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VIRTUAL LAF CONFERENCE

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SUBJECT: Is LAF Physiologic or Pathologic?

This Session of the CR is intended to delve into this question, especially via my recently published article in *Medical Hypotheses* (2007), v. 68, pp 281-287, on this very topic. Copyright stipulations preclude my posting it on the open internet. However, I can send it "to specific colleagues that you know". Such use is limited to educational purposes and/or non-commercial research. However, this is a username and password secured site and I feel that I already know many of you, especially after having posted in the CR since its inception. In fact my post on 1/2/03 in Hans' inaugural CR Session was the first. We are now in the 55th Session. THANK YOU HANS!!!!.

Quite a few of you participated in LAFS XI on which the hypothesis is based and which include the results of that survey. Our discussions have always been and will continue to be educational and non-commercial. Accordingly, I will be happy to provide a copy of the full text via email to those so requesting (just click on my initials above), provided you abide by the above restrictions and hopefully contribute to the discussion. Show the article to your MDs. Educate them, e.g., that digoxin is contraindicated in LAF, that amiodarone may not be necessary and that coumadin is definitely not, that prognosis is good and in fact better than normal.

As LAFers, we are all concerned about the origin of our arrhythmia. Prognosis hangs in the balance. Is it the harbinger of doom, just another ailment incidental to aging, or a manifestation of some controllable physiologic abnormality? If it is the latter, LAF is a mixed bag nonetheless. Indeed there appear to be no test(s) that might definitively differentiate LAF from pathologic AF. Although most MDs probably ultimately regard LAF as an early manifestation of a pathologic process, I beg to differ.

Although the results of LAFS XI and several of Hans' previous surveys suggest that insulin sensitivity and hypoglycemia may play a significant role in most cases and that MVP may also be involved, these mechanisms do not appear to be all inclusive. There are clearly others with LAF that do not fit neatly into this schema. Your comments especially are solicited in the ensuing discussion to help further refine it.

ABSTRACT

Atrial fibrillation risk has been strongly associated with increasing age and visceral obesity. These characteristics are strongly associated with diabetes, decreased heart rate variability, and chronic inflammation. Lone atrial fibrillation (LAF) on the other hand exhibits a predilection for the physically fit and the middle aged, especially males. Given these opposing features, it is postulated that pathologic AF is due to cardiac fibrosis and other age related changes, while LAF is due to physiologic neurohormonal changes related to autonomic tone, insulin sensitivity, and electrolyte imbalance and that pathologic AF and LAF can be reliably differentiated via an anthropometric approach using weight, height, hip, and waist measurements. An anthropometric study is undertaken from an LAF database to test this hypothesis. Such individuals in addition to being younger and predominantly male appear to be taller with less central adiposity v. those with pathologic AF. The ramifications of these findings with respect to insulin resistance, sympathetic tone, inflammation and hypertension, often associated with pathologic atrial fibrillation, are discussed. Speculation is

drawn about possible etiologic link with mitral valve prolapse (MVP), which is commonly encountered in the tall and thin and which shares multiple clinical features with LAF.
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=17005327&query_hl=2

PC

You've been busy. I seem to have elements of both scenarios that you've outlined. At the time of my afib discovery I was overweight and borderline high blood pressure. I was also training for x-country skiing and playing hockey in the winter and half Marathon training in the summer. Sounds like the worst of both worlds.

Would there be different approaches to treatment based on where one falls in your road to afib hypothesis or is it a case of it doesn't matter how you got there just that you are there.

Adrian

Hi Adrian,

That's an interesting combination.

The central thrust of the paper is that atrial stretch is at the heart of PAC generation (for both LAF and pathologic AF) and that blood sugar dips are at the heart of potassium imbalance. They both contribute to reversible substrate modification that provides fertile soil for initiation of an episode. And, of course, dehydration and stress aggravate the potassium shortfall and stress also generates PACs.

Do you have increased PACs and, if so, for how long. I'll bet that you had them long before your AF ever reared its ugly head. From the survey data your LA dimension is increased. Whether it is due to borderline hypertension or occult mild mitral regurgitation is difficult to say. Have you had a TEE? As tall as you are, you're definitely a candidate for MVP.

Your overweight category translates to less vagal tone and more sympathetic tone (and inflammation and insulin resistance). If this is so, then your episodes should not have been typically nighttime and should have been more stress related. In adrenergic LAF stress and its associated catecholamine induced automaticity may contribute to PAC generation, whereas in VMAF atrial stretch is the primary culprit in this regard. I've always suspected that vagal maneuvers trigger LAF because 1) gravity retards atrial emptying (=> more atrial stretch => increased PACs) and 2) the vagal tone (=> shortening of AERP) makes for fertile soil.

The real problem is trying to decipher how much each of these various factors contributes to episode initiation, even assuming we're on the right track in that regard. Even within the same individual the composition of factors can change and presumably their weighted contribution as well. And some LAFers are going to be more afflicted with one factor than another v. another LAFer, making attainment of a comprehensive classification schema a real conundrum.

And if that's not sufficiently challenging, there's glutamate, GERD, spinal misalignment, etc.

PC

PC;

I read your paper with great interest and I must admit I feel like the "poster child" for your hypothesis. I fit the anthroprometric model quite accurately. In addition, I have been diagnosed with mild MVP and when I was in my early twenties, I was also diagnosed with reactive hypoglycemia.

I started jogging at age 30 (I'm now 59, still jogging) and the symptoms of the hypoglycemia totally disappeared after I became fit. I believed that the running increased my ability to store glycogen and therefore fixed the reactive hypoglycemia. I went through 2 major growth spurts when I was young. During the first one, my vision went badly myopic and my guess is at this time the seeds were planted for MVP. MVP also has a preferred anthroprometric model

and my MVP was predicted before the tests were run to confirm it. I also have long arms; my wing span is larger than my height. As you would expect I am a pure vagal afibber. I am keeping the AF under control presently with mag, K and taurine supplementation. I have also backed off my jogging mileage in an attempt to lessen my vagal tone. Thanks for the effort you have invested in this board.

Larry Zajdel

Hi PC, the best I've been able to come up with is that I have (had!) 'anatomical AF'. I think the only peculiar thing that's been measured in me is that I have overly large pulmonary veins. I'm guessing this large size has just predisposed me to AF and no adjusting of any hormone or change in diet has done anything to changed this physical problem. Maybe an MRI should appear earlier in the diagnosis for LAF? It may at least help to spot those individuals where ablation might be bumped up to second or even first line procedures.

I've always thought of ablations/MAZE as a bit of a crazy solution to AF but for those individuals with an anatomical abnormality perhaps there is little else that will fix the problem?

All the best,
James D (NSR since second ablation Feb 2006 and loving it!)

29 years old, 6'2" , 200lb and pretty fit when AF hit.

Re: "...it is postulated that pathologic AF is due to cardiac fibrosis and other age related changes, while LAF is due to physiologic neurohormonal changes..."

Using this terminology, I'm concerned that LAF tends to become pathologic AF as we age due to induced cardiac fibrosis from LAF.

Bob K.

I think that it is environmental (OK, Physiologic). Our environment has been changed by industrial, agricultural and military practices so that some of us are no longer fit for life in this polluted planet.

I posted something relevant to our interest in the forum to this effect, unfortunately the language (theoretical sub-nuclear physics) is too much for me, and apparently for anyone else so far.

William

James,

Thanks for your post. A series of anecdotes can eventually become statistically significant.

The dilated PVs certainly explain the PAC production, which you probably had for years. This covers the trigger component of the Dual Substrate Concept. But what might have created the fertile soil?

I neglected to include in the text of my article the explanation for the correlation between venoatrial stretching and PAC production. I didn't want to overload the readers and dilute the message even further. So, I'll overload you CR readers.

Atrial stretch also stimulates specific ionic channel remodeling (a sodium channel called the “funny current”) that enhances pacemaker activity in the area of atrial stretch.
http://www.springerlink.com/content/ngc66792ml325am9/
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=10413374&dopt=Abstract
http://www.ionchannels.org/showabstract.php?pmid=15485684

FYI the funny current I(f) = the queer current I(q) = the hyperpolarizion current I(h).

This particular ion channel is the one that most characterizes nodal cells (P cells) and essentially means that pacemaker-type cells are now firing from the PVs.

Also, based upon clinical info similar to yours from another respondent requesting the article I am beginning to suspect that tall more than thin may predict MVP.

PC

Hi Bob,

That's a realistic concern that IMHO is more dependent on factors under your control, e.g., weight, BP, ... Clearly AF begets AF and even LAF is causing inflammation and remodeling and a significant percentage go on to become permanent LAFers. But AERP shortening (v. fibrosis) is a key requirement for maintenance of an LAF episode. This lengthens as we age and should help many (?most) LAFers turn the corner, all else being equal.

But there are clearly others like James D (and Hans) where, no matter what is done (besides ablation), the beast will continue to raise its ugly head.

The main players in pathologic AF that cause these probably irreversible changes are aldosterone and cortisol. These two are more associated with hypertension, obesity, sympathetic tone, inflammation than features seen in most LAFers.

Another word about adrenergic LAF, which I also didn't include in the article.
Hans started as a typical adrenergic type, yet his HR response to swallowing on the Freezeframer indicates strong vagal tone. You might rightfully ask how can that be?

Hans is not only a giant amongst us, he is also over 6 feet tall. IMHO mild mitral regurgitation, which Hans has, is causing increased natriuretic peptide secretion in response to the secondary atrial stretch. This means he has a low blood volume (characteristic of those with MVP) and has to compensate in some way to maintain perfusion of his organs. Although he is not hypertensive, he is hyper-responsive to catecholamines (a lesser amount causes a greater response). This means that any given stressful situation causes more adrenergic symptoms including increased PAC production. Catecholamines increase automaticity.

As an aside, it might be worthwhile considering the possibility that intracellular magnesium testing amongst LAFers might be a specific marker for increased natriuretic peptides => those LAFers with MVP. It is not generally appreciated in the medical literature that ANP and BNP both cause not only urinary sodium (and water) wasting but also urinary magnesium wasting.

I took over 800 mg of aqueous elemental magnesium per day for a year and my intracellular Mg went from 34.2 to 34.3. Clearly I'm a urinary magnesium waster. I too have mild MVP and mild MR.

PC

William,

I can't really disagree with you, except to say that it is more likely multifactorial.

Early on I was like you - some specific thing was causing this. Like Hans I searched and searched. We all have.

The Dual Concept Theory of AF is gaining in popularity and for good reason. Researchers are moving away from the idea that some specific ion channel is the culprit. That's not to say that ion channels are not the culprit in many familial cases nor am I claiming that there is no genetic component. MVP is genetic.

You're absolutely correct on the physiologic component. The fact that the majority of single episodes of LAF are due to holiday heart syndrome (drinking on an empty stomach => hypoglycemia => a kind of stress => increased blood

cortisol => urinary potassium wasting) underscores the role of low blood potassium in the genesis of LAF. And the number two reason is probably urinary potassium wasting due to diuretic usage + dehydration.

I've had a long running discussion with Jackie about the role of inflammation in LAF. Clearly it is a major player in pathologic AF. However, the fact that an episode of AF itself causes an increase in ROS (reactive oxygen species) and therefore inflammation makes a definitive statement about this difficult. IMHO inflammation follows LAF but does not precede it, unlike in pathologic AF.

PC

More anecdotes: My LAF began after flying to Europe and a half a bottle of white wine (A binge for me). The second episode was three months later, the first time I had wine again and the last episode was December 10th last year, 36 hours after a single swallow of tequila. What has been very obvious and troublesome is what happened to my body after the first episode which has been the onset of PACs mostly between midnight and 6:00 AM and GERD symptoms. I am 5'11" and was 189lbs when this all started and am now 173 lbs, the wt. loss stemming basically from cutting out evening snacks and trying to eat less bread. I never drank much, but for the past year I have had no alcohol.

I stopped exercising vigorously for a while but I started again. I seem to be fairly strictly vagal with a resting pulse of 48-50. I am almost 60.

I feel strongly that the process is pathologic as the change to frequent PACs happened right after the first episode of LAF.

My echo was read as normal except for a whiff of mitral regurg. No sign of Atrial enlargement. There were no PACs on the stress test, but PACs on the Holter and mainly at night.

My guess is that the first episode (which only lasted 11/2 hours, self converted and was a typical holiday heart) did some degree of remodeling. The result has been that anything which increases vagal tone is a potential trigger for my PACs and on two occasions the AF. The first episode of AF started with no warning, but was obviously AF.

I do think my GERD symptoms started after the first episode of AF. The symptoms consisted of a post prandial hoarseness and coughing. They have resolved with the use of a PPI, head elevation of the bed, decreasing tomatoes and no large meals in the evening.

I am curious why the GERD started after the AF and I also have a suspicion that the PPI is playing some role in the one year vacation I have had from the AF.

I continue to think that we could and should use the power of this board to survey all who become participants, in terms of their size, shape, triggers, and treatments. The number of people who visit this site with real motivation to find answers is large and I think we should try to add people data to our theories.

BillE

Hi BillE,

Thank you for your contribution and I heartily agree with your parting comments.

Regarding the onset of PACs after your first episode, I might insert a useful anecdote. While a practicing pathologist, I never ceased to be amazed at the large number of testicular masses with clinical history of trauma that I subsequently diagnosed as cancer. Clearly testicular cancer is not caused by trauma, but it sure did seem so from this experience. Obviously the tumor was there pre-trauma and then the trauma led to its discovery. The problem with this explanation was that many of these tumors were definitely not small. Was this denial or just unadulterated unawareness?

Unless you had a 24 hour Holter pre first episode showing considerably fewer PACs, most MDs would have a hard time explaining your observation.

Regarding your GERD, IMHO its correlation with LAF rests with the concomitant stimulation of the vagus nerve and not with local inflammation, but who knows? Vagal tone per se should not stimulate PACs. It only shortens the AERP (=> fertile soil), but associated vagal maneuvers usually translate to increased left atrial volume and stretch due to position related changes in the effects of gravity (=> more PACs). Bedtime is the major vagal maneuver of the day. Furthermore, the nadir of blood K+ is midnight. Ectopics have been correlated with low blood K+. Associated leakage of intracellular K+ also causes shortening of the AERP.

Could the stress and anxiety associated with your first episode have contributed to your subsequent increase in PACs and GERD?

PC

Re: "Catecholamines increase automaticity."

I tend towards mild hypothyroidism but I am also an adrenergic afibber. I had thought that these were somewhat contradictory symptoms. However, to compensate for the mild hypothyroidism, my system may be producing more catecholamines to keep my activity level up and the extra catecholamines may be increasing automaticity in my heart cells resulting in afib.

Bob K.

PC, it is definitely sufficiently challenging. It's difficult to say whether I had increased PACs prior to initiation of afib. I do remember rare instances of skipped beats while sitting quietly but they were few and every time I checked my pulse it was normal. My resting heart rate has decreased over the last ten years from ~72 to now ~62. I know this because we always checked it in first aid class. My afib seems to occur when my pulse slows down to 60 and lower which allows for the generation of more frequent PACs that eventually lead to afib. Shortened AERP? Sounds vagal eh!

My personal opinion on atrial stretch is that it occurred due to the physical exertions that I was undertaking at the time. In Aug of 2002 I had a echocardiogram that listed the left atria at 43 mm (19-40 mm) and the right ventricle at 31 (9-27 mm). The right atrium was also enlarged with the left ventricle at the upper limits of normal. Wall thickness and global systolic function were within normal limits. The four cardiac valves were structurally intact. No significant Doppler abnormalities were present (what's a Doppler abnormality?).

In February 2003 I had a TEE. I don't have the numbers but I will summarize the diagnosis I received. The study demonstrated normal cardiac chamber dimensions and function. In particular the right atrium and right ventricle were of normal size. The atrial septum was intact without evidence of atrial septal defect or patent foramen ovale. Cardiac valve structure and function were also normal. This result demonstrates no morphologic abnormality to suggest a pre-exposing situation for his atrial fibrillation.

In the six months after afib onset I stopped almost all of my athletic activities. If one is to believe the two successive cardiac reports that I received then my enlarged heart chambers must have shrunk a bit, ergo the enlarged heart chambers were probably the result of said physical activity.

Having said all that, I was overweight. My brother, who takes after the other side of the family (mom's) but was about the same size and weight as myself ended up as a type 2 diabetic about the same time as I was becoming an afibber. He is one year younger. Was I becoming insulin resistant? Perhaps although there is no proof of that. My blood sugar was always normal, fasting or otherwise. I've never seemed to benefit from potassium supplementation and my tests have always been mid-[normal to high.

I've resigned myself to the fact that I don't fit the mould. That is why I am trying the Hoffer protocol for a few months to see if that helps. Even a blind squirrel finds a nut once in a while.

Cheers

Adrian

As a physician also, I feel quite certain that I never had (unless they were silent) episodes of PACs similar to the ones I now have. For me they are quite obvious and easy to palpate. I don't think that anxiety plays a role in the PACs. They are mostly at night at rest and go away with motion and stress, although at times I will feel them as the stressful situation abates. I will also go long periods without them and feel that my heart has almost returned to normal and then seemingly out of the blue I will start having them again. I hold to the idea that something clearly happened with that first episode that makes me now react to increases in vagal tone with PACs and at least on two occasions with AF.

Although the statistics tell me that years of exercise played a role in this, I do have two sisters with MVP and frequent ectopy, with similar triggers to me. I believe that a genetic predisposition and a perfect storm lead up to the first episode and then like a old fashion phonograph record one has created a pathway for the easy initiation of ectopic activity. My perfect storm for the first episode was very high stress for about a year with an issue at work, the endurance training, and the holiday heart.

It is interesting to me but since I returned to training about six months ago, in general my ectopic activity has been less. I have the feeling that just as people report that an episode of AF gives them some respite from the next episode, that a very high heart rate through exercise helps to reset the heart. The real question though is whether a short term benefit will lead to long term worsening of the LAF.

All just ramblings, but nice to have participants. It has become a boring topic for the home crowd.

BillE

This morning I became aware of new info that appears to be relevant to what causes our problem, and maybe what might be done. All new to me, and IMHO worth an intensive read.

http://www.newmedicine.ca/overview.php

Plus my post today in the bulletin board entitled "eSens in Clear(er) language"

Enjoy

William

Larry - the best way to resolve hypoglycemia is through exercise - especially that which uses the large muscles...so through your jogging, you created ideal insulin sensitivity and circumvented the hypoglycemia - as advertised. This is the classic way to both resolve hypoglycemia, improve insulin sensitivity but more importantly, ward off diabetes.

Good job.

Jackie

Bob - When I was researching for the CR topic on Cardiac Fibrosis, I learned it is common with aging - it may be more so in a fibrillating heart due to the irritation involved. (This is how I became so interested in the use of proteolytic enzymes to reduce fibrosis).

Do a google on "cardiac fibrosis aging" - there 's a huge amount of data there along with studies. Dr. VanWagoner of the CCF has an excellent article on cardiac fibrosis and the link is at the CCF site or in the CR topic.

Quote: "In humans, cardiac fibrosis is universal in the aging heart."

Source:

Gene Therapy for Repair of Cardiac Fibrosis
A Long Way to Tipperary

(Circulation. 2005;111:391-393.)

http://www.circ.ahajournals.org/cgi/content/full/111/4/391

Jackie

PC - Thanks for posting and fielding the interesting questions popping up here.

Your last statement, magnesium waster, brought a question to mind since in the regular BB, I just posted to Dean about gut dysbiosis and inability to absorb nutrients, treatments etc. Have you gone to the trouble of investigating your functional gut health?

Best to you,

Jackie

BillE - I'm sure you are aware of this but you said:

"The symptoms consisted of a post prandial hoarseness and coughing"..... these are classic signs for food allergies or sensitivities. So is sneezing or elevated pulse after food consumption.

While the PPI may lower stomach acid, if you have food sensitivities, you will do nothing to correct that issue with the use PPI, just avoid the immediate symptoms. The problem lies in what the circulating antibodies do in the body, especially the intestinal lining - long term.

Jackie

Hi Jackie,

Mahalo for your posts.

My unproven conclusion that I am a urinary magnesium waster is based on the premise that the only other way to lose it is through the stool. In which case this should be decidedly loose. This is the basis on which laxative magnesium preparations are based. My BMs are much softer while on a diet rich in nuts (lots of Mg) than they ever were on aqueous magnesium (800 mg per day). Therefore, I must have been absorbing it => urinary wasting, since IC levels essentially unchanged.

The other reason for this conclusion is that it fits physiologically with the hypothesis on several levels.

1. BNP is increased in those with LAF and is independently associated with left atrial diameter
"B-type natriuretic peptide levels in patients with paroxysmal lone atrial fibrillation"
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=16715186&query_hl=2

The situation for ANP and LAF is less clear.
'Discordant Atrial Natriuretic Peptide And Brain Natriuretic Peptide Levels In Lone Atrial Fibrillation"
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=15629379&dopt=Abstract

In humans ANP is considered strictly atrial and BNP is considered strictly ventricular in origin.
However, there are some studies that suggest that early LV dysfunction is associated with increased BNP. And, at least in dogs, this seems to be produced by the atria.
'Differential atrial and ventricular expression of myocardial BNP during evolution of heart failure"
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=9612380&dopt=Abstract

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2. ANP is secreted by type B atrial baroreceptors (afferents or sensory fibers). These receptors are stimulated by atrial filling or distention and are located near the venoatrial junctions (PVs and SVC and adjacent atrial tissue). Ablation studies have demonstrated dilated PVs to be the predominant source of ectopic activity triggering AF. The role, if any, that these receptors play in generating that activity is not clear, but ANP secretion should be stimulated in such individuals. Studies of those with MVP show blood volume to be uniformly decreased and to be inversely proportional to ANP levels.

ANP has a half life of 2-4 minutes, while for BNP it's 20-40 minutes and Mayo Clinic charges about $200 for either. Both attach to the same natriuretic receptor sites in the renal tubules. So, differentiating between the two in LAF may be moot.

It is not generally known that natriuretic peptides cause urinary magnesium wasting. I kind of stumbled onto this fact while researching a related issue.

I think it is mighty curious that 7/7 respondents to LAFS XI that underwent intracellular mineral analysis were all low in magnesium.

This raises an important issue. Might it possible to selectively identify a large component of LAF that might be causally related to MVP by looking at their IC Mg? Perhaps I could approach Dr. Burton Silver at Intracellular Diagnostics (www.exatest.com) about this. Perhaps we could generate another paper on LAF.

PC

Thanks for your comments, Jackie,

Your hypothesis is reasonable, but all post prandial symptoms cleared up rapidly with the PPI, elevation of head of the bed, and avoidance of large meals in the evening. In addition when I stop the PPIs the GERD symptoms return in a mild form. I don't think food sensitivities would clear in the same way. I have to admit my own first dx was food sensitivities, but these are also classical GERD symptoms and it did not seem to matter what food I ate.

I actually would not pay too much attention to these symptoms, if I was not aware of the connection of GERD and AF. I have been rigid about my anti-Gerd regime since my last AF episode, a little over a year ago, and I do feel it has played a role in my AF free time period. This is further supported with a study in Italy which showed treatment of GERD to decrease AF episodes.

I believe that it is even possible that there is something else about PPIs (besides treating GERD symptoms) which may play a role in decreasing AF episodes.

BillE

BillE,

Proton pump inhibitors obviously inhibit gastric secretion of H+ ions.

Not only should PPIs cause less GE junction irritation and/or vagal stimulation but less of a meal related alkaline tide. For those non-MDs out there this means that gastric acidity is accompanied by a mild blood alkakosis (H+ into the gastric lumen => OH- in to the blood), required to maintain electrical neutrality. To maintain blood pH the kidneys will

preferentially secrete K+ (instead of H+) into the urine, whenever an anion is excreted in the urine.

Perhaps this other role of PPIs in decreasing AF rests with improved K+ balance.

PC

PC;

Your statement "Not only should PPIs cause less GE junction irritation and/or vagal stimulation but less of a meal related alkaline tide" prompts the following question.

I have used a heart rate monitor for the past 5 years when running. I run at a pace to maintain a 130 bpm rate. I have observed that when I burp during my run, my pulse rate drops from 5-10 bpm and then returns to 130 over a 15-20 second period. The more burn sensation that is associated with the burp, the deeper the fall in pulse rate. I believe that the pulse rate data drop is real and not some kind of artifact of the HR monitor.

Recently as an experiment, I have started taking a dose of Maloxx before I run. This has produced far fewer burps/run and when I do burp, the fall in pulse rate is only about 3-4 bpm and there are no burning sensations. My thought was that if the gas coming from the stomach was accompanied by some stomach acid due to the stomach contents agitation of running, I could be irritating/stimulating some aspect of the vagal nerve.

Is the burping causing vagal stimulation via acid irritation? And once irritated, would the vagal nerve be sensitive for some period of time after the run? The vast majority of my AF episodes occur in the early morning following a run the evening before.

Larry Zajdel

Larry,

Thanks for posting your personal history and meticulous measurements. It is a most fascinating question.

As I see it, there are two possible explanations.
1) Inflammation at the GE junction extends to involve the very near vagal ganglia (less likely)
2) Cross stimulation of cardiac vagal fibers by adjacent impulses in GE junction vagal fibers (more likely) - they connect to ganglion cells in the same plexus

In the latter the burping and/or burning stimulates vagal afferents (sensory fibers to midbrain) that reflexively stimulate vagal efferents (motor fibers) from the midbrain that result in GE junction smooth muscle contraction. However, some of these signals may crossover to fibers to the heart. Given the very close proximity of the GE junction and cardiac ganglionic plexi in the epicardium (outside surface of the heart), this crossover (kind of an "innocent bystander" phenomenon) simultaneously stimulates a slowing of the HR.

This crossover phenomenon happens all the time, e.g., HR increases with swallowing, HR decreases (and facial muscles contract) when face is immersed in ice water, etc., and is due to the fact that motor control passes from voluntary (skeletal), i.e., chewing and swallowing, to involuntary (autonomic) in the upper esophagus. This is presumably why swallowing a cold fluid can trigger AF, as it did for Akeem Olajuwon during an NBA game. That's why IMHO #2 is the more likely explanation.

This question of epicardial vagal ganglionic plexi and GERD is intriguing.

I've posted previously on some of this in CR Session 50 at
http://127.0.0.1:4664/cache?event_id=61460&schema_id=2&q=Pachon+2005&s=w9ZB1QpccmkGxgbM2sM9NX8lw0E

It would appear that these AF nests, as described by Pachon in the above cited article, might be central to the genesis of LAF. In fact I wrote to him about the one patient in his article in whom AF could be elicited but who had no history of AF. In his response he stated that this patient had strong vagal tone. One recent Cleveland Clinic ablatee asked Dr. Natale about vagal reflex sites (?= AF nests) and Dr. Natale responded that he thought they were related P cells (=pole cells = pacemaker cells).

If it ever comes to that, you would probably be in the ablatee group with extra pulmonary foci that experience a 99% success rate with PVI.

PC

I did not realize either that PPIs would act to improve K+ balance.

An interesting survey question would be to try and discover how many people on PPIs have noted some improvement in PACs and/or LAF (and how many have not). Of course at first blush we might only be re-demonstrating that treating GERD improves LAF, but if the numbers were significant, we might try looking a little harder at PPIs and their effect on K+ balance etc.

My pediatric career was spent convincing patients that they did not need medication as often as they thought they did and it is ironic to me that I am worried about going off of the PPIs, but my instinct is that they are playing some role that is more helpful than only treating the mild GERD symptoms that I had.

BillE

While reading these exchanges and thinking about them later, i believe i need some clarification about the terms physiologic and pathologic. I googled them both and what i came up with is that physiologic refers to the normal workings of the body, while pathologic seems to refer to disease processes of one kind or another.

PC, are you using these terms as opposites? Are they true opposites?

Is aging physiologic or pathologic? Lone afibbers are most often over 50 [not always, of course], is why i ask this.

When you call LAF physiologic, do you mean that it is due to some normal body process? [Aging, maybe?]

We have often said here and in the regular bb that afib is a symptom, not a disease in itself, and that LAF is a symptom of some disease processes not yet detectable by modern medicine. How do these terms relate to that concept, if at all?

All my life people have said i ask too many dam questions, and i have to believe there is some truth to that. Never have been able to help doing it, though.

PeggyM

Hi Peggy,

Don't ever apologize for asking questions. Although Mark Twain once said, "better to keep your mouth shut and be thought a fool, than to open it and remove all doubt," I subscribe to the old Chinese proverb - "He who asks a question is a fool for five minutes; he who does not remains a fool forever."

Your question is an excellent one. The short answer is: Physiologic and pathologic are not opposites.

As you stated pathologic means caused by a disease. What is that? Many purists would not classify a genetic abnormality as a disease, preferring instead to call it a disorder. But this is denying the fact that all disease in the ultimate analysis is, at least partially, of genetic origin. Clearly, physiology (normal and abnormal) is determined by genes. However, I'm using the term disease to mean age related changes, changes would one not expect to see in middle age, although, depending on genes, the dividing line is not clear cut. At what age does the onset of dementia

cease to be Alzheimer's and become senility? So, even though they are not opposites, that is not a bad way to think about them. (long answer)

I believe that if one were to graph number of patients with AF on the y-axis and age at onset of AF on the x-axis, you would get a bimodal curve. One with a mean at 50 years and the other with a mean at least 20 years higher. This would imply two different populations. But what differentiates the end of one and the beginning of the other? For me age at onset of AF over 60 years of age should not be considered LAF. Shortening of the AERP is vital in the genesis of LAF and AERP increases with increasing age.

I suspect that ultimately differentiation of LAF from pathologic AF will be done via a scoring system that will incorporate age at onset, blood pressure and other anthropometric measurements, GERD or not, intracellular Mg++, ?MVP, thyroid disease or not, etc. Indeed an objective, easy-to-administer test for vagal tone may become available in the future. I don't consider one time episodes of AF associated with holiday heart syndrome, diuretic use, etc., to be LAF. However, this might represent a group at higher risk for developing LAF or pathologic AF.

As you know, I feel that development of risk factors for pathologic AF (hypertension, diabetes, age, ..) in one who has already been diagnosed with LAF may/will eventually result in pathologic AF. LAF may precede appearance of some of these risk factors.

"Latent Arterial Hypertension In Apparently Lone Atrial Fibrillation"
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=16177847&dopt=Abstract

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Is that coincidence or is there a connection? Most MDs (see above article) think that there is a connection and that LAF is just early pathologic AF yet to be diagnosed with structural or organic heart disease. The Mayo Clinic study (ref #44 in the article) clearly shows that this is not the case. And that in a nutshell (high magnesium nut only) is why I wrote the article.

PC

So is it correct to say that in your estimation, LAF [but not pathological AF] is the product of an abnormal physiology, that is, a genetic disorder?

PeggyM

Both LAF and pathologic AF are genetic disorders. Indeed there may be one set of genes that govern onset (shared by both LAF and pathologic AF) and another that govern maintenance. Furthermore, the expression of these genes is to some extent determined by environment - diet, exercise, ...

It's just that IMHO pathologic AF is due more to age related changes, which we call disease.

PC

Aloha PC,

Regarding the AF v. LAF question from my own point of view, I've got to say that when last checked (and, indeed, when often checked) my BP is kinda in the region of 140-50/80-90. As such, would you say that my AF episodes are rather more towards AF as opposed to LAF? (And this mild hypertension in spite of eating celery most days, mostly avoiding salt, no processed food, moderate alcohol, reasonable exercise levels etc.) I'm accordingly contemplating trying valsartan 40mg caps one a day. My only concern as a quite obviously vagal AFer is the possible vagotonic effect of the valsartan as regards its effects on potassium levels. My serum K is typically quite high at 4.8/4.9. OK, I know you aren't MY GP, but given your background and obvious grasp of many of the issues involved, do you think positive effect of the valsartan on my BP will far outweigh the possibly albeit slightly negative effect upon my vagal tone (as in increasing it....)?? On a more general note, is it possible or even likely that in my case lowering BP will reduce both

ectopy and AF episodes?

I'd be most pleased to hear/read what you think.

Kind regards (and mahalo for all your ongoing endeavours!),

Mike F.

PC - would you classify the genetic disorder as one being born with the defective gene or that a polymorphism occurred as a result of lifestyle insults or some of both? Various opinions on percentages - but some say 75% of gene expression is modifiable with the remaining fixed and unrepairable. Garry Gordon MD is a strong proponent of repairing the 'broken' gene factor and I know Jeff Bland PhD thinks that we can definitely modify gene expression to create health and has proven his theories with his functional medicine approach. This latter would seem to be what many afibbers have been able to accomplish through nutritional support and intervention.

Jackie

While relief from afib is certainly that which we have all been seeking at one time or another, I would hate to see wholesale recommendations for PPIs given the downside that reducing or blocking stomach acid is found to increase risks for pathogens to flourish. Given the escalation of E.coli outbreaks, we need all the stomach acid we can get to help fight off invaders like E.coli, C.difficile – note reference #4.

Certainly in cases of ulcers or esophageal erosions, modifying stomach acid during healing is obviously critical, but even then, PPIs aren't the only solution. The use of DGL is safe and effective.
http://www.doctormurray.com/articles/pdfs/DGL.pdf

Jackie

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Mike,

Perhaps high vagal tone has been given a bad rap. I daresay any nonLAFer would be wanting more of it, since it is an independent prognosticator for longevity. In fact vagal tone may be at the heart of the difference in mortality between LAFers and the normal population (ref #44). At the very least, I would say it is more important to treat the hypertension than worry about any possible concomitant increase in vagal tone.

Valsartan is an ARB (angiotensin receptor blocker) and is IMHO the best choice of drug class for treating hypertension, at least for an LAFer. Not sure I'd include such an individual in the category of LAF and there are many experts that do not so include, but that's another complex question I'm not prepared to answer.

I've experimented with an ACEI (lisinopril), even though I don't have hypertension or prehypertension, and it seemed to aggravate my LAF. Angiotensin II type 1 receptors (increased in left atrium in LAF but not in normals or those with CHF) mediates atrial stretch and associated remodeling (upregulation of the funny current).

I don't remember your height and weight, but I do recall that you are a urinary Mg++ waster. If natriuretic peptides were/are causing this, then you should be hypovolemic with a low BP, albeit possibly hyperresponsive to stress, excitement, etc. If your BMI is significantly over 25 then you shouldn't worry about increasing your vagal tone.

Lowering BP over time should reduce venoatrial stretch and hence PACs. It should also retard the age associated changes that cause pathologic AF.

But, like you said, I'm not your MD and this is not medical advice. I'm retired. Having a good time is now my job.

PC

Jackie,

Genetic disorders (including polymorphisms) are predominantly a congenital thing. Certainly lifestyle contributes to the degradation of genes and cellular function but I think it contributes much more to their expression or not. For example, an individual with a strong family history of heart disease can sometimes overcome this and live to a ripe old age by diet and exercise. On the other hand, physico-chemical factors can trigger certain latent genes into causing disease, e.g., solar exposure and melanoma. There's not a mutation only the turning on of a switch. Control of the latter type factors in LAF are probably what you're citing.

LAFers are different out of the gate. It just takes quite awhile (more for some than for others) to finally express LAF. IMHO it's not a mutation or broken gene but an expression of a gene already there that was awaiting a certain environment to announce its presence. And there appear to be myriad genes that are involved in the LAF story, some more prominent than others. Indeed there may be one group that impact initiation of episodes and another group that concern themselves with maintenance of AF. LAFers probably share the former with pathologic AF but diverge from those with pathologic AF on the latter. After all, PVI is equally efficacious for LAF v. pathologic AF.

Ablation of vagal reflex sites improves the efficacy to 99% (Pappone's figures). I have a hard time believing that those with pathologic AF have vagal reflex sites or that ablation of them, if they did have them, would be so successful. Because PACs play a larger role in the genesis of LAF v. pathologic AF, ablation of all such sites should be more efficacious. But the maintenance (arrhythmogenic) substrate of LAF remains unchanged postablation. Fortunately as we age and avoid the damage of recurrent episodes this arrhythmogenic substrate recedes.

I feel strongly that my family history of pathologic AF (dx after age 70 in both parents) and my LAF dx at age 51 mean that my endurance sporting history fast forwarded the expression of AF by offering my genetically determined "initiation substrate" (PACs) a physiologically modified maintenance substrate. Hopefully the elimination of PACs by the PVI and the pursuance of a "good" lifestyle (retarding development of a pathologic substrate) will enable me to maintain NSR.

PC

Many thanks for the input.

My height is 6' 4" and BMI is 29. I intend to reduce the BMI to 25/26 over the next 6 months. Although my BMI is obviously a bit high, you wouldn't think me fat if you met me - waist is 36" and chest 50". 'Wingspan' is 78". Not many folks would have me over in an arm wrestle! My CoG is high and my legs are a bit lanky and skinny but strong enough.

I'll give the valsartan a try and see what transpires. You know, as an aside, the thing I tried which gave me the biggest batch of ectopy I've ever had in one evening was a tablet of spironolactone..... Similarly, when I had the MgSO4 IM injection as part of my 24hr Mg-loading urine retention test (result mild deficiency), I had a wonderfully calm day with no ectopics (at least that I was aware of) but i had a hellish evening of them afterwards....

Kind regards and thanks again for taking the time out from your hectic schedule of relaxation and leisure to answer my questions!

Mike F.

I think an opened minded approach to all improvement stories is mandatory. I would love to find a natural way to completely cure my heart, but because I seemed to worsen on supplements and PPIs are possibly playing a role in my improvement, I believe they also need to be considered. I also understand that my improvement could be purely the natural course of the condition for me and/or the diet changes I have made.

The number of people contributing to this board and ending up with ablations suggests to me though, that we have not yet found the answer.

I was extremely impressed with this month's Afib report and the survey reported in terms of the amount of information derived and the numbers responding. I was disappointed though that there was no survey data for supplements taken prior to the procedures.

The faithful contributors to this board, particularly Jackie and PeggyM have played a vital role in making the board popular throughout the world, but if we don't collect data on all the stories out there, this beast will continue to run rampant.

I believe also that many if not most of the chronic disease afflicting the developed nations are greatly worsened and often caused by diet and lifestyle and can often be cured by improvements in the same and it greatly distresses me that medical research in America is mostly funded by drug companies and appears to be basically new drug oriented.

Having said that though I also spend a fair amount of time, in this phase of my life involved in the medical affairs of poor nations and I am extremely impressed by the strides we have made in the developed nations in terms of vaccinations, antibiotics, and the acute treatment of trauma and cardiac or respiratory failure. Much of what I see in the third world is never seen (anymore) by a physician practicing only in America.

Bottom line for me is that LAF will only become a condition of the past if the research on cures looks in all directions and I believe we should continue to use the power of the internet to turn multiple stories into meaningful statistics.

BillE

BillE,

Your suggestion about gathering data on the effect of PPIs on PACs is commonsensical. Indeed I was trying to do a similar thing wrt PACs in the tall, esp. those that participate in endurance sports. My plan was to attend both the start and the finish of the Honolulu Marathon to achieve concentration of the target group. However, the HM authorities forbade this, citing liability and other issues. So much for the goal of improving the human condition, something in which we should all be interested.

Then I thought about targeting basketball players. But attaining sufficient numbers would be difficult, even assuming

permissibility.

The real problem encountered when tackling PACs is obtaining objective data. This requires something more than personal impression. They must be measured by an instrument and this reduces to either a Holter monitor and/or a Polar HR monitor. Getting data over a 24 hour period, the usual reference standard, is quite time consuming, esp. if statistically significant numbers (and results) are to be obtained => about 50 individuals minimum. Then there are always confounding factors. What's a PAC and what's artifact? Getting a control group with which to compare may also present a problem. Even if one does establish a correlation, this does not equate to cause and effect.

Getting such a study out there is somewhat difficult. I tried about 10 medical journals before getting the green light on my article. Most of them want multi center studies on something, not a patient survey. Someone has to finance the former and that translates to Big Pharma. There's IRB (institutional review board) meeting that must approve any study involving hospital patients and/or their medical records. I'm sure you know the drill. HIPPA and other federal regulations preserving patient privacy appear to supersede any benefits that might accrue from such efforts, even amongst willing patients.

So, in short implementation of such suggestions presents a formidable challenge.

PC

I agree the task is formidable, but I think that there may be a loophole that at least lets us open some lines of reasoning. Quality of life surveys seem to be gaining some credibility and there are large groups of people who have decreased quality of life found in the MVP and AF chat groups. Although Holter monitoring will add statistical power to any study, I, for one, find my quality of life to be markedly different when I am having PACs (much worse with AF) then when I am not. When I am free of them, I feel great and when I have had a rough night, I am sleepy and somewhat depressed, fortunately for me I only occasionally have them during the day. The PACs and the AF also provide a fairly quick feedback loop as opposed to the course of ASHD etc. where you need to track for some endpoints such as MIs.

For example, I just had a root canal and although I asked to have the epinephrine removed from the lidocaine, the dentist forgot. I had two days of more PACs than I have had ever. Now because it is only a single story, it suggests a connection but is far from conclusive, but if we collected 20 similar stories, we have the ability to give the idea some weight. (I am actually not positive there is connection because epinephrine is rapid acting and I had no PACs noted during the procedure or actually until that night.)

One last thought on physiologic vs pathologic, if a male lives long enough the odds are very high that he will get Prostate Cancer, this statistic is certainly increased with a family history. Despite this common side effect of living a long life, I would never think of prostate cancer as not being pathologic.

As I go through my second holiday season, not drinking and asking my hosts what a dish is made, I find it hard to believe that my new compulsive and somewhat hypochondriacal behavior is not pathologic in itself.

Just some thoughts.

BillE

BillE,

Thank you for your continuing contributions to the topic at hand, esp. the anecdotal information.

In your most recent post you again mentioned MVP and AF. You had previously mentioned that you had two sisters with MVP. It would be interesting to know what a TEE (trans esophageal echocardiogram) on you might show. You really ought to read the full article, as well as visit the CR Proceedings at http://afibbers.org/conference/index.htm and read Sessions 14A, 14B (see abstract of article on Mg and MVP) and 25.

It has been well documented that epinephrine and catecholamines in general deplete serum magnesium primarily via

urinary Mg++ wasting. Your history of stress related episodes and episodes that appear out of the blue suggest, at least to me, that your most recent flurry of PACs many hours after your encounter with the dentist may have been related to low intracellular Mg++, aggravated by catecholamines.

In the normal population the intracellular Mg++ concentration is more than 20 fold higher than the extracellular concentration. If the IC component is already low, then it will be even more difficult to staunch the leakage across this gradient, since Mg++ is required for any ATP pump activity – kind of a Catch-22.

This process of post dental Mg++ equilibration with subsequent and ongoing K+ leakage (ratio of 30:1 EC v. IC) might have required many hours, and manifested at night because midnight represents the diurnal nadir for blood K+, not to mention vagal tone. Low blood K+ is certainly associated with ectopic activity.

Several posts back I suggested the possibility of a survey on intracellular magnesium in selected LAFers, specifically those with MVP, orthostatic hypotension, leg cramps, migraines, etc. But no one has expressed any interest in this. Unlike PACs and their measurement, measurement of intracellular Mg++ (at the heart of the potassium problem in many if not most with LAF) might represent an easier route and a more definitive correlation. It might more readily explain the apparent potassium shortfall correlation as well as many other LAF symptoms.

PC

BillE - I can relate to your dental experience with epi in the local. Very early in my AF career and before I had even begun to think much about triggers or any other type of biochemistry, I had RCT and almost immediately upon introduction of the local anaesthesia, my heart set off on irregular gallop. I became short of breath and had to sit up from the reclining position. I thought I might have to reschedule the appointment. Fortunately, the tachycardia did not turn into AF and settled down enough to allow the endodontist to proceed, but I was a bundle of nerves over the potential with my heart (not the procedures).

Fortunately, I've only required a couple of other minor dental procedures in the past 15 years, but always reminded them not to use the epi.

I worked in a dental office for 22 years and my operatory was right next to the dentist's so I could hear all the interactions. Most patients typically don't have the tachycardia reaction to local anaesthesia that I did. Only after afib had entered my life was I so sensitive to the epinephrine.

Jackie

PC,

Thanks for posting this interesting CR topic. I've been pretty busy so have neglected reading.

Bill,

One poster who has had the most success keeping his afib in remission by suppressing GERD is Dean. He also swears that by eating 50g of natto food at least 5 day/week, his PAC's reduced to zero. You might wish to search on Dean and natto in the regular board and also in the archived boards.

Another poster, Fran, who did not react well to supplements did get relief with Epsom salt baths as another means of putting Mg++ in the system. You might consider this.

On an unrelated topic - but showing you can be surprised, I suggested my wife try taking 5-Htp for her migraine headaches after a post on this by Jackie (thanks!). She did & also told her functional MD about what she was doing. The MD suggested she take L-Tryptophan, and she gave her a Meyer's cocktail injection (see below). My wife has continued with the supplements and has had a total of 2 Meyer's cocktail injections over a one month period.

Now here is the surprise. She went into menopause at 38. She is now 53 and has had minor cystocele (urinary

leakage) issues for several years. Since she started the supplements and the Meyer's cocktail, this cystocele issue has completely resolved. Of course the question is - which supplement is the "active" one and we don't know she started with all of this at about the same time. BTW this has not resolved her hormone related migraines, however she generally feels much better.

Here is info on the "generic" Meyers cocktail (I don't know the specifics of her injections):

http://www.diagnose-me.com/treat/T16780.html
The Meyer's Cocktail is an intravenous vitamin and mineral protocol developed in the 1970s by a physician at Johns Hopkins University in Baltimore, Maryland. It contains magnesium chloride hexahydrate (5cc given), calcium gluconate (2.5cc), vitamin B2 (1000mcg/cc; 1cc given), vitamin B5 (100mg/cc; 1cc given), vitamin B6 (250mg/cc; 1cc given), the entire vitamin B complex (100mg/cc; 1cc given), and vitamin C (222mg/cc; 6cc given). Solutions are further customized by other doctors. The solution is slowly injected over a 5-15 minute period.

I've wondered is if this "cocktail" might be of benefit for afibbers, and have any different/better effect than the common MgSO4 injections for magnesium. Just a random musing.

George

PC states above:

"Several posts back I suggested the possibility of a survey on intracellular magnesium in selected LAFers, specifically those with MVP, orthostatic hypotension, leg cramps, migraines, etc. But no one has expressed any interest in this. Unlike PACs and their measurement, measurement of intracellular Mg++ (at the heart of the potassium problem in many if not most with LAF) might represent an easier route and a more definitive correlation. It might more readily explain the apparent potassium shortfall correlation as well as many other LAF symptoms."

FWIW, my leucocyte Mg was right at bottom of range when tested 3 years ago at 44 (range 44-62 from memory). Interestingly - and after many years of gobbling Tums - my IC Ca was also right at bottom of range (I seem to think you had a similar outcome yourself IC-Ca-wise and with similar TUM-guzzling history in this regard). I also get quite a lot of muscle twitching and jumping in my eyes, eyelids as well as various parts of my arms and legs etc. When falling asleep I often jolt quite violently for no reason. I also get cramp quite readily in my calfs, second toes (!) and under my chin when yawning! And I startle VERY easily. All that said, my serum K is usually towards upper range at 4.8/4.9. (Lordy, just had a run of 6 fastish regular ectopics as I typed that though!) Whilst leaning towards hypertension rather than hypotension, I can feel light-headed when jumping up out of the chair quickly, but then I'm guessing pretty well anyone and everyone can. I don't suffer from migraine headaches but I do get quite a lot of recurrent nasal/sinus problems and am on a daily basis clearing my throat as a result of a post-nasal drip.

Cheers and seasonal best wishes,

Mike F.

Your theory on the possible reason for an increase in ectopy hours after the injection (actually there were two nights of PACs) is very interesting and makes a lot of sense. I have also gone on to read the conference proceedings you suggested.

I am very impressed with the logic and knowledge presented. My concern about Mg supplementation is again anecdotal. The only episode of AF that I had that required cardioverting occurred during the time that I was faithfully taking Mg supplementation. I was also taking carnitine, B complex, CQ10, and Omega 3's. I have also noted with interest the number on this board taking Mg supplements and how many have gone on to have ablations. I have also watched over the years as some supplement, such as beta carotene, seems to make sense (and seem very safe) until we get enough data.

I think we have to agree that for significant numbers these supplements don't work too well and I think we have accept the fact that for some they might even worsen the situation.

It is also possible that people who continue to follow the board tend to be those who are not doing too well and that possibly large numbers have done very well and "graduated". I don't think we can actually draw too many conclusions on the clinical effect of Mg supplementation until we collect more data.

Please comment.

Note: my real name is Lee, Bill is just a nickname and life got a little more complicated as I tried to make it BillE (as there is a growing number of Bills on the board). For simplification, I am going to sign off from now on as Lee.

Lee

Lee - while the testimonials here and the other BB are anecdotal, they simply confirm what the experts in the field of magnesium research have said for years about the importance of magnesium to cardiac functioning. The fact that at least 80% of the population is magnesium deficient certainly makes their conclusions plausible. Mildred Seelig, MD, MPH - world recognized magnesium researcher for over 40 years has written a whole book about the consequences of magnesium deficiency - about 400 pages including references. (The Magnesium Factor). She and her fellow researchers -including the Alturas and Dr. Durlach, agree. (lots of study references at the website of www.mgwater.com)

Mike F posting here is obviously suffering from lack of magnesium as his symptoms are classic esp. the muscle fasciculations.

Now the interesting thing about magnesium deficiency is that in some cases, one never can replete and others may take a few months or several years. A lot depends on what causes a drain on the stores.... like constant stress, continual sugar or alcohol consumption, malabsorption, etc. (long list). It's like trying to fill a sink to overflowing when the stopper is cracked open a bit. The sink will never overflow. The same goes with magnesium deficiency.....according to Dr. Seelig.

Once optimal magnesium IC stores are achieved, then the addition of potassium makes sense and in many cases, as the testimonials indicate, afib is relegated to the back burner... surfacing only very occasionally if all conditions are right.

Additionally, I found that adding taurine to the mix, helped greatly - thanks to George Eby's website referencing taurine which was a reminder of what I had heard about 10 years earlier by one of the pioneers in alternative therapies - taurine was essential to cardiac functioning. Taurine acts like a traffic cop directing the electrolytes in and out of cells as required. Many of the people on the list have indicated taurine has made a tremendous difference in eliminating or greatly reducing afib.

So, rather than just magnesium, it is magnesium first, then potassium and taurine or magnesium and taurine and then potassium.

Obviously, because of the biochemical individuality factor and the extent of depletion in any of these nutrients, everyone will respond differently... such as the case of Mike and Fran and others, who are unable to take supplements at all to correct the magnesium deficiency. While Fran did it with foods, not everyone is willing to go to the special effort or discipline.

A final comment on the taurine issue, Eric Braverman, MD, in his book "The Healing Nutrients Within" says: "Some patients with mitral valve prolapse..... have been found to have depressed levels of heart muscle taurine. This inborn error underscores taurine's importance in the heart and suggests there may be some cases of the common diagnosis MVP that respond to taurine therapy."

Leo Galland, MD FACN, (Director of Foundation for Integrated Medicine) on the other hand, writes about the role of Magnesium deficiency in MVP. So, it could be a combination. http://mdheal.org/magnesiu.htm

Now, just to make this post a bit longer, I'd like to offer my own testimonial based on recent experiment of my own.

I take at least 600-800 mg. magnesium glycinate plus food content. My IC levels were on the low side and I've tried to maintain optimal before and after PVI in 03. Recently, I decided to cut back to see if I had managed to maintain IC repletion. After about a month of taking only 300-400 mg a day, I started to experience muscle aches and pain. First, the dull aches in my upper back muscles became persistent. Then I began to experience calf leg muscle cramps while sleeping. Then I progressed to calf and thigh muscle cramps just when sitting at rest but pointing a toe downward. During the night, I was getting ectopy and short runs of tachycardia.

I then took 400 mg. magnesium glycinate at bedtime and slept well with no cramps; the pain in the upper back was gone the next morning; and after the next daily dose of 400 mg. more of magnesium, about 95% of the muscle aches are gone.

I've proven to myself that I do not retain magnesium efficiently. I'm sure that same scenario could be duplicated with testing to verify low IC levels.

The bottom line is that some individuals, even though taking magnesium, may never reach optimal IC stores or sustained repletion. People who have had ablations and are still complaining of ectopy need to address the need for continual attention to all three nutrients - magnesium, potassium and taurine. Non-ablated individuals may find success but it may take heroics in the supplement department or strict food intake and it will be an ongoing priority to take magnesium in and eliminate magnesium depleters.

Living with Passion in NSR

Jackie
8 years vagal afibber
Age 70, PVI - '03 (Natale)

Hi Jackie,

Great post.

I'd read Dr. Galland's article on Mg-D and MVP sometime ago (? after your CR Session 25 on MVP and magnesiuim deficiency) and used many of the same references in my article. It's amazing how rereading an article reveals many little tidbits that were missed on the first read.

Unfortunately I personally believe that Dr. Galland is on the wrong track in trying to equate Mg-D as the cause of MVP. IMHO MVP is part of MASS (mitral, aortic, skeletal and skin abnormalities) disorder (genetic), while Mg-D and all of its symptoms (MVPS = mitral prolapse syndrome), including LAF are secondary. As far as I can determine, no one has implicated natriuretic peptides (ANP and BNP) in this process. These latter cause not only urinary Na+ wasting but also urinary Mg++ wasting.

MVP is often associated with mild (trace to 1+) regurgitation. Over time this leads to well documented mild atrial stretching, esp. of the very venoatrial area in the left atrium where natriuretic secreting cells are located. The stretching triggers its release. The stretching of this same area is responsible for upregulation of the very ion current that determines pacemaker activity and the ectopy that triggers AF.

PC

Thanks PC - for the critique. We are probably closer to the afib story than Dr. Galland - at least that's my educated guess.

On the BNP, I recall seeing that referenced before you addressed it in your article, but I can't for the life of me recall where it was. I just remember thinking... hmmmm that should be something worth investigating further and then I got busy with other things - probably wouldn't have understood it anyway. ;)

BTW, very early in my AF diagnosis when I switched to a CCF cardiologist who specialized in rhythm disturbances, he noted on my echo that there was a very insignificant mitral valve involvement. He said it was so trivial it wasn't worth noting on the record; could not hear it (but then everyone hears differently). It didn't change in all my years and wasn't mentioned on the last echo.

Be well,

Jackie

Hi Jackie,

Thank you for your very informative post. As you can tell from my posts, i believe also that anecdotes can be very powerful. I also know that all pathologic disease processes that wax and wane can lead any of us to large number of conclusions, some of which are correct and some of which are not.

There is the ability with this board to take the next step regarding all the healing methodologies discussed here and that is to collect the stories in sufficient numbers to create meaningful data. As George and PC have shown, meaningful data and well written presentations can make it into the medical literature.

I believe one step that could be very informative would be to send a follow-up questionnaire to all who participated in Han's recent Ablation survey. I am very interested in knowing what supplements, if any, all of those who went on to ablation were taking prior to the procedure. It might be interesting to ask what meds also, but I think asking that question would decrease the return rate and we know the available meds are not yet miracle drugs.

Hans has suggested that our "List" project should start with a letter to each on the list to see if their success has held and that project has returned to an active phase.

Lee

Lee,

I have to agree with your assessment about supplements and their ultimate impact upon subsequent ablation or not. But I don't think a survey is necessary to validate this impression. Everyone that reads the BB and/or the CR is aware of the value of supplements and yet "the List" is rather short, relatively speaking.

I personally feel that those on "the List" either jumped on the LAF before any measurable atrial dilatation developed or have a variant of LAF unrelated to MVP, i.e., perhaps more related to controllable factors governing Mg++ and K+ homeostasis. Who knows? But MVP related LAF is relentless. One will have greater success controlling hypertension than controlling the increase in LA pressure due to MVP.

Just more food for thought.

PC

Most of my life I've had all the symptoms of Mg deficiency (twitching, cramping, migraines, constipation). For five years, since a-fib appeared, I've been supplementing with Mg, but the amount has been slowly decreasing to avoid soft BMs. For the last few months very soft BMs continue in spite of no Mg supplementation. Coincidently, a few episodes of cramping have occurred which is suggestive of Mg deficiency reappearing. Any thoughts about non Mg related reasons for the soft BMs?

I would certainly participate in a survey to determine IC Mg.

Wil

Wil, when you figure this out please let me know, because i am having very similar problems myself. I have even resorted to taking immodium with my Mg glycinate to keep things stable enough for the Mg to get time to be absorbed. Those leg cramps are vicious. I need to be taking at least i mg glycinate tab a day to keep them away.

PeggyM

Wil,

A few thoughts, for what it is worth.

Since stool softness issues are related to oral supplementation with Mg++, how about either injection of or transdermal supplementation with $MgSO_4 \cdot 7H_2O$ (epsom salt bath) Mg chloride. You might have your MD daughter look into the Meyer's cocktail that I posted about above. It includes mag chloride and there are many positive anecdotes about its use.

I've read that too much sulfate from the epsom salt baths is not good over an extended period. However, food grade Mg chloride is used in making tofu, so may be available in quantity for soaking purposes (in a bath).

Here is a little PDF on MG chloride:
http://www.arthritistrust.org/Articles/Magnesium%20Chloride%20Hexahydrate%20Therapy.pdf

The Mg chloride used in tofu making is known as nigari (almost, but not 100% Mg chloride). Here is a quote for it @ $1.89/pound US: http://www.simply-natural.biz/Nigari.php

Where I live, here in Colorado, they put massive quantities of mag chloride on the roads as a deicer. Maybe naked snow angels in the slush would work instead of bathing in it (but a bit chilly). However, it unfortunately is probably not food grade.

George

The reason I brought this up here is that PC suggests in his message above (12-13-06 17:46) that he is experiencing a similar problem. This may be a consequence (possibly desirable) of long term Mg supplementation, but, if so, it eliminates the only feedback mechanism we had to set Mg dosage.

I am sure that the symptom appeared while I was supplementing with WW, so the glycinate isn't likely a factor.

Wil

Wil - do you consume a considerable amount of fruit or anything else that would contain fructose? What you describe is often seen with too much fructose consumption not only from fruit sources but anything that contains high fructose corn syrup.

If that's not the case, consider also a type of parasite - easy to get if one eats considerable raw foods. Remedy is easy - add daily drops of GSE
(Grapefruit Seed Extract) by Nutribiotics - kills pathogens and also tends to create a minor degree of fecal matter concentration.

If all else fails, the addition of capsules of apple pectin is an economical and easy remedy. Any health food sure should have this. Or another solution is to take a small amount (200 mg. or so) of calcium citrate or calcium carbonate which competes for magnesium and tends to be constipating....a small amount could balance out supersaturation of IC stores if that may be the case (which I doubt) as magnesium leaves the cells fairly easily.

As you know, what you really want to avoid is losing electrolytes through a diarrheal type symptom.

On the magnesium supplementation, do you take the patented form by Albion Labs - magnesium glycinate? That's formulated not to create bowel issues unless one gets to fairly high levels.

Just some random thoughts. I forgot to add - it's fairly easy to reach bowel intolerance with the WW.

Jackie

Jackie, does the Doctors Best Mg glycinate from iHerb use Albion? That is the one i am using.

PeggyM

PeggyM: The back of your Doctor's Best label notes the glycinate is supplied by Albion.

Jackie: I normally ingest two: apples, or pears, or peaches a day. I used to drink fresh squeezed grapefruit juice, but haven't done that roughly since the problem began. Perhaps the fresh squeezed grapefruit juice, with the smaller seeds included, was suppressing or eliminating some parasite. I eat nothing containing corn syrup. I do have the Albion Mg glycinate and had also considered the calcium idea. I think the first step will be to restore the grapefruit juice and see if the problem goes away. I'm sorry to hear about the WW. It was easy to include it when cooking.

Wil

Aloha Wil,

There appear to be so many LAFers that derive considerable benefit from Mg++ and K+ supplementation, be it WW (waller water = aqueous Mg++), magnesium glycinate, MgSO4 or MgCl2; p.o., bath, IM or IV. Why is that such continuous/ongoing supplementation or replenishing is required to prevent episodes? Clearly there are a number of LAFers, Hans being one, that run borderline low blood levels of K+. Any circumstance that negatively impacts this can trigger an episode. But there appear to be an even larger subgroup of LAFers whose blood K+ routinely runs above 4.5. What is their problem? (Note: Mg++ is required for the ATP pump that maintains both intracellular K+ and Mg++) The GI tract for the most part is a very nondiscriminating organ for disposal of specific ingested nutrients. The kidneys are much more discriminating. Diuretics but not stool softeners are associated with triggering AF episodes.

So, it seems only natural to me to look to the kidneys as the leading culprit in this prominent subgroup. The first two CR Sessions delved into the K+/Mg++ question and natriuretic peptides were recurring players. At that time we thought that ANP was desirable because it caused retention of K+ at the expense of urinary Na+ wasting (to maintain electrical neutrality). However, it is now known that both ANP and BNP also cause urinary Mg++ wasting. Is it just a coincidence that natriuretic peptides are increased in LAFers?
B-type natriuretic peptide levels in patients with paroxysmal lone atrial fibrillation. (5/2006)
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=16715186&query_hl=2

Plasma brain natriuretic peptide concentrations in patients undergoing pulmonary vein isolation. (11/2006)
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=16740921&dopt=Abstract

"Median plasma BNP concentrations were significantly higher in patients with lone AF than in controls (patients with supraventricular tachyarrhythmias)"

and in those with MVPS?
Atrial natriuretic factor: a possible link between left atrium, plasma volume, adrenergic control and renin-aldosterone in the mitral valve prolapse syndrome.
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=2952778&dopt=Abstract

Furthermore, dehydration induced MVP can be seen in 10% of males that are not tall and thin, features typical for MVP. This is called functional MVP and is associated with greater elevation of BNP than in organic MVP. Could this explain in part the predilection of LAF for endurance athletes?

It also appears that natriuretic peptides levels decrease after successful cardioversion and/or ablation and this decrease is in fact a predictor of procedure success. Could this be the flip side of the "AF begets AF", i.e., NSR begets NSR?

What I'm implying is that magnesium loss via natriuretic peptides may be the primary determinant or final common pathway by which LAF manifests for a large subgroup, perhaps the majority, of LAFers.

Wouldn't it be interesting to determine pre and post ablation IC Mg levels in those for whom such data is available? Perhaps another survey correlating symptoms of Mg deficiency (muscle cramps, fasciculations, migraines, etc.) with IC Mg might prove elucidating. Now that we've got limited data published (7 of 7 LAFers under going IC Mg++ were found to be low or at the very lower limit of normal), Intracellular Diagnostics (www.exatest.com) might be willing to underwrite a study on this.

Wil, you're the first volunteer on this. Great! We might be able to run a carefully crafted symptom survey and then run the test on respondents. Hopefully Hans will allow this and perhaps I can persuade Burton Silver, CEO of ICD, that another paper might result.

PC

PC, i think you may have answered this question and i am just too dense to understand your answer. I hope if this is the case you can break it down a little so i can understand it. Here goes:

How come both Wil and me are getting diarrhea from minimal doses of Albion process Mg glycinate, while still getting loud symptoms of Mg deficiency like leg cramps and eye twitches for me, and migraines for him? Apple pectin capsules and 4 grams a day of taurine notwithstanding, this has been developing over the course of about a year for me, and is getting worse.

Also, what can i do about this?

PeggyM

While considerable effort has been expended on this web site analyzing possible physiologic consequences of low Mg/K/Taurine/etc., we have ignored some basic questions: i.e. we have not determined the cellular concentration of Mg/K/Taurine/etc. in a-fibbers (except for the limited data PC quoted), and we have not determined what techniques are useful for adjusting those concentrations.

Assuming that PeggyM and I are experiencing some kind of reaction to long term oral Mg supplementation, and that is a huge assumption give the limited data, our situation has exposed some questionable assumptions we have believed in: (1) maximum oral dosage of Mg supplementation can be determined by BM consistency; and (2) maximal oral doses of Mg increases IC Mg concentration.

PC's intention to obtain more measurements of IC Mg concentration in a-fibbers is a needed first step. Since we will likely find the IC Mg concentration in a-fibbers is very low, the next step will be determining what techniques are useful for raising IC Mg concentrations. Count me in.

Wil

My personal belief, without evidence i can quote except for a general impression gained thru reading, is that an iv of one of the generally accepted Mg solutions is my best bet. Too bad i do not have health insurance, and in any case have no access [the wilds of central Maine, remember] to naturopaths or to much of anything but my overscheduled,

overworked GP. An epsom salts bath seems next best, but i fear that if i get down into a bathtub i will not be able to get out of it. Too soon old and too late smart, indeed. I think an epsom salts footbath is the most likely alternative.

PeggyM

Here are some random thoughts. As one of the 7 who've undergone IC Mg++ and was at the very lower limit of normal, I'm interested in the absorption and wasting/excretion of electrolytes. I think it has been known for some time that Wil's assumption (2) (maximal oral doses of Mg increases IC Mg concentration), has been suspect. Jackie has made previous posts to the effect that those with low IC Mg++ have a difficult time absorbing Mg++ from the gut and may need to rely on other approaches, trans dermal or IV, to increase magnesium IC levels.

Two recent studies reported: "Our studies imply that differences in our gut microbial ecology may determine how many calories we are able to extract and absorb from our diet and deposit in our fat cells."
http://www.medicalnewstoday.com/medicalnews.php?newsid=59760 (abstracts appended at end)

These studies relate to caloric extraction from food, but is it possible that gut microbial ecology may also have an effect on mineral absorption?

In LAFS XI, PC asked questions about height, weight, waist size & etc. that would lead to information about body fat/type. From the results of the survey, it was implied that LAF'er tend to have lower BMI's than normals. As studies imply that one source of LAF is chronic fitness, one might conclude that these low BMI's are a result of chronic fitness. In a recent article, "Are you an invisible fatty?" People with low BMI's are described with large amount of internal/organ fat. A question - would you expect LAF'ers to have low BMI's and large amounts of organ fat.
http://www.dailymail.co.uk/pages/live/articles/health/dietfitness.html?in_article_id=423578&in_page_id=1798

Along the lines of the above, I recently learned that the US military uses height and the difference between abdomen and neck measurements (and also hip measurement in women) to estimate body-fat percentages. Would this approach be able to identify those with low BMI's and large amounts of organ fat? I've appended some references to this approach at the end. Perhaps in future studies we could gather the neck & hip info & see if it gives any more information.

And lastly, a post by Dean on the regular board: Bacillus subtilis supplement to replace natto food? B subtillis "cures" dysentery in N. Africa for German military in WWII:
http://www.afibbers.net/forum/read.php?f=4&i=22263&t=22175

Could B subtillis help in mineral absorption - is this a possible reason for Dean's success staying afib remission? For someone who would like to try this, here is a reference to a source of B subtilis (besides natto food):
http://www.afibbers.net/forum/read.php?f=4&i=22336&t=22289#reply_22336

George

References:
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Army body fat
http://usmilitary.about.com/od/theorderlyroom/a/bodyfat.htm

Body-Fat Procedures

The Department of Defense formula to compute body-fat percentage is somewhat complicated. For males, the formula is % body fat = 86.010 x log10(abdomen - neck) - 70.041 x log10(height) + 36.76, and for females, the formula is % body fat = 163.205 x log10(waist + hip - neck) - 97.684 x log10(height) - 78.387.

Detailed measuring instructions here: http://www.armyg1.army.mil/hr/weight/r600_9_1Sept.pdf

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Nature. 2006 Dec 21;444(7122):1027-131.
An obesity-associated gut microbiome with increased capacity for energy harvest.

* Turnbaugh PJ,
* Ley RE,
* Mahowald MA,
* Magrini V,
* Mardis ER,
* Gordon JI.

Center for Genome Sciences, Washington University, St. Louis, Missouri 63108, USA.

The worldwide obesity epidemic is stimulating efforts to identify host and environmental factors that affect energy balance. Comparisons of the distal gut microbiota of genetically obese mice and their lean littermates, as well as those of obese and lean human volunteers have revealed that obesity is associated with changes in the relative abundance of the two dominant bacterial divisions, the Bacteroidetes and the Firmicutes. Here we demonstrate through metagenomic and biochemical analyses that these changes affect the metabolic potential of the mouse gut microbiota. Our results indicate that the obese microbiome has an increased capacity to harvest energy from the diet. Furthermore, this trait is transmissible: colonization of germ-free mice with an 'obese microbiota' results in a significantly greater increase in total body fat than colonization with a 'lean microbiota'. These results identify the gut microbiota as an additional contributing factor to the pathophysiology of obesity.

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Nature. 2006 Dec 21;444(7122):1022-3.
Microbial ecology: human gut microbes associated with obesity.

* Ley RE,
* Turnbaugh PJ,
* Klein S,
* Gordon JI.

Washington University School of Medicine, St Louis, Missouri 63108, USA.

Two groups of beneficial bacteria are dominant in the human gut, the Bacteroidetes and the Firmicutes. Here we show that the relative proportion of Bacteroidetes is decreased in obese people by comparison with lean people, and that this proportion increases with weight loss on two types of low-calorie diet. Our findings indicate that obesity has a microbial component, which might have potential therapeutic implications.

After I posted the above, I recall that when I discussed Mg++ supplementation, my EP suggested a product named like "Mg B". I further recall that B6 supplementation with Mg is indicated for better absorption.

A quick Google of magnesium & B6 yielded this, which is unrelated to afib, but might provide an insight:

Biol Psychiatry. 1985 May;20(5):467-78.
Vitamin B6, magnesium, and combined B6-Mg: therapeutic effects in childhood autism.

* Martineau J,
* Barthelemy C,
* Garreau B,
* Lelord G.

This article reports the behavioral, biochemical, and electrophysiological effects of four therapeutic crossed-sequential double-blind trials with 60 autistic children: Trial A--vitamin B6 plus magnesium/magnesium; Trial B--vitamin B6 plus

magnesium; Trial C--magnesium; and Trial D--vitamin B6. Therapeutic effects were controlled using behavior rating scales, urinary excretion of homovanillic acid (HVA), and evoked potential (EP) recordings. The behavioral improvement observed with the combination vitamin B6-magnesium was associated with significant modifications of both biochemical and electrophysiological parameters: the urinary HVA excretion decreased, and EP amplitude and morphology seemed to be normalized. These changes were not observed when either vitamin B6 or magnesium was administered alone.

George

George, it does not seem to be a shortage of B6 with me, because i take 2 of the NOW brand B-50's every day, one at night and one in the morning, each of which provides, among the other B vitamins, 50 mg of B6 as pyridoxine HCl.

Likewise i would expect since i am definitely fat, my gut flora are probably the fat peoples' variety rather than the skinny peoples' one. But i think it cannot hurt anything to try the B. subtilis approach. I think i will get mine from the bottle, though, rather than straight from the horses' ..... errrr..... never mind.

PeggyM

Hi Peggy and Wil,

Who knows what the answer to your question is.
However, I do know that, as we age, magnesium absorption decreases and ANP secretion increases (along with urinary Mg++ wasting) - a double whammy. A good discussion of this can be read at
http://www.mgwater.com/aging.shtml

As far as the solution, I remember talking to Dr. Burton Silver at ICD (and reporting on the BB) that maximal Mg++ absorption is obtained by taking as many different chelates as possible. Each form has its own absorption mechanism, which is easily overwhelmed, except for possibly aqueous Mg++. Therefore, it would appear that the best course to pursue might be to take not just Mgglycinate but also Mgcitrate, Mggluconate, Mgaspartate, etc. Dr. Mansmann recommended using fiber to counter any laxative effects. Imodium was high on his list as well. I've emailed some info to Peggy on this, but didn't want to clutter up the CR. Perhaps she can pass on that which she feels might be of general appeal.

George,

Your mention of Mg supplementation combined with Vit B6 is interesting. It may not necessarily be the absorption, but the biochemistry. Pyridoxine (B6) is a cofactor for 116 enzymatic reactions in the body. All of these require Mg++ as an additional cofactor. Mg++ is required as a cofactor for about 350 enzymatic reactions in the body => about a third of these require B6.

Regarding "external" body fat v. that around intestinal organs, I'd say that they both follow the same rules. In all the autopsies I performed over 30 years I don't recall any in which there was an obvious discrepancy between the two.

PC

Hi PC,

So, if I interpret your autopsy body fat statement correctly, and assuming the information posted in the story about the MRI scans is correct, someone could have a low BMI and still have a relatively high amount of internal and external body fat, is this true?

George

George,

It doesn't make a whole lot of sense to me that fat cells should behave differently in different parts of the body, at least outside the gender induced differences.

Even the author of the article, however, appears to back off a bit from this problem and offers no explanation.

"So should we all have MRI scans? Dr Little believes that the best indicators are still BMI and waist measurement - which give an indication as to how much excess body fat a person has - blood pressure and blood sugar levels, and cholesterol and triglyceride levels -which indicate how much mobile fat there is in the blood stream.

"If you're slim and there are no other risk factors involved, there is really no reason to worry too much," he says. "If you want to stay healthy you should be eating a balanced diet - including plenty of fruit and vegetables, not too much salt or sugar, high fibre and whole grain food, and not too much fat - and exercising regularly, whether or not you are fat or thin, inside or out."

I think the more important consideration is proper nutrition (v TOFI or not), i.e., who cares whether ones lifestyle is creating TOFI, when there is increased CRP, hypertension, diabetes, etc., all because of that lifestyle. Dr. Little raises a valid shortfall of BMI. A body builder can have an increased BMI, while another junk eating non-exerciser can have a low BMI. Clearly, the latter is at greater risk. Skeletal muscle has greater density than fat and leads to this dichotomy.

Another problem I have with the article concerns alcohol.

"Alcohol has been found to increase internal fat - and smoking is unlikely to help, although we've not studied this yet."

One constant I've found doing many hundreds of autopsies at a large County Hospital is that "alkies" have distinctly less internal body fat. In fact their arteries are notoriously clear of atherosclerotic plaques. This is because the majority of their caloric intake is in the form of alcohol and one does not gain weight on an alcoholic diet.

Just my opinion.

PC

PC,

Thanks!

I suppose "alkies" would therefore tend to have low total cholesterol levels. Is the reason why epidemiological studies suggest that low total cholesterol is indicative of poor health, cancer (they probably smoke, too) & etc? I'm guessing a normal eating the SAD diet would generally not have low total cholesterol (& would have plenty of atherosclerotic plaques).

George

George,

Yes, they would.

The NIH for decades tried to tie high LDL cholesterol levels to diet and was largely unsuccessful in this effort. Genes are the dominant determinant of blood cholesterol, but yet the low fat diet persists. Athletes generally have lower LDL cholesterol and higher HDL cholesterol, but their dietary cholesterol and fat is usually well above the norm.

I think the TOFI category corresponds to "cachectic obesity", relatively thin individuals with very little muscle mass (often seen in association with Rheumatoid arthritis). Their BMI is not elevated, and they eat a nutritionally deficient diet. Their genes keep them from showing much weight gain, but they are very much at risk for premature CV disease.

PC

Wil - I'm responding to this post but it actually blends in with the other speculation as to why the magnesium may be at the root of the loose/soft stools.

Your fruit intake isn't all that high so I'm thinking not much risk of the fructose intolerance.

But, I've been reading much more on the impact of gluten in sensitive people. You've commented on your own experiment and I, on mine. What is said about the gluten sensitivities manifesting either inside or outside the intestine or both prompts me conclude that in individuals who have gluten sensitivity - not full blown true Celiac where even a hint of gluten causes a huge and dramatic impact - but enough irritation in the gut lining so that, in time, there is some measure of reduced nutrient absorption. If this is the case, then (to me) it is logical that magnesium that is unable able to be absorbed would be dumped/excreted and is likely to cause the loose stool/diarrhea symptoms. I doubt that this would indicate IC saturation or optimization - quite the contrary. It could well be that inability to absorb leaves the host low in IC stores but at the same time, allows the body to dump unused magnesium as it is supposed to do when it senses extra magnesium. In some people (you and Peggy, perhaps) that may be the case.

One of my reference lists on consequences of gluten sensitivity (due to malabsorption) indicates deficiencies in calcium, iron, zinc, magnesium, potassium, selenium, vitamins A, D,E, K, folic acid, B6, B12. Many of these we try to shore up to help prevent arrhythmia.

It also could be possible in your case that steps need to be taken to heal the gut. You are on the right path thinking grapefruit seeds as a killer of parasites - but you'll need the concentrated GSE (grapefruit seed extract). It wouldn't hurt to try - and it's slightly constipating as well when taken in therapeutic doses. (GSE from Nutribiotic - iHerb or any vitamin store should have it - very inexpensive - small bottle lasts forever and it's great for warding off cold, sore throats, flu, purifying water, etc.)

Healing the gut after years of insult from circulating antibodies to gluten or dairy or any other allergen takes some time - maybe years. And, if you've read that blurb I did on the 4R's - you know the first step is 'Remove.' When we consume fruit, it's fairly easy to pick up parasites and an occasional dose of one of the effective killers like GSE or Oil of Oregano is always recommended. Along with that goes adding back in the good bowel flora via probiotics. Also very important for GI tract health.

While I'm not discounting PC's hypothesis, it could be that your issue is really with the gluten which manifests as I've suggested or a combination of both. As he says in another response about cofactors and magnesium, it just makes sense that many enzymatic and cellular functions will not be performed or will be impaired with gluten running interference.

I'm still working on the gluten update refinement and just as soon as I get the Iodine report finalized, I'll be doing more on the gluten issue as well.

This makes for interesting speculation because most likely gluten affects most of us to one degree or another.

Jackie

Hi Jackie,

If your hypothesis is correct, then it would seem the near term solution to the Mg++ problem would be transdermal or IV Mg, as the gut is not an issue with these methods.

George

"It could well be that inability to absorb leaves the host low in IC stores but at the same time, allows the body to dump unusused magnesium as it is supposed to do when it senses extra magnesium. In some people (you and Peggy, perhaps) that may be the case. "

This is not a disagreement but, i think, a better explanation. Unused [unabsorbed] magnesium does not require a dumping mechanism but seemingly provides its own. Magnesium attracts water by hygroscopic action while lying in the gut, counteracting the gut's normal function of removing water from stool.

You could very well be exactly right about any degree of sensitivity to wheat radically decreasing the gut's ability to absorb magnesium. I know from another minor trouble wheat gives me [gerd] that i do have some kind of wheat sensitivity.

PeggyM

Hi Peggy,

You and I had discussed the value of increasing intake of dietary nuts. There was a post in the BB that perhaps you'll remember on the effect of hazelnuts on cholesterol. Can't remember who posted it, but thank you. It mentioned an article entitled

Effects of hazelnut-enriched diet on plasma cholesterol and lipoprotein profiles in hypercholesterolemic adult men.
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=PubMed&cmd=Retrieve&list_uids=16969381&dopt=Citation

IMHO the reason for this effect is magnesium. In Mildred Seelig's book The Magnesium Factor she points out that the first enzymatic step in the metabolism of cholesterol requires Mg++ as a cofactor. This means that if you are Mg deficient your cholesterol level will be higher all else being equal. So, Mg is a natural "statin".

In Bill Sardi's most recent newsletter there is some interesting info on magnesium and cholesterol that relates to the above and which can be read at
http://www.knowledgeofhealth.com/report.asp?story=The%20good%20and%20the%20bad%20cholesterol%20or%20is%20it%20cholesterol%20at%20all&catagory=Drugs,%20Heart%20Disease

PC

P.S. Replenishing magnesium primarily via nuts may not be without some drawbacks. Hans has mentioned that there are a lot of omega 6s in almonds. As in all things, moderation may be the watchword. Replenish magnesium, potassium, ... via as many different foods (and supplements) as possible.

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