibly a combination of alcohol and magnesium consumption is a dual cause.
Do you have any data or history in this area?

I have had three episodes in less than a week during which time I was just starting magnesium supplement therapy. Have since discontinued.

Bob

Bob we have a Lady (Fran) who cannot take any supplements, it might be the fillers in them or the gelatine caps as they contain MSG.

For me I got great improvement in taking 6 magnesium glycinate tablets in divided doses a day, but then we are all different, I never drink alcohol so can't comment on that.

Ella

To all with GERD and LAF,

Most invoke vagal irritation to explain the association.

I would like to suggest a possible alternative explanation to the association.

For the past 7-10 days I've been drinking my daily 1.5 liters of Waller water without neutralizing it first. I've limited my WW intake to no closer than 30 min before a meal or two hours after a meal. This has definitely resulted in less bloating.

In addition to what I posted on the regular BB I also noticed that my bowel tolerance threshold for Mg has definitely increased. I can drink more without ill effect.

Why is that?

Perhaps it is due to the increased pH. Mg++ is absorbed in the distal small bowel most effectively at a pH of 6-7. The

more alkaline WW may result in enhanced Mg++ absorption and less is then available to cause loose stools.

Many who suffer from GERD have excess acid production. Perhaps this retards Mg++ absorption. Furthermore, perhaps it also impacts K+ absorption. Everyone knows that as we grow older our ability to absorb needed nutrients becomes compromised.

Perhaps this is one of the mechanisms for many of the highly touted benefits of an alkaline diet. Perhaps in those with GERD AF is potentiated via electrolyte imbalance.

PC