**THE AFIB REPORT**
**Your Premier Information Resource for Lone Atrial Fibrillation**
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# VIRTUAL LAF CONFERENCE

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## SUBJECT: GABA & GLUTAMATE – Part 2

PC,

I wonder if your atypical AF episode at the movie could have been a touch of AMAF. I don't know what you were seeing, but several times movies have set me off. A tense or scary scene in a movie can cause a big release of adrenaline and off I go. I have to be a little careful what I see or in a especially tense scene to look away & remind myself that it is only a movie.

Just another thought.

***Rick S.***

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Fran and Rick,

Thanks for the observations and suggestions. I definitely think that some episodes in VMAF are triggered by catecholamines. It has been several weeks since I've had a typical VMAF episode, i.e., vagal maneuver triggered. This I think is because of the "megadose" supplementation with B6/P5P. As previously stated my HR and HRV underscore the vagolytic effect of P5P. Accordingly, I think that this most recent episode may have been an "adrenergic" episode.

I may have dropped the ball a bit. I've been really pushing P5P and Mg and I think they are definitely key. However, although I've mentioned proper hydration on several occasions, I don't think I've been implementing this very well. I've always been very bad at drinking just water. For a former marathon veteran, this is ill advised in particular. Much can be gained by looking at the dietary habits of those that have become ex-LAFers. Hans posted this information gleaned from one of his surveys in the AFIB Report two or three months ago. These individuals differed from the rest of us in drinking much more water. They averaged 3.5 l/d.

I think that my last two episodes, which occurred at almost exactly the same time of day and while doing absolutely nothing, except sitting in the movies or sitting at my computer, were triggered by catecholamines. They were triggered because I was dehydrated and several hours from the last meal (?early hypoglycemia).

We all know what catecholamines do to K and Mg (cause them to move into cells). They also

increase automaticity, i.e., stimulate more PACs. Dehydration also stimulates the RAAS (renin angiotensin aldosterone system). This also causes increased urinary excretion of Mg and K. Renal tubular epithelium (cells in the kidney that secrete or absorb cations), which are loaded with Mg and K (courtesy of the catecholamines), pour this into the urine in exchange for Na (courtesy of RAAS). Hypoglycemia (very commonly encountered in LAFers) further enhances this by stimulating more catecholamine release.

I think dehydration is more important in the genesis of some episodes for several reasons.

Firstly, nocturnal leg cramps wax and wane for me, but they never really go away. They seem to always be present the night before an episode. Shortfalls in P5P, Mg and K are well known causes of nocturnal cramps. This was/is despite much more than adequate intake of these to alleviate any problem. However, I may have been overlooking the obvious. The experts all recommend good hydration to rectify cramping.

Secondly, dehydration/hypoglycemia episodes for me almost always occur on the weekends, especially Sunday. These are the very days that I am more likely to be out in the sun during its zenith, e.g., golf with buddies, jacuzzi with the kids.

Thirdly, GABA is secreted by the beta cells of the pancreatic islets (along with insulin). Although GABA helps wrt GERD, it also enhances the action of insulin. See my previous post on this.

Fourthly, at 2 PM on the day of this most recent episode I'd upped the dose of GABA for the first time.

Fifthly, my poor sleep pattern has also persisted. I know this is related to Mg and P5P. They are both critical in the production of both serotonin and melatonin. Dehydration is also associated with insomnia.

Sixthly and most importantly, always listen to Fran. Her feel for things related to LAF is uncanny. It is all about balance. Too little GABA and GERD can be a problem. Too much and hypoglycemia becomes a problem. And there must be many other tradeoffs that lurk just beneath the surface.

Unlike a vagal episode which is precipitated by a specific movement at a specific time, an adrenergic episode develops slowly. K and Mg deteriorate slowly with worsening PAC status.

So I think I'm going to:

- increase water intake to at least two liters of WW/d
- maintain other sources of Mg to maintain at least 700 mg/d
- add Zn - 90 mg/d as well
- return to my previous dose of 25 mg spironolactone/d (K sparing antialdosterone)
- maintain at least 300 mg of P5P a day and take Fran's advice and at least temporarily put GABA on the back burner.

Will report back on the next ugly head rearing.

***PC***

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Hi Fran,

I apologize for the link. Go to this site and then scroll down to www.orion1..... and click on that. At the bottom you can click on biotechnology.

http://www.ionsource.com/links/AAlinks.htm

Thank you for your heads up on cysteine. I won't be taking any of these, until I have an amino acid panel, so I know where I am. Boy, you are definitely avoiding glutamate, like a plague. I don't think I can throw anything at you, concerning glutamate, that you don't already know, but I'll continue trying. HA! You are the glutamate queen.

Hi PC,

Thank you for getting back to me. No need to apologize, if it takes you a while to reply. We all have lives to live. I'm sorry to hear of your episode with AF. I just wish we could figure this out. Anyway, it seems to me that there has to be something that carries the free glutamate out of our systems. Maybe that would be to keep adding more B-6 and p5p until all of it is metabolized. I just don't know if adding GABA is the solution, as that is much like putting a bandaid on the problem. We need to metabolize the glutamate and open its path to GABA, rather than add GABA. I understand what you are trying to achieve and that you are experimenting, however, and I appreciate your efforts. I do feel you are very close to figuring this out. Maybe adding a lot more enzymes to our diets, as was shown in Erling's post, by Dr. Rosedale, would help. It seems that the actions of breaking down glutamate are specifically done with different enzymes, but I don't know if they're available for purchase, or how we achieve getting them in our diets. Besides, p5p being a cofactor for certain enzymatic functions and Mg. being needed for others, maybe there is something else we're missing, that we can add. You stated that p5p was responsible for 112 enzymes and Mg for 300, so you only have about 3400 to go. HA! I posted on the BB about cholrides and the electrolyte structure of sea water. Read that and let me know what you think. Have you ever had your serum PH tested? Thank you, PC.

***Richard v57***

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PC
Between your last post and the post of Rick I have been thinking. Never quite sure how I get to my reasoning but bottom line... Could it be that the problem for vagals is too much free glutamate and the problem for adrenergics is too much GABA. Or would this be too simplistic? It would make sense if it was as they are both different sides of the same coin. And I have always thought adrenergic and vagal were the same but opposite.

Richard
Thanks for the link. Yes I do avoid free glutamate like the plague and I am sure this is my success. But there is so much more to learn.

***Fran***

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Fran, good thought. Maybe the adrenergic converts glutamate to too much GABA, as you said, and maybe the vagal converts glutamate, to too much AABA. Hmmmm.

***Richard***

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Fran,

It's a reasonable thought. It would certainly fit with the higher incidence of hypoglycemia seen in AMAFers (see previous posts). The role of GABA in the brain and peripheral nervous system is much less clear than for glutamate. However, glutamate is the main excitatory NT for the ANS (PNS and SNS). Although the vagus efferents to the SA node involve glutamate, the signal delivered is inhibitory. To my knowledge glutamate driven signals to the SNS end organs are not inhibitory. If AMAFers had less glutamate and more GABA v. VMAFers, then they should exhibit less adrenergic activity than VMAFers. About all I know for sure about GABA is that more is good for curing GERD but bad for hypoglycemia (see previous posts).

As usual, the closer you look, the more complicated it becomes.

***PC***

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Richard,

Although I've not tested my plasma pH (this used to require arterial blood and was usually run with pO2), I have run my serum CO2. This is equivalent to HCO3 and is an indirect measure of how much buffer the body has to use to maintain the rigidly required range of 7.35-7.45 blood pH. I watched my CO2 slowly increase while I was taking unneutralized WW. Needless to say, I neutralize my WW these days.

***PC***

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PC,

In your post earlier today, 4/6 at 13:37 you mentioned: "the vagolytic effect of P5P". Would this mean P5P should probably not be supplemented by AMAFers? Or at least at the levels you are trying?

Don't AMAFers want to decrease the SNS tone and boast PNS?

***Rick S.***

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Rick,

That's the great thing about P5P. It only goes where it's needed. If your glutamate decarboxylase has low affinity for it, the larger dose will overcome the problem and a VMAFer might become an ex VMAFer. If there is no such problem, as might be the case for an AMAFer, then there is no vagolytic effect. But there are other areas where P5P might help an AMAFer. See my post on B6 and stress.

***PC***

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Thanks PC. It certainly seems to support the case for what happened to me. It seems I have managed to up GABA through avoidance of free glutamate which swung me to hypoglycemia. Obviously the amount of GABA I produce is not enough to produce an adrenergic AF run, but who knows what would happen if I took a supplement of GABA. Maybe an adrenergic run. And maybe for people with too high free glutamate sensitivity this is why GABA does not work. Also

thinking back to Erling’s post about the man who experimented with GABA - he got fast heart rate etc. Just musing again

***Fran***

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Fran,

Your comment about Erling's post correlates well with my thinking on GABA and HR, as previously posted. Unfortunately it also seems to cause hypoglycemia. It would be interesting to know your GLP-1 level after a light carb breakfast. That should tell you whether your PRH is due to correction of Mg deficiency or increased GABA.

***PC***

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PC

How do I get a GLP-1 test done? IS that what I would ask for? Would a standard Dr do it for me, or would I need to send a blood sample away to a lab and pay for it (in NHS land). I think I asked this before but got side lined. I take it GLP-1 would be low if it was due to corrected Mg deficiency and high if it was due to increased GABA.

Thanks
***Fran***

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Fran,

Increased GLP-1 or increased GABA would both cause hypoglycemia.

Magnesium deficiency causes gastric dysmotility, just as it manifests as esophageal and bowel smooth muscle problems (difficulty swallowing and constipation respectively). With the gastric distension and slower gastric emptying secondary to Mg deficiency GLP-1 levels should be lower than normal (see article on PRH).

So if your "cured" VMAF was mostly secondary to Mg repletion relatively speaking (v. B6 or P5P) than the PRH of long term NSR should be associated with elevated GLP-1 (more Mg means more effective gastric emptying means higher GLP-1)

If your "cured" VMAF involved improved function of glutamate decarboxylase mostly through repletion of B6 (or P5P) relatively speaking (v. Mg) then the PRH of long term NSR is probably due to elevated GABA (more GABA from beta cells in pancreas means less glucagon and more insulin).

GLP-1 levels are available in the US, but not generally so. I've found one lab that tests for this. But as we discussed previously, the result means little unless it follows the appropriate kind of carbohydrate meal (see PRH article).

***PC***

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Hello PC,

I'm still afibbing, but have eliminated all meds, so we'll see what happens. Tomorrow I will eliminate all supps. for a day as well. Now to my question.

I hope my question doesn't seem stupid, and maybe I'm not thinking straight, as I'm just pretty darn tired of thinking, but why are we trying to eliminate an excitory amino acid, such as glutamate? It would seem that we are parasympathetic dominant and what I'm understanding is that we want to get the sympathetic to kick back in, or excite, so to speak. I went back in the previous posts in the CR, and it seems that VAFer's body's are too relaxed. By that I mean, that we don't digest our meals, we need to urinate more often which is maybe the sphincter of the bladder being too relaxed, our esophogeal sphincter's stay open which causes GERD, so it just seems like we need to excite our bodies. I'm not saying free glutamate is good to eat, I'm just a bit confused and hopefully you can shed some light for me. I'm not a type A personality and do sleep very well, sometimes too much. It just seems that maybe excitement is what my body needs. (No, I'm not talking about your favorite AF trigger) I know what your thinking. Anyway, I would appreciate you clarifying this for me, when you get a free moment. Thank you.

***Richard v57***

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Richard,

This has been a confusing issue for me all along. I have considered myself to be adrenergic. Does that mean my sympathetic side is "over active" or is the parasympathetic side "under active". Which side is the problem?

***--Kent***

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Richard,

Let me add an important piece of information that might make the excitatory/inhibitory view a little more clear.

As Fran has repeatedly stated the neurotransmitters glutamate (and aspartate) are excitatory, while GABA is inhibitory. This means that the former depolarizes the nerve (impulse is more easily triggered) by facilitating intracellular movement of Na+ into the "receiving" nerve. GABA, on the other hand, hyperpolarizes (impulse is more difficult to trigger) by facilitating intracellular movement of Cl-.

However, the nerves that they stimulate do not necessarily excite or inhibit their target organ or nerve respectively. In fact the vagal efferent (motor) nerve to the SA node stimulated by glutamate is in fact inhibitory. I've also found out that GABA is the neurotransmitter in one of the three reflex baroreceptor pathways involved in controlling HR and BP. It inhibits a nerve in the RVLM (rostral ventrolateral medulla) that inhibits another nerve (via glutamate) in the sympathetic ganglia that also controls HR. This latter is why I think GABA supplements decreased the number of dropped beats I was experiencing while on large doses of P5P (it increased AV node conduction velocity making it less likely that the P5P induced SA node increase in HR would encounter a refractory state in the AV node).
So you can see how complicated this can become.

Hope there's no brain cramp on this.

***PC***

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Thank you PC for taking the time to explain that. It's still a difficult process to understand and pathways for breaking down amino acids are truly an amazing process. Thank goodness, we have you here to understand and translate this difficult process. I hope all is well for you. I don't remember if you posted that you went back into NSR after your bout on Sat. Did you?

***Richard***

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Richard,

Thanks for asking. I feel I'm gaining more and more control over these episodes. You may laugh, but I'm actually looking forward to the next one so that I can continue to learn. How demented.

Happy to read that you converted.

***PC***

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Good morning PC,

In light of feeling like a guinea pig, to the point I'm "weeting" everytime my wife opens the fridge, I decided to start taking cal/mg 2-1, Tues. morn, and twice again, early eve and late eve. Along with this, and yours, my wife's and Fran's suggestion, I stopped meds. beginning Mon. eve. I woke up this morn. in normal sinus rhythm. I'm a happy little guinea pig, at least while it lasts. I think the extra B6 played a crucial role in this, as well. What possessed me to take the calcium, was when I awoke yesterday morn, and upon stretching, got muscle cramps for the first time. My wife has been taking Mg., as well, but supplements a little with Ca., but last night she got a toothache, and she has never had one before, and it's pretty painful to her. Now is this power of suggestion, from the article at ithyroid.com, just a coincidence, or a sign that we need more calcium. I do feel we're being guided in mysterious ways that I can't explain. In my case, I believe I needed more Ca., because of converting to normal SR. As for now, I will continue with the 2-1 ratio of Ca./Mg. and continue monitoring my success, along with the other regimen of diet change, and supplements, however today, I'm eliminating everything, except C, B's, and Ca/Mg. What do you think?

***Richard***

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Richard,

Just don't take your Vit C with your B vits, esp. B6. See Dr. Mansmann website recs at

https://secure.salu.net/cgi-perl/get.cgi?pub=50460&ext=doc

Also be sure to read his other wonderful articles available through
http://magnesiumresearchlab.salu.net/

You might consider adding Zn, since research reveals this to have more affinity for P5P than Mg at least in the brain.

***PC***

PS Congrats on getting off the meds. Ca is OK as a supp just so long as you don't take in too much thru diet, i.e., dairy products. Also be sure to drink plenty of water.

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Richard

Just because we are parasympathetic dominant and our heart rates can be slow, I have never felt that I am really laid back. In fact quite the opposite. My brain is always on the go, I have abundant energy and am always looking for things to stimulate me. If there is no stimulating stimuli around I can get quite bored and have to do something energetic to get rid of it. But the excitation caused by internal excitotoxins do not have this same effect. They change the way the body reacts. These will in some cases literally stimulate nerve cells to fire themselves to death. Here is a link that Erling posted a while back on the effects of excitotoxins.

http://www.academyofwellness.com/excitotoxins_cellular_energy.htm

This is part of an online book so read the rest. This is chapter 4.

Also, and nice and simple is this site here from which I have cut and pasted. Note that MSG also stimulates the pancreas to over produce insulin.

**What exactly is MSG**? from http://www.msgtruth.org/whatisit.htm

MSG, or Monosodium Glutamate is a salt of the amino acid - Glutamic Acid (glutamate). A salt is the chemical name for a molecule held together by opposite charges. Basically one (mono) sodium atom is "stuck" to the amino acid glutamate.

**What is an amino acid**?

Amino acids are often called the building blocks of life because it takes many of them linked together in a chain to create a protein. DNA tells the body how to make the chain and in what order the amino acids must line up. Some amino acids must be eaten because the body cannot make them (essential), some the body can make (non-essential), and yet others are able to be made during some times, but not others (conditionally essential). The life processes are all dependent on proteins which play critical roles in the body as structure, messengers, enzymes, and hormones.

Proteins are globular and clumpy because the amino acid chains fold in on themselves. This is how the immune system recognizes proteins. They are large compared to single amino acids, and they are uniquely shaped. The immune system does not recognize tiny MSG as an allergen. However, trouble can begin because the body can attack the larger enzymes like GAD, responsible for turning excess MSG into GABA.

**What is an enzyme**?

Enzymes are simply proteins with interesting day jobs. Enzymes help make things happen by helping to create other proteins and by helping break them down too. Enzymes are not straight

chains, they are globular and clumpy, because they are folded into intricate shapes like other proteins. It is these shapes that help them create and break down other proteins and compounds.

**What is a hormone**?

Hormones are extremely potent protein based messengers that travel around the body connecting the lines of communication between glands of the endocrine system. These glands direct important functions like metabolism, growth, and sexual development. It has been found lately that smaller amounts of hormones are more effective than larger amounts because the body has feedback mechanisms that don't take kindly to overdoses of hormone. Things shut down because it is considered a trouble signal if there is too much hormone present. By affecting the part of the brain - the hypothalamus, that controls the master gland of the body - the pituitary, MSG may affect hormone production in the body.

**Glutamate - Protein Building Block and Excitatory Neurotransmitter....**

Glutamate is just one of many amino acids used by the body and linked into the chains of protein in the body. However some amino acids are free to float around by themselves as well as being found linked into proteins because they serve vital functions - some are neurotransmitters which carry nerve cell impulses throughout the body. Amino acid neurotransmitters are like chemical messengers carrying news from nerve cell to nerve cell. Some amino acid neurotransmitters like glutamate trigger nerve cells to fire, others like taurine and gamma amino butyric acid tell those firing nerve cells to cease firing. It is a delicate balance. An important balance. Researchers are finding out just what happens when that balance tips. In patients who suffer a stroke, for example, an excess of glutamate in the brain causes the nerve cells to die from over-stimulation. Glutamate blocking drugs are being used to prevent some of this damage.

For more info on neurotransmitters see:

http://artsandscience.concordia.ca/psychology/psyc358/Lectures/transmit2.htm

For information on glutamate and excitotoxicity:

http://www.memantine.com/inhalte/s2.html

How does the body usually deal with excess amino acids?

Most amino acids if not used right away, are not stored as amino acids. The body has elaborate means of changing extra amino acids into other amino acids, and removing nitrogen and changing amino acids into fuel to be stored. There are processes such as "transamination" and "deamination" which occur mostly in the liver. In patients with compromised livers, however, they may have trouble transaminating cysteine, for example, into taurine, the amino acid that acts counter to glutamate. Also, an excess of the amino acid aspartate (found in Nutrasweet) may result in excess glutamate, since the body can convert aspartate directly to glutamate. Aspartate and glutamate affect some of the same receptors. In a different example, there is an enzyme that the body uses to convert excess glutamate into another neurotransmitter called GABA. In many patients with Type II Diabetes, their bodies view the enzyme responsible for turning MSG into GABA as an enemy and create antibodies to attack it so that it cannot do its job. This is a problem. The body is compromised in its job of getting rid of excess glutamate. It again is a question of balance, and what tips it.

**Is manufactured MSG a problem?**

According to some MSG opponents the glutamate added to foods is "bad" and the natural glutamate in our bodies is "good". MSG sellers argue that MSG is exactly like the glutamate in the

human body, therefore it must always be "good". It is not so simple. There are contaminants in processed MSG. An analogy that can be used is that there are right-handed amino acids and left handed ones. They are like mirror images of each other. Processed MSG contains not only the kind of amino acids the body is used to handling, but mirror image ones too. This may cause problems because it is like putting the wrong glove on your hand. It's not quite the same. We don't exactly know what problems this may cause. On the other hand (so to speak) the fact that glutamate the body is used to handling is also in MSG may present a problem because an excess of naturally occurring glutamate is well known by neuroscientists to be a problem in many disease states. Natural glutamate can cause problems we already know about. The reason food processors "free" glutamate from its bound form, is that it acts as a neurotransmitter in its free form. The food industry's claim that free glutamate is as harmless as bound glutamate is disingenous at best. If it was exactly the same, they wouldn't need to hydrolyse vegetable protein (split the amino acids apart).

**Why do food companies add MSG to foods?**

There are several reasons:

MSG acts as a drug like caffeine. It affects the body by stimulating the nerve cells in your tongue. It is not a "meat tenderizer". It is not a "preservative". It barely has a taste of its own. The food industry is trying to confuse the issue by focusing on the "fifth" taste sense they call umami. However, the truth is, they are using the very same neurotransmitter that your brain uses. It directly affects processes in your body. It changes your perception of taste by altering you.

MSG stimulates the pancreas to produce insulin. The food industry has found their own "anti-appetite suppressant". It's a convenient way to keep consumers coming back for more. The blood sugar drops because of the insulin flood. And you are hungry an hour later. Sound familiar?

The body changes excess glutamate to GABA. GABA may be addictive. It is calming and affects the same receptors in the brain as valium.

***Fran***

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Thank you Fran, for sharing all that info. I think I have much more reading in my future. I definitely do not think free glutamate is good for us, esp. after reading all you and PC have shared. The difference I see in myself, is that I'm not a highly energetic person, and never have been. I have found it much more difficult to get motivated, so I was just feeling that maybe my body was just too relaxed. I have been an exerciser in the past, but not to any extreme. I've actually taken most things in moderation and do like to relax, and don't get bored in doing so, therefore I was just confused, more on the issue of excitory vs. inhibitory, but that has been clarified on the issue of glutamate. I might add that phenyalanine keeps haunting me, in that maybe that is what I'm lacking, but I'm still reading on the amino acids and the roles they play. I'll know more in the days to come, as my tests come to fruition and I will definitely share what I learn. I won't be taking any extra aminos, either, until I learn more about my body's deficiencies.
Thank you again,

***Richard***

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Richard

Not quite what I was looking for but something to be getting on with. Very much in the same vein as glutamate. There is no doubt you will be getting enough Phenylalanine in your diet, however

the problem arises with Phenylalanine hydroxylase (PAH) the enzyme which metabolises it.

Found this on the way about nitrogen and glutamine, pheylalaine etc, and need to read it yet. Could only call it up on cached version though.

http://216.239.51.100/search?q=cache:Qs6ZDBACLAUC:www.indstate.edu/thcme/mwking/nitrogen-metabolism.html+phenylalanine+and+late+onset++errors+of+metabolism&hl=en&ie=UTF-8

Phenylketonuria is a disorder where by the liver does not produce the enzyme Phenylalanine hydroxylase. This disorder is usually picked up with the heel prick test after birth and is very severe. However, research I read and have still not located suggests that there are other lesser severe forms of PKU (not truly PKU) and are described as late onset. There are other things which can cause Phenylalanine not to be metabolised and this was linked from one such site.

Hepatic encephalopathy is caused by disorders affecting the liver. These include disorders that reduce liver function (such as cirrhosis or hepatitis) and conditions where blood circulation bypasses the liver. The exact cause of the disorder is unknown.

The liver cannot properly metabolize and detoxify substances in the body. Accumulation of toxic substances causes metabolic abnormalities that lead to damage in the central nervous system (brain and spinal cord). One substance believed to be toxic is ammonia, which is produced by the body when proteins are digested, but normally is detoxified by the liver. Many other substances also accumulate in the body and damage the nervous system.

In people with otherwise stable liver disorders, hepatic encephalopathy may be triggered by episodes of gastrointestinal bleeding, excessive dietary protein, electrolyte abnormalities (especially decrease in potassium, which may result from vomiting or treatments such as diuretics or paracentesis), infections, renal disease, and procedures that shunt blood past the liver.

The disorder may also be triggered by any condition that results in alkalosis (alkaline blood pH), low oxygen levels in the body, use of medications that suppress the central nervous system (such as barbiturates or Benzodiazepine tranquilizers), surgery, or possibly a coincidental illness.

Disorders that mimic or mask symptoms of hepatic encephalopathy include alcohol intoxication, sedative overdose, complicated alcohol withdrawal, Wernicke-Korsakoff syndrome, subdural hematoma, meningitis, metabolic abnormalities such as low blood glucose.

Hepatic encephalopathy occurs in approximately 4 out of 100,000 people. It may occur as an acute, potentially reversible disorder or as a chronic, progressive disorder.

***Fran***

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Thank you Fran,

Are you sure your not type A. Just kidding. You just seem to be such a busy go-getter. Anyway, I will be sending in my amino acid panel on Mon. so we'll see if I'm lacking or overabundant in anything. I'll read your links and see what I find. Another day of strong solid heartbeats, so I feel pretty darn good. No more cooked tomatoes for me!!

Appreciate your search,
***Richard***

PC
Thanks for taking the time to relay the intricacies of GLP-1.

From what you have said, and my following the paleo diet, I don't now seem to fit in with any scenario you stated above. So it would seem reasonable to assume that I have fixed it. On thinking more about my family health problems it would seem to me that free glutamate, rather than vit B6 is the root of the whole problem. A high free glutamate diet will also result in an unbalance of minerals, enzymes and vitamins (think of what it does to calcium channels). Even if you are eating or supplementing vitamins minerals etc in abundance it won't fix the problem because of the way that free glutamate acts on the cells of the body. I mean by this that a diet high in free glutamate is not natural. The body was not meant to deal with the high levels in modern diet. For this reason supplementation, in my mind, is a poor second to good nutrition. It seems like the easy option, but is full of unseen problems.

My interest now is more in understanding the reasons why. In a silly way I am still looking for a magic pill, but in my heart of hearts I know that it is continued elimination that is required.

From what you wrote I personally feel my PRH was probably due to elevated GABA and hence the drop in anxiety, the wild ectopics, but good sleep I now got - but not enough to set in an adrenergic episode.

Since discovering the Paleo diet I do not suffer PRH anymore. I do if I eat grains and potatoes (never did get on with beans). I have by no means a perfect sinus rate. It is quite slow but at 50 - 60 at rest not worrying. I assume I still have high vagal tone. I often have early beats and the odd missed one. But I never get the trigiminy, the runs of ectopics, the long pauses, the slurring and turning etc. It is by the ectopics I get from time to time that I monitor my diet - what is good and what is bad for me. I never get them when active, but often at rest - the digestion period.

I would like to say I am cured. As opposed to you thinking I am "cured". The reason being that I know I will not get another episode unless I go back to my former dietary habits and eat free glutamate. Free glutamate acts like a drug on the body (there are references for this), so its elimination will stop its effect. In the same way you know you will not get drunk unless you drink some alcohol.

And thinking on - It may be that high free glutamate with good levels of B6 makes for adrenergic AF - and high free glutamate with low B6 and Mg makes for vagal AF (depletion through exercise). Those with a mixed form may be swinging from one extreme to the next. And those with high vagal tone who don't get AF may not be so unbalanced by free glutamate.

***Fran***

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Fran,

Elimination of dietary MSG, at least in SoCal, has to be nearly impossible, without becoming some kind of recluse. Like many others I enoy dining out, especially with my wife and kids. If nearly complete avoidance is required before LAF can be banished, I must confess that LAF would be the lesser of the two evils (fortunately I'm blessed with a relatively slow ventricular response rate). However, I sincerely hope and believe that LAF can be "cured" through a combination of avoidance (to the extent possible without withdrawing from society) and judicious supplementation based on the biochemistry and physiology of glutamate/GABA. Only time will tell if this is possible. There are a ton of people walking around out there with diets much higher in

free glutamate that exhibit no evidence of LAF. That's not to say they aren't killing neurons left and right, but they are different from us VMAFers in being able to handle free glutamate. Why is this? I think there is an answer and it's knowledge will allow a much less disciplined approach to "curing"/curing LAF. I want to be able to eat chocolate (in moderation) and pizza and dine out and in general be able to free myself of this preoccupation with LAF. The solution to this riddle exists and you are proof of it.

Regarding adrenergics and glutamate, I'm more inclined to think they are more sensitive to decreased GABA and we vagals are more sensitive to increased glutamate (see post on 4/4 10:22AM for pertinent article). But this is just supposition.

***PC***

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Hi PC

I can understand the predicament. I forget that my life style is so different from most other people. It is not that I am a recluse (far from it), I just live in a very rural situation. I am not surrounded by restaurants or take-aways. My work takes me deeper into the wilds so there is no chance of temptation setting in and I take my own food. I do big huge two weekly shops in the 'big city' (where temptation is always a problem). In many respects I am so lucky as I live in a fishing/crofting community so prime meat and fish is always available and I can grow a lot of my own veggies. A bit like back to basics with a computer etc. As I have a lot of spare time in the evenings I tend to experiment in the kitchen and find that I can create better versions than restaurant food, but that does not have the same implications as a social night out. I really enjoy it.

I am also very much of the opinion that shopping and eating out should be consumer led - and am noticing in more and more that companies and restaurants are advertising no additives, MSG etc - pure and natural sources (they just haven't understood the whole scenario yet). I want them to keep this up. Eating out should be for all. Think of the other health problems that actually prohibit certain foods and customers out. I would love to open a good food restaurant myself. Only problem is there are not enough people to justify one here.

As you say the problem is eating out, and I do feel I am missing out here. My husband stopped drinking so the pub is not a threat now, in fact I would run a mile if he went. If my colleagues and I go after work then I always just have water and a slice of lemon - boring.....

If you find out what it is that remedies this and I can take it I would love to have the odd temptation. But the fact remains that I got so frightened by my near death experiences that I am too scared. To date no-one can tell me why and all I know is they stopped along with the free glutamate and the AF. I always thought that this would be how I went, and I am not ready yet!

Having said that, I do cheat. Today - a hospital visiting trip to my sister with a sideline shopping trip - resulted in a packet of 2 chocolate eclairs 'mysteriously' ending up in my trolley. In the car were three of us. It was to be my treat to them, but before I knew it I had eaten half of one. Boy it was soooooo good. But I'm skipping now, and know that will be me until next time..... But still free glutamate is not the main problem here..

Richard

I would be sceptical of phenyalanine. I can't remember exactly why but I will go digging again. Many people cannot process it and on many aspartame products it says a source of phenyalanine so those who are sensitive know. It is something to do with vit B again and inborn

errors of metabolism. Hopefully I am wrong but I have a list of no-nos that I have formulated over the years and this is one of them.

***Fran***

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PC –

I'm very late in participating on this GABA topic but I have been reading diligently and with great interest. I wanted you to know your presentation resonated with me. Between you and Fran, you have explained the cascade of events that could very well be contributory to afib.

At the very least, you have opened the door to discussions of how the polymorphisms are going to affect our future when it comes to diagnosing risks for disease and quite possibly the key to understanding if polymorphisms are influencing factors that lead to afib. This is an extremely important and monumental moment in finding answers to the cause of conditions and disease.

We are on the cutting edge of this through genomics and that statement was made very clear to me last night when I attended a mini-seminar on the role of Functional Medicine Today by my MD. In one facet of explaining how she makes a functional diagnosis, she touched briefly on the exciting testing done with the variations in genetic makup called Single Nucleotide Polymorphisms commonly called SNPs, pronounced "snips."

She explained, while they don't cause disease, SNPs are associated with almost every human disease. But, the expression of one's genes into an actual disease isn't inevitable, as many people think. The genetic variations which make a person particularly susceptible to a specific type of disease most often do so when exposed to certain, often MODIFIABLE factors, such as environment, diet and lifestyles.

Detecting the SNPs make it possible to play around with potential risk and allows the patient to take an alternate route to health and avoid the risks.

Genetic testing shows what specific genetic factors might pose a potential problem for you. Testing for your family, as well, then allows everyone to change course toward a healthier future. It isn't the fate of your genes or even SNPs to cause disease. But they need all the help they can get and that's by being identified and then by having their "environment" modified.

25% of our genes are fixed and 75 % are modifiable and this can be determined by blood testing. Genomics is the new buzzword and is going to crack the medical model of open how we treat disease – by modifying the gene expression before turns to a full blown disease state and not treating the condition too late with drugs that don't work.

In giving examples, she said in people with the gene expression that does not allow them to metabolize and benefit from Vitamin B6, then P5P is the safest form to take and the only form that allows complete functioning. There is little toxicity with P5P.

Lastly, she said the easiest way to determine if you are B6 deficient, is to assess your sleep. Do you dream? If so, then you are metabolizing B6. If not; take P5P.

So thanks again, PC, Fran, et.al. for the huge amount of research and effort you have all contributed to this particular topic. The doors are opening!

***Jackie***

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Jackie,

Well put, as usual. I was beginning to think people (except Fran and a very few others) were intimidated or really put off by the term polymorphism. I sincerely hope that others begin to experience some of the improvements I have with B6. And not just in the realm of LAF.

I can't tell you how excited I was after starting WW. Its benefit was twofold. My PACs disappeared and my episodes went from q 36 hours to 3 weeks right off the bat (thank you Erling). But more importantly it opened the eyes of a conventionally trained nutritionally ignorant MD. My interest and participation in this BB took a giant step forward. Unfortunately the WW and Mg thing didn't last. However, I am even more excited about P5P. It's connection with Erling's beloved Mg and Fran's beloved glutamate is a perfect fit. Only time will tell, but I sure get the feeling that we are successfully "unravelling the ball of yarn", as Hans once put it.

Looking forward to further CR posts from you.

***PC***

PS This thread may set a record for number of posts in the CR. That portends well for the importance of this topic in LAF.

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Jackie

Brilliant post. And now I know I must be metabolising B6. I dream, and dream and dream. Even when I had AF I had dreams, dreams of a type that weren't too nice. We are all so different.

As to polymorphisms, I have had genetic testing done, but not for AF. For narcolepsy. I have the gene, but not the disease. My dad had the disease. Whatever triggered it to develop in him has escaped me thus far. I find genomics absolutely fascinating. Interestingly narcolepsy is now known to be a brain disorder where specific neurons (can't remember their name off hand) die and scar the hypothalamus. The hypothalamus keeps cropping up in AF too.

***Fran***

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PC

Do you know anything about 4'-O-methylpyridoxine, an anti–vitamin B6 neurotoxin? Just came across it in an article about glutamate with no explanation. Did a search on Google and it is found in Gingko seeds and leaves.

***Fran***

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Fran,

I know absolutely nothing about 4'-O-methylpyridoxine. Sorry to be so ignorant.

***PC***

---

Hi Fran,

It appears to only be in the nut, not the leaf.

http://www.guardian.co.uk/Print/0%2C3858%2C4266068%2C00.html

You can read here.

Have a good weekend, all.

***Richard***

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No it's in the leaf too, in much smaller quantities though. Don't know if it can be found in any other foods or seeds though. It seems to be following the principal of some seeds and grains having anti-nutrients in them as a survival mechanism - to stop them being eaten.

I don't know what part the ginkgo tablets are made from but know a few on the board take it - and if B6 is a problem in AF it maybe that gingko is not such a good supplement, unless the benefits outweigh the bad.

***Fran***

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I shouldn't really post about supplements as I know very little about them. But this one struck me if intracellular calcium is a problem. It will be if consuming lots of free glutamate. The name of the supplement was mentioned on a hypoglycemic forum as an antidote to eating sugar and I thought to do a wee search on it.

This was a press release in 1998.

http://www.mext.go.jp/english/news/1998/11/981116.htm

Through the Mikoshiba Calciosignal Net Project, which is part of the program for Exploratory Research for Advanced Technology (ERATO), the Japan Science and Technology Corporation (JST) has discovered and clarified the mechanism by which the inositol 1,4,5-trisphosphate reseptor (IP3R) that is responsible for discharging calcium ions from the calcium store in a cell plays an important role in the development and growth of nerve cells. On the tip of the process of a nerve cell, IP3R covers the organ that stores calcium ions responsible for information transmission. When the organ is stimulated externally, the cover opens to discharge, calcium ions and the process extends. This mechanism was also clarified. This achievement by Kotaro Takei, Kunio Kato, et al was published in journal "Science".

The research used a chromophore-assisted laser inactivation (CALI) method to inactivate local particular molecules, and clarify where the functional molecules of a live cell work actually. When antibodies with coloring matter combined with protein were irradiated with laser light, the coloring matter was activated and the targeted protein was broken. Since the molecules without antibodies and the part not irradiated by laser were not affected, the molecular functions of nerve growth can be elucidated by targeting particular molecules. This achievement should be useful in

clarifying the control mechanism of growth and recovery of nerves and the method of treating abnormal development and damage of nerves.

and for me who doesn't like taking supplements I am relieved that it comes in food form too.

Inositol is a simple carbohydrate that was originally thought to be essential to good health, but has since been demonstrated not be a vitamin. In the body, inositol is metabolized into phosphatidylinositol, which then acts as a second messenger system to stimulate the release of calcium from its intracellular storage site in the endoplasmic reticulum. The sugar has also been implicated in improving the transmission of neural signals in individuals afflicted with diabetic nerve damage and numbness. Major sources of inositol include beans, citrus fruit, nuts, rice, veal, pork, and wheat germ. There are no known deficiency symptoms in humans.

***Fran***

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Several weeks ago I began the Paleo Diet...not to lose weight but to improve my heart (hopefully reduce ectopics). My AFIB always had a vagal emphasis hence eating was a real trigger so I found myself eating less and my weight gradually dropped from 200 lbs. (I'm 6'4") to my current weight of 175 over a several year period. Now I'm trying so very hard to gain weight but this diet, as good as it may be for helping the heart, is damned hard on which to gain weight. Anyone have any suggestions how I can stay on the Paleo Diet but, at the same time, gain weight? Am already eating a ton of veggies, fruit and nuts daily but they just don't pack much of a wallop in terms of calories and one can only eat so much meat/fish. Anyone had the same problem? Thanks!

***Wade***

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The inability to gain weight on a Paleo-style diet is not usually considered to be a problem by those of us who eat that way. In fact, the reduction in weight (fat) and the maintenance of healthy weight levels are primary reasons for many of us to eat that way.

A person at normal weight (no excessive body fat) should be quite pleased by the results. An efficient body ingests and uses the energy it requires, and it keeps insulin levels low, and fat storage at a minimum. Blood pressure is most often normal, and diabetes risk is reduced or eliminated, along with any significant risk of heart disease.

If you've lost your body fat by being on this diet, then you must be aware of what your normal Lean Body Mass is. If you have zero body fat (hence, your weight is what you consider to be minimal for your size), then you have a choice to make, it seems to me: A) stay at this weight by eating your regular diet; B) gain weight by eating more, or by eating those foods that will add body fat. Fat will be gained when we eat either far too much sugar, or far too much protein. You won't gain excessive weight by eating too much fat under MOST conditions. If you find that you're hungry, eat more food. If you're not hungry and you just wish you were heavier, you can increase your calories, or you can exercise much more, adding muscle mass to your existing body mass. You'll gain weight, and the added muscle will require more calories (mostly from protein) to sustain itself.

Good luck.
***Jerry***

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Hi Wade

Tis me. As you know I have a similar problem. I could do with a few extra pounds now but know this is the way my body should be. As a child, teenager and young adult I was always painfully thin. A school nurse thought I was malnourished. The art teacher described me a young colt - all arms and legs (with knobbles on the knees). When on meds I started to put weight on, (round the tummy and thighs - spot the insulin resistance) even though I was hardly eating anything. Like you my AF was so related to eating that I kept putting it off.

Just by stopping meds and free glutamate my weight started dropping back, even though I was eating three or four times as much as in the past. You are probably like me and have very fast metabolism. Do what I do and enjoy your food. Don't worry about it, there are a million people out there who would give their eyeteeth to be able to eat and eat.

***Fran***

---

Hello PC,

Just a quick post, to see how you're coming along on P5P and if your AF has quieted down or changed in any way.

Thank you for your response.
***Richard***

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Richard, thank you for asking. Sorry to be so late in responding, but I've been quite busy at work.

Here's what's been happening. As you may know, I have an episode of AF every 6-8 days now. Since starting the mega doses of P5P my PACs have dropped off the chart, but my dropped beats at night are very frequent. These are not a risk factor for AF. My episodes always occur on the weekend and this most recent one was no different. Even though I've not had a typical "vagally" mediated episode for over three weeks, the other variant types appear to have increased.

This past Sunday evening I had a late dinner, partly because it was convenient and partly because I wanted to see what would happen (again I am most fortunate to have a slow ventricular response rate). I have found that taking P5P in divided doses during the day essentially removes the precipitating PACs and slow HR that usually trigger an episode. Unfortunately peak levels are reached fairly quickly and drop off rapidly thereafter. This means that it is impossible to prevent an episode while sleeping. Monday at 1 AM AF appeared. This was 4 hours after dinner and 3 hours after hitting the sack. I discovered it upon arising at 7 AM. The most interesting aspect of it was that there wasn't a PAC to be seen in the preceding 30 minutes and my HRV wasn't that high. I immediately took 50 mg of P5P with no apparent effect. However, after an hour I took another 50 mg P5P and 200 mg GABA (with 1000 mg inositol) and converted after 20 minutes. Whether this was coincidence or not remains to be seen. I could certainly live with weekly night time episodes that can be terminated upon arising with P5P and GABA, especially since I believe that the tail should not be wagging the dog wrt dining out, occasional dietary indiscretions, etc.

Besides looking closely at the P5P/GABA AF terminating potential, I'm really studying what it is about weekends that triggers AF for me. I have long thought that dehydration/hypoglycemia were key in this regard, but this past weekend both were under excellent control. The GERD/GABA connection did occur to me but the literature states that LESRs (lower esophageal sphincter

relaxations) associated with GERD secondary to "GABA deficiency" (GABA B receptors) does not occur at night. This is not to say that nocturnal acid reflux does not exist, far from it. I just think that there's something else there on the weekends (?outdoors with more direct sun exposure and more physical activity later in the day ?hypothalamus mediated). Alternatively I could be on the cusp and this could even be the beginning of the end (ha-ha). You never know what that might mean coming from a pathologist. This is a work in progress, or at least so I think.

***PC***

---

I just love the way you experiment with your self. A very personal journey of self-discovery for the benefit of all.

Just a very long shot PC

Could it be that you are making too much vitamin D at weekends. According to http://www.acu-cell.com/acn.html "changes in serum calcium provide important information about various hormonal or organic disturbances, including excessive Vitamin D status".

As far as I know there is no connection with GABA or glutamate here, but that does not mean there is not.

I know that some people with certain disorders like sarcadoisis have to avoid the sun as their bodies make too much vit D. They often have heart irregularities.

***Fran***

---

Fran,

Thanks for the input. I also briefly considered Vitamin D. Actually that's why I stopped my Vit D supps sometime back, given my sunbelt location. Although Vit D enhances the absorption of both Ca and Mg, it is more so for the former.

I'm presently looking to find a link between bright light earlier in the day and darkness, e.g., indoor movie, in the afternoon. We already know about physical activity followed by rest in toning up the vagus. It would certainly make sense since the diurnal variation in vagal tone is tied to daylight. The problem I'm having is that these new episodes are accompanied by few if any PACs and my HRV is not that high. I can't help but think that GABA is involved somehow (as a neurotransmitter not as an oral hypoglycemic).

It's hard to separate the effects of outside physical activity from just being outside.

***PC***

---

I'd like to chime in on the weekend-factor.

Something like 80% of my episodes take place on Sunday morning while I'm cooking breakfast. I wonder how many other folks have found an increase in symptoms on weekends.

I've investigated the following possible explanations for this significant statistic:
- I tend to drink a beer or two on weekends.
- I tend to snack more on weekend nights, usually while watching a movie with the family or something.

- I tend to go to sleep later on weekends
- I tend to get up later on weekends
- I'm much more relaxed on weekends

I've tried elimination technique to try to rule any of these out and the only one that seems consistent is the last one. I tend towards low HR - perhaps on Sunday mornings I'm so relaxed that my HR gets low enough to trigger an adrenaline response and thus the afib.

Any thoughts?

***David***

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David

Did you happen to cook on a gas stove? That is when my afib first started it turned out 1 pilot light was always off, and gas was leaking out.

On pesticides I was at the theatre one night watching my granddaughter play the lead in the Music Man, within 15 min I was in afib it turned out they had sprayed the carpet after having had a dog show the day before, so my afib is definitely and overload of toxins.

***Ella***

---

CR aficionados,

While doing more research on the P5P/GAD polymorphism hypothesis, I discovered that B6 is essential for the proper absorption of Mg and for formation of HCl (hydrochloric acid). Deficiencies of magnesium, zinc, and riboflavin (B2) can also affect B6 levels. So there is a definite interdependency between B6 and Mg (not to mention digestion). It is also the most crucial of all the B Vitamins in maintaining a healthy immune system. High protein diets increase the need for B6. Vitamin B1, B2, B5, C, magnesium, potassium, sodium all have a positive effect on B6 physiology. Vitamin B6 deficiency has been associated with food processing, alcohol, estrogen, alkali, and ULTRAVIOLET LIGHT.

This latter may in part explain the frequency of VMAF, at least for me, on weekends after outdoor physical activity. Once I retire indoors to a darker place and sit or lie down, the stage is set, especially if coincident with postprandial state. All this glutamate (from diet, from peristaltic vagal signals, from baroreflex signals and from changes in ambient light) overwhelm the available P5P (already compromised by the UV light). Fran, the Vit D suggestion was good. It was just the wrong vitamin. And by the way, smoking depletes or prevents the uptake of B6, as well as the other B vitamins. So your healthy diet is only partly responsible for your ex AF status.

For those of you yet to be convinced of the many benefits of P5P (v. B6) or for those concerned about toxicity, the following website might prove informative. It also underscores the B6/Mg connection and states:

“Also notable are the clinical findings of increased cellular magnesium when vitamin B6 is administered. Individuals with poor magnesium retention may need B6.”

http://www.nutrition4health.org/NOHAnews/NNSp99B6.html

Hopefully the technical explanation is more understandable than what I appear able to convey. He concludes by saying “to my knowledge no sensory neuropathy has ever been reported with use of P5P”.

For those of you considering or actively pursuing amino acid levels (Jerry and Richard) elevations of alanine, aspartate, glycine, serine, tyrosine, valine, leucine, isoleucine, homocysteine, cystathionine, alpha-aminoadipic acid, or beta-alanine, just to name a dozen B6 sensitive amino acids, can signal increased need for coenzyme P5P.

Since initiating a daily regimen of at least 250 mg per day of P5P, I have noticed my MTD (maximum tolerated dose) of Mg to steadily increase (now at 1350 mg without any anticholinergics). I have watched my HRV and HR respond in a vagolytic fashion to these timed doses of P5P (50 mg q4h or so). The effect definitely wears off after 2 or 3 hours, depending on what I'm doing and where I'm doing it. This weekend will be the big test. My next predicted episode should be Sunday or sooner. However, now that I know about the UV connection (on B6 degradation and vagal stimulation) I feel that a judicious increase in and appropriate timing of P5P dosage should successfully address the issue.

Will report in on Monday or Tuesday.

***PC***

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A shot in the dark guess because my episodes increase markedly during the weekend also. It appears that it maybe caused by my lack of food intake on the same schedule as during the work week. I run around too much without eating. Is this a possibility for you too? Surprisingly, sometimes eating will stop an episode. (On the flip side, I get strong missed beats right at the end of eating or right afterwards also.)

What's the difference between a PAC and a "dropped beat"? I'm defining a PAC as a "missed beat" but with a regular pulse rate. Is this not correct?

What are the brand names of the P5P and GABA that you are taking? And what's inositol?

Are you on just aspirin as a blood thinning product?

***Kyle***

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Hi Kyle,

I stopped taking ASA some time back, since I started taking gingko biloba, fish oils, Mg, CoQ10, Vit E, etc.

The brand of P5P and GABA is Bio-Recovery, Inc. They're on the Internet.

A PAC is a beat that originates from an ectopic focus in the atrium. Some of these are nonconducted, when vagal tone is very high (they don't get past the AV node). Most reset the SA node via the retrograde wave from the ectopic focus to the SA node. Those that do not reset the SA node can be differentiated by the variation in the R-R interval (time between beats).

Dropped or skipped beat = one R-R interval that is exactly twice normal.
Nonconducted ectopic beat = one longer R-R interval that is longer than normal but less than twice normal
SA node reset = one R-R interval shorter than the normal R-R interval
No SA node reset = one shorter followed by one longer. This occurs when the ectopic beat retrograde wave is late in the cycle and meets the SA node impulse, which gets cancelled by the retrograde impulse.

This is easy for me to differentiate because I use a {Polar S810) and have learned through biofeedback the difference. It gives me HRV in real time as well.

Thank you for the weekend trigger suggestion. I don't think there is any question about it. Different food, often at restaurants, contributes to the increased glutamate load. I also think that being outside contributes to the problem, because upon going inside and sitting/lying down there is a greater vagal rebound and more glutamate is released. This rebound is not only due to lessening of physical activity with a vagal maneuver to boot but also to the abrupt increase in darkness (brain thinks it's time to sleep). This is similar to the vagal rebound postprandial or post exercise. In the former it is due the vagotonia of the digestive process and in the latter it is the vagotonia of the baroreflex. Both cause increased levels of glutamate.

Fran is absolutely correct in stating that matching P5P intake to glutamate levels to coordinate processing is a difficult proposition. However, I don't think this is insurmountable.

I've always noticed that I can terminate AF in the morning if I'm on the golf course much more easily (usually within 20 minutes) than I can by getting on or off some piece of exercise equipment indoors (I think non-steady state sympathetic activity is much more effective than steady state). I've also noticed that when I'm out in the sun my HR is definitely higher and my HRV is definitely lower all else being equal. For me AF is more easily triggered in sunny HI. By this I mean that AF is more likely once the rebound sets in (going inside and sitting down, especially if in the dark). It means that any subsequent vagal maneuver (bending over/lying down or eating/swallowing) is potentiated. This was the situation for me pre P5P/Mg. Since initiating the latter, much of this has changed, because I am better able to cope with the glutamate load. My most recent episodes attest to the fact that supply does not always meet demand.

I have found that episodes now are not triggered by any vagal maneuver. It seems that I slowly slip into a susceptible state, e.g., sitting in the movies after physical activity in the sun or during sleep after a late meal. Both situations represent a slowly increasing glutamate processing load that eventually exceeds the processing ability through P5P/Mg and expresses itself as an episode of VMAF. I've also found that those episodes that occur during the day are more easily terminated by sympathetic activity than those that occur during night (besides the fact that you're asleep for the latter). This is because the net vagal tone is lower for the former and cardiac conduction velocity can more easily be increased. This means the atria can sustain less wavelets of Moe (wavelet wavelength = conduction velocity x refractory period). Six or more are required for critical mass. I don't mean to cause a brain cramp with all this technical detail, but that is what I think is happening. In pursuit of indirect and anecdotal proof I hope that I can prevent daytime VMAF by P5P dosing relative to anticipated activities. It gets into the system relatively quickly (10 min or so) but peaks in around two hours with a half life of about 8 hours. I'm coming to the conclusion that postprandial episodes are not related to GERD, although GERD can definitely trigger AF (probably through insufficient GABA in some). They are caused by an overload of glutamate both from dietary intake and from vagal stimulation (and glutamate release) during GI peristalsis. Eventually the battle should become less difficult as B6, Mg, Zn, etc. levels are replenished.

VMAF was first described in the late 80's by Dr. Coumel, the French cardiologist. It is such a dramatic disease that it most assuredly would have been described long ago had it been in existence to any significant degree prior. Therefore, it seems most reasonable to ascribe its birth to the deteriorating Western diet, esp. Mg and the B vitamins. It's expression in a relatively select few VMAFers may be due to a glutamate decarboxylase polymorphism that exposes this dietary deficiency. This polymorphism was selectively favored in evolution because of its antidiabetic properties.

***PC (brain cramp specialist)***

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PC, I follow your posts closely (I may have to read it a few times slowly but I appreciate the knowledge) because your symptoms very closely parallel mine if not exactly. For instance, what you said about non-steady activity was right on. I discovered that riding my exercise bike terminated an episode but hasn't worked since those two times. What has worked (sometimes) was walking home from the grocery store with groceries. It didn't work walking TO the store. My guess, the uneven walk and uneven strain of caring groceries vs. the steady walk just going to the store. But I can't duplicate this indoors (via jogging in place slow/fast/ etc.) What you added in your observations about "subsequent vagal maneuver" and especially your comment about "episodes that occur during the day are more easily terminated …" also exactly describes my feelings because at least during the day (and awake) we have a shot at doing something to stop the episode.

What is frustrating is that just when I think I'm on the right path and something worked (riding the exercise bike, stopping all carbs, etc.) – it only worked temporarily and never (not yet) again. It's interesting what you said about the B vitamins. Just when I was going to supplement with them, I then noticed that the multiple vitamin that I take already had 150% of the daily recommended requirement so I have ok with the B vitamins all along even without knowing it. But, no effect on my afib.

Your comment about "eventually the battle should become less difficult as B6, Mg, Zn, etc. levels are replenished" was interesting but many of us have been on these supplement for many, many months now, if my case about a year so wouldn't you think there would be some results by now?

Anyways, I am following closely your posts and will try the P5P and GABA supplements. One question, should we go above the daily recommended requirements for stuff like B6 and Zn to be effective and if yes, how high should we go?

Thanks for your posts,

***Kyle***

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Kyle,

I started taking Mg as MgO. Then I got informed and starting taking 600 mg per day of elemental Mg as glycinate and various chelates like citrate. Nothing happened. Episodes became more frequent (q36h for 12 h per episode). Then I went to aqueous Mg and immediately went three weeks without an episode. I felt fabulous. Then it gradually deteriorated to once a week. One can't deny that Mg has/had a role in the process.

Then I started doing intense research on glutamate and Mg. All the while I was taking a Vit B complex with 50 mg of B6 (25 times the RDA) as well as 700 mg of elemental Mg per day. No improvement. Can't tell you how much I relate to the "being on the right path" only to encounter a dead end. Finally I stumbled onto the polymorphism phenomenon and tried taking even larger doses of B6 and found a curious decrease in vagal maneuver related episodes. Switching to P5P and Zn made this even more pronounced. Finally I'd made a breakthrough that has stayed with me - vagolysis by P5P mega doses - and one that was secondary to intellectual effort v. trial and error (I'm no Thomas Edison wannabe).

I'd forget about the GABA for now and put it on the back burner, especially since it appears (at least for me) that night time episodes are probably not GERD (probably GABA deficiency related in many) whether or not there is a late dinner.

Don't be afraid to bump up your P5P intake to 250 mg per day and add a B complex containing another 50 mg of B6. Since Zn is probably better than Mg as a cofactor for brain GAD, I'd add it. Maintain Mg intake. Zn competes with Cu (copper) and you can induce Cu deficiency with large

Zn doses, although the reverse is more likely to be the case without supplementation. I take 30 mg Zn with 2 mg Cu tid (GNC makes a Zn supp in this combination). If no improvement is seen within a week or two, you're probably not going to see it. I immediately perceived an improvement in my skin that has persisted. I have no idea what enzyme if any is involved, but it does say to me that I was effectively deficient in B6 even at 25 times the RDA. The B's are all water soluble and the peripheral neuropathy that can sometimes be seen with mega doses of B6 appears to only be in those on more than 300 mg per day for many months. Furthermore P5P, because it is the active form and requires no further processing in the liver, cannot build up in the body. The neuropathy probably occurs in some because the P5P is a required cofactor in its own production. So don't be shy, give it a whirl. Let me know what you find. Try 100 or more mg P5P immediately upon arising in the AM with VMAF and then walk to the store. Try the above daily regimen and I bet at least the circumstances of your episodes will change. That alone should tell you that you're close to the source. For daytime episodes on large doses of P5P holding my breath is very effective. I guess stimulation of all those pulmonary stretch receptors serves to suppress the vagus (HR slows during end exhalation via brief vagal toning).

***PC***

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Kyle,

Forgot to answer your other question about inositol. Please see Fran's post at the bottom of the page.

***PC***

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Good morning, PC,

It's nice to hear from you. Seems you're detecting many differences in your new regimen. Very interesting that you converted by taking the p5p, gaba, and inositol. I thought Lynn's post under "Can somebody tell me" in the BB was of interest. She used to live on a golf course, as did I, and they used organophophates for pest control. She also stated that when chemicals were sprayed by neighbors it would activate an episode. Could it be that when you're outside, these chemicals could be playing a role. I think she stated that she no longer has the enzyme, paraoxenase, to break down or eliminate the toxins and that Clem Furlong at the UW in Seattle can test for this. I wonder if he could test for the enzyme that converts glutamate to gaba. Maybe I will check into that. Are you aware if there is a test for this?

I would also like to add, that the day I ate spag. sauce, which I thought was the culprit, on 4/1, was a day I played golf, as well. They were plugging the course that day. I have had AF since, with the exception of 2 days. I'm still off my meds, but tried flecainide on demand and a toprol, to no avail. I have not been ingesting any free glutamate to my knowledge; eating only grass fed beef, organic chicken, halibut, organic veggies, salads, sardines, and nuts.

Thank you for responding and keeping us abated of your experiments with yourself. I hope you get to the bottom of this.

***Richard***

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Richard,

It seems that golfers are more prone to develop ALS (amyotrophic lateral sclerosis or Lou Gehrig's Disease). Tom Watson's former caddy and another PGA tour golfer, who's name escapes me right now, are two that I know of.

ALS has been associated with glutamate toxicity.

http://www.ninds.nih.gov/health_and_medical/pubs/als.htm

Perhaps we VMAFERS are all just ALSers in transit. Better start looking at VMAF as a warning sign that others lack.

Brain GAD is a very hard thing to measure. It may be normal in amount. It's the activity per unit that is deficient.

Organophosphates are cholinesterase inhibitors, i.e., they're vagotonic. There may very well be other ingredients in the pesticide mix that impact GAD.

So Richard, make sure you don't lick your golf ball to clean it.

***PC***

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PC, you mentioned your nightly episodes. Going with the free glutamate thread, evidently there are more being secreted at night to trigger the afib. What do you think these triggers are? Lying in the horizontal position, the slower heartbeats, the lack of activity, etc.?

***Kyle***

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Kyle,

I believe that Fran is right and that VMAF is caused by an inability to metabolize free glutamate as quickly as required. B6 and Mg deficiency aggravate this because they are required (together they constitute the required coenzyme) for the rate limiting step (glutamate decarboxylase) in this processing of free glutamate. I also believe that sometime eons ago there was a small mutation (in a common ancestor to all VMAFers) causing a single amino acid substitution in the critical glutamate decarboxylase enzyme that essentially gave us immunity to Type I diabetes (insulin dependent) and that it why it has persisted over all these millions of years (natural selection). However this same amino acid substitution resulted in decreased affinity of the enzyme for the coenzyme. Up until recently (last 20 years or so) this was no problem because almost everyone was getting plenty of Mg and B vitamins in their diet. To further contribute to the VMAF expression of this genetic abnormality in some yet to become VMAFers, the popularity of endurance sports entered the picture. These individuals with compromised, yet to be expressed ability to process free glutamate develop increased glutamate receptors in their NA (nucleus ambiguus) that drives RSA (respiratory sinus arrhythmia). This makes them even more sensitive to free glutamate released by vagus sensory nerves (from the baroreceptors in the neck and the stretch receptors in the lung) that interact with the motor nerves in the NA that control HR. The resulting excessive vagotonia creates the required conditions for VMAF - shortened refractory period, increased dispersion of refractoriness and decreased cardiac conduction velocity.

Unlike Fran I believe that this shortfall in free glutamate processing can be remedied by enhanced prolonged Mg and P5P intake. My view is that naturally released glutamate as a neurotransmitter substance is more of a problem than dietary free glutamate. The latter clearly can cause a problem. It sure has for me. I believe that vagal maneuvers and their immediate triggering of an episode are clear evidence of this. We can process glutamate but not when there's a flood of it. Vagal maneuvers create more of a sharp increase in it than does diet where it slowly bleeds into the system. I've eliminated the vagal maneuver triggered episodes but those that occur without any identifiable movement (sleeping, watching a movie) represent a situation in which the increase in free glutamate from the vagus is SLOWLY overwhelming processing

capability. I think that I can address this by increasing my P5P intake in anticipation of this. Up until now I just didn't realize that light and dark have such an effect on stimulating my vagus. Hopefully months of this will slowly enable depleted stores of B6 (even though some of the medical literature states B6 cannot be stored, other sources state that over 30% of the population is B6 deficient) and Mg to be replenished.

That's the long answer to your question, but I suspect that you're only interested in the short answer and that is, you're correct. Lying down increases the hydrostatic pressure of blood flow in the neck (carotid baroreceptors) and this is sensed by the vagus nerve and it then slows the HR and force of contraction. Unfortunately it goes overboard on this because of the above and VMAF results.

***PC***

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PC,

This is a very interesting take on Boron and how it reacts with B6, zinc, copper, etc. and how it causes increased calcium absorption. Boron reduces B1, thiamine, which is needed for cholinesterase production and relief of dermatitis. I personally have high copper and low zinc and believe that organophosphates are playing a role in inhibiting my cholinesterase enzyme, which I was trying to find a source that would increase this enzyme. I wonder if there is an active source of B1.

***Richard***

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Richard,

I believe that the active form of thiamine (B1) is TPP (thiamine pyrophosphate). In this regard it is similar to P5P. There are many enzymatic reactions that require TPP as a coenzyme. A Google search will tell you much more than I ever could about them. I believe one of them is required in producing P5P.

Also Mg is required by cholinesterase in order to break down Acetylcholine. So Mg deficiency translates to increased vagal tone.

***PC***

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Richard,

The following site talks about an available oral form of active thiamine (thiamine tetrahydrofurfuryl disulfide) and also talks about genetically abnormal forms of enzymes with decreased Km constants, i.e., decreased affinity of the enzyme for its TPP coenzyme and resulting disease. All this is very similar to B6 and I'm sure other B vitamins have similar situations. All this is a testament to biochemical diversity.

http://www.uic.edu/classes/phar/phar332/Clinical_Cases/vitamin%20cases/thiamin/case_report3.htm#thiamin1d

***PC***

Work backwards from this specific web address for a great website on other vitamin deficiencies.

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Thank you for the link. I just got back from the movies and am ready to dig in. Did you read my link. I forgot to put the link on the first post, so the link is on my second post. Here it is again, so you don't have to go back if you missed.

http://www.1abcweb.com/products/boron.html

In thinking today, this is my take on what is going on with me.

AF began when living on golf course. (pesticide - organophospates)
Organophospates block cholinesterase.
Too many antacids and Prevacid, therefore improper digestion of amino acids and inability to absorb Mg.
No Mg. to produce enzymes for cholinesterase and glutamate to GABA or for assimilation of B's, in particular B1 for cholinesterase and B6 for GABA.
Lack of B1 may indirectly effect dopamine and serotonin.
Tested high for copper which could mean high boron as boron causes this.
Copper also blocks cholinesterase.
Tested low in zinc. Boron has indirect link to reducing zinc.
Boron also causes calcium retention.
Ate apple, went into AF - #1 culprit for organophospates and good source for boron.
Played golf on day of pesticide application on golf course - went into AF 4/1 and have been out ever since, with the exception of 2 days.

Anyway, just a quick overview of what I see right now.

Thank you for replying and for the link, once again, PC,

***Richard***

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PC,

This is a very interesting take on Boron and how it reacts with B6, zinc, copper, etc. and how it causes increased calcium absorption. Boron reduces B1, thiamine, which is needed for cholinesterase production and relief of dermatitis. I personally have high copper and low zinc and believe that organophosphates are playing a role in inhibiting my cholinesterase enzyme, which I was trying to find a source that would increase this enzyme. I wonder if there is an active source of B1.

***Richard***

http://www.1abcweb.com/products/boron.html

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Richard,

The boron thing is interesting. I think that it's important to remember that we LAFers are different in some unique way (I think it's related to GAD). If boron was that important, there would be many others with LAF. That's not to say that it can't aggravate the central problem. But the simpler the explanation the better. I'm just not very good at translating medical jargon.

Just keep throwing in your thoughts.

***PC***

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Good morning, PC,

Be careful what you wish for, in regards to more thoughts. Hopefully Fran will read this, as well, as it pertains to her.

Organophosphates block cholinesterase. (pesticide used in tobacco farming)
Nicotine competes for acetylcholine.

I know Fran mentioned that the only last vice she had was smoking. My wife smokes, as well. In the case of Fran smoking, it would seem that the cigarettes could possibly be controlling the functions of these two components.

Here is a story of organophosphates on tobacco farmers: it speaks of sheep dipping and epilepsy, as well.

http://www.christian-aid.org.uk/indepth/0201bat/bat2.htm

This article shows the different compounds that compete for acetylcholine:

http://www.uky.edu/~holler/neurotox.html

I don't know how glutamate would play into all this, esp. in regards to Fran, but maybe the Kreb's cycle is disturbed by ingestion of pesticides, therefore rendering the enzymatic action of glutamate to GABA nil, and is obviously disturbed by imbalances of nutrients. There just seems to be an epidemic of diseases relating to acetylcholine and cholinesterase, which I know you have already covered with a fine-toothed comb. In all regards, it definitely gets down to enzymes and their actions or inactions, Mg., B vitamins, zinc and C. Enzymes are the workhorses and they need the tools to work and in some cases may have to be re-taught how to do their job.

As for me, I think I'll try to take the active form of B1, as well and find out what my boron levels are, somehow.

***Richard***

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Richard,

Pantothenic acid (B5) is also involved in the production of acetylcholine and its active form is called Acetyl Co A. It would be nice to find a B complex that contained all these active forms of the B vits without having to resort to the individual approach.

***PC***

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Hi, PC.

I'm taking 15 mg of zinc daily, but I take no copper at all. What do you think the implications or effect(s) of that intake might be? Should I consider adding some copper, or is that not necessary at my level of zinc ingestion?

I'll keep you posted on the results of my amino acid testing, especially the phosphoserine result.

Thanks in advance for your help.

***Jerry***

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Jerry,

Good to see your name on a CR post.

I'd tend to agree with your "not necessary at that level" thinking. I believe people are Zn deficient much more frequently than Cu deficient.

I just came back from a noon lecture by some MD from UCLA on osteoporosis in males. There was nary a mention of Mg. In fact when asked, he said "Who's Mg deficient?". This from an "expert" who speaks all over the country. I needn't mention that a pharmaceutical co. was footing the bill.

Thanks

***PC***

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Hiya.

If it weren't so troubling, the irony of your anecdote about the physician-lecturer would make me laugh out loud. Instead, it saddens me. In my younger life, I was trained as a military corpsman, then I scrubbed in an OR on my return from active duty. [I was a lucky one--Fort Bliss, Texas rather than Korea or Nam.] What I found among the surgeons and residents with whom I worked was an amazing knowledge of the mechanics of the human body. And it came to pass that I worshipped at the altar of traditional medicine. After all, who would not be awed by the power of placing your hands on, as well as inside, the body of a living being, witnessing the rhythms of active life first-hand? I still have an abiding respect for physicians, even though I wonder why they've decided to enter a profession upon which more and more cultural changes and demands continue to be imposed.

When I worked in the OR, I marvelled at the amount of schooling and training required to become a physician, and I realized that there were innate abilities needed in order to fulfill that educational and vocational mission, abilities I knew I did not possess. I learned many things from the men [yes, all men in 1970] I assisted in the OR. More than anyone else, though, a third-year resident taught me something I'll never forget, when he said: "We haven't learned anything about the biochemistry of the human body in med school."

I was stunned at the time, but I've since learned to accept that the complexity that comes with the task of comprehending the human body in order to treat the patient demands that shortcuts in the four-year curriculum be found. And the process has only gotten more complex over time, given the technological advancements that have followed. Still, I would be remiss if I didn't say that the patient has suffered because of the physician's lack of awareness of less complex aspects of health, such as magnesium supplementation, that can have so many far-reaching effects.

Perhaps we'll have better luck in the near future, now that the genome has been fully mapped and the supporting role of RNA has come to the fore. We may well be able to break the stranglehold that pharma conglomerates have had on the medical profession. Won't it be fantastic when my daughters will be able to visit a pediatrician's office and read their children's genome and the likelihood of disease that will be evident there? Interventions will soon come fast and furious, I'm convinced of it. Of course, the pharma companies will be replaced by the gene companies, whose stocks are already soaring. But if they provide real treatments and interventions that will disconnect the disease or disorder, won't we come to depend on them as much as many patients now rely on drug companies?

I'll leave that to the ethicists. For now, I'm looking for some intervention.

Thanks for writing back.

***Jerry***

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How about that, I've doing something right for a change. I've been taking B6 for general health reasons for along time now, but really haven't impacted my afib. PC I'm at 150% of the RDA for B6, should I go higher? Maybe my body has built an immunity since I've taking it for so long.

***Kyle***

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Kyle,

Don't be afraid to experiment with higher doses of B6. It is reported in the literature that 50 mg of P5P is equivalent to 200-300 mg of B6. P5P has not been associated with neurotoxicity. Try it for two weeks to see if there is any improvement in LAF or any other organ system. If not, drop it.

***PC***

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PC:

I'll be waiting until my amino acid profile comes back--especially as it relates to the phosphoserine level--before I begin any P5P. My research shows that proper phosphoserine would mitigate against P5P deficiency.

Don't want to fix what ain't broke.

All the best,

***Jerry***

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CR As,

I have to confess failure, but it is only a temporary setback.

Friday PM I went out for dinner and a movie appropriately laden (internally and externally) with P5P. Finished dinner at 6:50 and started the movie at 7:30. All went well. Then I returned home and started to watch a Harry Potter movie with my 6 year old daughter. One hour into the movie while half asleep and within one hour of my last dose of P5P, there was an ugly head rearing.

Because I had some leg cramps and fasciculations (muscle twitching), I think I must have outrun my Mg/Zn coverage with the extra P5P. The cofactorless (no attached Mg or Zn) coenzyme out competes the complete coenzyme (P5P + Mg or Zn) for binding on glutamate decarboxylase. In addition the excess P5P with or without the attached metal is quickly excreted in the urine, diluting my onboard supply of Mg/Zn. This even though I was drinking my WW with Mg supplements throughout the day. At least this is my working spin on the incident.

Just goes to show you how delicate the balance is.

Awaiting NSR

***PC***

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PC,

Will you check this product out for me and all of us, it almost sounds too good to be true but it might have all the B's you are looking for in an easily absorbed liquid:

http://www.myseasilver.net/rmf/about.html

It is only available in the US.
Thank You,

***Ella***

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Ella,

Thanks for the post and link.

I was just looking at SeaSilver the other day. Liquid is definitely better than solid when it comes to absorption. But active is better than inactive too and from what I could discern, SeaSilver is nothing special in the latter category. Besides one of its amino acid components is glutamate.

Would love to find a P5P with Zn or Mg supplement in liquid form.

***PC***

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Hello All,

I found this to be a most enlightening article, that hopefully has not already been posted here. It speaks of thiamin, zinc, glutamate, etc with the importance of methyl-cobalamine B-12, in the brain. It is based on one doctor's theory in regards to his research and involves the cytochrome P450 system. He also thinks that it is very important to take active forms of certain nutrients, which are covered here.

http://www.orthomed.org/links/papers/tigalc2.htm

***Richard***

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PC,

I know you're busy, but I would like your opinion on the above link. I have located some forms of active B12 and allithiamine at the source below. I am particularly interested in the methylcobalamin vs. cyanocobalamin, which is cobalamin attached to cyanide, a strain on the liver that needs to be avoided, in my opinion. It appears that lack of thiamine, or the inability of the liver to break down thiamine to its active form, plays a part in glutamate neurotoxicity, as well as, considerably reduces acetylcholine. Thiamine is needed for blockade of dopamine oxidation to hydroxy-dopamine, which is a neurotoxic agent for the noradrenergic neurons, as well. Anyway, here is my link to a supplier:

http://www.healthyhotline.com/1010.shtml

In the search box, enter thiamine - scroll down to Cardiovascular Research for allithiamine. You can click on that to get ingreds.

In the search box again, enter B12 - scroll down and look at Neutraceuticals - Neurobolic - SPRAY - ingreds. b12 500mcg/ coenzyme b12 500mcg/ ATP-10,000 mcg/ alphaglycerylphosphorylchol 10,000mcg.

Also look at R & D Formulations here, as a sublingual of 5000mcg. of methylcobalamin.

I think maybe these combined with P5P may help, and is my utmost priority come Monday morn.

I have also come to the conclusion that the microsomal cytochrome P450 system is playing a huge role in the ability to break down different components of nutrients, as well as eliminating toxic by-products, therefore whenever we can take active forms of vitamins, as you are doing with p5p, all the better. It was the doctor's theory in the above post and link, that the main aim was to normalize the altered microsomal liver enzyme activity. It only makes sense, that our livers are suppressed on many levels, due to all the outside influences of our environment, whether it be drug or pesticide induced, or just being nutritionally deficient.

Thank you for your time,
***Richard***

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Richard,

Those are very good websites with lots of meat. Thanks. And as long as we're talking about autism, here's one for you.

http://www.autism.org/vitaminb6.html

***PC***

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Good morning PC,

I am very sorry I missed this. I cleared the cookies on my computer and it messed me up. This was very interesting, in regards to the dosages of Mg. and B6. By my weight, I should be taking 1350 mg. of B6 and 495 Mg. I'm certainly not getting that much B6, but my Mg. is probably about right. I'm still trying to increase Mg. dosage. Thank you for that link. I do think there is much to learn about ourselves by looking at other diseases.

***Richard***

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http://www.dietsexercise.com/l-theanine-pms-dopamine-Text1.htm

So to increase GABA it would seem that all you had to do was drink loads of green tea. But it also increases dopamine (no good for me).

"Theanine may protect against glutamate, an essential brain chemical that is toxic in high amounts. Although essential to brain chemistry, too much glutamate kills brain cells. The most common cause of glutamate overload is insufficient blood supply. If the brain doesn't get adequate blood flow, glutamate surges, calcium increases, and free radicals damage cells. Theanine is structurally similar to the amino acid, L-glutamic acid. The similarity enables theanine

to physically block glutamate (which is a version of glutamic acid). Although researchers aren't positive how theanine works yet, they theorize that theanine blocks the NMDA receptor which is the doorway that glutamate uses to enter cells. Because of the similar structure, theanine can also fit in this doorway, blocking access to glutamate. But, although it can fit in the doorway, theanine does not have the same effect on the cell as glutamate does. Rather than causing damage, theanine acts like a shield against damage."

***Fran***

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Drat

Theanine also heightens dopamine. My dopamine levels are too high anyway.

http://www.itmonline.org/arts/theanine.htm

***Fran***

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