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SUBJECT: Hydration & Aldosterone

Aloha Fellow Fibbers,

Long time no post. Sorry for its length. I have been (and still am) on an extended vacation here in HI. In addition my computer died secondary to the ferocious corrosion. I have now been able to repair it (took forever to get the required computer part – slow boat to HI and then to China, I guess). Furthermore, I have developed a severe case of HI narcolepsy, AKA HI sleeping sickness. Symptoms include heavy eyelids and flaccid muscles 24x7. Nonetheless, I have been perusing all the excellent and erudite posts and have been especially intrigued by those about LAF and endurance sports, salt, fibrosis and, of course, my favorites Mg and K. During my respite I've continued pondering the LAF puzzle and experimenting accordingly. I have some experimental results to report and more theory. This post is directed especially to endurance athletes with LAF.

As you may know, last year I experienced a tremendously positive result ingesting 1 to 2 liters of aqueous Mg (waller water) per day (please refer to Erling Waller's recipe using carbonated water plus milk of magnesia). I personally add the juice from a lemon to neutralize the 8+ pH and avoid any possibility of hypokalemic alkalosis. I immediately went from AF episodes q36h to a 3 week interval without AF. Then I fell into the usual trap – if a little is good then a lot must be great. So I started increasing the concentration of elemental Mg in the water and decreasing the amount ingested (since the 2 AM bladder calls became somewhat tiresome). The result - BMs that were too loose. Ironically the end result was loss of K and Mg in the stool. The drop in water intake was compounded by this water loss in the stool. Over the ensuing months the magical waters of waller seemed to lose their potency. Episodes gradually increased in frequency until I was back to my usual one-week intervals between episodes (my pattern for the previous two years – usually lasting 18-20 hours). The time frame during which I was experiencing episodes of AF q36h started during last summer's visit to HI (episodes for me seem to be more frequent here -? due to greater humidity/temperature). This was aggravated by an associated increase in flecainide on demand (never took more than 100 mg). I had stopped all flecainide for two weeks at the time I started the Waller water trial.

By June 2003 after terminating my Waller water regimen and flirting with various oral Mg supplements (see https://secure.salu.net/cgi-perl/get.cgi?pub=50136&ext=doc), vitamins and other supplements to no noticeable improvement, I returned to the pharmaceutical trough. The only med I'd been taking was a homeopathic dose of spironolactone (Aldactone 25 mg per day). This is a K (and Mg) sparing diuretic (weak). This seemed to help but this was not clear-cut, even though I've always limited my salt intake. My thinking on spironolactone has always been predicated on the supposition that aldosterone is integral in triggering many episodes of LAF. Hopefully you'll recall Hans' play (see Session 2 of the Proceedings of the Conference Room – LAF and the Hormone Connection) in which aldosterone plays a prominent role. Aldosterone is vital to the renin angiotensin aldosterone system (RAAS). It causes the urinary secretion of K and the absorption of Na with concomitant water retention. Its secretion is especially stimulated by dehydration (or the equivalent), increased K or decreased Mg, and stress (or the equivalent, e.g., increased ACTH). Spironolactone is most beneficial wrt to aldosterone blockade in those with fibrosis, as measured by

procollagen type III peptide. Perhaps LAFers, 100% of whom have abnormal heart tissue on biopsy, would benefit most from this blockade. Aldosterone induces cardiac fibrosis and vasculopathy, especially in the kidney and the brain. Aldosterone also causes inhibition of fibrinolysis (increases blood clotting). Please see http://www.medscape.com/viewarticle/422919_3

In June 2003 I experimented with disopyramide (AKA Norpace, Rythmodan). Disopyramide afforded great vagolysis. Taking 125 mg q6h resulted in an HRV that was almost always less than 10 ms. There was the usual dry mouth, blurred vision and urinary retention, but that was tolerable. This worked great for about a week (PACs went from two or three per minute to one per 15 to 30 minutes). However, the first day I forgot my noon dose of spironolactone resulted in a short episode of AF. So I dropped the disopyramide and concentrated on spironolactone. Aldosterone levels peak around 8 AM. Spironolactone peaks in two hours with a half-life of 10-20 hours. So I continued with this regimen taking the 25-mg dose at 6 AM. However, after a week AF again reared its ugly head. This coincidentally was a day I had sushi for lunch. Seaweed (wrapped around the rice) is very high in free glutamate. See Fran's (the glutamate queen) numerous posts on the ill effects of this dietary item. She has waged a nearly single handed battle, educating us all. Another MD has recently posted on this BB that diligent removal of this dietary item has resulted in a record AF free run.

Why did the spironolactone fail me? Perhaps even in the face of no more than mild dehydration the body will secrete enough aldosterone (no matter how much spironolactone is onboard) to maintain total body water. Perhaps no matter how much you restrict salt intake the body will secrete sufficient aldosterone to maintain its prerestriction levels. Mercola (thank you Fran) has recently exposed the irrationality of mainstream medicine in preaching dietary salt restriction for all. This latter recommendation is valid only in the salt sensitive. If you are normotensive, then you are probably not salt sensitive. Jerry has experienced excellent results wrt LAF by limiting his salt intake and ingesting plenty of K rich foods. Is his variant of LAF different from mine?

With this in mind I returned to a daily intake of at least 1.5 liters of neutralized (with one quarter of a small lemon) waller water. I used a concentration such that this would result in about 600 mg of elemental Mg per day. The result? During this ongoing summer visit to HI and after initiating better hydration, I have experienced only one episode of AF in five weeks, lasting between 5 and 8 hours (v. the usual 18-20 hours). I am now approaching a longtime barrier to my AF free interval (I truly believe it will be no more than an interval). Furthermore, during this period I have increased my workouts and pursued activities that are reliable triggers for me, e.g., sexual activity and snorkeling. However, I must admit that I have not tried both simultaneously. Furthermore, I pulled a Hans Larsen in HI and have stopped all vitamins and supplements. One benefit of this is the ability to detect early dehydration by urine color now unadulterated by the strong tinctorial qualities of excreted B vitamins (very yellow). I have continued to dine on known sources of free glutamate even after 8 PM.

Perhaps my initial very positive response to waller water (three week AF free interval) last year was not due to Mg, as assumed, but due to improved hydration. Recently my HR seems to be slightly higher than usual throughout the day. HRV, however, is still as high as ever. I have no physiologic explanation for this apparent contradiction. Hans in his book quotes a British study that showed an increase in sympathetic tone after drinking 500 ml of water. It seems to me that improved hydration would initially caused an increase in vagal tone (greater hydrostatic pressure sensed by the carotid sinus), followed in days to weeks by a drop in aldosterone and blood pressure. Although I still have plenty of PACs, I also have many dropped beats. Their appearance is all tied to increased vagal tone, which often appears unpredictably. Actual AF on the other hand appears to be in retreat for the time being.

Why should this happen? Perhaps it is better to remove the stimulus for a hormone's secretion (mild dehydration – I only drink when I'm thirsty) than to block its action (spironolactone is an aldosterone antagonist). Is this result simply due to the effect of better hydration on aldosterone or does the Mg have something to do with it? After I switched from the dilution and volume of aqueous Mg recommended by Erling Waller (more Mg and less water), my AF free intervals between episodes began to shorten. This shortening of the intervals slowly continued during subsequent oral Mg supplementation, according to the regimen recommended by Dr. Mansmann. So Mg would seem to be less important. Could the constant supply of highly bioavailable aqueous Mg potentiate glutamate decarboxylase (breaks down glutamate, the neurotransmitter substance for activity of the SA node and the vagus nerve)? After all it is a required cofactor. Or is the well-published ability of Mg to facilitate the maintenance of intracellular K (ATP pump and/or membrane stability) more critical? Perhaps increased Mg removed some stimulation for aldosterone secretion. Whatever the explanation, it is working for me and perhaps such a regimen might work for some of you.

Consequently it would appear that for me low intracellular K for whatever reason and high vagal tone are both required for VMAF. Only a little of one and a lot of the other (or a moderate imbalance in both) may be required to trigger an episode. Neither alone is sufficient. GERD (gastroesophageal reflux disease) may represent a condition that can cause both hypokalemia (alkaline tide) and vagal stimulation.

Increased vagal tone can independently cause all the changes required for AF, e.g., shortening of the atrial effective refractory period (AERP), increased dispersion of refractoriness, slowing of cardiac conduction velocity, increased PACs. Increased catecholamines (adrenergic LAF) are also associated with shortening of the AERP and cause increased automaticity (PACs). Cardiac fibrosis can cause increased dispersion of refractoriness. This is why I think the incidence of AF increases with age. Hypokalemia and hypoglycemia (see below) can both cause shortening of the refractory period.

VMAF is highly associated with years of participation in endurance sports. Perhaps the well-documented enhanced vagal tone in such individuals is also accompanied by chronic low-grade dehydration. Could this latter cause an increase in aldosterone receptor sites and their affinity for aldosterone to correct for this chronic condition? Hormones and neurotransmitter substances all operate through receptor sites. Acutely any shortfall is addressed by increased production of the hormone or neurotransmitter. In the chronic state activity is generally increased via upregulation of the receptors. There is an increase in the number of receptor sites in or on the cells of the end organ (target tissue) and an increase in receptor affinity for the hormone or neurotransmitter in question, be they renal tubular cells (aldosterone) or neurons in the brainstem (glutamate). This latter upregulation generally takes days to weeks. You'll recall that glutamate is the neurotransmitter for the Parasympathetic Nervous System and increased glutamate results in stimulation of the SA node. HR then slows and PACs begin to appear. Although the blood brain barrier is impermeable to dietary glutamate, the CSF (cerebrospinal fluid) is not. Glutamate can diffuse from blood vessels into CSF and from the CSF only to the nearby neurons in the brainstem (as well as the hypothalamus). Those that control the SA node and vagal tone just happen to be amongst those neurons so located.

A strong association between hypoglycemia (v. hyperglycemia and diabetes) and LAF was demonstrated in Hans second survey. Several articles in the medical literature have shown a strong association between AF arising in the left atrium and hypoglycemia. This is caused by shortening of the AERP and appears to be mediated by insulin. Could the endurance athlete also possess an increase in these cellular insulin receptor sites to help regulate the large blood glucose swings both before and during a workout? After all such individuals have made the phrase "carbohydrate loading" famous. The cellular receptor site for glucose uptake mediated by insulin also facilitates the simultaneous cellular uptake of K. This is why diabetics receiving insulin can become seriously hypokalemic. Could this association between LAF, hypoglycemia and insulin all be secondary to hypokalemia?
Proarrhythmic effects of reactive hypoglycemia, Rokas et al., Pacing Clin Electrophysiol 1992 Apr;15(4 Pt 1):373-6
Susceptibility of the right and left canine atria to fibrillation in hyperglycemia and hypoglycemia, Vardas et al., J Electrocardiol 1993 Apr;26(2):147-53

In the past OJ and/or a banana just before going to bed may have precipitated several of my episodes. Ironically instead of increasing blood K this may have caused a transient drop secondary to the insulin release triggered by the slight increase in blood glucose. Perhaps it would be wiser to replenish K without an accompanying carbohydrate infusion, e.g., green leafy vegetables (asparagus, cucumbers, collards, spinach, pickles), mushrooms, blackstrap molasses. But I still like bananas, just not before going to bed. Please visit the below hyperlink for a great listing of foods by mg of K/kcal
http://members.tripod.com/~charles_W/table.html

In the past on occasion I have been able to terminate an episode by holding my breath (voluntary apnea). I have never been able to do this in the evening. Perhaps afternoon episodes are triggered predominantly by insufficient intracellular K and some minor vagal maneuver (v. PM episodes being triggered predominantly by high vagal tone and only slightly low intracellular K). The former theoretically might be more easily reversed by a maneuver that decreases vagal tone. Pulmonary stretch receptors activated by full inspiration would do this. The latter (PM episodes) might respond more to a large glass of OJ with added K, reported as effective for some (definitely not me). This is all pure speculation. With this model there are myriad combinations of low intracellular potassium, hypoglycemia, cardiac fibrosis, stress, vagal tone, predisposing autonomic wiring, etc., that might trigger an episode in any given individual.

Aldosterone induced fibrosis and vasculopathy is mediated by a reduction in NO (nitric oxide). Mineralicorticoids produce superoxide radicals, which degrade NO. Dr. Louis Ignarro in 1998 received the Nobel Prize for his work with NO as a signaling molecule in the cardiovascular system. Viagra works as a result of this, as Richard has previously posted. This is one highly probable benefit a regimen of vitamins and supplements rich in antioxidants may specifically have on LAF. An antioxidant that is especially active against superoxide radicals is bioflavonoids (proanthocyanidins or procyanidins), such as in grape seed extract. Hans has expressed considerable interest in NO. Perhaps he can further educate us all on the NO connection.

Aldosterone also opposes fibrinolysis, i.e., is procoagulant and this is the last thing a LAFer needs. So whether aldosterone's effect on K is paramount and NO is a bit player in triggering LAF, aldosterone remains at the heart of the controversy. Aldosterone (and its tendency to lower total body K) may very well represent the missing link between VMAF (frequent dehydration) and adrenergic LAF (in addition to catecholamine release, stress results in excess mineralicorticoid (cortisol and aldosterone) activity). If aldosterone (presumably through K imbalance) is proven to be integral in the genesis of LAF, then there may be associated cardiac inflammation and fibrosis, given the presence of aldosterone receptors in the heart. Other causes of transient perturbations in blood K, e.g., alkaline tide, dysinsulinism, may be less worrisome on this count. My CRP (C reactive protein, a serum marker for inflammation) levels are slightly elevated, putting me at greater statistical risk for heart disease and stroke. Could this be due in part to aldosterone?

As Jackie has repeatedly posted, biochemical individuality (we are all different) makes a one size fits all approach likely to fail. But nothing ventured, nothing gained. It's just water and magnesium. Perhaps there is a subset of LAFers out there that drink only when thirsty, skimp on their vegetables and love their sweets, like me. I believe that most of the BB posts on vitamins and supplements are well advised wrt general health. Their direct connection to specifically improving LAF is less clear, at least to me. I've given up specific targeting of possible vitamin shortfalls, e.g., B vitamins, esp. B6. The mechanisms regulating fluid and electrolyte balance are very complex. Delving in depth into amino acids and related enzymes wrt LAF would surely put me into overload. Call me simple minded, but LAF is so prevalent in those that eat properly and so infrequent in those that do not.

Von Wagoner of the Cleveland Clinic, who has done extensive research on the physiology of AF, recently wrote an article on its molecular basis. Its contents basically recapitulate the biochemical individuality arguments. "Infrequently AF has a monogenic cause. But whatever the cause(s), reentry is required."
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12875431&dopt=Abstract

There are many, many "healthy" people that eat (and hydrate) far worse than LAFers. Many have lower heart rates or more stress in their lives. Yet they don't have LAF. Why is that? I believe that the atrial "wiring" in LAFers is the key difference. The vagus nerve does more than just control HR and HRV (through the SA node). It also causes shortening of the AERP and increased dispersion of refractoriness (variability in signal propagation). This must be caused through its intracardiac nerve fibers (present only in the atria). Cardiac autonomic nerve fiber pathways in LAFers may be such that greater dispersion is enabled even in the absence of high vagal tone. This one feature (dispersion) greatly enhances the phenomenon of reentry (an impulse encounters refractory tissue in such a way that it ends up traveling in a circle). Increased parasympathetic and sympathetic tone both shorten the refractory period and this may enhance the likelihood of AF in one with such atrial "wiring". This wiring is hereditary. My father and uncle had long standing AF and died of massive strokes. My mother is still alive with AF. ALAFers and VMAFers can avoid stress, hydrate, K replenish, and carbohydrate control "to their heart's content", but will always remain susceptible to AF. Only an ablation (or the Maze Procedure) will effectively rewire the atria and remove this predisposition. But in the meantime, I believe we do have some anti-AF weapons in our arsenal. We can control the some of the modifiers (see above). Various polymorphisms can predispose us to LAF (see Jackie's previous posts on "snips" or single nucleotide polymorphisms). As an example, there could be a mutation in the DNA that codes for glutamate decarboxylase, causing greater vagal tone secondary to excess glutamate. More Vitamin B6, a required coenzyme for this decarboxylase, might help it degrade the glutamate.

At the very least have some "green" with every meal, don't let your urine become any darker than a hint of yellow and avoid sugar snacks, especially in the afternoon (the vagus is beginning to tone up). For those with strong vagal tone avoid free glutamate, especially at night (I'm not very disciplined on this). Consider sitting down before lying down in bed and try sitting up in bed before completely lying down (20 minutes or so transitions). I too have trouble lying on my left side. According to Miyamoto (Reference #8 in Hans play), lying on the right side is paradoxically more vagotonic. I still eat late and dine out with regularity, especially including sushi and Chinese food, often laden with MSG. For those

prone to the “Chinese restaurant syndrome” you might consider disopyramide prophylactically (in advance) or “on demand” (when you detect the telltale increase in PACs). Therapeutic levels can be attained in less than an hour (except for the SR or slow release version). Consider spironolactone or the new anti aldosterone drug eplerenone. Also remember, there are many equivalents to dehydration. For example, prolonged standing (as in “a good walk spoiled” on the golf course) causes peripheral pooling. Throw in a skipped meal with just a carbohydrate snack on a hot day and you are ripe (for an episode of AF that is).

So don’t despair.

This is all personal opinion and is not meant to apply to all LAFers. However, this regimen might allow some of you to reduce LAF symptomatology enough to delay that date with the electrophysiologist long enough for improvement in current ablation techniques. I have halved my AF episode length and at least doubled my AF free interval. I have not one shred of objective evidence to support this model (except intracellular K and Mg levels that were for me at the very lower limit of normal, despite repeatedly normal blood K levels). It only fits the experimental results so far for me (N=1). However, I have tried to investigate it further. An email to Burton Silver, Ph.D., founder and CEO of Intracellular Diagnostics, inquiring about a study of intracellular K at LAF onset and termination, initially received a positive response. However, my follow-up email to him about a specific proposal has received no response. At this point I plan to continue with the status quo. Perhaps at some point I’ll request another intracellular mineral analysis to evaluate K, Mg and Ca.

For those of you that are now in status brain crampicus, please read my three part report on Mg and K in LAF in the April, May and June 2003 issues of the AFIB Report. If you have not subscribed to Hans’ monthly AFIB Report and his International Health News, please reconsider. They are not only a tremendous resource on AF but also a cogent review of the medical literature on timely topics of general interest (in very understandable language).

P.S. Richard, hope you are no longer in brain cramp withdrawal. Also, I’ve decided to do the hula full-time.

Mahalo
PC v54

PC,

I suddenly have this mental picture of you wearing a grass skirt, coconut cups and all. Sure is nice to hear from you and good to know you're enjoying your vacation. I think I need a more extended vacation, so I can sound as relaxed and more at ease with our situation, as you seem to have become, yet I can tell your wheels are still spinning. Yeh, I did miss your brain cramps, and yes I will have to go back and re-read your post, but happily.

For me, it wasn't excessive exercise that caused my problem. Even though I know you've mentioned aldosterone before, I must go find out what aminos have to do with it and get a better understanding of just what it does. If you defined before, I have forgotten. I just don't have enough gigabytes of memory any longer.

Thank you for the informative post and all your thoughts, and remember one thing. Don't swing the hips too much, as that could be vagolytic, at least under the Hawaiian moon.

I'll be back.
Richard

Aloha PC,
as ever I'll need to re-read your post a few times to digest it.

One thing that sprung to mind was your comments on hereditary wiring.

Do you have a theory as to why people with a genetic component to AF usually still get AF later in life?

Do you think it is likely that anatomical changes in heart structure compound any wiring problem? -and are such changes reversible without getting the soldering iron out:)

Anyone know why I can hear a ukulele in my head :)
--
James D

---

hi PC

you're very good aren't you? glad to hear you are well , AF free and back with us. Don't leave it so long next time we have all missed you!

after reading your post i picked up on a couple of things.

all my AF episodes have been directly linked to dehydration. most are before my evening meal when i have forgotten to drink for most of the afternoon and gone about 5 hours since my lunch. its hard sometimes to remember to drink even when you are not thirsty also after having alcohol when i'm dehydrated.

another thing i picked up on was about alafers being prediposed to AF when i was two they picked up a heart murmer which just disappeared. the only other problems i have had until the AF started was during puberty i used to faint and get really light headed can't remember whether i was getting skipped beats at the time . Then after the birth of my daughter it started , i was 22. I think its been there all along just waiting to happen. I must have some kind of defect they haven't picked up on.

anyway i've been very well until i watched Bo selecta the other night i laughed so hard i nearly went into AF . This is a must for anyone in the UK just be careful you don't set yourself off.

tonigirl 29 alafer xx

PC
I am relatively new to this board, so please be patient with a few questions.

Totally agree about dehydration being a probable contributing factor in LAF.

As a matter of fact, during a severe heat wave several years ago my physically fit and totally healthy husband suddenly collapsed several times at home, (I pounded on his chest and shook him to revive him), got him to the local ER, where his heart stopped (again). He was resuscitated, diagnosed (quickly) with SSS or Sick Sinus Syndrome and a pacemaker was installed the next day after tests showed nothing wrong with his heart. Like LAF, SSS is another "lone" cardiac/neurolical disorder. In other words, his heart was and is healthy.

We are now both convinced that all that was really wrong was that he was seriously dehydrated from the heat wave. Back then, neither of us thought about drinking water, even during a heat wave. So, dehydration may be an explanation for SSS as well as LAF. In the crisis of his cardiac arrests, the doctors apparently never considered something as simple as dehydration during a heat wave.

ARe you vagal, mixed or adrenergic?
Why haven't you eliminated MSG?
How about your caffeine intake?
Lots of hard science speculation in your recent post, but how about the "soft" sciences of stress management?

For the continuing record, after 11 years of increasing episodes of LAF, ever since implementing high intakes of magnesium and potassium, I have been three weeks without a single episode - only occasional PACs, when I first lie

down at night. Now the PACs don't develop into Afib. This is a record for me. Hope it holds.

Am also drinking lots of water and have cut way back on caffeine.

Carol

PC

So glad you are having a prolonged vacation. Great to hear from you again and read another of your fabulous brain cramps. How eloquent.

Just today I was thinking of the importance of keeping hydrated. We are in the middle of a heat wave. I usually drink about two litres of mineral water a day. However, yesterday and today I have exceeded this and am nearer 3 litres already, and it is only 7.30 pm.

I really think your scenario above warrants further exploration. I love the dehydration and Aldosterone theory. It fits right into my 'gut instinct'. Not very scientific, but is also another of the things I have done for my AF free diet - the hydration part. The colour of Pee is a great indicator of hydration. It should never be more than straw coloured.

The one thing I would take issue with (for my type of AF) is that I still can not take free glutamate - as discovered with my short run after a burger king a month or so back. But then it might have been the lack of sleep, stress or the lack of not eating properly that contributed. Now why could I not have just introduced just the one factor at a time- but life is not like that.

Or maybe it is my wiring....

I suspect that you will always seek the alternative to giving up MSG, given your love of food, and I admire this as maybe, just maybe, you will find the alternative enabling someone like me to pig out on junk once in a while.

Can aldosterone be measured? How difficult (and expensive) would it be to set up a double blind study to study this theory with all the other scenarios eg glutamate, mg, K, etc. One that could be published and recognised by main stream medicine. Would you be able to lead something like this?

Keep swigging that bottle..... of water

Fran

PC,

Good to read your posts again..... and, almost worryingly..... no brain cramps! PLENTY of good points/stuff in your post. But my favourite bit??.....
"sexual activity and snorkeling. However, I must admit that I have not tried both simultaneously."
LOVE IT!

Kind regards and continue enjoying your vacation,

Mike F.

PC,

It would seem, through what reading I have done, that dopamine has inhibitory effects on aldosterone. In my case, I believe the pathway for Phe. to tyrosine isn't being carried out, therefore I must be low in dopamine, but I am also low

in tryptophan, arginine, and methionine, and a few other important aminos. Have you ever thought about trying a balanced blend of free form aminos, to see what effects you would have? Check them out at www.jomarlabs.com

Here are some studies:

Autonomic functions, such as increased sympathetic and parasympathetic activity and the brain's suprachiasmatic nucleus, higher nervous centres, depression, hostility and aggression appear to be important determinants of heart rate variability (HRV), which is, itself, an important risk factor of myocardial infarction, arrhythmias, sudden death, heart failure and atherosclerosis. The circadian rhythm of these complications with an increased occurrence in the second quarter of the day may be due to autonomic dysfunction as well as to the presence of excitatory brain and heart tissues. While increased sympathetic activity is associated with increased levels of cortisol, catecholamines, serotonin, renin, aldosterone, angiotensin and free radicals; increased parasympathetic activity may be associated with greater levels of acetylecholine, dopamine, nitric oxide, endorphins, coenzyme Q10, antioxidants and other protective factors.
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12653178&dopt=Abstract

CONCLUSIONS: These data show that, at least in compensated cirrhotic patients, the stimulation of systemic NO production and the increased dopaminergic function may be mechanisms preventing renal perfusion, GFR, and fractional excretion of sodium from precocious reductions.
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12358261&dopt=Abstract

CONCLUSIONS: Two subtypes of APA were defined according to their responses to metoclopramide during salt manipulation. On HS, the nonsuppressible subjects, with less dopaminergic inhibition of aldosterone secretion, had less urinary DA excretion and greater BP elevation. The renal and adrenal dopaminergic activities are regulated in a parallel fashion.
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12118908&dopt=Abstract

Norepinephrine, dopamine, and prostaglandin E2 can inhibit the antinatriuretic effects of VP, and changes in the actions of these autocrine and paracrine regulators may also be involved in abnormal regulation of Na+ reabsorption.
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12110505&dopt=Abstract

Inhibitory agents

Dopamine inhibits aldosterone secretion in humans by a mechanism that is independent of the effects of prolactin, ACTH, electrolytes and the renin-angiotensin system (18). This inhibitory effect may involve binding to D2 receptors on glomerulosa cells (19). Atrial natriuretic peptide (ANP) directly inhibits aldosterone secretion and blocks the stimulatory effects of angiotensin II, potassium and ACTH, at least in part, by interfering with extracellular calcium influx (20).
http://www.endotext.com/adrenal/adrenal24/adrenalframe24.htm

This is an index for a lot of info. on the endocrine system.
http://www.endotext.com/adrenal/index.htm

I'm still studying. Happy Hula!!!
Richard

Fran,
See below. And yes I love to dine out. My LAF will have to get a lot worse before I cease and desist that.
If you measured the aldosterone, I don't think it would be abnormal (except in the acute period). After development of a chronic state of dehydration albeit mild, receptor sites for aldosterone are upregulated and become more sensitive. This means that less aldosterone is needed and therefore less will be secreted. In my opinion in this whole process the critical action is on or inside the cells. Measuring something in the blood, e.g., K, is insufficiently sensitive and not very informative. Heck, my efforts to illuminate the intracellular K picture in LAF secondary to ANP and aldosterone have met with little success. I'm sure someone somewhere is working out the physiology. Once it is published, some smart guy somewhere else will make the correlation to LAF.

James,
I think it's the effect of hereditary wiring on shortening of the refractory period but especially dispersion of refractoriness that is the key. Autonomic tone encompasses more than just heart rate, heart rate variability or heart period variability. There are contributors beyond the SA node and the AV node. There are also intracardiac autonomic nerve fibers (predominantly vagal nerve fibers) that contribute. These are never perfectly uniform (homogeneous) in their distribution within the heart. Their pattern is unique to the individual and form a kind of genetic cardiac fingerprint. The contribution of this component to autonomic tone can only be measured by such things as refractory period, dispersion of refractoriness, conduction velocity, etc. Indeed intracardiac nerve fibers are not alone in controlling these things. There are also circulating hormones (predominantly adrenergic) in the blood that can target receptor sites that are also intracardiac. Aldosterone has only recently been added to the list. It's hard for me to believe that its presence is only negative (fibrosis). There must be some positive benefit that is yet to be discovered. The distribution of these receptor sites are also unique. Typically as we age there is also progressive fibrosis (?due to aldosterone amongst other causes). The deposition of this collagen should not be perfectly uniform either and therefore should further enhance this inhomogeneity, esp. wrt dispersion of refractoriness. So, as I see it, if the wiring were sufficiently inhomogeneous (more nerves in one area v. another), then this might predispose to AF at an earlier age (triggered no doubt by some of these modifiers – see below). In others perhaps this threshhold for AF is not reached until later in life after sufficient fibrosis has accumulated to cause the necessary inhomogeneity or dispersion. The fibrosis, as far as I know, should, like any scar, be irreversible.

Tonigirl,
I'm missing my chocolates. Are you? Have to agree with you that it is very difficult sometimes to remember to drink when you're not even thirsty. Also, although I'm not sure about the murmur, the history of lightheadedness and fainting, esp. associated with PACs, go right along with possible AF later in life.

In my opinion this increased dispersion of refractoriness secondary to the atrial wiring is not necessarily abnormal or something that has been missed. Its presence in the general population should be a bell shaped, with LAFers toward one end. Some of those in the general population that have enhanced their vagal tone through exercise, that underhydrate, that experience more stress, that have developed cardiac fibrosis NOS (not otherwise specified), etc., may then increase their dispersion to the point that they develop AF. Those with reactive hypoglycemia, low dietary Mg and K, etc., also experience shortening of the atrial refractory period. I think that one's incidence of PACs is a direct measure of one's innate refractory period (again this should form a bell shaped curve, since everyone gets PACs). The shorter the refractory period, the more susceptible any heart cell is to an ectopic impulse, i.e., more PACs. Only autonomic tone secondary to intracardiac autonomic nerves is genetic. These other factors are all modifiable. Glutamate sensitivity may be both genetic and modifiable, e.g., some polymorphism. A polymorphism is a genetic mutation that is measurable and that is present in less than 5% of the population. Sometimes a modifier is required for its expression, e.g., Mg deficiency.

Carol,
I would be very careful about SSS. This is generally considered to be of pathologic origin. Sometimes the ability of modern medicine to detect early disease is overrated. I've spoken with a number of people that have been given a clean bill of health after an extensive check up only to experience a heart attack within a few months. That is not to say that your husband's heart is diseased.

V54 means vagal 54 y/o

You and Fran are absolutely right. I should be more aggressive in eliminating free glutamate from my diet. However, as she pointed out, I would have to severely limit my dining out. Glutamate definitely causes a marked increase in PACs for me. Although this is somewhat uncomfortable, it has not triggered an episode under my present regimen. I'm attacking this disease in a stepwise fashion. It's all about quality of life and both LAF and glutamate restriction compromise it. Until the former gets worse, the latter will be half hearted. Caffeine intake on the other hand is minimal. No coffee and no soda.

Richard,
You ARE the amino man. Just don't put me into overload.

PC v54

Carol said" during a severe heat wave several years ago my physically fit and totally healthy husband suddenly collapsed several times at home, (I pounded on his chest and shook him to revive him), got him to the local ER, where his heart stopped (again). He was resuscitated, diagnosed (quickly) with SSS or Sick Sinus Syndrome and a pacemaker was installed the next day after tests showed nothing wrong with his heart".

I know it is not quite the same thing, but often when you hear of healthy peak condition athletes, footballers etc dropping dead during the course of a game, it makes you wonder more and more about the effects of dehydration.

I'm glad it turned out so well for your husband.

Fran

PC,

I hope it doesn't seem to be, that I'm putting you in overload, just sharing info. that may spur thoughts. I'll just have to treat myself with aminos, and see what the outcome is.

In regards to your statement: "The fibrosis, as far as I know, should, like any scar, be irreversible."

As you may have read, but I'll repeat, the scar on my butt is almost completely gone, from taking L-lysine. I am amazed and I had it for several years.

You may find this of interest, in regards to fibrosis. I think James D. shared it. http://www.bodybewell.com/fibrosis-article.html

Enjoy life and be well!

Richard

PC,

Great stuff again in your replies to everyone. I particularly like the comments you made to tonigirl with regard to fibrosis, PACs, dispersion etc. I know that these are VERY crude and basic questions, but I have visions of many AFrs reading this board and deducing the following questions:

1. How could one find out if one had/has developed a degree of cardiac fibrosis? Or is the degree required to produce AF too subtle to detect with testing? (I do note your comment about homogeneity - makes sense to me.)
2. How can one modify (lengthen) ones refractory period? (So as to reduce/prevent PACs.)
3. How can one reduce ones dispersion of refractoriness?

All the above would surely help in no small way to reduce or eliminate AF for many. Are there any simplish and easy-to-take-on-board and implement answers to these questions?! (I suspect that regrettably there are may not be.)

Also, did you read Richard's website suggestion regarding fibromyalgia and fibrosis and a possible supplementary way to tackle it? If so, what do you think - could this protocol or something similar reverse fibrosis to a significant degree/helpful extent?

Lastly, having read your excellent replies as aforementioned, I wonder if you have any sort of medical take - endo/hormonally-speaking - as to how childhood and adolescent stress could make one particularly prone to PACs and AF as an adult? Adrenal burnout--->chronically-stressed hypothalamus--->hormonal changes---->anxiety & PACs & AF???

Thanks again for your continuing contributions to this excellent forum,

Mike F.

Aloha PC,

What a terrific summary of your research and experience with LAF! I have just learned from Dr. Van Wagoner at the Cleveland Clinic that they, in cooperation with Vanderbilt University, have several projects underway to investigate the role of oxidative stress, inflammation and aldosterone in AF. They apparently have received several NIH grants to do this and will also take a look at NO and its role in AF. Very, very encouraging; they certainly have access to more resources than we do!

I tried spironolactone (25 mg/day) myself for about a week, but all it seemed to do was to produce a whole lot of skipped beats, so I discontinued it.

I believe you are right on with the need for constant hydration - 1.5 to 2 liters a day is probably the minimum, and magnesium - probably to bowel tolerance.

It seems to me that the situation with potassium is a bit more complicated. Certainly hypokalemia is a cause of AF. However, if you try to increase your potassium level by supplementing won't you also tend to increase aldosterone production? In other words, is this a catch-22 situation or is it definitely more advantageous to get lots of K and not worry about the increased aldosterone? Are some ways of increasing K levels better than others insofar as aldosterone production is concerned? Anyway, your results with the Waller water regimen are indeed impressive so I am going to have another go at being consistent about my hydration - it is so easy to lapse!

Nevertheless, there is always the possibility that there is something "magical" in the air in HI!! The first possibility that comes to mind is a vast surplus of negative ions due to the proximity of the ocean, the wave action, and the moist tradewinds. Pure speculation of course, but the ion environment certainly would be fundamentally different from that found in your average office with its fluorescent lights, computers, and phtocopiers.

This thread is turning out to be extremely interesting and is already becoming "old news", so I will be sure to include it in the Conference Room Proceedings when it has run its course.

In the meantime, may Kanaloa be with you and your wa'a be swift!

Hans

Hans said

"The first possibility that comes to mind is a vast surplus of negative ions due to the proximity of the ocean, the wave action, and the moist tradewinds."

A lovely thought, but don't think it can be the case.

Fran, free of AF whose house is within 30m of the ocean with the gulf stream running through - and loads of negative ions. But who also lived here right throughout her AF career.

Fran

Mike,

Again I wish I was better informed.

Regarding some of your questions, be sure to visit the article on aldosterone hyperlinked above. I believe it was you that first pointed this BB in its direction. In it Dr. Struthers talks about procollagen III peptide as an indicator of fibrosis. We don't offer it in our lab and I'm not sure that it is commercially available. Short of an ill advised cardiac biopsy I know of no other way to directly measure cardiac fibrosis. Dr. Frustacci's findings on LAF and abnormal hearts by biopsy (100%) were posted somewhat recently here by Hans I believe. I know that info is available on this website somewhere outside the BB as well. Accordingly, you ought to consider the recently available high sensitive C reactive protein blood test (hs CRP) as a screening test for inflammation.

Increased vagal (parasympathetic) and increased adrenergic (sympathetic) tone both shorten the refractory period. Hypokalemia and hypoglycemia also do this. Increased vagal tone also increases the dispersion of this refractoriness (kind of a two in one "bonus"). Cardiac fibrosis also increases this dispersion. Anything that compromises the homogeneity of the nerves fibers should increase dispersion. I'm sure there are other etiologies, but control these modifiers and you should be right mate.

Haven't read (or more likely forgot the contents of) Richard's recommended site. Please repeat it for me.
AF is so hard to comprehend in the present. Delving into ones distant past would be nearly fibrillogenic for me.

Good luck

PC

Hans,

I guess there's a reason that the Cleveland Clinic is numero uno (Natale, Von Wagoner, etc.). And I always thought it was because that outspoken Clevelandite Jackie was continually singing its praises.

Your response to spironolactone is interesting. Like you, I also experienced PACs after ingesting K supplements. But I never did with spironolactone.

Your comments on increasing K intake and aldosterone are right on, as usual. I take the spironolactone at this point more for its presumed blockade of aldosterone receptors in the heart with attendant decrease in fibrosis. If increasing K intake increases blood K, then aldosterone secretion will increase. However, perhaps poor absorption and/or urinary K wasting compromise the ability of some to maintain intracellular K. As far as I know, K, unlike Na or Ca, has no dedicated neurohormonal mechanism that protects against a K deficiency. But just in case I'm underestimating the aldosterone stimulating effects of increased K intake, at least I've limited my spironolactone to an almost homeopathic 25 mg per day. But most importantly I take no K supplements. I've never heard or read anywhere that a diet high in K is harmful wrt aldosterone. So its lots of apple bananas for me here in HI.

HI is indeed a magical place. It has been much more so for me this summer, given the marked reduction in episodes. I don't know whether the tradewinds and the gentle sound of the surf has been positively affecting my episodes, but these certainly make one sleep like a baby.

In a few more nights it looks like there will be a full moon. Moonlight (while walking on the beach) is so bright over here that when it re-emerges from behind a cloud it is as if someone has just turned on the lights.

Hans, thanks again for your dedication and devotion to the fibrillating mass.

PC

PC,

I had a CRP test done on 5/14/03. The results were 0.6 and this fell into the lowest risk group of <0.7, therefore I'm under the assumption that inflammation is not my problem. Have you had this testing done?

This was the site on fibrosis, that I believe James D. or William shared. I can't now remember for sure.

http://www.bodybewell.com/fibrosis-article.html

Richard

Richard,

I don't remember the numerical result of my CRP. I do know that our lab breaks results down into related risk for stroke and/or heart disease either by quartile or ?thirds (can't remember exactly). But I do remember that I was in the second quartile or middle third. I'll have to follow this as Jackie suggested. Not exactly sure what I'll do about it other than worry.

Visited your posted website. I didn't wade through the whole thing, but there were a number of factual errors. Fibrin is not scar tissue (collagen is scar tissue). How can one compare absorption of a delicate protein like an enzyme with Salmonella protected by a very hardy cell wall? There were many others.

Regarding the reversibility of fibrosis. Perhaps certain kinds are. Hyalinized scar tissue (usually only seen years after the collagen was deposited) would be a very unlikely candidate for this.

PC

I'm going to ask a really daft question:

Is there a known connection between fibrosis and fibromyalgia. Could a muscle that has fibromyalgia be biopsied?

And a quick thought about K inducing PACS. Would this not have something to do with hypoglycemia. I have read in numerous articles that K is bad for reactive hypoglycemia.

Fran

Fran,

Although we send out most of our diagnostic muscle biopsies, I have never seen or heard of a biopsy from anyone with a diagnosis of fibromyalgia. Muscular dystrophy, polymyositis, etc., are commonplace. Fibromyalgia literally means pain in muscle and connective tissue. I know of no pathology textbook that describes abnormal microscopic findings in fibromyalgia. This is basically a wastebasket clinical diagnosis without specific histopathologic changes.

Hypoglycemia and hypokalemia are often connected. Insulin causes the intracellular transport of both, lowering blood levels. Hypokalemia occurs more often clinically and this is usually independent of low blood glucose. I know that they both cause shortening of the AERP, but I don't know exactly how this happens biochemically.

If you have an article stating that ingestion of K is bad for PRH, I'd sure like to see it. Only low K should be bad for PRH.

PC

Hi PC

This is one of the mentions I have read about high K and hypoglycemia (its more to do with it reducing zinc and chromium). I posted this and others a while back. There is more about hypoglycemia and high K on this website. Also

the beneficial aspects of sodium on hypoglycemia. You can look around.

http://www.acu-cell.com/znk.html

Likewise, many weight loss formulations contain rather large amounts of potassium to take advantage of its diuretic properties. Some people benefit from additional potassium and will lose water weight,
however those with a tendency for hypoglycemic (low blood sugar) episodes may end up worse as a result of that extra potassium reducing manganese and chromium levels, which help stabilize blood
sugar, and also as a result of potassium reducing sodium, which is an insulin antagonist. In addition, lowering chromium too much following long-term high potassium intake can contribute to osteoporosis of trabecular bone (end-part of a bone or spine).

And for interest..."Total cholesterol levels are somewhat affected by potassium levels, whereby low cholesterol is often found in the presence of low potassium, and total triglyceride levels are somewhat affected by zinc levels, whereby low triglycerides are frequently found in the presence of low zinc. Some exceptions apply, particularly when high calcium and/or magnesium levels are involved, which have a lowering effect on total triglycerides (calcium) and total cholesterol levels (magnesium) also."

As to fibromyalgia, if biopsies have not been taken how can they say fibro is a wastepaper basket syndrome. It certainly isn't for the sufferer.

You said "Although we send out most of our diagnostic muscle biopsies, I have never seen or heard of a biopsy from anyone with a diagnosis of fibromyalgia."

If biopsies are not done how can they truthfully say "This is basically a wastebasket clinical diagnosis without specific histopathologic changes".

There is so much to learn!

Fran

Fran,

First of all, "wastebasket" is not meant to demean the suffering of any patient with a "wastebasket diagnosis". It refers to the fact that we don't know enough about the disease to explain the symptoms. In fact many times a wastebasket diagnosis is later found to contain several distinct diseases.

If a patient with fibromyalgia were to have a biopsy and it showed inflammation than it would be called myositis, as in polymyositis or dermatomyositis (inflammation of skin and muscle). The same for arthralgia/arthritis. Not all pain originating in a particular organ has a distinct microscopic finding. That certainly doesn't mean that there is therefore no pain. That certainly doesn't mean that fibromyalgics haven't had muscle biopsies. Oftentimes it is easier to detect inflammation by blood tests, i.e., CRP, autoimmune tests, etc.

PC

Fellow Fibbers,

I've been reading with dismay some of the recent posts regarding untoward results of ablation. The purpose of this one is to promote some confidence in the alterative approach.

About a month ago I submitted a post on Hydration and Aldosterone. It was rather long. This one is not and is merely a followup.

Since commencing the hydration regimen, my AF free interval between episodes has lengthened from its usual of about a week to (in order) 15 days, 16 days and now 23 + days (still waiting for the next epsiode). PACs are definitely

not decreased, which makes this result all the more remarkable. Although I try to avoid obvious sources of glutamate (Fran I've finally given up sushi, i.e., seaweed, at least for now). Just underscores not only how multifactorial LAF is but also that improvement is within our grasp. As I stated earlier, I still think that for some LAF may be "cured" but the propensity will forever lurk in the background. In addition the episodes themselves have shrunk from 18-20 hours to less than 4 hours using a single dose of disopyramide "on demand". I've only taken it twice in the last two months. I've just returned from two months in Hawaii where my episodes are usually more frequent (this latter observation led to the increased emphasis on hydration). Actually I feel rather stupid to have only recently stumbled onto the extreme importance of this rather obvious oft preached healthful habit. Duh! What a dullard. But better late than never.

In the beginning I found it quite easy to forget to drink at regular intervals throughout the day. So then I set my watch to alert me every 30 minutes. Now it's become second nature. The color of my urine has been straw colored at most throughout the day. I stop drinking 30 minutes before and don't restart until 60 minutes after major meals. I'm now at 2.5 liters/day on the average and eat all the salt I want. I'm more concerned about losing electrolytes through the hydration induced diuresis, so I'm concentrating on the supply side and more intake (more fruits and vegetables).

What I would like to know is has anyone alse been exploring in the same direction with similar results. Am I just a freak? (don't answer that one) Are any other VMAFers noticing any improvement? Francois, what are your hydration habits?

Please review my post of 8/5/03 for theoretical details, especially if a brain cramp is desired.

PC v54

PC: I for one have adopted the taking of more water. It hasn't cured my afib, but has certainly changed the nature of the episode, making it milder, and slightly extending the period of normal rhythm between episodes. I am sufficiently impressed as to continue the treatment. For many years, I had prided myself on being able to go without liquids for long periods. I now see this as possibly misguided.

Jeff

I was going to post some remarks on the topic of coffee, electrolyte depletion and afib, but I think I can make the points usefully here.

Over the past couple of months I have been drinking more coffee than I had during the previous 6 months. About three weeks ago I started noticing the return of PACs or PVCs and very short runs of rapid heartbeats, perhaps once or twice a day. At that point I was on a trip during which I was doing a lot of driving and consequently drinking perhaps three cups of coffee daily to keep my alertness up while behind the wheel.

This past three weeks have also been very warm here in California and in the Puget Sound region (Seattle) where I was traveling. I am not very good at drinking water when I am active in warm weather.

At any rate, whether it was the increased diuresis from the coffee drinking, or other electrolyte-wasting properties of coffee or the effects of dehydration from being active during very warm weather, my PACs or PVCs were definitely on the increase. I did not, however, experience any directly- afib-related symptoms. I did experience short runs of perhaps half-a-dozen rapid heartbeats on occasion, for example just after drinking an ice-cold smoothie.

So a week ago I cut back drastically on my coffee and made a point of drinking a couple of liters of my favorite mineral water (Pelligrino--I am Italian) a day. I reduced my coffee intake to less than one cup a day.

As a result of my improved hydration or reduced dehydration or diuresis, the PACs or PVCs and the occasional runs of rapid heartbeats have disappeared.

I should note that I have never stopped my daily magnesium, potassium and B-vitamin supplements. I take 800 mg daily of magnesium glycinate.

It is quite clear to me that electrolyte depletion and/or dehydration are important factors regarding my afib.

And now I am off to make a mango smoothie just to test my vagal response.

Michael in SF

PC

Does your 2.5 liters/day include your WW?

Rick

Rick,

The 2.5 liters is only ww (=waller water for Erling Waller, who perfected the formula of MoM (or Milk of Magnesia) mixed with soda water (containing bicarbonate) and introduced it this BB). Any other fluids are in addition.

My personal opinion on LAF at this point is that there are two most important ingredients, intracellular K imbalance and increased dispersion of refractoriness. The former is mediated by aldosterone either from stress and/or chronic dehydration (also including equivalent conditions like peripheral pooling of blood, e.g., secondary to prolonged standing or endurance sports). The latter would be mediated by vagal tone (endogenous from endurance sports or exogenous from dietary glutamates) and/or perhaps early cardiac fibrosis (from aldosterone and/or the natural aging process). Although it's probably not quite as simple as this, I think if we were to look closely at our collective LAF episodes, this might be the pattern.

PC

Before my 'cure' I tried just about everything including hydration. I had 12/15 hour sessions of AF every other day. I tried altering my diet, supplements, hydration, Unique water, acupuncture, Chinese Medicine and visits to the chiro'. Most of these had an effect to start with but eventually the AF settled down to the previous depressing level. The best effect was Unique Water (Magnesium Bicarbonate) which reduced the sessions to once every 12 days. The effect lasted about 3 months. Hydration worked for about 10 days or so. I don't wish to stop anyone from trying these possible remedies and in some cases where the events are far apart it may actually stop them but my message is 'don't get your hopes up too much if you get an improvement at first as it is just so depressing if the dreaded af returns'. Best wishes to you all.

Rod V54+p/m Tasmania

Rod,

By "cure" I presume you mean ablation.

I too experienced a tremendous improvement during the Waller water trial. I immediately went to 3 weeks from every 36 hours between intervals. Being an inveterate experimenter I started fooling around with the amount of water and Mg. I thought the benefit was due to the Mg and perhaps some of it was. However, when I started taking less water (hated those early AM bladder calls) containing more Mg, this benefit gradually deteriorated finally culminating in my usual 7-8 days between episodes.

Accordingly, your point is well taken. I've had so many false starts that I can really relate to your sentiments on this. I could relapse tonight. However, hope and optimism springs eternal. I think that the benefits of better hydration are myriad (including less likelihood of stroke) and maybe the intervals will continue to lengthen. The episodes are certainly

much more mild and shortlived.

PC

P.S. I also stopped taking spironolactone recently to see what effect that might have had on all this.

I'm taking 600 mg of magnesium - how much more will 2.5 liter (using PC's intake volume) add and is this too much?

People are concerned about losing electrolytes - isn't this something that Gatorade is supposed to be rich in? Is there anything in Gatorade that's bad for us?

Gregg

PC
I agree entirely that none of us may be able to entirely cure our afib. When we are in "remission," afib still seems to lurk in the background ready to spring at us perhaps as a warning signal, telling us that the body/mind has been thrown off balance by the stressors of dehydration, depletion of mg and potassium, mental/emotional stress, etc.

Even though I have had a couple of break-through afib episodes since beginning increased mg. (now tolerating well 700mg.), that were clearly brought on by emotional factors, I am ECSTATIC that after suffering for 11 years from afib, I am now essentially afib free. I can live with these occasional break-throughs and actually learn from them. It was the almost constant afib episodes that were intolerable. I have my health and confidence back. Afib can take that confidence away.

If on occasion I feel vulnerable to afib (i.e., wake up to PACs at 4 am, when after 7 hours of no water the body is almost certainly dehydrated) , I avert an episode by drinking a glass of water. I am then able to go back to sleep.

Carol

PC: Where does one find this WW (Waller Water)? Do you think the same effect could be had from the same amount of water and slow released magnesium ie: Slow Mag? Water with MOM? Milk of Magnesia? Are there other calls besides bladder calls? I also believe that hydration and magnesium are very significant in the treatment of afib., thus the alcohol afib connection.

Thanks
Pam

Michael in SF,

I posted a discussion about the effects of caffeine on afib a while ago, but it never seemed to get off the ground. Hope your contribution will get a response.

I believe that there are substances besides caffeine in coffee that are equally deleterious to afibbers.

I would advise anyone about to reduce caffeine to do so slowly. An abrupt reduction or what amounts to withdrawal from the drug caffeine can bring about severe , long lasting headaches, nausea, passing out from sudden blood vessel dilation and contraction in the head, which occurs from the reduction.

I discovered that a brand labeled "high energy" green tea that contained other herbs was much too energizing for me. It precipitated some pacs, which stopped, when I switched back to regular green tea - 1 cup a day.

Carol

Carol: I take Slow Mag., and was prescribed 1 daily which is 64mg. of Magnesium Chloride. What form of Magnesium are you taking? Do you feel like that in itself is what has helped you?

Pam

PC,

Your post, and thoughts on this subject, have really made me think, in regards to what is going on with the number of incidences of AF. We, as a society, love our caffeinated beverages, alcohol and salty foods, not to mention the easy, processed crap, we call food. I, for one, have not been much of a water drinker, and never have. Yes, I would hydrate, after a game of basketball, but during golf, I would find myself reaching for a beer or a pop, where AF seemed to hit me most often.

When drinking that cola or beer with a hot dog or any other sodium concentrated food, that so many of us do, not only are we consuming caffeine or alcohol that disturbs our ocean within our bodies, but we are not balancing the sodium with potassium. It is quite a simple concept. The effects of doing just these two wrong things in our diets, day in and day out, are exponential, in their effects of the mechanisms of our bodies. This is the one thing, more than likely, that we all share as a commonality, however the damages done, may be more far reaching for some than others, and this is why they won't see lasting results, until they stick with it for the length of time it takes. Patience.

The diet must change, to more wholesome foods and hydration, and then in order to help the process along, one could take the vitamins and minerals, and probably should, unless they react to them. All alcohol and caffeine should be eliminated in this healing process. I, personally, still feel that aminos will help in my case, but until I put this to the test, I cannot fully represent it, but will continue to do research on it, and post.

Thank you for the update, PC, and your ongoing, guinea pig style of research. Wheet!! Wheet!! Didn't Blockbuster have a hula skirt on that guinea pig? Ha! I wish you continued success, and I hope your stay in Hawaii will have wonderful memories. I do think you have hit the nail on the head.

Richard

Gregg,
Although I take about 2.5 liters per day, I try not to exceed 600 mg of aqueous elemental Mg total per day. So just dilute the ww concentration you're presently taking to increase daily water intake without increasing the Mg++.
Regarding electrolyte loss with all that water intake, the problem is probably overblown. The human body's ability to regulate homeostasis via neurohormonal controls is underappreciated. A shortfall in water, on the other hand, is a horse of a different color.

Carol,
I used to have to worry about triggering an episode when drinking water in the early AM. No longer.
Health and confidence are a wonderful thing. Like you, I'm very content to live the rest of my life with such mild, infrequent and shortlived episodes. As Rod says, I'll have to wait to see if it lasts.

Pam,
If you keep the total elemental Mg intake per day to around 500-600 mg, you should never have to worry about early AM type 2 calls. The formula for Waller water should be available somewhere on this website. In short if you mix MoM with soda water (to its saturation point - I separate a liter of refrigerated soda water into two 1 liter containers and then pour in enough MoM for it to bubble back up to the container's opening. Cap immediately and wait for the sides of the plastic container to cave in and for the mixture to separate with the excess MoM at the bottom. This latter can be reused for the next batch) This results in a mixture that contains 1500 mg of Mg++ per liter. So mix about a sixth of a liter of this concentrate with enough drinking water to take the volume back up to a liter. This will result in a daily intake of about 500 mg Mg++ when drinking 2 liters of this final brew. However, there are other ways to create aqueous

magnesium (much more bioavailable than that taken in tablet form).

Richard,
I'm glad you mentioned patience, which is definitely not my strong suit. I just hope everyone appreciates that improvement is not immediate. It takes weeks for neurohormonal mechanisms to downregulate. This was pointed out by Hans in "the play".

Like you, many of my episodes occurred upon sitting down after walking 18 holes of golf. This was definitely more frequent during the summer, when the weather was warmer and fluid loss more appreciable. I also play a lot of golf in the winter and episodes triggered in this manner seemed to disappear.

Regarding the hula, as stated early, I plan to do it full time next year. However, for now I've shed my grass skirt and changed back into long pants.

PC

PC I just had to repeat your sentence

"As I stated earlier, I still think that for some LAF may be "cured" but the propensity will forever lurk in the background"

This is the case for me. I believe I will always have the propensity for AF. But I know how to avoid it and will continue for the rest of my life.

I always put my AF free staus down to avoiding free glutamate. But what I also did at the same time was learn to drink lots of water (just mineral water with a higher Mg ration to calcium) and eat lots of fresh veggies, nuts etc (for potassium and Mg). So maybe it is just as you say - and it makes good sense.

When I forget to hydrate it makes my heart feel sluggish and slurry, which brings on lots of PACS and PVC's. Would hate to think what a dose of free glutamate would do to me in this condition.

I hope this works long term for you too.

Fran

PC, I thought your concern about drinking water in the early a.m. came from the swallowing process, not from the water, that might act as a trigger. Have you changed your mind about that swallowing?

Gregg

Gregg,

No, I haven't changed my mind on that. You're right. It's not the water, it's the act of swallowing that often used to trigger episodes for me during times of high vagal tone. However, now that my intracellular K seems to have improved (I plan to run an intracellular mineral analysis to actually evaluate this soon), my recent threshhold for triggering a vagally mediated episode appears to be higher, i.e., such episodes are less likely. During the past 24 AF free days I've invoked all the usual triggers (snorkeling, sex, 5 hour plane flight, golf, early AM swallowing, etc.) without "success". This affliction in some LAFers appears at least partially controllable. However, short of an ablation, which denervates the heart autonomically, controlling dispersion will always be our cross to bear.

PC

Dear Carroll,

i am absolutely flabbergasted and filled with joy to read your little note..... i have been suffering with a-fib and tons of pac's since 1986..... i have been thru tons of meds and the only one that worked for many years was TAMBOCOR.... but in the last thee months that has seemed to change.....
two weeks ago i had the worst and longest episode of a-fib ever..... it was at the point where i prayed to god to either fix me or kill me.... needless to say i was at the end of my rope..... the doctor upped my tambocor as a last ditch effort before wanting to do a radiofrequency ablation.....the part that flabbergasts me is that there seems to be some validity to the use of magnesium and water and I HAVE NEVER BEEN TOLD THIS !!!!!

at this point i don't care why i just want to learn more about it... where did you get yor information and what do you do that leaves you virtually a-fib free..... how does one find out if their magnesium level is to low or does that have anything to do with it, are there any other little changes such as this that can help,,,, because i think, besides a ton of stress in my life that my diet has something to do with my episodes,,,,, where do i go from here

thanks
Paul

Hi, Pam
I take Solgar brand Chelated Magnesium glycinate. Each tablet is 100 mg.
I take two in the morning, three at lunch, two at dinner = total 700 mg. I tolerate 700 without gi upset. I may try to increase it to 800 once I have completely established this level. I always take supplements with a meal.

You have to start at the average dose, 400 mg. and gradually increase it.

Glycinate is considered, I believe, the best form.

Good luck.
Carol

Hi, Michael
See you are still at 800 mg. Magnesium Glycinate.
How much potassium are you now taking?
Carol

Dear Paul,

I hope that what has worked for me will help you. There are a number of other individuals on this BB, who appear to be traveling this route. They will probably have further suggestions. Hans has a lot in his book about alternative ways of handling afib. I am not an expert, but have done a lot of studying and thinking and experimenting.

One has to learn to work closely with one's mind and body. Listen, be kind to yourself.

It takes a long time to reverse the mind/body chemistry, which has been insulted over a long period of time by a number of bad health practices and situations. So PATIENCE, vigilance and discipline are necessary. You mustn't get discouraged. You are just taking extra good care of yourself. Think of being good to yourself, the way you might take care of a fine car or yacht or your house, whatever.

Try implementing these new habits slowly. Take one thing at a time. For example, it is amazing how you CAN eliminate bread from your diet. Instead of a sandwich, discard the bread and have the filling as a salad, for instance.

Eat only vegetables, fruit, fish, poultry, meat and some whole grain cereal, as ORGANIC as possible. I eat organic, low fat red meat only occasionally. Eat lots of sardines and salmon. Eating organic and salmon are expensive, but so are

drugs and ablation, which can damage your health.

NO JUNK FOOD - you know what that is. No soft drinks, potato chips, snacks, candy, cake, bread (yes, bread) , crackers, cookies - and no processed food or most of the stuff stuffing the shelves of supermarkets.

Cut down or eliminate white carbs - rice, potatoes, bread.

No added sugar.

I happen not to eat cheese or cream, because about ten years ago, I developed an intolerance to lactose, but I do use lactaid organic milk on my whole grain cereal, which has berries or fruit. I add lecithin granules to my cereal.

Be aware of possible food allergies - milk, wheat, citrus, etc. They may be implicated.

Are you sensitive to tyramine? Do a search on the subject.

NO MSG. It is added to food in many guises. Read up on it, do a search.

I use lots of olive oil, very little butter, no margarine. Mayonnaise made from canola oil.

Eat almonds and raw pumpkin seeds for snacks.

Magnesium glycinate. Start at 400 mg. and work your way up to about 600 or 700 mg. slowly. Take all supplements with a full meal.

Take Norwegian fish oil in capsule form.

Take Flax oil or grind flax seed in a coffee mill.

I take Taurine, 60 mg.CoQ10, L-Carnitine. Basic - B vitamins, a full multi vitamin, L-lysine, Buffered Vitamin C, Daily Ginger. I take aspirin and vit. e only when I have had an episode. Potassium - I try to get it through food, but when I think I might be low, I take a few 99 mg. tablets of potassium.

NO ALCOHOL. NONE. Alcohol also affects the blood vessels adversely. Sulfites in wine can be a real problem.

8+ glasses of water. That is water, not juice.

Limit caffeine to one cup and eventually 1/2 cup. If you are a real coffee drinker, cut back on caffeine, very slowly. If you reduce it to quickly, you can get severe withdrawal headaches, which just goes to show you how strong a drug caffeine is. Caffeine causes the blood vessels in the head to restrict and dilate. (I wonder if the same caffeine mechanism is not operating in the pulmonary vein, triggering off afib attacks.)

8 hours of sleep.

Walk every day, filling your lungs, stretching your legs. Also consider working out at a gym, but slowly and easily. Do not over exert.

Do breathing exercises - there are lots of books out there on this subject. Dr. Weil has a good summary of them.

Stretching exercises. There are books on this subject. Yoga is wonderful.

Find and address hidden and more obvious stressors with an intelligent, truly caring, totally interested, perceptive therapist. Find one with whom you feel totally comfortable and at ease. I have a therapist who practices integrated, wholistic therapy in which Reichian principles relating to breathing is central.

Eliminate the stress of violence and conflict on TV and in movies.

Read everything you can find on current thinking in nutrition. Become an expert on nutrition. Pride yourself on it.

Keep "T.P.'s (my expression for toxic people!) at arm's length in your life and find people with whom you feel totally relaxed and comfortable.

Since I had vagally mediated afib, I found that taking it easy or winding down at the end of the day was important. I try to socialize and entertain in the middle of the day. This is not always convenient for others, however.
This is something like working with your vagus nerve, which if you are vafib, is highly sensitized to begin with.

It all takes time. Remember all these changes are excellent for your general health. This is probably the way everyone should be eating. Hopefully these changes will stop the monster afib in its tracks. It has worked for me.

I try to keep four hours between eating a LIGHT meal at night and going to bed.

Prop a soft pillow under your left shoulder/back for sleeping if sleeping flat or on the left side brings on afib. This may not be necessary as your body/mind begins to heal.

Take charge. Take care.
Carol

Solgar doesn't make a GLYCINATE magnesium, that I know of. They make a GLUCONATE form, which is different than that found from Carlson's or Metagenics, both of which use GLYCINATE. The Carlson's is a good buy, in that each capsule contains 200 mg, rather than the 100 found in Solgar and Metagenics.

Jerry

Carol--

Regarding potassium, recently I've run out of my potassium tablets but am eating lots of fruit, including bananas, which I assume are more than sufficient for potassium intake. Besides all the ripe peaches and cantaloupes I am eating taste better than any tablet.

Michael in SF

Hi, Paul,
More suggestions to keep Afib at arm's length:

With respect to the "modern medicine" not informing you about the benefits of magnesium, when I had my first episode of Afib 10-11 years ago, I started on my rounds of doctors. Not one mentioned magnesium, diet, hydration, etc. When I brought up the subject of magnesium on my first visit, the cardiologist at Massachusetts General Hospital quickly dismissed the idea. Partly because of her response I believe I spent ten years needlessly in afib. ?!#!!! What more can I say?

I assume that none on this BB smokes!

Turkey is good for you. Organic turkey is best.

Fried foods are bad for us, as PC reminded us.

Best, ***Carol***

Carol:

Great job! I'm going to copy your post. I think there is something in it for everyone (probably everything in it).

I have such a hard time taking out all carbs. No bread or crackers or pasta. Then I wind up filling up on meats. Whatever it is, chicken, whatever. Snacks, I love Triscuits, and buy the reduced fat variety and, according to Adkins you can subtract the fiber from the carb content and then I don't feel so guilty.

My cardiologist recommended to me the Mediterranean diet. Do you know anything about that? and how close do you think it is to your plan?

Pam

Carol

(very quiet voice). I smoke. Wish I didn't but just don't seem to be able to quit. I often wonder if the nicotine helps me? Nicotine patches have been recommended by some with vagal AF and I think there is something in it. When I did give up many years ago my AF got worse. I remember thinking that if ciggies were bad for the heart - then why when I gave up did mine get worse.

All told though I have stopped AF through diet alone. No supplements or anything. But maybe the nicotine gives this an extra edge?

I'm not recommending it.

Fran

Paul, if you want to get tested for magnesium and other Electrolytes go to

www.exatest.com

They have a process that tests the intracellular levels of magnesium potassium calcium sodium etc... As opposed to the normal electrolyte tests that are done by blood serum tests.

As only 1% of mg. and 2%of K are in blood serum it's really not indicative of your body's true levels.
If you get tested let us know.

Have any Canucks out there been tested yet and what are the costs?

Adrian v49 (frugal not cheap)
