

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

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SUBJECT: Baroreceptor Sensitivity & Aldosterone

Hello All! Time for a little brain cramping again. PC has torn himself away from his paradisiacal existence and come up with a most fascinating hypothesis regarding the effect of baroreflex sensitivity and aldosterone on afib. I believe we may be on the right track here (not again :-))

I and at least two other afibbers have noted that minimizing salt intake makes things worse. Others of course find a high potassium intake to be beneficial - both pointing to the crucial importance of maintaining a proper electrolyte balance at all times.

The Conference Room is open for your contributions. Hans

Baroreceptor Sensitivity (BRS) and Aldosterone

While continuing to experiment on my AF and ponder its mechanism I thought I would post some anecdotal thoughts on the above. Perhaps others have experienced something similar.

Since relocating to HI, I've had several short episodes (less than two hours) that appear to be related to golf and associated fluid loss in this humid environment. They occur an hour or two after the round, while relaxing. Fluid replacement during the round had been Waller water (aqueous magnesium) without any added salt. This happened despite increasing my disopyramide (Norpace, Rythmodan) dosage from 750mg (sufficient on the Mainland for many AF free months) to 1 gm per day after I'd noticed an increase in PACs. However, then I started adding half a teaspoon of sea salt to 1.5 liters of WW and have had no noticeable PACs or AF, despite sweating profusely. It has now been almost two months since my last brief episode. Furthermore, I've been able to decrease my dose of disopyramide to 750 mg again and am about to try 625mg, a daily dosage that I could never reach even on the Mainland.

Much has been written on the BB not to mention the general medical literature on the pros and cons of dietary salt. Little has been written regarding a possible specific role for salt in the genesis of LAF. Sodium physiology and the baroreceptor (carotid sinus) are inextricably entwined.

Because of this rather unexpected result with sea salt, I would like to make the following suggestion. Perhaps the defective substrate of LAF is not actually the heart itself but the baroreceptor (carotid sinus) that exerts so much influence over it. And, as Hans has repeatedly written, aldosterone plays a major role.

"Aldosterone not only impairs the baroreflex response but also exerts a tonic inhibitory effect on cardiac vagal control."
"There is evidence that in humans sodium restriction may impair the arterial baroreflex."

Aldosterone Blunts The Baroreflex Response In Man.

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=retrieve&db=pubmed&list_uids=9831693&dopt=Abstract

What Is The Influence Of Aldosterone Blockade On Vagal Tone?

http://www.ishne.org/scientificnews_06_04_05.shtml

Baroreflex Impairment by Low Sodium Diet in Mild or Moderate Essential Hypertension

<http://hyper.ahajournals.org/cgi/content/abstract/29/3/802>

I think that maybe during a round of golf on a humid day I may become slightly dehydrated, but more importantly I'm not replacing lost sodium by drinking WW alone.

Sodium: The Forgotten Nutrient

<http://www.gssiweb.com/reflib/refs/247/SSE78.cfm?pid=96&CFID=787633&CFTOKEN=46243323>

Either one of these conditions would stimulate the secretion of aldosterone. This would cause vagolysis and impaired baroreflex activity. This situation persists for awhile after cessation of exercise. I've noticed this on my Polar HR/HRV monitor. It is well known that BRS is reset during weightlessness and then again during return to earth. Similarly during exercise (or other form of "postural stress") there appears to be a resetting of BRS.

Resetting Of The Carotid Arterial Baroreflex During Dynamic Exercise In Humans

<http://jap.physiology.org/cgi/content/full/87/1/332>

So this vagolytic/impaired baroreflex state conspires (with the vagolytic effect of exercise itself) to create a vagotonic rebound during the post round relaxation period that appears to be able to overcome even the vagolytic effect of disopyramide. Indeed, the disopyramide induced vagolysis during the round may ironically also contribute to the rebound. Accordingly, I've started delaying my morning disopyramide dose (peaks in five hours, half life is about 12 hours for slow release) to coincide with the post round vagotonic rebound. My daily regimen for disopyramide depends on the activities scheduled for the day.

"There is also evidence that those with high carotid baroreflex responsiveness accommodate lower body negative pressure (LBNP) via renin (and aldosterone). Those with low responsiveness do not." LBNP simulates the postural stress of prolonged standing. This underscores a relationship between aldosterone and the baroreflex in those with high vagal tone.

Hemodynamic Strategies In Blood Pressure Regulation During Orthostatic Challenge In Women.

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=9263619

Perhaps the baroreflex may represent the "missing link" for vagally mediated and adrenergic LAF. Perhaps all LAFers owe their episodes to the vagus. Hans' adrenergic LAF transited to typical VMAF during spironolactone therapy. Spironolactone is a direct aldosterone antagonist.

"Moreover, sympathetic and vagal activity do not exclude one another; both may be active at the same time. In fact, vagal effects are more pronounced in the presence of high sympathetic tone and vice versa ("accentuated antagonism") (Levy 1971). When a premature atrial beat hits the atria at a point in time when autonomic tone has created favorable conditions for atrial arrhythmia to occur (in particular shortened refractoriness), both initiation and perpetuation of AF are facilitated. In addition to the above, vagal stimulation produces a marked variability in atrial refractoriness, which also facilitates AF (Liu 1997)."

<http://www.ub.rug.nl/eldoc/dis/medicine/a.e.tuinenburg/introduction.pdf> p. 24

"Fluctuations in autonomic tone, with a primary increase in adrenergic drive followed by a marked shift towards vagal predominance, have been noted just before the onset of AF in some patients.[13-17]"

Autonomic Tone Variations Before the Onset of Paroxysmal Atrial Fibrillation

http://www.medscape.com/viewarticle/462561_3

"In patients with focal ectopy originating from the pulmonary veins, sustained episodes of atrial arrhythmias are mainly dependent on variations of autonomic tone, with a significant shift toward vagal predominance before AF onset."

Fluctuation In Autonomic Tone Is A Major Determinant Of Sustained Atrial Arrhythmias In Patients With Focal Ectopy Originating From The Pulmonary Veins.

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=11294170

So, for typical vagally mediated episodes vagal tone appears to be the only player. However, in mixed or pure adrenergic episodes vagal tone appears to make an appearance only just before onset of the episode. In either situation one can make a strong argument that enhanced BRS makes this possible.

Some of us are predisposed genetically to AF (both my parents have/had it) via enhanced baroreceptor sensitivity (BRS). This becomes more of a problem as we age due to nutritional shortfall, especially K and Mg, poor hydration habits, physical and emotional stress, etc. Although BRS decreases as we grow older, perhaps in LAFers that probably start life with enhanced BRS the natural decline with age is eclipsed by dysfunction secondary to the above factors.

"Cardiovascular recovery from stress is associated with increased vagal modulation despite residual sympathetic activation. Vagal rebound may be involved in mechanisms resetting the baroreflex sensitivity at the onset and offset of stress."

Vagal Rebound and Recovery From Psychological Stress

<http://www.Psychosomaticmedicine.Org/Cgi/Content/Full/63/4/650>

Vagal Tone: A Physiologic Marker Of Stress Vulnerability

<http://pediatrics.aappublications.org/cgi/content/abstract/90/3/498>

"Baroreflex sensitivity (BRS) is significantly higher in men than in women. BRS decreases as we grow older. Age and gender are the most important physiological correlates of BRS."

Age And Gender Dependency Of Baroreflex Sensitivity In Healthy Subjects

<http://jap.physiology.org/cgi/content/abstract/84/2/576>

This dovetails nicely with Hans' survey results that show that VMAF is usually male and younger, while adrenergic LAF is usually female and older. The nearly complete absence of diabetes and the frequency of hypoglycemia (v. the general population) set LAFers apart. Many are physically fit with high vagal tone. From Hans' survey it would appear that the BMI (body mass index) of the typical LAFer is well under the norm.

The obvious question is how and why is the BRS of LAFers different.

"Wide intersubject variability in BRS is not well explained by cardiovascular risk factors or life style, suggesting a genetic component responsible for the variation of BRS. Common genetic polymorphisms in the aldosterone synthase ... are associated with interindividual variation in BRS, especially in the younger population."

Baroreflex Sensitivity And Variants Of The Renin Angiotensin System Genes.

<http://dilib.inist.fr/bin/dilib/AppIiPs/docPs.cgi?/dps/public/dps/sdv/hypertension/Server/EN.Psbase.Genes.wsh+000465>

"BRS is heritable; the variability can be explained by genetic influences"

Genetic Influences on Baroreflex Function in Normal Twins

<http://hyper.ahajournals.org/cgi/content/full/37/3/907>

Furthermore, some women have posted on the BB a correlation between LAF episodes and their menstrual cycle. This can also be nicely explained by BRS.

"Our results indicate that baroreflex control of HR is altered during the regular menstrual cycle, and estradiol appears to exert cardiovagal modulation in healthy women."

BRS (and estradiol) is highest during preovulation.

Influence Of Menstrual Cycle On Baroreflex Control Of Heart Rate: Comparison With Male Volunteers

<http://ajpregu.physiology.org/cgi/content/full/285/5/R1091>

Hyperthyroidism appears to reduce parasympathetic activity and hypothyroidism appears to increase it.

Thyroid Status Influences Baroreflex Function And Autonomic Contributions To Arterial Pressure And Heart Rate

<http://ajpheart.physiology.org/cgi/content/full/280/5/H2061>

And thyroid hormones are known to cause urinary K and Mg wasting.

Other well known associations exist between swallowing and AF and between GERD and AF. Both appear to work through the carotid sinus.

Swallow Syncope Associated With Paroxysmal Atrial Fibrillation

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=11888795

Deglutition Syncope Associated With Carotid Sinus Hypersensitivity

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=9309758

"Swallow syncope is a rare disorder caused by hypersensitive vagotonic reflex in response to deglutition."

Swallow Syncope.

<http://www.blackwellpublishing.com/abstract.asp?ref=0147-8389&vid=26&iid=3&aid=22&s=&site=1>

Autonomic Regulation in Asthmatics With Gastroesophageal Reflux*

<http://www.chestjournal.org/cgi/reprint/111/1/65.pdf>

Furthermore, it would appear that runners are particularly prone to develop LAF, not only on the basis of increased vagal tone but also GERD.

Gastroesophageal Reflux Induced By Exercise In Healthy Volunteers.

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=2724505&dopt=Abstract

And then there's sleep apnea and atrial fibrillation. This too appears to involve the carotid sinus.

"... bradycardia, and a decrease in blood pressure, all of which are usually seen in central sleep apnea patients, are associated with increased vagal tone.

Sleep Apnea -- A New Approach?

<http://www.healthandage.com/PHome/115!gm=2!gid2=1760>

The baroreceptor (carotid sinus) is stimulated not only by MAP (mean arterial pressure) but also by pulse pressure (difference between systolic and diastolic pressures).

For me (and others) sex was/is a potent trigger for AF. It provides potent stimulation for dopamine secretion. My pulse pressure is markedly increased. Dopamine markedly increases systolic pressure via alpha 1 receptors, causing reflexive relative bradycardia via the baroreceptor (carotid sinus). This enhanced vagal tone (shortened atrial effective refractory period) creates very fertile cardiac soil for AF. Perhaps epinephrine operates in a similar fashion in adrenergic LAF.

Furthermore, although the vast majority of my episodes were typically vagal, occasional episodes appeared to be adrenergic. Dopamine appeared to be responsible for some but others occurred during brief "sprints", e.g., at the end

of a long jog or while playing soccer.

Aldosterone plays a dual role. On the one hand it impairs BRS. On the other it contributes to hypokalemia. I believe that in adrenergic LAF low potassium may be integral whereas in VMAF baseline vagal tone may be integral. However, BRS may be the final common pathway for triggering both variants. Potassium is decidedly helpful for me but vagal tone rules.

PVI ablates vagal fibers that all enter the atria via the pulmonary veins. This procedure appears effective for both ALAF and VMAF.

Although this is perhaps an oversimplification of a very complex process, it is a view that appears to me quite plausible.

PC

Yeah

I have always noticed that I need salt. If I have too little salt - echoing and loud semi blocked ears, whooshing in ears when moving, dizziness and unsteadiness. The worst time for this to happen was around the time of my menstrual cycle.

I now eat a sea salt cured kipper every morning for breakfast followed a wee bit later by fresh squeezed orange (not from a carton). For the rest of the day I add salt if my body says I want it. If I ignore it (as I often do if table salt is only available) my ears go to pot (or at least the symptoms come from my ears - despite nothing wrong being found there).

Despite not falling into the category of "... VMAF is usually male and younger, while adrenergic LAF is usually female and older (age at onset 22 and female).

But falling into this category " The nearly complete absence of diabetes and the frequency of hypoglycemia (v. the general population) set LAFers apart. Many are physically fit with high vagal tone it would appear that the BMI (body mass index) of the typical LAFer is well under the norm"

My BMI 2 weeks ago - 20.7 (estimation for my height and weight 24.2)

Perhaps PC's realisation of why sex triggers VMAF is a potent reason why I have no sex drive. It is a preservation to my well being.

Well done. I will go with that as a baseline of why AF. Now the question remains WHY this predisposition?

Glad to see you back at your best

Fran

Furthermore, some women have posted on the BB a correlation between LAF episodes and their menstrual cycle. This can also be nicely explained by BRS.

"Our results indicate that baroreflex control of HR is altered during the regular menstrual cycle, and estradiol appears to exert cardiovagal modulation in healthy women."

BRS (and estradiol) is highest during preovulation.

Influence Of Menstrual Cycle On Baroreflex Control Of Heart Rate: Comparison With Male Volunteers

<http://ajpregu.physiology.org/cgi/content/full/285/5/R1091>

This bit suggests why slapping on an oestrogen patch during menopausal palpitations and irregular beats brought my hb back to 'normal' within hours.

Joyce

I forgot to include my thoughts on MSG and free glutamate in the genesis of LAF.

Once when I was looking for a low glycemic between meals snack I tried dried seaweed strips. They were quite tasty and late one afternoon I ate quite a few. Although I didn't go into AF, I had an unbelievable number of PACs. Fran was kind enough to educate me on this.

I've posted previously on the BB on how specifically the baroreflex works. Please visit http://www.yourhealthbase.com/forum/read.php?f=3&i=14501&t=14421#reply_14501

and the role of the NTS will become more apparent.

"Microinjection of glutamate into the NTS mimics activation of the baroreflex and produces a decrease in arterial pressure and heart rate."

Glutamate In The Nucleus Of The Solitary Tract Activates Both Ionotropic And Metabotropic Glutamate Receptors

<http://ajpregu.physiology.org/cgi/content/full/275/6/R1858>

Unilateral Blockade Of Excitatory Amino Acid Receptors In The Nucleus Tractus Solitarii Produces An Inhibition Of Baroreflexes In Rats

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=1678142

Cardiovascular Changes Induced By The Local Application Of Glutamate-Related Drugs In The Rat Nucleus Tractus Solitarii.

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&list_uids=2575008&dopt=Abstract

"These observations suggest a resetting of the baroreflex, attributable to neonatal administration of MSG."

Monosodium Glutamate Neurotoxicity: A Sex-Specific Impairment Of Blood Pressure But Not Vasopressin In Developing Rats.

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=3756544&dopt=Abstract

The NTS is immediately adjacent to the fourth ventricle in the brainstem from which CSF laden with dietary MSG can easily diffuse - please read 10/28/03 post at

http://www.yourhealthbase.com/forum/read.php?f=3&i=1115&t=1082#reply_1115

I can clearly trace many of my episodes to the unintentional ingestion of glutamate/aspartate about 5 or 6 hours prior. Depending on the amount ingested (acutely and chronically), excitatory amino acid sensitivity, etc., this time frame may vary for others. To my knowledge the only effect that free glutamate exerts on the heart is through the CNS. To test this hypothesis about the effect of free dietary glutamate and its possible/probable correlation with LAF through the baroreflex I'd like to try GABA during my next MSG related episode. The problem is that, like glutamate, it does not easily cross the blood brain barrier. However, some pharmaceutical companies are experimenting with ways to overcome that problem.

Inhibition Of Baroreflex Bradycardia By Ethanol Involves Both GABAA And GABAB Receptors In The Brainstem Of The Rat.

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=1325357

I'd also like to throw out for your discussion two other observations wrt to aldosterone and episode termination.

Aldosterone, a known vagolytic, has a diurnal pattern. It is highest just after dawn and this coincides with our bodies going vertical. Hydrostatic pressure decreases in the renal JGA (juxtaglomerular apparatus) and this stimulates the release of aldosterone via the RAAS (renin angiotensin aldosterone) axis to increase BP. Angiotensin is also vagolytic.

Perhaps this is why VMAFers are most successful in terminating their episodes just after getting up in the morning.

Significant intake of potassium just before and/or immediately after an episode has been triggered appears to terminate the episode for some LAFers. Could this also be due to aldosterone? The blood K/Na ratio is a potent stimulator of aldosterone secretion. If the former increases, then so does the latter.

Also, I forgot to add that in obstructive sleep apnea CPAP (continuous positive airway pressure) has been found to be quite efficacious. Could CPAP perhaps also add a vagolytic element that prevents LAF as well. Expanded lungs cause the HR to increase due to vagal respiratory arrhythmia. This is a normal evolutionary physiologic adaptation by mammals to optimize gas exchange. With pulmonary inflation not only is vascular resistance lower but also more O₂ is available. So an increased HR is more efficient. This occurs through vagal withdrawal during inspiration.

Breath holding (voluntary apnea) was a great fib buster for me. I'm sure the pulmonary stretch receptors were instrumental in providing just enough vagolysis. However, this only worked for the infrequent afternoon episodes (and then only at the very onset of AF) and never for the PM episodes when vagal tone was much stronger.

I think it is important to remember that adrenergic LAF is quite different from paroxysmal atrial fibrillation. As we age and especially as cardiac disease begins to manifest itself, vagal tone decreases. It begins to play less and less of a role in initiating episodes. Where LAF ends and AF begins is difficult to know, as we've discussed on the BB. In fact I'd be willing to bet that if any of your episodes are in any way triggered by vagal maneuvers you're probably still in the "lone" category.

PC

As I have already posted a while ago, a very low salt diet made me having PACs and bradycardia on a daily basis. Incidentally I discovered that having more like normal salt intake my PACs and brady would go away. Someone on BB advised me to eat sea salt instead of regular table salt, and that is what I have ever since, and I'm doing well. For me, sodium is another very important electrolyte, even if I couldn't come up with a clear explanation for it, as PC did. Thank you all for contributions.

Cornelia

I'm still going to all of the links because what has been posted so far describes me pretty closely. But two initial questions:

1. I've been avoiding salt for the last couple years. Maybe I need a little more sodium. So, so much is enough? PC adds sea salt to his WW, should just add 1 or 2 teaspoons of sea salt to water and try that? And how often during the day?
2. The part about swallowing is especially intriguing. When I wake up in the middle of night, as thirsty as I am sometimes, I don't drink water because many times it triggers an episode - especially if I take big gulps. Sometimes after a couple of golf and take a big gulp, even this sometimes triggers an episode. I went to link posted but all it was one paragraph. It said there was a simple cure but it didn't say what. Any ideas or is the answer in one of the other links?

Gregg

Hi Gregg,

I just add sea salt once to my drinking water at the beginning of the day and drink it throughout. I'll make up another batch later in the day, if fluid loss and intake is more than usual.

I once triggered nighttime episodes identical to yours.

To which hyperlink are you referring? I certainly don't know of any simple cures.

PC

Your Body's Many Cries For Water , by F.Batmanghelidj

In this book he suggest increasing your water & salt intake. He doesn't address AF in his book, but he says hypertension, angina pain & a lot of other health problems can be cured with water therapy. Asthma can be cured with water & SALT therapy. www.watercure.com

Todd

PC, I misspoke, it wasn't "easy" but curable per the swallow link, **Swallow Syncope Associated With Paroxysmal Atrial Fibrillation**
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=11888795

... "Swallow syncope associated with paroxysmal atrial fibrillation.

Gordon J, Saleem SM, Ngaage DL, Thorpe JA.

Swallow or deglutition syncope is a very unusual potentially lethal but treatable disorder. "

But it doesn't elaborate on the treatment.

Also, how much salt do you add to your water, roughly in terms of teaspoons?

Gregg

Hi Gregg,

The treatment to which they refer was probably surgical or medical treatment of his GERD, but I don't really know.

As stated, I add about 1/2 teaspoon per 1.5 liters of water. This translates to just under 2.5 grams of sea salt or 16 gms per liter (1.6%), which is just over the concentration in normal saline IV solutions (0.9%).

The only way that I can surmise that swallow syncope might be potentially lethal is through ventricular asystole associated with vagotonic control of the AV node. That would seem to be a rather extreme outcome.

PC

PC, 1/2 teaspoon per 1.5 liter is pretty small. Would this amount really have an impact on anything?

Fran,

re: For the rest of the day I add salt if my body says I want it.

How does your body tell you this?

Gregg

Gregg,

You're absolutely right. Two and a half grams of Na isn't very much. But, if the loss occurs over a relatively short time in a susceptible individual, then perhaps it's not so little.

The following two passages are lifted directly from "Sodium The Forgotten Nutrient" at

<http://www.gssiweb.com/reflib/refs/247/SSE78.cfm?pid=96&CFID=787633&CFTOKEN=46243323>

"In warm to hot conditions, most adult athletes lose between 1 and 2.5 liters of sweat during each hour of intense competition or training. Notably, sweat rates over 3.5 liters per hour have been observed with some well-conditioned, world-class athletes who were competing in very hot and humid climates (Armstrong et al., 1986).

Well-conditioned athletes who are fully acclimatized to the heat often have sweat sodium concentrations in the range of 5 to 30 millimoles per liter (i.e., 115-690 milligrams of sodium per liter of sweat) (Wenger, 1988). On the other hand, athletes who are not acclimatized to the heat typically lose much more sodium from a given volume of sweating (e.g., 40-100 millimoles or 920-2300 milligrams per liter). However, some athletes can have relatively high concentrations of sodium in their sweat, no matter how fit or heat acclimatized they are, which suggests a strong genetic influence. Sweat sodium and chloride concentrations also vary with sweating rate (Wenger, 1988). As sweating rate goes up, the concentrations of these minerals in sweat usually increase as well. Again, given the impressive sweat losses that athletes often incur, it is not difficult to imagine that a sizable sodium deficit could readily develop, especially if the athlete follows a low-salt diet (e.g., <2,500 mg of sodium intake per day). For example, a sweat sodium loss of 2,500 to 5,000 milligrams per hour is common among many athletes with high sweating rates (e.g., 2.5 liters per hour)."

For LAFers it would appear that it's not the Na itself that's the problem but the aldosterone that its loss stimulates.

PC

Good to see that 'ol Doc Braincramp is back! From PC's posting above:

"Hans' adrenergic LAF transited to typical VMAF during spironolactone therapy. Spironolactone is a direct aldosterone antagonist."

About 6 months ago - after having read all the CR discussions re. aldosterone at that time - I tried some spironolactone..... after one daily dose I had just about the worse day of ectopy (and a real close call with an AF episode at 3am that night) I've ever had. Didn't take another spiro. tablet again. Has PC or anyone any simple surmisal to offer up to me explain my adverse reaction to the spiro.?? I am still a little unclear as to all involved here: could I be LOW in aldosterone and the spiro tablet made the situation worse?? Would increasing salt intake help my situation??

I'm very interested in the salt issue. At age 17/18 I had an embarrassing problem with REALLY excessive sweating from my armpits - even in winter thru a leather jacket large salt-rimmed circles would form on the outside of the jacket. After about a year, this problem thankfully resolved itself as quickly as it came. Whether or not that indicates I have some sort of residual salt imbalance issues I do not know.

I absolutely crave and adore salty food. I have over recent years however, tried to minimise salt intake. Should I add a little more to my diet??

Mike F.

PC et al: just had a look on the Web and came across the following (apols if you've already cited/quoted from it):

Home About ISHNE Membership Education Journal Fellowship Links News

5. What is the influence of aldosterone blockade on vagal tone?

*Fletcher J, Buch AN, Routledge HC, Chowdhary S, Cooté JH, Townend JN.
Acute aldosterone antagonism improves cardiac vagal control in humans.
J Am Coll Cardiol. 2004 Apr 7;43(7):1270-5.*

The authors of the present study examined the acute effects (<45 min) of aldosterone antagonism on heart rate variability and baroreflex sensitivity, markers of cardiac vagal control, in 13 healthy subjects. Evidence for the beneficial effects of aldosterone antagonists comes from studies showing increased survival rates following their addition to standard heart failure therapy. Many mechanisms have been suggested for this action, including effects upon the autonomic nervous system. Heart rate variability and baroreflex sensitivity were examined 30 min following the administration of potassium canrenoate (intravenous) (aldosterone antagonist) or saline (control).

Active treatment reduced resting heart rate (-6 +/- 1 beats/min [mean +/- standard error mean]) compared to control (0 +/- 1 beat/min) ($p < 0.001$) and increased measures of high frequency (HF) heart rate variability. Root mean square of successive RR interval differences increased by 21 +/- 5 ms versus -6 +/- 5 ms control ($p < 0.001$); HF power increased by 1,369 +/- 674 ms² with aldosterone antagonism compared to -255 +/- 431 ms² following saline infusion ($p < 0.01$). Baroreflex sensitivity (alpha-HF) was increased after active treatment (+4 +/- 2 ms/mm Hg vs. 0 +/- 1 ms/mm Hg control [$p < 0.05$]). No changes in plasma potassium levels were observed. The results of the present study provide evidence that aldosterone antagonists acutely improve cardiac vagal control, irrespective of any diuretic effects, and may in part explain their beneficial effects in treatment of heart failure.

Comment: The independent relationship between impaired cardiac autonomic control and prognosis in disease states such as heart failure suggests that high levels of sympathetic activity and reduced vagal control may exert direct deleterious effects. Clinical studies have demonstrated that in heart failure patients, chronic treatment with spironolactone improves cardiac autonomic control measured by heart rate variability. These studies could not, however, exclude the possibility that the improved heart rate variability was secondary to the beneficial haemodynamic effects of spironolactone rather than any direct neuronal or receptor-mediated effects. The present study demonstrated that, acute administration of the aldosterone antagonist potassium canrenoate results in an increase in HF measures of heart rate variability and an increase in baroreflex sensitivity, suggesting that aldosterone exerts a tonic inhibitory effect on cardiac vagal control. These results cannot be attributed to changes in circulatory volume, blood pressure, or potassium concentration, or by chronic effects on vascular fibrosis and compliance at the arterial baroreceptors.

(Polychronis E. Dilaveris, MD, FESC)

I'm particularly intrigued by the line:

"suggesting that aldosterone exerts a tonic inhibitory effect on cardiac vagal control."

Could that explain why the Spiro tablet gave me lousy ectopy??

Mike F.

Hi Mike,

That article you quoted is a good one and the second on my list above concerning aldosterone and vagal tone. I think it's noteworthy that the cited article encourages the search for increased vagal tone, precisely what LAFers don't need. In fact spironolactone and one's reaction to it could be a kind of provocative test for differentiating lone AF from paroxysmal AF in general.

I'd have to agree with your analysis of the PAC/spironolactone connection.

In increasing dietary salt just don't neglect potassium. It's the balance and the relative numbers between the two more than the total amount of either.

PC

PC,

Apols if I'm missing something obvious here, but I'm still a little confused. In your response to Gregg you state:

"For LAFers it would appear that it's not the Na itself that's the problem but the aldosterone that its loss stimulates."

This implies that aldosterone production is - as often discussed in the past - problematic. I therefore can't quite get my head round why suppressing aldosterone production with spironolactone is problematic for LVMAFrS as per our previous discourse.

I'm essentially struggling to reconcile:

Spiro as an aldosterone antagonist increases LVMAFrS susceptibility

with:

Aldosterone production is a problem for LVMAFrS

Given that you are obviously a bright chap, I can only conclude that I'm missing something here.... could you be kind enough to I suspect I may be trying to over-simplify things.....

Cheers,

Mike F.

Hi Mike,

I don't think you're missing anything except the following, which is only my opinion and not an absolute fact.

It's all about rebound. Vagal maneuvers trigger LAF. That's the bottom line.

Aldosterone becomes a problem because it heightens the vagolytic state. For me this occurs during a round of golf, which is vagolytic in and of itself, as is any exercise. This conspires to produce an accentuated vagotonic rebound. LAFers probably have enhanced BRS and this combination often triggers an episode, especially if you throw in a little aldosterone induced loss of K.

PC

"...for differentiating lone AF from paroxysmal AF in general."

PC, i thought lone af and paroxysmal af were synonyms. What is the difference between those 2 conditions?

PeggyM

Hi Peggy,

LAF is a subset of paroxysmal AF. I don't know the actual breakdown. Perhaps Hans does. But many, if not most paroxysmal AFers have associated cardiac disease. LAFers, of course, as we've discussed on this BB before, have no discernible cardiac disease.

PC
