

**Your Premier Information Resource for Lone Atrial Fibrillation
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VIRTUAL LAF CONFERENCE

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SUBJECT: MVP, ANP, P Cells and Hydration in Relation to LAF

Some may consider what follows as a stretch of the imagination, if not a load of something else. Apologies in advance for any perceived yet unintended complexity in terminology or concepts. Just skip to the summary if you feel overwhelmed. Much of the material represents recently reported findings. It seems that oftentimes the research physiologists don't read the research results electrophysiologists publish and vice versa.

MVP AKA mitral valve prolapse has been previously (February 2004) discussed in the CR (<http://www.afibbers.org/conference/session25.pdf>). Jackie at Hans' suggestion presented a comprehensive discourse on it. Thank you Jackie.

However, MVP need not be present for you to benefit from reading what follows. If you are tall, have long arms or PACs upon vagal maneuvers, light headedness upon standing (orthostatic hypotension), bradycardia, leg cramps, migraines or a family history of AF, then read on.

I did not participate much in the first MVP discussion, despite mild MVP and trace to 1+ mitral regurgitation having been noted on a 2D echocardiogram done in 1/01. A subsequent independent pre-ablation TEE in mid 2005 reiterated this finding. In years past the diagnosis of MVP (primarily by auscultation) has been overdone, at least until the emergence of echocardiography. Nonetheless MVP is fairly common with estimates of about 10% of the population being commonplace. Revisit your previous echo reports and read that portion which pertains to your MV. Revisit Jackie's CR post for the typical features of MVP syndrome (MVPS), e.g., pectus excavatum, myopia, visual migraines. The typical patient is generally taller and often female. Although the discussion is really about mitral regurgitation (MR) as a cause of increased left atrial pressure, MVP is the most frequent cause of MR.

MVPS and LAF

LAFS-11 underscored not only the predominance of LAF amongst males but also its affinity for the tall and thin. LAFers responding to the survey are almost three inches taller than their normal counterparts. MVP is an affliction of the tall. Most frequently their arm span exceeds their height. Sounds like a good basketball player to me (Bill Bradley, Hakeem Olajuwon have LAF). Marfan's Syndrome is considered one end of the MVPS spectrum. Abraham Lincoln is purported to have had Marfan's. Could unappreciated and undiagnosed MVPS be the explanation for greater height amongst LAFers? PM Tony Blair developed LAF prior to receiving a diagnosis of MVPS. His measured height of 6 feet falls far short of his real stature.

At some point one must ask the obvious question: Why is LAF so much more common in males, especially since MVPS is more frequently encountered in females? The explanation for this may rest with estrogen. Estrogen downregulates AT1 expression, a prime determinant of atrial mechanical stress. AT1s also mediate ANP release. One study of LAFers demonstrated increased AT1 receptors in the left atrium, but not the right atrium compared to normals and those with pathologic AF. Further gender disparity is created by mitral regurgitation, which is much more common in males with MVP than in females with MVP.

According to a 2006 article entitled "The Relationship Between Stature and the Prevalence of Atrial Fibrillation in

Patients With Left Ventricular Dysfunction”, the latter are at least an inch taller than their normal counterparts (v. two to three inches for LAFers). However, they are not only shorter than their LAF counterparts but also more plump (mean BMI of 28.7 v. 25.9). In fact the connection between increased sympathetic tone and central obesity appears to rest with baroreceptors. In pathologic AF (increased sympathetic tone) there is more abdominal respiration, whereas in LAFers the breathing method is thoracic respiration. The latter creates greater pulmonary distention and cardiopulmonary baroreceptor stimulation => greater vagal tone.

Dual Substrate Theory

Many AF experts, e.g., David van Wagoner at the Cleveland Clinic, have subscribed to the Dual Substrate hypothesis – one substrate for triggering AF and another for maintaining it. All AF episodes are preceded by at least one PAC and greater numbers appear to be required to trigger LAF than pathologic AF. Increased autonomic tone seems to create the substrate for maintaining LAF, while fibrosis is the most likely candidate for maintenance of pathologic AF. The former is a transient condition, while the latter is ever present. But perhaps their shared height provides a clue that they share the substrate for initiation of AF. The vagal withdrawal (Bainbridge reflex – see below) that accompanies venous return (greater in the tall) could easily stimulate atrial ectopic activity. Furthermore, there is no law stating that tall and thin LAFers with MVPS must remain tall and thin, as they age.

Atrial Natriuretic Peptide (ANP)

Atrial distention or stretch AKA mechanical stress is essential to the development of all AF. Hypertension is the greatest risk factor for future development of AF, thereby underscoring the role of stretch. Although aortic and/or mitral valvular disease are also prominent players in creating atrial stretch, only mitral valvular disease does this without involving the ventricles. For me this is a distinguishing feature for LAF v. pathologic AF. The key is ANP. Established pathologic AF has increased renin angiotensin system (RAS) activity and decreased ANP activity, whereas in LAF this pattern is reversed, at least for LAF secondary to mitral valvular disease. For this reason those with the former have increased blood volume and those with the latter have decreased blood volume. This explains the orthostatic hypotension and low HR and BP seen in MVPS. It also explains myriad symptoms due to Mg deficiency. Consider the following:

- 1) ANP inhibits aldosterone secretion via RAS, ACTH or blood K⁺/Na⁺
 - 2) ANP promotes urinary sodium and magnesium wasting
 - 3) Aldosterone and angiotensin both have vagolytic properties => ANP is vagotonic => bradycardia, increased HRV
 - 4) Low blood volume and bradycardia => increased catecholamine secretion to maintain BP
- (Note: Caffeine and alcohol => diuresis =>? LAF trigger)

The finding of low or low normal intracellular magnesium (range 30-35 mEq/liter v. normal range 34-42) in all seven LAFers responding to that question in LAFS-11 coincides with the frequent finding of magnesium deficiency in MVPS.

Vagal Reflexes

Ion channelopathy research as a cause of most cases of AF appears to have taken a back seat to that on vagal reflexes, which are presently a hot topic with many investigators. Please see pp. 90-91 of Hans' LAF Latest Research – 2004 for a discussion of vagal reflexes and PVI. In fact there is an upcoming conference at the Fairmont in San Francisco June 7-10 by the International Society for Minimally Invasive Cardiothoracic Surgery (ISMICS) (<http://www.ismics.org/abstracts/2006/18.cgi>) that underscores this. In fact channelopathy appears to have been discarded as a cause of AF in general. "Atrial Fibrillation: A Question of Dominance?" discusses this latter point and is a good review of the present state of affairs. http://www.hcs.gr/Keimena/6_2004/eng/2004_6_345.pdf

These vagal reflex foci are often the source of the dominant frequency (DF) triggering AF episodes. When ablated atrial fibrillation cycle length (AFCL) increases. For most people, especially older patients, these sites are located in the PVs and are effectively eliminated by PVI, thereby terminating AF. However, in younger patients, especially those with persistent or permanent AF, extrapulmonary foci are often present and must also be ablated to terminate AF. If such sites are identified and ablated, PVI success improves dramatically. In males these sites extrapulmonary foci are most often located on the posterior wall of the left atrium near the PVs. In females such sites are much more common in the SVC.

Ablation of the vagal reflex focus requires elimination of the vagal ganglion cell (receiving the type B receptor impulses – see below) in the immediately subjacent epicardial fat pad. This not only eliminates the trigger substrate but also should beneficially modify autonomic tone, the maintenance substrate for LAF.

Type B Atrial Baroreceptors

ANP is secreted by type B atrial baroreceptors (afferents or sensory fibers) similar to baroreceptors in the lung. Both are Group C fibers, which means that they are not insulated with myelin. In the heart they are located near the venoatrial junctions (PVs and SVC and adjacent atrial tissue). Type A fibers are stimulated by atrial contraction and type Bs by atrial filling or distention. These fibers (v. type A baroreceptors) discharge with high frequency at around 10 times per second (10 Hz) but the rate is directly proportional to the degree of distention. Release of ADH is tonically inhibited by afferent B-receptor activity => decreased ADH secretion (pituitary) in MVP, which further aggravates ANP induced low blood volume. Interestingly during pregnancy, when blood volume is increased 40-50%, type B fiber discharge is reduced. This would then represent the exact opposite situation to MVPS. And there appears to be a connection between the high frequency discharge of type B receptors and the high frequency component of HRV (controlled by efferent or motor fibers). The magnitude of high frequency afferent receptor discharge follows a gradient with the highest frequencies in the left atrium, decreasing as one proceeds to the right atrium.

Furthermore, type B atrial baroreceptors mediate two reflexes – the baroreceptor reflex (identical to the more potent carotid and aortic arch baroreflex) and the Bainbridge reflex (a subset of type B fibers that are myelinated determine this reflex). The BB reflex involves vagal and sympathetic activity and can only be elicited at low HRs. When HR is low and venous return is enhanced (vagal maneuver), the BB reflex => increased HR. Consequently at low HRs these two reflexes are antagonistic. A vagal maneuver should increase HR via the BB reflex but decrease it via the baroreceptor reflex. At very low blood volume the baroreceptor reflex should predominate, resulting in a vagal reflex (Behold-Jarisch reflex), e.g., bradycardia, asystole, AV block (manifestation of the Behold-Jarisch reflex).

P Cells

These type B receptors are afferent or sensory fibers, but should theoretically be able to trigger an impulse in an immediately adjacent pacemaker type cell. Until recently pacemaker type cells (P cells) had only been described in the SA and AV nodes. However, the Cleveland Clinic was the first to demonstrate ectopic P cells in PVs (histologic exam, pharmacologic challenge) and to implicate them as the probable trigger substrate. Others have also indentified them in PVs (histologic exam, HNK immunostaining, leu-7 immunostaining) and the SVC (histologic exam), as well as sites within the atrium, e.g., coronary sinus (histologic exam, HNK immunostaining). It appears that about 10% of the population carries these ectopic P-cells. Whenever these cells are found in areas identified as the source of AF, the tissue appears to be locally dilated => mechanical stress. Their discharge rate as the site of DF is similar to that of the type B atrial baroreceptor that are also associated with areas of mechanical stress. Could the 600 cycles per second receptor cell discharges be transmitted in the vulnerable 10% to their immediately adjacent ectopic P cells? The fact that these type B receptor fibers are not insulated is rather provocative for this scenario. Alternatively the “cross talk” could occur in the intracardiac vagal ganglionated plexi that receive input from efferent sympathetic fibers (motor) as well as afferent fibers. Thus, both discharge frequency and anatomic location seem to incriminate both type B atrial baroreceptors and ectopic P cells in the generation of DF foci (vagal reflex sites).

Summary

ANP appears to be integral to the development of LAF. The vast majority of the time this appears to be due to MVPS and mitral valvular regurgitation, seen in a large segment of the population. The former is seen primarily in females and the latter primarily in males. The discrepancy appears to be due to the protective effects of estrogen. The height and BMI findings of LAFS-11 and the features of typical MVPS indirectly support this interpretation. Depressed intracellular magnesium levels found in LAFS-11 coincide with the well described magnesium deficit so frequently encountered in MVPS.

Therefore, LAF appears to be due to low blood volume and HR, whereas pathologic AF appears to be due to high blood volume and HR. Vagal tone with catecholamine sensitization (adrenergic LAF) characterizes the former, while sympathetic tone with catecholamine desensitization characterizes the latter. The combination of bradycardia and low blood volume conspire to trigger atrial ectopics due to two antagonistic reflexes.

Ablation of vagal reflex sites, critical to the success of PVI, represent foci where atrial stretch receptors have become activated due to chronic local mechanical stress secondary to MV regurgitation or the equivalent, and these rapidly firing sensory nerves have come into close proximity to previously existing pacemaker cells. The latter are not nerve cells but specialized cardiac cells that represent the end target for efferent or motor signals from either arm of the ANS. Such ectopic P cells are seen in about 10% of the population and in this model may represent the rapidly discharging

cells with the dominant frequency (DF) that drive AF. Ablation of the vagal ganglion cell (receiving the type B receptor impulses) in the immediately adjacent fat pad is essential to termination of AF. This not only eliminates the trigger substrate but also should beneficially modify autonomic tone and the maintenance substrate in LAF.

Dual Substrate Model for AF

The first two conform to the rotor theory of AF AKA focal AF and the last to Moe's multiple wavelet theory (>5 wavelets required). Trigger substrate for I. and II. requires ectopic P cells present in about 10% of the population. PACs precede all episodes.

I. Isolated single episode

A. Trigger substrate - low blood potassium due primarily to aldosterone secondary to B.

B. Maintenance substrate - significant hypovolemia, e.g., dehydration or diuresis due to alcohol, caffeine, "water pill" (hydrochlorothiazide) for BP.

II. Lone atrial fibrillation (both vagally mediated and so-called adrenergic)

A. Trigger substrate – low potassium or vagal maneuver (Bainbridge reflex)

B. Maintenance substrate – increased vagal tone, e.g., MVPS, aerobic fitness.

III. Pathologic AF

A. Trigger substrate – low blood potassium

B. Maintenance substrate – atrial fibrosis secondary to prolonged RAS activity (high blood pressure) or ischemic heart disease.

LAF

MVPS type - Low blood volume and HR characterize early VMAF. With time and increasing mechanical stress these parameters worsen, requiring an increase in catecholamines to maintain BP. The Bainbridge reflex becomes more prominent with resulting increased PACs at first only with vagal maneuvers, but later triggered by catecholamine sensitization, e.g., stress, exercise. More frequent episodes mean more ANP secretion and a vicious circle => "AF begets AF".

Mild hypertension type – episodes are determined more by trigger substrate than maintenance substrate, but still driven by mechanical stress and increased ANP production. Although blood volume may be low, HR is not. Potassium balance is more critical in triggering episodes.

Pathologic AF

If LAF is then combined with weight gain (central adiposity) and/or an increase in BP, then LAF => AF over time. Such individuals not only eventually develop insulin resistance but also catecholamine desensitization. Consequently blood levels of both hormones increase to attain the necessary result. RAS activity also increases thereby contributing to atrial fibrosis.

Adrenergic v. Mild Hypertension

Adrenergic LAF is probably a variant of typical VMAF. They both probably share low blood volume, and during episodes low blood volume induced catecholamine secretion probably results in increased conduction velocity and eventually episode termination. They appear to have greater vagal tone than normals, as evidenced by Hans' prominent HR acceleration on swallowing (swallow reflex). Although most commonly stress and occasionally exercise can trigger PACs and episodes, so can vagal maneuvers.

LAF associated with mild hypertension is probably less frequently triggered by a vagal maneuver. There should also be some BMI differences with slightly higher values in those with mild hypertension. Perhaps the "big pee" in adrenergic LAFers may be another discriminant. And if all else fails in differentiating the two, just take a BP reading.

Additional Questions

Can early LAF be arrested by an assiduous regimen of Mg, K and hydration?

Would ARBs (angiotensin II type 1 receptor blockers) prevent ANP secretion or reduce mechanical stress in worsening LAF?

Does GERD or the equivalent cause LAF via direct fiber "cross talk"? or does it cause a BB reflex and PACs similar to

the swallow reflex?

PC

PC,

Ke aloha nā!'

Nice work on the afib GUT (Grand Unified Theory)!

This certainly seems to fit me:

- A. Trigger substrate - low potassium
- B. Maintenance substrate - increased vagal tone, e.g., aerobic fitness

"Can early LAF be arrested by an assiduous regimen of Mg, K and hydration?"

I've certainly thought this was the case in my situation - though I'd add taurine into the mix as it was during its absence that I had my 2 most recent afib episodes (now over a year ago).

Mahalo,

George

Aloha George,

You've ruined it for me. Now I can no longer conduct a soliloquy. This conversation now has to be expanded to include someone else - you.

Given the frenetic exchange of ideas on this topic I thought I might try to wedge in another post. Clearly the first one needs some translating. You are apparently the only one that can read hieroglyphics.

To simplify my model, let me rephrase it.

All recurring AF is associated with increased atrial stretch. And stretch begets more stretch. Anything that backs up the plumbing - any degree of hypertension, heart valve disease, etc. - will increase atrial stretching and ANP. The ANP will then lower blood volume in an attempt to address what the stretched atria perceive as too much blood volume.

Renin and angiotensin are vagolytic. ANP inhibits both => ANS balance is shifted toward more vagal tone. Increased vagal tone => lower HR. So, now we have low HR and low blood volume and that causes a problem. Only under these conditions are these two antagonizing atrial receptor reflexes brought into play. One is increasing vagal tone at the same time the other is increasing sympathetic tone. The former is predominantly juicing up the maintenance substrate (shortening the refractory period) and the other is juicing up the trigger substrate (PACs).

This antagonism is like an ANS tug of war. There are some in whom the sympathetic arm appears dominant (adrenergic), while in others the parasympathetic arm is top dog (VMAF). Because there are usually 3 times as many baroreceptor reflex fibers (vagal) as Bainbridge reflex fibers (sympathetic), stimulation of this area during ablation causes a vagal reflex. Some appear to be more sensitized to catecholamines, i.e., stress (adrenergic) and others more susceptible to vagal maneuvers. But it's a sliding scale.

If we stopped here, there would be no AF. However, it appears that in at least 10% of the population sinus node tissue can be found around these atrial receptors. And it is through these cells that the PACs (and reentry) are expressed.

That's it.

We are now experiencing an epidemic of AF. Could it possibly be related to the obesity epidemic and associated increase in blood pressure?

The Nat'l Academy of Sciences says that 80% of Americans are short on Mg. Could this be related to urinary magnesium wasting induced by this epidemic of ever increasing ANP? Perhaps isolated episodes associated with alcohol, coffee and diuretics, esp. those that waste K, are mediated by ANP.

There are plenty of people that can control their AF with K+. A shortage shortens the refractory period and increases PACs, a double whammy. After his successful ablation Hans' blood K improved dramatically. No more episodes => less ANP => less urinary Mg wasting => enhanced K retention.

For a couple of years while avidly monitoring my episodes via my Polar HRM I'd noticed that post episodes my HRV was always depressed and my HR was always elevated v. normal. In other words my vagal tone was low. It appears to me that the explanation is the vagolytic effect of the renin and angiotensin. During an episode there is even more atrial stretch and even greater secretion of ANP. The big pee early on further depletes blood volume. Light headedness is even more pronounced. The RAS cannot be suppressed forever and increased ANP production cannot go on forever. Catecholamine secretion is likewise being increased to support BP on the dwindling blood volume. Once this limit is reached the episode terminates. There is no more stretch and ANP secretion drops precipitously. RAS and catecholamines are unopposed => increased HR and decreased HRV for awhile post ablation. PACs are also nonexistent for a few hours while the body equilibrates to the new environment sans ANP.

I know that there is a sensory connection between ablated ganglion cells on my heart and those to my GI tract, because almost immediately post ablation I experienced right upper quadrant discomfort that resolved within a few days. I mentioned this to Prof Haissaguerre, but he said nothing. Upon my return to HI I then read a confirming research article on this connection. I'm sure Dean and others with GERD or the equivalent know that there is a connection between the two. It is possible because both arms of the ANS are in close proximity in these discrete epicardial sites and their connecting fibers from there to the endocardium are not insulated.

Hopefully we've gone from hieroglyphics to at least Greek.

PC

I'm sure a few that have waded into the ... and may feel that this discussion is nice and all, but so what?

The what, is the fact that this might open up new treatment modalities. With this burgeoning epidemic of AF the ablation centers can't possibly keep up, not to mention Medicare that will be underwriting much of it.

First of all, anyone with hypertension should leave no stone unturned in an effort to bring it under control. Increased K intake is only part of the recipe for this. Aerobic activity is another, although I'd go light on the marathons.

Secondly, those with mitral valvular disease cannot so easily control ANP. Perhaps a new drug approach might work. Although AT1s are very much associated with ANP production and ARBs might improve the situation, there are others that might be even more successful in this regard.

Endothelin A receptors are more closely related to ANP production and there are ET A receptor antagonists available. Might it help alleviate recurrent AF? No one would dare prescribe such a drug for AF, because that would represent severe medicolegal liability.

Investigation into ablating intrinsic cardiac ganglia is hot right now. Perhaps with better mapping techniques ganglia governing these vagal reflexes can be isolated and ablated without actually entering the heart, i.e., epicardial approach. An approach similar to mediastinoscopy might be used. This might be safer with a shorter learning curve.

Who knows? But understanding the mechanisms is more than just an idle intellectual pursuit.

PC 3 Others 2

"But understanding the mechanisms is more than just an idle intellectual pursuit."

Of course it is. The reason we are all here is that clearly our doctors do not understand why this is happening, and if it were better understood we might not have to put up with this the rest of our lives. Ablation and the maze operation eliminate the symptoms and do nothing about the cause of the symptoms. I am not convinced that shooting the messenger is a smart thing to do.

About mitral valve prolapse, that seems, like afib, to be closely associated with, if not caused by, magnesium deficiency. PC, do you go along with that idea?

PeggyM

Hi PC,

As you have reminded me, I know you did not have good results with IV mag chloride. However the only documented success in increasing intracellular Mg++ levels is here:

<http://www.magnesiumforlife.com/relevantresearch.shtml>

He talks about "Magic Oil", but I believe it is just mag chloride in solution.

I've found a fairly inexpensive source (looking for food grade mag chloride) and it is also used in the making of tofu. So here is the source:

<http://www.herbalhut.com/detail.aspx?ID=5328>

My thought would be to order the 25 pound quantity and use it like an epsom salt bath & see what happens.

As an aside, I looked at my echo results from 9/04 and a trace of mitral regurgitation was reported.

Cheers,

George

Peggy and George,

The number of posts is beginning to overwhelm me.

Peggy,

The chicken M(gD) or the egg (MVP) - which comes first? I personally think that Mg deficiency is caused by the widespread increase in ANP production. As George now reports, his echo revealed a trace of mitral regurgitation (MR). Mine showed trace to 1+ and I believe Hans' showed the same.

MR is medspeak for an incompetent MV wherein blood leaks back into the left atrium during contraction of the left ventricle. MVP is the most frequent cause of this. This leakage slightly increases intra-atrial pressure and thereby stretch => slight increase in ANP.

As we all know MVP and even trace MR are incredibly common, so much so that it's most often just overlooked. The relentless increase in BP due to the obesity epidemic has to be exerting upward pressure on MR. Once AF rears its ugly head the ANP problem is then put on the fast track.

It is very hard to overlook the urinary magnesium wasting of properties of ANP. However, low Mg and K can certainly contribute to hypertension and in that way aggravate MR and increased ANP. Although Mg deficiency might directly aggravate AF, I think it's more an effect of ANP and MR than a cause of either.

All I can say is that you and George better keep to the straight and narrow wrt Mg and K and perhaps taurine. The beast will always be lurking, at least in the absence of a successful ablation.

PC

PC, can you hazard a guess as to what the taurine is actually doing that is helpful in preventing afib episodes? Maybe facilitating the best use of whatever electrolytes are available? Also,

"I personally think that Mg deficiency is caused by the widespread increase in ANP production. "

Not by deficiency of intake?

PeggyM

Peggy,

"can you hazard a guess as to what the taurine is actually doing that is helpful in preventing afib episodes"

See the paper at the bottom of this page:

<http://www.coldcure.com/html/taurine.html>

It is ironic that one of the great sources of taurine is chicken, I guess that KFC is not a great way to get it.

My first exposure to lack of taurine and heart rhythm problems was 20 years ago. I had a 5 yo cat with cardiomyopathy. It was at the time when they just discovered that many commercial cat foods did not have enough taurine in them. We saved the cat with nitroglycerin and digoxin. However, he lived another 14 years & I'm convinced it was because of increased taurine in his diet, not the digitalis that I gave him for the rest of his life.

PC,

"All I can say is that you and George better keep to the straight and narrow wrt Mg and K and perhaps taurine. The beast will always be lurking, at least in the absence of a successful ablation."

I would agree wholeheartedly. There have been a few nights when I've lain down in bed and then remembered I've not gotten my nightly dose of supps. I get up and take them. I think I've gone one morning in 19 months without taking them - trips, camping & everything. I also tend to sample my PVC/PAC rate 2x a day when I meditate, every day - just to make sure something isn't sneaking up on me. Even if I'm sleeping in the backcountry or a snow cave without a "current bush" (electrical outlet) around, I manually take my pulse & count the beats between ectopics. If it is too frequent, I take some more K+ & taurine. I don't add Mg++ so as not to create bowel intolerance.

I'm now looking at my echo results. In addition to the trifle regurgitation, there is no mitral stenosis. It also shows relatively normal left atrial size, may be slightly prominent but almost within normal limits.

George

Peggy, wrt intake v. loss I can only venture opinions. Clearly we're not doing very well on the Mg intake side, but that is so much easier to control and monitor. Mg loss is another story.

I have a hard time believing that those seven respondents on LAFS-11 were all Mg deficient due to inadequate intake, esp in view of the high profile K and Mg carry on this BB.

To address your question on taurine will require a more technical discussion.

Cyclic GMP (cGMP) is a second messenger and mediates the effects of ANP in target cells, i.e., enhanced natriuresis. Adenylate cyclase (for cAMP production) requires Mg, whereas guanylate cyclase (for cGMP production) requires Ca. Consequently, in Mg deficiency the intracellular cAMP/cGMP ratio, normally between 10 and 100 to 1, is reversed. ANP induced natriuresis (and further urinary Mg wasting) is enhanced => a vicious circle.

Jean Durlach, M.D., Editor-in-Chief, Magnesium Research, President of the International Society for the Development of Research on Magnesium, and author of Magnesium in Clinical Practice, suggests that taurine may effect this turnaround (less cGMP and more cAMP). Catecholamines and insulin favor cellular influx of taurine. Taurine is a powerful membrane stabilizer. It also chelates Ca, a Mg antagonist, facilitates maintenance of intracellular K and opposes the undesirable cellular effects of insulin and catecholamines. Taurine plays an important role in Mg deficiency. Ingestion of monosodium glutamate (MSG) can lead to taurine deficiency, since glutamate competes with cysteine (required to make taurine) for cellular uptake.

So, your statement about the colonel and MSG may not be wrong.

PC

George,

You're my idol on keeping to the straight and narrow.

Mitral stenosis is much less common than MR and would suggest past rheumatic heart disease. It should cause much greater atrial stretch and a much higher frequency of AF.

Forget about an echocardiogram giving you any useful information on local stretch, which is what we're talking about. It's like measuring the speed of light with a stopwatch - much too crude.

PDC

PC,

"I have a hard time believing that those seven respondents on LAFS-11 were all Mg deficient due to inadequate intake, esp in view of the high profile K and Mg carry on this BB."

At the time of my Exatest, 10/15/04, I was two months into my 2.5 month chronic afib episode. I also was not nearly as conversant with what to take with supplements as I am now. I really hadn't taken many supplements, to speak of. I've not repeated the test because, as with your results, I'm pretty sure it has not changed. When I can no longer take my 0.8 grams of Mg++/day without bowel issues, it will be time to retest.

It did, however, convince me that Mg was part of my problem, (along with transient low serum K levels). I then proposed to my EP that he:

- 1) convert me;
- 2) I'd try to stay in rhythm with a supplement program (at the time I conceived of this, I was thinking mostly Mg); and
- 3) write me a script for flecainide on demand, as backup if 2) failed.

I was:

1) surprised when he agreed to my suggested protocol (he had previously suggested I just stay out of rhythm as per the AFFIRM trial); and

2) shocked when a loading dose of flecainide (300 mg) converted me after 2.5 months of continuous afib (despite studies showing that you need to take it very shortly after you go into afib).

I then thought to myself, "oh shoot", now I really have to come up with a supplement program! It was then that Hans

published the Results of LAF Survey VII – Part III in the Afib Report. In this survey, there was an "elite" group of 10 afibbers who had observed at least a 99% improvement (average of 99.9%) who had effectively managed their afib for at least 6 months and who had, during the time their protocol had been effective, experienced, on average, less than one episode per six months. This gave me real motivation to research a program for myself. I read all the back issues of the Afib Report, most of the CR archives, Hans's 1st book and spent a lot of time searching the archives of the 3rd board (the 4th had just started). Unfortunately, I was yet to be aware of Peggy's "The List." The synthesis of this led me to my program. It has evolved a bit over time, but the core elements of K+, Mg++ and taurine have been there since the beginning.

George

George,

I think that Mg supplementation might be especially helpful right after termination of an episode, when ANP levels are dropping and hence less magnesium is being wasted in the urine.

But if episode frequency and duration increase, then benefit from magnesium supplementation becomes a much tougher road to hoe.

PC

P.C.

I had an echo taken in Dec. of 96 (my first one)--I had two events of afib, the beginning of my afib career--the echodoppler showed very mild aortic insufficiency and mitral insufficiency but no evidence of shunt and mild left atrial enlargement, everything else checked out fine.

Over the ensuing years I have had a couple more echos, in fact, the last one was taken in Jan. of this year. Everything remains the same, no change in the aortic insufficiency, ejection factor is very good at 65. The past couple of years I have had a lot of afib episodes, but with the pill in the pocket they didn't last a long time.

My grandmother had what they used to refer to as a heart murmur, I don't know if she had afib, but she died of heart failure. My mother, in her later years- 80s- had this murmur, she also had permanent afib in her 80s and died of congestive heart failure at 92.

I want to add I don't completely fit your profile, I am just under 5'3", I am not thin, but not overweight either, I could lose 10 lbs., I do exercise a lot by working in my yard and garden, so I am fit, but you know what, my arms are not short, they are long --oh well, maybe I was meant to be tall. I was a little myopic until cataract surgery corrected that.

About a month ago I resumed taking Propafenone at night before bedtime (as I am vagal), and have gone about a month without fib, I was starting to get it every week or sometimes more.

I do have a comment about the shortening of the refractory period as one of the requirements for afib---doesn't the refractory period vary throughout the day for everybody---lengthening and shortening.

I have increased my mag., am taking taurine, l-arginine which is helping greatly with my palps, they are almost non-existent, I don't take any K supplements, because every time that I have had blood work done, my levels are towards the high side, mag. down towards the low end.

Oh yes, I also have gotten visual migraines--however, I believe the increase in mag. has been helping those as well as they have diminished. Well so far so good, whatever happens it has been a good month.

Liz

Hi Liz,

Thank you for your post.

ANP may not be as prominent a player for some as for others and MVPS is not the only cause of increased ANP by a long shot. As I recall, hyperthyroidism was a significant factor wrt your AF.

"Atrial fibrillation occurs in 10 – 15% of patients with hyperthyroidism. Low serum thyrotropin concentration is an independent risk factor for atrial fibrillation. Thyroid hormone contributes to arrhythmogenic activity by altering the electrophysiological characteristics of atrial myocytes by shortening the action potential duration, enhancing automaticity and triggered activity in the pulmonary vein cardio myocytes."

"Atrial Fibrillation and Hyperthyroidism"

Indian Pacing Electrophysiol. J. 2005;5(4):305-311

<http://www.ipej.org/0504/jayaprasad.htm>

You're absolutely right about AERP changing throughout the day, but all it takes is one period of transient AERP shortening coinciding with increased PACs and instant AF.

Continued good luck.

PC

"Ingestion of monosodium glutamate (MSG) can lead to taurine deficiency, since glutamate competes with cysteine (required to make taurine) for cellular uptake."

I think that is what happened to me yesterday, all right. This is how come heavy taurine supplementation makes me able to eat the Colonel's cheap, tasty crap, because i am supplying plenty of taurine to keep the glutamate from making a deficiency. You know, i have a vague recollection of Fran posting something on this topic a long time ago. She was convinced, and rightly so, that msg and other excitotoxins were what had done her in along with a whole lot of other people. I am going to go back and re-read George Eby's site, too [www.coldcure etc.], as George suggests.

PeggyM

Hi all,

1. I am not myopic - my vision was 20-10 & I could read very fine print till aging and presbyopia took hold.
2. I added in l-arginine for a while, but could not tell any difference.
3. I've never had a migraine (thankfully) and rarely have headaches.
4. Here is an interesting comparison - the lower graph is of my pulse this morning while meditating - 1 PAC & 30 PVC's, a bit ratty, but not a problem. Ten minutes after ending meditation, I did some mild exercise on a lateral, downhill, ski trainer for 20 minutes and a treadmill for 10. I did not hydrate or take any supplements or food in between. In fact this was all done after a 12 hour fast. You will notice the very smooth character of the upper - exercise graph. I suppose this is because of changes in the AERP, or because of the higher heart rate?

http://home.att.net/~g.e.newman/med_ex.jpg

George

PC;

Many sources recommend drinking approx. one half your body weight in oz's of H2O everyday. Is this going to exacerbate Mg wasting even when not in AF? Any idea what would be about the right amount of daily water intake to

balance Mg wasting with proper hydration and toxin elimination?

Larry Zajdel

Hi Larry,

Thank you for your post.

In answer I'll regurgitate my post to Mark on the BB a few days ago. Mark Heyer had noticed that he "was able to completely control the PAF by simply drinking a half liter or so of water at bedtime and again in the AM. In fact, I could turn the PAF on and off like a switch."

http://www.afibbers.net/forum/read.php?f=4&i=8669&t=8652#reply_8669

I had the same experience several years ago. At the time I was going into AF at least every week for about 24 hours. Then I tried drinking 2.5 liters of aqueous magnesium daily. Up until that point in time I'd been drinking only about 1.5 liters per day, so I just diluted it a bit to attain the 2.5 liters. I then immediately went 15 days and then another 16 days and then 33 days without AF. But unfortunately I had a difficult time maintaining it and eventually relapsed to the more frequent AF program. Retrospectively clearly it was the water, not the Mg and not the alkalinity.

Even with straight unadulterated H₂O there shouldn't be much electrolyte loss in the increased urinary output. Of course, continuing to supplement with Mg and K is a good idea.

PC

The book *The Water Cure* suggests drinking 16oz of water 30 min before meals to take stress of the liver.

Todd

PC;

Thanks for the response, sorry for making you backtrack to a previous post.

I am however, a bit confused. Why would copious amounts of water help terminate or prevent AF. To my simple way of looking at this, the extra blood volume created by the extra fluid intake would raise BP and create additional atrial stretch which in turn would release ANP and therefore urinate out the excess fluid. Sounds like a close loop system attempting to maintain control around a set-point range. I'm an electrical engineer and I need to visualize this in terms I am comfortable with.

Larry Zajdel

Larry,

You're absolutely correct in your understanding.

The key point is the BP. Hydration will do two things:

It'll transiently increase blood volume, which will then decrease catecholamines previously required to maintain BP.

This should then block the development of an episode, which requires both low BP and low HR (probably unchanged by hydration). Only under such conditions can these two antagonistic reflexes conspire to create PACs (trigger substrate) and AERP shortening (maintenance substrate). But unless the hydration regimen is diligently pursued, increasing ANP will eventually assure an appearance by the unwelcome visitor.

As far as terminating an episode, hydration probably prolongs it. The only thing that terminates an episode is increasing catecholamines and renin/angiotensin in response to the increasing ANP (big pee => even lower blood

volume) during the episode. At some point ANP loses. The increasing catecholamines increase cardiac conduction velocity so that the wavelet wavefront catches its refractory tail => no more wavelet. So, theoretically dehydration should hasten the end of an episode. But it'll also thicken your blood and possibly increase stroke risk.

PC

Thank you, PC. I always had kind of an inchoate idea that dehydration predisposed me to afib episodes, but i did not know why. I am the kind of weirdo who always wants to know WHY things are. It is a form of mental disease, i think. People who don't have it seem to me to lead happier lives, all told, than us questioners.

And i have another "why is that" question to do with my recent short episode. I thought that it was the gram of taurine i took that hastened the end of that episode [one of the 4-5 shortest ones i ever had, incidentally]. Surely the potassium i took was helpful, but it was within half an hour of taking those supplements, with the taurine among them, that the episode terminated. I remember some discussion on the regular bb a while ago where somebody else [George, maybe?] downed some taurine and the afib converted within some fantastically short time, like maybe 15 minutes. There was some inconclusive discussion about whether the taurine could possibly have acted so quickly. Could it have been the taurine, and should we be trying taurine for a conversion technique?

PeggyM

George, thank you for jogging my memory about that taurine article. I just read it, and i have a vague recollection of having read it quite some time ago, but i did not then know some stuff i have run across since, and that article explains a lot of stuff that has been happening with me. Again, thank you.

PeggyM

Hi Peggy,

Great memory! Here is the post you are thinking of - however I wasn't in afib, just more ectopics than I like:

=====
http://www.afibbers.com/forum/read.php?f=6&i=23974&t=23890#reply_23974

Author: George (---.hln.qwest.net)

Date: 01-18-06 06:31

Hi Bob,

I recently sent my Polar monitor (S810) in for a new battery, so I haven't been monitoring my ectopics as regularly - manually for a minute before I retire & every once in a while with my Freeze Frammer. Yesterday evening the monitor returned in the post. So this morning I monitored my ectopics for 25 minutes as I meditated, as is my normal habit. I had 35 PVC's & 2 PAC's in this time. This is high for me, so hooked up for a minute on the Polar & got 6 PAC's & a PVC in this one minute. As a check, I put on the Freeze Frammer (which is better for real time display). It was giving readings like the Polar. Now these ectopic rates are not good, so I went and took 1.5 grams K citrate (15 tablets), 200 mg magnesium glycinate (1 tablet) and 1 gram taurine powder. I immediately went and sampled my pulse with the Freeze Frammer (no more than 1 minute after ingestion, in fact I was still swallowing the last K pill. The change in character in my pulse was instant. I was now down to 1 PVC in 4 minutes (the length of my sample). Also the pulse was really smooth.

I really have to attribute this to the taurine, as I can't imagine the tablets dissolved fast enough to act in this time. In fact, the Mg tablet is supposed to pass through the stomach & be absorbed in the small intestine.

So, yes I would say my results are basically instantaneous.

George

=====

I've not repeated that experiment as the ectopic rates aren't alarming, but more than I'd like. Also I usually sample 12 hours after ingesting supplements, so it is a "worst case" scenario.

I do think I'm making some progress overall as I just got back a fasting blood test and my 8:30 AM serum potassium level was 4.2 mmol/l. This compares with another fasting test a year ago of 4.1 mmol/l. A year ago, I'd been on my supplement routine about 6 months. Two years ago, the reading was 3.4 mmol/l. This was two months before my first afib episode.

On the day of my first episode, the reading was 3.2 mmol/l.

This goes along with PC's statement above: "Trigger substrate – low potassium." As long as I keep my blood K up, I seem to be in good shape.

My hypothesis is that in the morning, 12 hours after fasting and ingestion of supplements should be a "worst case" for my serum K levels. About a year ago, I tested my serum K at 1 PM, about 4 hours after eating and taking my morning supplements. My serum K at this point was 4.8 mmol/l. I figure this should be a daily high point.

The body has many puzzlements though. The link below compares meditation and exercise readings for yesterday (on the left) and today. I have not been exercising hard recently, however you will notice that my heart rate was in the 60's & 70's during meditation yesterday. As well there were many PVC's. Today, my heart rate (after settling down from putting the cat outside to puke) was in the upper 40's. Yesterday during mild exercise, all of the PVC's completely dropped away. Also I should have been more vagal today as evidenced by the lower heart rate, but my ectopic count was a small fraction of the day before.

http://home.att.net/~g.e.newman/med_ex1.jpg

Always thinking & wondering :)

George

This thread has turned in an interesting direction - what triggers the ectopic sinus node tissue.

Although I've mentioned this before, I'm still working on it and was hoping that perhaps you K and taurine empiricists might make a few comments.

I've been going round and round in my mind about a science fair project for my soon to be 8th grade home-schooled son. The goal is to demonstrate in an objective manner, similar to George's approach, that K decreases PACs (and PVCs).

Originally I thought that this would require a Holter and that other preteens would provide the most relevant message - eat your fruits and veggies. However, a Holter is overkill and smacks of too much help from Dad. Furthermore, a 24 hour Holter on my son revealed no PACs or PVCs (youth is wasted on the young).

So, I think that George is absolutely correct in his approach. The Polar S810 can provide a very acceptable record of ectopics and quick visual inspection enables easy differentiation of PACs from PVCs. Clearly I need a population (other than preteens) and a method to select a population more at risk ectopics. Obviously I can't stress them or give them MSG. Should I target coffee drinkers or perhaps employ some vagal maneuver. I'd get a baseline reading or two, initiate the ectopic producing behavior and take another recording, and then give them say 500mg K (?taurine) and repeat the recording after ? minutes.

Remember, I'd like to keep it somewhat simple and not include the taurine, but ... Targeting a population that might exhibit ectopics over a short monitoring interval may be difficult. But I've been thinking of using the Freeze Framer and selecting anyone that demonstrates a strong "swallow reflex", increased HR upon swallowing => strong vagal tone.

Your suggestions would be most appreciated.

PC

Hi PC,

How about master athletes - runners, bicyclists, swimmers, triathletes & etc? Some of them may have Polar S810's themselves & from all that we know they are more at risk for afib (& probably ectopics) than the general population.

It would also be good, because if you found someone with a lot of PAC's & PVC's, they might benefit from knowing about K+, Mg++ & taurine supplementation on a prophylactic basis.

As to how long to wait, you'd need to study a few by getting a baseline, then monitoring them while they took the supplements and seeing how long it took to act. Obviously powders could be absorbed more quickly than tablets. So you might make up a KCl solution or K gluconate. If you use taurine, use the powder form. I was astounded how quickly my pulse settled down in that January experiment.

You may have not seen my response to Gunnar's post on the board. He asked someone with a Polar to record while they were laying on their right side, left side & standing. Here is my post, but the interesting thing is the heart rate on my left side was noticeably slower. Is this a vagal response?

=====
Author: George N (---.hln.qwest.net)
Date: 05-20-06 19:16

OK,

So, I lay on my back for 4 minutes, then went to my right side for 3 minutes, then left side for 3 minutes, then standing for 3 minutes.

There was some contact problems with the chest strap when I was on my back, so I'd pretty much ignore everything before 4 minutes.

In general up spikes are PAC's and down spikes are PVC's. Starting at 4 minutes - in the right, left and standing section, there are no PAC's, only PVC's. This is not surprising for me as I rarely have PAC's. I have felt strange beats on my left side, but rarely.

This sample was taken about 1 hour after my evening meal and about 12 hours after my last supplement dose.

I'm not sure I'm a great subject for this as I haven't been in afib for 13 months and my heart is pretty stable now.

I'm not sure who has a Polar that hasn't had an ablation. Everybody I'm familiar with - PC, James in the UK, ArtSD have all had ablations.

I think this would be a good experiment for someone who has a less stable situation than mine.

http://home.att.net/~g.e.newman/r_l_st.jpg

Here is a zoom of just the right/left section:

http://home.att.net/~g.e.newman/Rt_Lft.JPG

With vertical scale exaggerated:

http://home.att.net/~g.e.newman/rt_lt2.jpg

You will notice that the left section is a bit lower heart rate (more vagal?).

=====

Unfortunately none of the rest of us have the background to intelligently debate your afib GUT, however at least it is no longer a soliloquy and has multiple posts.

George

George,

That's a good suggestion, but will make it a little more difficult to attain sufficient numbers. I'm not sure the local B-ball coach would accommodate such a request. However, it might certainly provide a good contrast with those of higher BMI.

Your other comments are also much appreciated.

Regarding the vagal tone question and HR changes lying R v. L side, I'll repost my post from January 2005 on this point. But the bottom line is that I think it depends - on time of day of the experiment, time wrt eating, baseline autonomic tone, ...

Right Side v. Left Side

There have been numerous posts on the BB indicating a connection between lying on ones left side (left lateral decubitus position) and triggering AF. I have also read separate reports elsewhere claiming this for either side. So which is it? And why?

A couple of years ago Hans turned me onto an article out of the Japanese medical literature (2002) entitled "Effects of Right Lateral Decubitus Position on Plasma Norepinephrine and Plasma ANP Levels in Patients with CHF". It states that "in the right lateral decubitus position, sympathetic tone decreased, whereas parasympathetic activity increased". But his was in CHF patients and not LAFers.

Then I happened upon another article (1997) entitled "The Effect Of The Lateral Decubitus Position On Vagal Tone". By using spectral heart rate variability analysis they found that "cardiac vagal activity is greatest when the right lateral decubitus position is adopted".

Both of these articles seem to go against the grain of the LAF experience, at least based on the posts in the BB. So in the wee hours of one recent morning I performed a little test. Around 3AM I was awake and became aware of increased PACs. I happened to be lying quietly on my right side at the time. I counted them silently and remained motionless. Slowly I switched sides and waited a few minutes to attain a sufficiently "quiet" state and began counting PACs again. Then I repeated the whole process again on the right side (guess I'd better consider increasing my Mg and Vit B6 (the dream vitamin) to achieve a deeper sleep state at 3AM).

Two interesting observations were made: 1) PACs occurred only when lying on my right side (right lateral decubitus position); 2) PACs occurred almost exclusively at end expiration. This latter observation appears to be related to respiratory sinus arrhythmia (RSA). End expiration is precisely the point in time that RSA would predict this, the time when vagal tone maxes during the respiratory cycle. You'll recall that RSA is an evolutionary adaptation of mammals to maximize gas exchange in the lungs. When the lungs are inflated, alveolar oxygen content is highest and pulmonary resistance to blood flow is least. So the HR picks up slightly to exploit this condition. Inspiration stimulates pulmonary stretch receptors resulting in withdrawal of vagal tone (and increase in HR). This stimulation is least (and vagal tone highest) at end expiration, hence greater likelihood of vagally induced PACs.

So how does lying on ones right side affect RSA? Well, the left lung is slightly smaller than the right lung, mostly because it has to share the left thorax with the heart. In fact the left lung only has two lobes v. three on the right. This translates to greater lung volume on the right than on the left. I think that when lying on ones side the more dependent (lower) lung is less inflated due to its more restricted position. So, when lying on ones right side there is less stimulation of total pulmonary stretch receptors (and greater vagal tone) than when lying on ones left side.

Having said all this, I for one have definitely triggered AF when lying on my left side. How does this then happen, since my LAF is predominantly vagally mediated atrial fibrillation (VMAF)? Well, the stomach lies in the left upper quadrant of the abdomen. It empties into the duodenum, which lies immediately to its right. So, gastric emptying is enhanced when one lies on ones right. Lying on ones left impedes this emptying and leads to gastric reflux, i.e., GERD. Vagal nerves (vagal afferents) from the stomach stimulated by reflux lead to neurons in the brain (nucleus ambiguus) which are in very close proximity to those that receive signals from the pulmonary stretch receptors and the carotid sinus. Although it has not been definitively shown thus far, I think there is crossover stimulation, i.e., reflux causes slowing of the HR.

Thus, in conclusion VMAFers and those with GERD should not recline while they sleep. That's a joke. IMHO they should sleep on their right side for the first couple of hours after eating (longer for larger meals) and then favor their left side for the rest of the night.

ALAFers should always sleep on their right side.

And, if you don't have AF but only a tummy ache, then lie on your right side.

Discordant opinions and/or reports of differing experiences are most welcome.

PC

George,

I hadn't seen the post from Gunnar. Sorry for the delay. I'll copy this to the BB as well.

While using the polar monitor, the results were not quite what I expected in view of the above early morning PAC experiment. Perhaps this is because of my 10+ months ago successful ablation.

While lying on my back quietly for at least three minutes my HR settled in around 50-51 and HRV (rMSSDs) at around 20-21. On my right side the results were 57 bpm and 27 msec. On my left they were 52-53 bpm and 22-23 msec.

So, my results parallel yours, i.e., slight increase in HR while lying on my right v. my left. Perhaps this now means that I'm more likely to escape future episodes.

PC

PC,

Thanks for your response on the left v right question.

Are there any local health fairs? If so, could you set up a table and screen people there, then ask the ones who have a lot of ectopics to meet you at another location for further testing/data gathering at some future time.

Other thoughts might be to make a deal with the pastor of a local church to screen his congregation for ectopics. Any place where people gather regularly - rec center, senior center & etc.

George

Hmmmmmm. Sleeping positions. I sleep against a stack of pillows, and when i have access to a recliner i can sleep very comfortably there. I am considerably overweight, and my knees are in poor shape, and they are most comfortable either in a recliner or with a pillow under them. Needless to say, i do not sleep on either side, but always on my back. To the best of my knowledge, i do not get ectopics while sleeping, or at least not enough to wake me.

Why did i wake up with afib the other morning? I mean, why at 5am rather than any other time after that unfortunate KFC chicken and biscuit meal? I took my 1 gram taurine, 1 tsp K gluconate, 400 mg magnesium glycinate that evening.

Plenty of GERD, for sure. Carbs and msg mixed, GERD guaranteed. I was eating generic tums all evening, made for much more calcium than I usually get. Calcium and magnesium are antagonists, like salt and potassium, isn't that right? Hmmm indeed.

The amount of white flour in the extra-crispy breading and the 2 biscuits should have been counteracted by the K gluconate. Salt and K are antagonists, I just got thru saying. Plenty salt in KFC "food". I think I am answering my own questions here. That dose of K was set against not only the insulin produced by the breading and biscuits, but the unusual amount of salt in that "food".

That brings us back to the dose of msg, of unknown quantity. However much it was, the low dose of taurine was not equal to the task. According to that G. Eby taurine article, that taurine was busily chelating all that calcium [the generic tums] to try to keep calcium out of the cardiac cells, and Mg in them, too. And 5 am is quite a while after I took the evening dose, as well. In fact, I have usually taken my morning supplements by that time. No wonder my poor long-suffering system went into afib then.

OK, I understand that whole thing a lot better. Thanks, guys.

PeggyM

Peggy,

Here are some more abstracts on Taurine:

Author(s): Chahine R ; Feng J

Affiliation: Laboratory of Physiology, Faculty of Medical Sciences, Lebanese University, Beirut, Lebanon. Title:

Protective effects of taurine against reperfusion-induced arrhythmias in isolated ischemic rat heart. Source:

Arzneimittel-Forschung. (Arzneimittelforschung) 1998 Apr; 48(4): 360-4

Additional Info: GERMANY Standard No: ISSN: 0004-4172 (Print); NLM Unique Journal Identifier: 0372660 Language:

English Abstract: The protective effect of taurine (CAS 107-35-7) against reperfusion-induced arrhythmias has been investigated in isolated perfused rat heart (Langendorff method). Partial ischemia was induced by occlusion of left descending artery for 15 min, followed by 10 min reperfusion. Left ventricular pressure and epicardial ECG were continuously monitored before and during ischemia and reperfusion. A control group was submitted to partial ischemia without taurine treatment. Three groups were submitted to partial ischemia, under taurine (10 mmol/l) treatment in the Krebs-Henseleit perfusing buffer during ischemia only (group 1), at reperfusion (group 2) and throughout the experimental period (group 3). Malondialdehyde levels were measured as an index of lipid peroxidation and heart muscle damage. The incidence of irreversible ventricular fibrillation was significantly diminished from 83% (control group) to 36% in group 1, 42% in group 2 and 16% in group 3. The incidence of premature ventricular beats and ventricular tachycardia at reperfusion as well as malondialdehyde levels were significantly decreased under taurine treatment. The results indicate that taurine protects ischemic heart against reperfusion-induced arrhythmias, via both its properties as membrane stabilizer and oxygen free radical scavenger.

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Author(s): Tao L ; Rao MR

Affiliation: Department of Cardiovascular Pharmacology, Nanjing Medical University. Title: [**Effects of enalapril and**

taurine on left ventricular hypertrophy and arrhythmia in renovascular hypertensive rat] Source: Yao xue xue

bao = *Acta pharmaceutica Sinica. (Yao Xue Xue Bao) 1996; 31(12): 891-6*

Additional Info: CHINA Standard No: ISSN: 0513-4870 (Print); NLM Unique Journal Identifier: 21710340R Language:

Chinese Abstract: The effects of enalapril (Ena, 6 mg.kg⁻¹) and taurine (Tau, 30 mg.kg⁻¹) on left ventricular hypertrophy (LVH) and ventricular arrhythmia were studied in two-kidney, one clip renovascular hypertensive rats (RHR). From the 9th week after operation, Ena and Tau were given per oral daily for 9 weeks. These drugs significantly decreased the systolic arterial pressure and the weight of the left ventricle. Combination of both drugs was found to reduce the blood pressure further than either drug used alone. Arrhythmias induced by trains of electrical stimuli were more frequent in working hearts isolated from RHR than that from normotensive rats. Ena and Tau could decrease the incidence of this arrhythmias in RHR. The calcium content in the myocardial mitochondria in RHR was increased compared with that in

normotensive rats. Treatment with Ena and Tau reduced this increase significantly. These results suggest that chronic therapy with Ena and Tau can induce an attenuation of systemic arterial pressure and reduce the propensity of RHR heart to arrhythmogenesis by limiting cardiac hypertrophy and calcium overload of the myocardium.

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Author(s): Tao L ; Wang HX ; Rao MR

Affiliation: Department of Cardiovascular Pharmacology, Nanjing Medical University. Title: **[Effect of angiotensin and taurine on arrhythmia in cultured neonatal rat hypertrophic heart myocytes]** Source: *Yao xue xue bao = Acta pharmaceutica Sinica. (Yao Xue Xue Bao) 1995; 30(5): 326-30*

Additional Info: CHINA Standard No: ISSN: 0513-4870 (Print); NLM Unique Journal Identifier: 21710340R Language: Chinese Abstract: Angiotensin (Ang) II (1, 10, 100 and 1000 nmol.L-1) was found to increase spontaneous contractile frequency dose-dependently in neonatal rat cardiac myocytes cultured for 3 d. After exposure to Ang II (100 nmol.L-1) for 7 d, neonatal rat heart cells became hypertrophy with increased frequency, elevated APA, prolonged ADP50 and ADP90, and shortened SCL. Addition of ouabian (Oua) 50 nmol.L-1 to the hypertrophic myocytes caused more frequent arrhythmia. Taurine (20 mmol.L-1) was shown to inhibit these changes induced by Ang II. These results suggest that Ang II can increase autorhythmicity as well as sensibility to Oua in cultured cardiac myocytes. These effects might be related to the promotion of Ca²⁺ influx.

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Author(s): Shustova TI ; Mashkova Nlu ; Cherkashina EM ; Dokshina GA Title: Vlianie taurina na sodержanie kaliia, kal'tsiia i natriia v krovi i tkaniakh krys.

Translated Title: **[Effect of taurine on potassium, calcium and sodium levels in the blood and tissues of rats]**

Source: *Voprosy meditsinskoi khimii. (Vopr Med Khim) 1986 Jul-Aug; 32(4): 113-6*

Additional Info: USSR Standard No: ISSN: 0042-8809 (Print); NLM Unique Journal Identifier: 0416601 Language: Russian Abstract: Effect of taurine on content of electrolytes was studied in rat myocardium, liver tissue, skeletal muscles and erythrocytes. The balanced electrolytic state of a body was impaired by administration of adrenaline at arrhythmogenic doses, while content of potassium in erythrocytes was altered by means of addition of adrenaline or p-chloromercuribenzoate into the incubation medium. Taurine was shown to normalize the content of potassium and calcium ions in vivo and in vitro.

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Author(s): Franconi F ; Stendardi I ; Failli P ; Matucci R ; Baccaro C ; Montorsi L ; Bandinelli R ; Giotti A Title: **The protective effects of taurine on hypoxia (performed in the absence of glucose) and on reoxygenation (in the presence of glucose) in guinea-pig heart.** Source: *Biochemical pharmacology. (Biochem Pharmacol) 1985 Aug 1; 34(15): 2611-5*

Additional Info: ENGLAND Standard No: ISSN: 0006-2952 (Print); NLM Unique Journal Identifier: 0101032 Language: English Abstract: In isolated guinea-pig heart submitted to hypoxia in the absence of substrate and subsequent reoxygenation 1-20 mM taurine decreases LDH release and ventricular arrhythmias, and the recovery of normal electrical and mechanical activity is increased. The taurine effect is dose-dependent, and is not mimicked by beta-alanine. Moreover, taurine reduces the increase in calcium gain of reoxygenated heart.

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Author(s): Hernández J ; Artillo S ; Serrano MI ; Serrano JS Title: **Further evidence of the antiarrhythmic efficacy of taurine in the rat heart.** Source: *Research communications in chemical pathology and pharmacology. (Res Commun Chem Pathol Pharmacol) 1984 Feb; 43(2): 343-6*

Additional Info: UNITED STATES Standard No: ISSN: 0034-5164 (Print); NLM Unique Journal Identifier: 0244734 Language: English Abstract: The potential antidysrhythmic effect of taurine has been studied both "in vitro" and "in vivo". "In vitro" experiments were performed on a model of automaticity induced in the isolated right ventricle of the rat. The "in vivo" studies have been done on anesthetized rats, assessing the effect of taurine on the electrical activity of the heart by continuous ECG records. Taurine decreases the "in vitro" automatic ventricular frequency. "In vivo" reduces heart rate (P-P interval increases) and conduction through the ventricular myocardium (prolongs QRS interval duration). We conclude that taurine possesses experimentally antidysrhythmic activity "in vitro" and "in vivo", that would warrant further research.

=====
George

Many LAFers have associated dehydration as contributing to episodes.

Dehydration not only worsens MVP but can also induce MVP, esp. in females. In one study a diuretic (furosemide) induced MVP in 50% of women and 10% of men, where there had been none before. This was diagnosed by echocardiogram.

Carol Andrews on the BB

http://www.afibbers.net/forum/read.php?f=4&i=8763&t=8742#reply_8763

and Larry (see above) have both commented on this. I believe Peggy has implied as much in one of her above posts.

Most LAFers are not hypertensive, indeed many are at the low end of the spectrum. In those with low range BP and light headedness upon standing (orthostatic hypotension) should consider increasing their salt intake along with their hydration efforts. In fact those few LAFers attempting to lose weight should be sure not to lose proportionately more fluid. This will only exacerbate your LAF otherwise.

Below is a list of triggers for MVP available on several websites.

- alcohol
- caffeine
- dehydration
- emotional stress
- fatigue
- foods containing sugar
- illness
- menopause
- menstruation
- missing meals
- smoking
- stimulants, such as over-the-counter diet medications
- unusual physical activity

Many of the above are also associated with LAF.

Salt intake for nonhypertensive LAFers deserves reevaluation.

PC

First a comment on PC's above re: blood pressure. As I recall Peggy is hyper not hypotensive.

Also in thinking through my own situation regarding PC's hypothesis for LAFers:

"II. Lone atrial fibrillation (both vagally mediated and so-called adrenergic)

A. Trigger substrate – low potassium or vagal maneuver (Bainbridge reflex)

B. Maintenance substrate – increased vagal tone, e.g., MVPS, aerobic fitness"

I must have gotten to a situation where the trigger substrate was activated through low potassium. There is some indication of this in my historical serum potassium tests. As to maintenance substrate, there were many times in my life when my vagal tone was much stronger than it is today. This was indicated by my resting heart rate. I exercised much more vigorously than now. I conclude from this that the change must have been in the potassium, not the vagal tone.

George

Below, I extracted a few selected quotes from the paper that PC already referenced. Some may not have waded through the paper, but I thought these were worth repeating. Here are the quotes:

http://www.hcs.gr/Keimena/6_2004/eng/2004_6_345.pdf

However, both vagal stimulation and administration of ACh have been shown to result in AF. In experimental animal models, vagal stimulation results in sustained AF as long as the vagus nerve is continuously stimulated, and in dogs catheter ablation of the cardiac parasympathetic nerves abolishes vagally mediated AF. This has been attributed to the heterogeneous distribution of vagal innervation throughout the atria, which increases spatial dispersion of refractory periods.

Indeed, a very recent work from Pappone et al suggests that in patients with paroxysmal AF isolation of the pulmonary veins together with abolition of all evoked vagal reflexes around all pulmonary vein ostia significantly reduces recurrence of AF at 12 months.

In 1995, Wijffels et al in Allesie's laboratory developed a goat model of chronic AF in which the animals were connected to an external automatic fibrillator. The device was programmed to deliver a 1-second burst of electrical stimuli (50 Hz) as soon as sinus rhythm was detected. As such, the automatic fibrillator was able to maintain AF for 24 hours a day, 7 days a week. On day one of the experiment, the paroxysms of AF induced by the fibrillator were short-lived. However, in the continuous presence of high frequency excitation for days or weeks, the rate and stability of AF increased, thus demonstrating that "AF begets AF." Importantly, with the persistence of AF, the atrial effective refractory period shortened and the slope of its frequency dependence became flat or inverted, which suggested the occurrence of a process of AF-induced electrical remodeling in the atria of these goats. More recently, the Allesie laboratory showed that electrical remodeling was not significantly affected by changes in autonomic tone or ischemia and concluded that high frequency activation itself was responsible for the AF-induced changes in atrial effective refractory period. On the other hand, it was shown that the remodeling process is reversible and the effective refractory period normalizes completely within one week of resumption of sinus rhythm. The process of remodeling is reproducible in other animal models of chronic AF. Moreover, recent studies in humans have shown that changes in atrial electrophysiology associated with persistent AF are reversible after cardioversion, which provides convincing evidence for the existence of AF-induced remodeling in humans. However, to date investigators have been unable to rigorously correlate the electrical remodeling process to the molecular and ionic mechanisms underlying the perpetuation of AF.

On the one hand, extensive ablation that is thought to modify the atrial substrate can cure many types of AF, but it exposes the patient to a higher risk of complications and to unacceptable fluoroscopy exposure times; on the other hand, more selective ablations that target localized "triggers" are safer but may be less likely to cure the AF, which may become prone to recurrences.

Since in this study the pulmonary vein region was the fastest of all the regions in the atria, the findings strongly support the hypothesis that targeting the regions that show the shortest AF cycle length for RF ablation may be a good strategy for AF termination. As may be expected in patients with persistent AF, it would not be surprising to demonstrate that the region with the shortest AF cycle length lies somewhere other than the pulmonary vein or the posterior LA.

The highest dominant frequencies (& therefore the shortest cycle length) in the paroxysmal AF patients were located primarily in the pulmonary vein region (4/7) and never in the RA. Interestingly, in the persistent AF patients, the highest dominant frequencies were distributed evenly in the two atria and the coronary sinus and none was found in the pulmonary vein region. We concluded that, in that group of patients, paroxysmal AF was characterized by the hierarchical spatial distribution of dominant frequencies where the LA and pulmonary veins are always the fastest regions. In contrast, in persistent AF, a more uniform distribution of the dominant frequencies was observed, where the highest dominant frequency could not be found in the pulmonary vein region, indicating the loss of this region's predominance. This may have implications for the localization of a target for AF termination in patients.

"As I recall Peggy is hyper not hypotensive."

Yup. Mild hypertension, controlled very well at present [and for a while now] by bedtime aspirin plus half of a 10 mg lisinipril tab each morning.

PeggyM

Hi PC,

I located the results of the Barium Test I had in April 2002. I think you will find it interesting in regards to the stomach changing shape when I move. During the test I also had runs of ectopics while I was being moved about. In regards to the shifting or "wandering" stomach, what is the correct medical term for this as I was unable to find anything on google? The attending doctor said a small percentage (how many?) of the population have this anatomical feature of the stomach not being "connected". I have always thought that this is very significant for a vagal afibber. Is a wandering stomach more prevalent among afibbers? I have brought this subject up time and time again but no one seems to come up with any info or satisfactory explanation.

If your stomach changes shape when you roll over in bed surely this must cause a significant vagal event?
What do you think PC?

Barium Test 22/04/02

"Clinical History

Reflux of food. Fibrillation of the heart when bending down.

A double contrast study was performed initially with barium only followed by a barium impregnated marshmallow at the end of the study.

There was no obstruction to the flow of the contrast to the stomach but there is slow clearing of both the barium and the barium impregnated marshmallow through the mid and distal oesophagus. There is no evidence of a hiatus hernia and free gastroesophageal reflux was not demonstrated during screening.

Initially on screening the stomach the configuration was normal. No mucosal abnormality was detected in the fundus, body or antrum. On lateral views, after moving Mr Rutledge supine, prone and then back to erect, the configuration of the stomach altered with the antrum and duodenum on the erect view now lying above the level of the fundus. The duodenal cap distended well and no focal abnormality was detected. Normal duodenal loop.

Impression

- 1) Slow clearing of barium and barium impregnated marshmallow through the mid and lower oesophagus without evidence of mucosal lesion.
- 2) No gastroesophageal reflux was demonstrated during screening.
- 3) There is valvulus of the stomach with a change in the position of the fundus from supine to the erect position.
- 4) No mucosal abnormality visualized in the stomach, duodenal cap or loop."

Dean

PC wrote:

- >
- > Salt intake for nonhypertensive LAFers deserves reevaluation.
- >
- > PC

Yes I definitely feel the need for some sometimes - even to the point of putting a grain of sea salt on my tongue.

Joyce

Hi PC,

If K+, Mg++ and/or taurine helps someone convert are the minerals (amino acid) effecting the a) the trigger substrate or b) the maintenance substrate. I would postulate a) and thereby taking away a "driver." What do you think?

George

Hi Dean,

Thanks for your post and sorry for the belated response.

Whether it's a wandering stomach (a term I've not heard except thru you) or GERD or hiatal hernia, there are undoubtedly sensory fibers from the upper GI tract that network with the parasympathetic ganglia on the surface of the heart (epicardial). And it's probably a lot more common than we think. However, it doesn't mean a thing, at least with respect to a tachyarrhythmia, if you don't have sinus node tissue around the PVs to complete the loop (motor v. just sensory). For the vast majority positional changes and their effect on the heart are limited to no more than a slight change in HR. This may be the explanation for George's finding.

PC

Hi Joyce.

Thanks for your post.

For many LAFers an increase in salt intake might decrease and/or eliminate their episodes.

If vagal maneuvers are particularly problematic in trigger PACs and AF and your BP and HR tend to run on the low side, this I'm talking about you, especially if you get light headed upon arising from a sitting or supine position. These findings are more frequently encountered in those with mitral valve prolapse and/or regurgitation.

In about 50% of these individuals (MVPS), atrial natriuretic peptide (ANP or ANF) is increased. The others also have low blood volume, but this does not appear to be due to increased ANP.

Mayo Medical Labs is the only placed I've found that does this test and I have an email in to them about the specifics.

So, if the above applies, try adding some salt to your water and just see what happens.

PC

Hi George,

Here's my opinion for what it's worth.

I agree with you that K+, Mg++ and taurine are helpful because they address the trigger substrate problem. K+ supplementation markedly reduces PACs in many of us. Wil, you and I have objectively shown this on multiple occasions.

But there are many people out there that pay no attention to K+ in their diet and yet have no PACs (or AF). Why is that? IMHO it's because they don't have sinus node tissue in their left atria/PVs.

PC

"IMHO it's because they don't have sinus node tissue in their left atria/PVs."

PC, do autopsies confirm this?

PeggyM

Peggy

You asked for the time and I'll build you a watch.

The first two articles on this (both autopsy studies) were published in 2003 within a month of each other.

The first in August was by Natale's group. The second did not demonstrate a statistically significant correlation with AF, but the numbers were small. Testing is expensive. Furthermore, one must remember that many individuals with these sinus node like cells may go on to develop AF.

Pachon et al. suggested this in his article at

http://europace.oxfordjournals.org/cgi/content/abstract/6/6/590?ijkey=649f413c1265c0f5fb8d961446c35e4f6a3a5ab6&keytype2=tf_ipsecsha

entitled

"A new treatment for atrial fibrillation based on spectral analysis to guide the catheter RF-ablation"

He calls these cells AF nests.

"Numerous AF nests were found in 34/34 AF patients and only in 1/6 controls (only in this case it was possible to induce AF despite an absence of AF history).

"Evidence of specialized conduction cells in human pulmonary veins of patients with atrial fibrillation."

Perez-Lugones A, McMahon JT, Ratliff NB, Saliba WI, Schweikert RA, Marrouche NF, Saad EB, Navia JL, McCarthy PM, Tchou P, Gillinov AM, Natale A.

Department of Cardiology, Center for Atrial Fibrillation, The Cleveland Clinic Foundation, Cleveland, Ohio 44195, USA.

Specialized Conducting Cells in Human PV. Introduction: Depolarizations similar to those from the sinus node have been documented from the pulmonary veins after isolation procedures. We assessed the hypothesis that sinus node-like tissue is present in the pulmonary veins of humans. Methods and Results: Pulmonary vein tissue was obtained from five autopsies (four individuals with a history of atrial fibrillation and one without a history of atrial arrhythmias) and five transplant heart donors. Autopsy veins were fixed in formaldehyde and processed for light microscopy to identify areas having possible conductive-like tissue. Areas requiring additional study were extracted from paraffin blocks and reprocessed for electron microscopy. Donor specimens were fixed in formaldehyde for histologic sections and glutaraldehyde for electron microscopy. Myocardial cells with pale cytoplasm were identified by light microscopy in 4 of the 5 autopsy subjects. Electron microscopy confirmed the presence of P cells, transitional cells, and Purkinje cells in the pulmonary veins of these cases. Conclusion: Our report is the first to show the presence of P cells, transitional cells, and Purkinje cells in human pulmonary veins. Whether these cells are relevant in the genesis of atrial fibrillation requires further study.

J Cardiovasc Electrophysiol. 2003 Aug;14(8):803-9

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12890038&dopt=Abstract

"Expression of Leu-7 in myocardial sleeves around human pulmonary veins."

Kholova I, Niessen HW, Kautzner J.

Department of Pathology, Vrije Universiteit Medical Center, De Boelelaan 1117, Room OE16, 1007 MB, Amsterdam, The Netherlands.

BACKGROUND: Atrial fibrillation (AF) is the most common sustained clinical arrhythmia. Myocardial sleeves onto pulmonary veins (PVs) have been recognized as a frequent site of origin of focal triggers for this arrhythmia. Expression of Leu-7 has been hypothesized to correspond with abnormal atrial automaticity. **OBJECTIVE:** To evaluate a possible role of the Leu-7 immunoreactivity in AF patients, we studied Leu-7 expression in myocardial sleeves. **METHODS:** Leu-7 was studied immunohistochemically in paraffin-embedded specimens from 55 human autopsied hearts (mean age 69 years, range 42-94 years, 34 males, 21 females). Twenty-two of the subjects had previous history of AF. **RESULTS:** Myocardial sleeves were found in 151 out of a total number of 220 PVs (68.6%). Leu-7 granular cytoplasmatic positivity was observed in 15 (9.9%) PVs from 12 different hearts: 6 (15.4%) in the right superior, 4 (10.2%) in the right inferior, 4 (10.8%) in the left superior and 1 (2.7%) in the left inferior PV. This finding was revealed both in patients with and without the history of AF. **CONCLUSIONS:** Leu-7 positivity, hypothesized to correspond with abnormal atrial automaticity, can be detected in some myocardial sleeves around PVs. However, no statistical

relationship to previous history of AF was found.

Cardiovasc Pathol. 2003 Sep-Oct;12(5):263-6

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=14507575&dopt=Abstract

These cells have also been "identified" via pharmacologic challenge. Natale showed that in 34 of about 1600 successfully ablated AFers dissociated rhythm (i.e., contained by the PVI) was present. Pharmacologically they were able to demonstrate that these foci were behaving like sinus node tissue.

Taiwanese studies on dogs also "identified" sinus node like cells.

PC

PC et al, i am having trouble understanding these reports. Do they say that these cells have been found in people who did have afib in life, but not in people who did not have afib in life? Or do they say that people who had afib in life have these cells, but they are not sure whether they are also found in people who did not have afib in life? I kind of think it is the latter, but i find these abstracts somewhat difficult to read.

PeggyM

Peggy,

The long and the short of it is that these cells appear to be necessary but not sufficient for the appearance of AF. If you don't have them you won't get AF (lone or pathologic) at anytime in life. If you do have them, then you might get AF.

PC

OK, that makes sense. These cells have indeed been found in the cadavers of people who did not have afib in life, then. Thank you, PC.

PeggyM

Hi PC,

"If K+, Mg++ and/or taurine helps someone convert are the minerals (amino acid) effecting the a) the trigger substrate or b) the maintenance substrate. I would postulate a) and thereby taking away a "driver.""

So, my point is that the supps do not appear to change the maintenance substrate. In my case, I had 20 minutes this morning with an average heart rate of 43 (range 39-46). During this time I had 2 PVC's & no PAC's. I don't think my supps. have kept me from being pretty vagal (either that or I'd have otherwise had a heart rate of 20!). As you point out, you, Wil and I have shown the effect of these supps on PAC rates. Obviously, if you don't have a trigger to initiate afib, you won't get it. However, the ability of the supps to help someone in afib convert to NSR must be from reducing/eliminating the trigger, not by changing the properties of the maintenance substrate (at least as far as being vagal is concerned). But, my understanding of afib is that once initiated, the trigger was no longer needed, the maintenance substrate would still propagate the afib.

Now, obviously, these supps do not convert everybody, but they do help some convert. So is this conversion help from reducing/eliminating the trigger, or is there some change in the maintenance substrate that we aren't aware of?

Just pondering.

George

Hi George,

I wish I knew the answers to your very good questions. I can only offer unfounded speculation.

I think most episodes terminate because of changes in the equation

wavelength = conduction velocity x AERP

Increase either CV (e.g., mild exercise) or AERP (increased ANP => more RAS activity => increased angiotensin and aldosterone => vagolysis => less shortening of AERP and CV) and the critical mass of wavelets cannot be sustained.

A K⁺ shortfall, as you've said, appears to be more of a factor affecting trigger substrate. However, it can also cause the AERP to shorten. A larger gradient facilitates greater K⁺ conductance (more flow from inside the cell to outside the cell). This forces faster repolarization, hence a shorter AERP.

But K⁺ has additional avenues of input. A K⁺ shortfall also facilitates AERP shortening due to hypoglycemia.

Increasing blood K⁺ => increased K/Na => increased aldosterone => vagolysis.

I have no doubt that not having had an episode for many months makes a K⁺ shortfall even more significant in not only triggering an episode but also maintaining it. ANP receptor sites are probably less numerous and the RAS activity (due to decreased renal perfusion during AF) quickly changes the balance of power.

So, don't deviate from your electrolyte regimen. As you age, the natural increase in AERP should take you out of danger. But, if significant cardiac fibrosis develops, those sinus node cells stand ever ready to trigger PACs, reentry and AF.

PC

if significant cardiac fibrosis develops, those sinus node cells stand ever ready to trigger PACs, reentry and AF."

We are now back in territory covered when Erling used to post here, i think. PC, can you update me on what effect cardiac fibrosis has on those errant p cells that we have been discussing, the ones found in all afibbers and some non afibbers?

PeggyM

It's not the effect of fibrosis on the P cells (trigger substrate) that's problematic. It's the effect of fibrosis on the maintenance substrate. Reentry (and AF and tachycardia) is enhanced by fibrosis and fewer PACs are needed to trigger this (v. LAF).

The problem is that one never knows how much fibrosis is present, although this is increasing in everyone with age. Present lab tests to evaluate this are relatively insensitive.

PC

PC;

You have stated that salt intake may reduce AF episodes. I recently checked my AF journal entries and found that my last several early morning AF episodes were preceded by a salty meal the night before. I realize that several other factors were involved in these also. One factor in particular was that I also had a stressful event of some kind before bedtime in each case. What role does nighttime stress play in vagal AF?

Thanks....

Larry Zajdel

Hi Larry,

Most LAFers live on a knife's edge, at least wrt episode initiation.

IMHO far too much weight is given by us to what we eat. Clearly this has an impact on LAF but the vast majority of those without LAF have a much worse diet yet no AF.

So, I would say that it's the autonomic connection that is paramount. It's the change in autonomic tone, i.e., from vagal to sympathetic or vice versa, that triggers episodes. We all have sinus node tissue in the PVs and LA. Otherwise PVI wouldn't be curative.

MVPS afflicts the tall and thin. Minimal MVP appears sufficient for the development of LAF. The relationship between LAF and MVPS is speculative but strongly supported by LAFS-11. LAF is frequently encountered in the physically fit. Many endurance athletes are only of average height. In view of this for the average LAFer to be 2-3 inches taller than normal is even more striking. Furthermore, dehydration can cause MVP. The constellation of findings in MVPS includes dysautonomia at the top of the list. Magnesium deficiency is close behind.

What is your BP and resting HR? If both are low normal, then forget about salt as a trigger. Control the stress and concentrate on the K+ and Mg++ supplements.

PC

Dysautonomia. PC, have you been following CRob's posts about dysautonomia and the treatment the ANSAR group advises for it? Please can you comment on that?

PeggyM

Dysautonomia is a waste basket term for autonomic dysfunction, for which there must be a million manifestations and of which atrial fibrillation is one.

I am unfamiliar with the medications listed by CRob as in use by ANSAR, i.e., low-dose Elavil (plus Coreg and Proamatime). They may do something for some individuals, but rest assured they represent no panacea for LAF.

Who knows whether the dysautonomia is primary or secondary. Numerous hormones affect the balance between the two arms of the ANS. Disrupting any of them could cause problems more serious than LAF.

Sorry that I can't be more helpful.

PC

PC,

Here is an interesting situation I noticed yesterday wrt ectopics.

Generally, my ectopics are dominated by PVC's. My PVC rate is usually under 20/hour, but can range from 0 to 60/hour. My PAC rate is generally 0 to 2 per hour.

Yesterday, my morning meditation readings were normal - 0 PAC's and 12 PVC's/hour. Around noon, I went for a 2.5 hour bike ride. It was about 90 deg F out. My ride, while fairly long, was not particularly strenuous. I wasn't wearing a monitor, but I'm guessing most of my time was at ~ 120 BPM. There were a few stretches around 140 BPM. I hydrated while riding and drank a lot of water when I was done.

Several hours later, I went for a long meditation - 80 minutes. Interestingly enough, my PVC rate was 0 and my PAC rate was 40/hour. Now this is still not enough to cause me AF, but it was a clear change. Later, I ate supper and took my evening supplements - K+, Mg++ and taurine. After several hours, that included a 30 minute walk, I hooked up my

Freeze Framer. My PAC rate was back to 0/hour and my PVC rate was about 20/hour. This morning was again 0 PAC's and 25 PVC's/hour.

Can you fit this in your theory? My guess is exercise changing the electrolyte balance.

George

Hi George,

That's very interesting.

I'd have to agree with your surmise. However, I personally think that more than electrolytes were at work, otherwise why the change from PVC prominence to PAC prominence.

When I used to play golf on warm humid days here in the islands, I'd often go into AF about an hour or two after the round. Clearly electrolyte imbalance was at work, but I always suspected that hormones were also involved, because my HR was inexplicably low for the activity and time of day at the time of the episode.

My thinking is that all that time on your feet and the heart pumping against gravity (v. sitting down or supine) there is an increase in aldosterone production. This is vagolytic. Immediately after the round my RLX (HRV) on my Polar was inexplicably low. But slowly it began to rise and then came the PACs. And an hour or two later I was in a full blown episode. I think there is a rebound vagotonia and since those nerve fibers primarily affect the atria, that might explain your PAC (v. PVC) preference.

Dehydration cannot only cause physiologic MVP but it can also cause vagal rebound, as can any form of orthostatic challenge.

My latest thoughts on my son's science fair project are centered on this later point. The Honolulu Marathon is held in mid December and I was thinking that we could set up a few cots at the finish line and take some measurements. I have a Polar S810 and a Polar S810i and two holter monitors and we could test as many as possible for ectopics for a few minutes while supine after they'd finished and hydrated (without electrolytes). Some might even allow us to subsequently monitor their ectopic response to a potassium drink of predetermined amount. We could even get some non marathon runner controls. This way I can hit several questions with one stone - do endurance runners have increased ectopics and do they respond to potassium.

Let me know your thoughts on how long to monitor and how much K to administer. If we monitored for 4 minutes we could do about 50-60 in an hour. We could even get anthropometric data simultaneously.

PC

Hi PC,

I'll give some thoughts to your questions. I have an extra S810 or 810i and additionally several extra chest straps that I could lend you to help the people flow.

My K+ dose is always 1.5 grams, but who is to say what the correct dose should be. Four minutes out to be long enough to get a good sample and you obviously don't need to give K+ to those w/o ectopics.

George

I assume that anyone in the marathon will be presumed to have healthy kidneys (as regards to giving them K+ drink)?

It would be nice to do a trial at a local 10K and test a few people before the marathon. What you learn here may help you tweak your testing for the marathon.

By the way, I had a number of PAC's (& some PVC's, too) this morning on a 30 minute treadmill exercise. This was a slow ramp up - 80 BPM to 130 over 15 minutes, then increased to 140 for 15 minutes. However the PAC's occurred in the slow ramp portion (at say 110 BPM). This workout was preceded by a 30 minute meditation with only PVC's at a rate of 25 PVC's/hour. This was all ~ 12 hours past last supplement intake.

George

George,

My speculative comments on vagal rebound and PACs were predicated on the appearance of ectopics well after the cessation of exercise.

If your K+ status was good at the time of the slow ramp portion, then I have no idea.

I also forgot to add that one of the features of MVPS is low blood volume. About half are reported to have increased ANP. That's why the constellation of findings includes catecholamine hypersensitivity, orthostatic hypotension and palpitations. Several articles have shown that those with high vagal tone tend to hyperrespond with renin during orthostatic challenge. I think some with VMAF may experience exaggerated vagal rebound after prolonged orthostatic challenge.

Vagal rebound after exercise is a well known phenomenon.

PC

Hello Everybody,

My husband, Richard, is off enjoying some golf with his friends, and I've been studying about hormones. I've been doing this partly because of my daughter, and partly because of myself. I found this article by Dr. Lee and Dr. Jonathan Wright to be of interest. I read PC's article above (still at the brain burners..haha), and need to go back and re-read it. You all might find the following statements of interest.....

Dr. Lee: Well, estrogen and progesterone tend to have competing effects at many parts of the body. If you have estrogen dominance, all these effects tend to be the estrogen effects. For instance, estrogen wants the body to retain water, whereas progesterone is a natural diuretic, and helps to restore normal cell membranes to maintain the proper concentrations of sodium, magnesium and potassium, on either side of cell membrane. If you have a women who retains water, that would be one sign of estrogen dominance. Also, estrogen wants the body to convert food into fat for storage, whereas thyroid and progesterone want to convert the body fat into energy for life.

AND....

Well, this means a sudden surge of water retention, and to me that meant that estrogen was becoming dominant, and something, such as cortisol, was either blocking their progesterone, or there was a surge of estrogen. Without doing any measurements, I just asked these people to increase their progesterone in the 10 days or so before their period, and 85% to 90% of these women became the most grateful patients you ever saw. They would say they now have a handle on what to do to get through the month without having a terrible 10 days just before and around their period time, where they felt like killing their husband.

http://www.life-enhancement.com/article_template.asp?ID=34

Here's another link that you all might find of interest for saliva testing that you can do from the comforts of your home. This site is recommended by Dr. Lee and Dr. Wright, and has some interesting reading, as well.

www.hormoneprofile.com

Richard hardly has time to study anymore, and I hit and miss at it, but I have found the hormonal issue interesting. From what I'm gathering, progesterone is deficient in many of us, including men. Estrogen dominance could be an issue due to our food supply being inundated with it, and it could be the synthetic kind. In further reading, I have come to the conclusion about one thing. Many adrenergics are people that exercise in excess, and when you look at women, such as gymnast, you notice that most of them have very small breast. Could this be due to their hormones taking the pathway to cortisol rather than to estrogen for breast enhancement. Looking at the biochemical pathway from pregnenolone to the other hormones, it would appear to me that the heavy exercise is perceived to be a "fight or flight" situation, with the hormones going mostly to cortisol in this case. Adrenergics also have cyclic AF. I know Hans had it about every two weeks, which suggests a cycle almost like a woman's menstrual/ovulation cycle. Maybe all we needed was a bit more progesterone to help hold on to K+ and Mg. Just speculating.

Found this to be of interest, as well....

Unopposed estrogen (estrogen dominance) can interfere with thyroid hormone activity and is often a primary underlying cause of thyroid dysfunction.

Because estrogen and thyroid hormone have opposing actions (probably at the thyroid hormone receptor level) unopposed Estrogen will prevent the thyroid hormone from "completing its mission," resulting in "hypothyroid symptoms." This excess estrogen is often responsible for the symptoms of Hypothyroidism, despite normal serum levels of Thyroid Stimulating Hormone (TSH).

Natural Progesterone, however, re-directs the activity of estrogen by increasing the sensitivity of estrogen receptors and, most importantly, inhibits many of unopposed estrogen's undesirable side-effects, which includes interference with thyroid hormone activity.

Most women who are plagued by the symptoms of hypothyroidism have found that thyroid function has been normalized within a few weeks of using a properly formulated Natural Progesterone Cream, in conjunction with proven Dietary- Lifestyle modifications.

Because natural progesterone most often has a normalizing effect on a sluggish thyroid, the use of thyroid medication may over-stimulate the thyroid gland. Consult your health care professional if you are taking prescription thyroid medication.

In addition to a properly formulated Natural Progesterone Cream, many informed women have found that supplementing with ten kelp tablets per day has a nourishing effect on thyroid function. (We know kelp has iodine in it, which is good for the thyroid, but it is also full of free glutamate)

<http://www.progesterone.com/hypothyroidism.html>

Another observed symptom of hypothyroidism is.....INDIGESTION.

Rhonda
