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VIRTUAL LAF CONFERENCE

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SUBJECT: Pre- and Post-Ablation PACs

There have been several recent posts on the BB detailing persistence of PACs post ablation despite successful termination of AF. I too have experienced episodes of markedly increased PAC activity post ablation that was dramatic but sharply delimited temporally. They are often postprandial but not necessarily and I have occasionally noticed concomitant upper gastric upset, although exceedingly mild in nature. My most recent episode concerned about 220 PACs detected by Holter monitor over a 90 minute period about 45 minutes after eating some spicey food. Potassium supplementation did not help, although it has in the past.

Why do PACs persist after ablation? Although parasympathetic stimulation provides fertile soil for maintenance of AF, it should not actually trigger PACs, unlike sympathetic stimulation, which increases automaticity. Furthermore, this parasympathetic stimulation is markedly reduced via ablation, as evidenced by the lack of AF episodes. Yet PACs are not. Clearly there appears to be another mechanism responsible for triggering these PACs.

Each nerve has three parts – its dendrites that receive signals directly from other nerves, its ganglion that contains the cell's nucleus, and its axon (nerve fiber) down which travel impulses that transmit the signal to the next nerve's dendrites (or targeted muscle). Autonomic nerve fibers can regenerate, although their ganglia cannot.

Parasympathetic ganglia to the heart are located near the pulmonary vein orifices. While sympathetic fibers travel adjacent to parasympathetic fibers, their ganglia are not located in the heart. See http://europace.oxfordjournals.org/cgi/content/full/7/1/1 for a recent (2005) discussion and diagram.

This means that PVIs are successful (or not) due to their destruction of parasympathetic ganglia and not due to associated fiber disconnection, as is the commonly held belief.

Consequently, it seems plausible to me that pre and post ablation PACs may be due to cross stimulation of sympathetic cardiac nerve fibers caused by gastro esophageal (GE) junction irritation. The GE junction is immediately subjacent to the area ablated. Successful ablation eliminates a critical mass of parasympathetic ganglionic input that would otherwise provide the fertile soil for AF but does not impact PAC production.

For me the sharp temporal demarcation of my PAC episodes argues against inflammation and the ineffectiveness of potassium supplementation against electrolyte imbalance in their genesis.

My post ablation course was characterized by a blissful period of several weeks during which PACs were not noticeable. However, the PAC free bliss eventually ended and for the past six to seven months occasional interludes of increased PAC activity have marred my NSR. This would conform to the time required for sympathetic nerve fiber regeneration.

Extrapolation of the above reasoning suggests that all LAF is predominantly vagal in origin. The effectiveness of PVI for paroxysmal AF (50% is adrenergic, vagal or mixed LAF) and its diminishing effectiveness for persistent and permanent AF (80% is pathologic AF) is also consistent with the above viewpoint. In pathologic AF the substrate is rendered fertile due to fibrosis-induced heterogeneity v. autonomic induced shortening of refractory period in LAF. PVI

does nothing to lessen the former.

Other anecdotal input and ideas would be most welcomed.

PC

Hi PC,

I had my touch-up ablation at NYU in May, 2004. Over the next 8 months, I tried several times to wean off flec and/or atenolol. Each time I was met with a barrage of strange beats that I assumed were a less debilitating form of FIB. Each time I resumed the flec, the rogue beats ceased. In early 2005, I became resigned to the fact my ablation was not a total success. I decided to stay on flec (50-75 mg/day) and atenolol (18.75 to 25 mg/day), and ponder a future touch-up.

Last summer, there was much talk on the bulletin about PACs, and the complex beat patterns that could arise. Historically, I'd always had had pretty simple PAC patterns. I assumed more complex patterns were fib. But I began to doubt this was the case. On November 1, I decided to come off flec once more and stay off it. I did so for seven weeks. To my surprise, I had ectopics but no fib. Even on a halter. So I guess the ablation had been a success! Unfortunately, over the 7 weeks the ectopics worsened. And they were accompanied by episodes of bigeminy. By Christmas week, I'd estimate I was in bigeminy 1/3 of the time. This despite increasing my intake of magnesium and potassium. Reluctantly, I went back on the flec. Almost immediately the bigeminy dissipated. Within 24 hours, it, and almost all of the ectopics were gone.

In January, I went to see Dr. Chinitz at NYU. He said despite the lack of FIB, a third ablation might help if the bigeminy had a single, identifiable point of origin. But he agreed the risk equation had shifted. That is, since I no longer had fib, there was no risk in not having an ablation, but there was still a small risk of a screw-up during the ablation. However, he said there was also a risk in staying on the flec, though this was mitigated by the small amount I was taking. We made no plans to do another ablation. Honestly, I am not eager to do so. Who needs the inconvenience of the procedure and the recovery?

Right now I am taking 25 mg of flec every eight hours and 6.75 mg of atenolol every six hours. I am pretty much ectopic free. A short burst occasionally crops up when I lay down, but this doesn't happen all that often. Also, if I drink something cold, which I usually avoid.

I don't think any episodes have occurred after eating. But I tend to eat only small meals, anyhow. I do not seem to have any adrenergic episodes. However, I will say I am less aware of stray beats than I used to be. Occasionally, I barely notice ectopics until I feel my pulse.

At some point I may do a third ablation. I hate taking any drugs. Prior to fib, I seldom even took aspirin. And recently I have become more aware of drug interactions. I was prevented from taking an antibiotic because one of its side effects was a prolonged QT interval. So aside from the specific side affects of flec (most obviously visual distortions in my case), there is the added concern over drug interaction complications down the road. A good reason for the ablation if I could eliminate the flec and the atenolol.

BillB

PC. Count me in synch with your symptoms. I was symptom free for about six weeks after my Natale ablation as well, then came the PAC's (SPVC's) and an increase in PVC's from the before ablation days.

When I questioned the Marin General staff about these I was told that they are benign and may lessen with time. They certainly didn't tell me before the procedure that this might be an after effect. If I had known I probably would not have changed my mind about the procedure but it would have been nice to have full disclosure. They must know about this as so many of us seem to be experiencing similar symptoms.

I was told that the PAC's were normally the precursors of afib but now that the conduction path was cut off they could

no longer trigger the fib, which has been the case with me. The PVC increase is certainly still a mystery, though.

I have not noticed help from taurine and magnesium other than those supplements may help me to notice the symptoms less.

I'd like to try George Eby's arginine and taurine protocol to see if that helps but still being on warfarin I don't tolerate arginine as it jumps my INR too high. Have you tried that protocol and, if so, anything to report?

Gordon

Thank you Bill and Gordon for your input.

The above viewpoint is certainly not mainstream at least wrt atrial fibrillation. So, take it with a grain of salt. However, the current explanation for the efficacy of PVI in AF just falls short, at least for me.

If nerve fibers regenerate, then how can AF be kept at bay after the few weeks or so that it takes for this regeneration to occur? Ablation induced fibrosis can never completely isolate the vagal ganglia. Full thickness fibrosis would almost require perforation during ablation. My HR has increased v. preablation by about 10 to 15 beats per minute after nine months (I love that). Parasympathetic ganglia were therefore clearly eliminated by the procedure.

Another point concerns vagal reflex foci. This is discussed on pp 87, 90-91 of Hans' second volume on LAF. Such reflexes include sinus bradycardia, asystole, AV block and hypotension. The bottom line is that the success rate of ablation is significantly improved if these foci are identified and ablated. It would appear that this results in elimination of parasympathetic ganglia outside their usual location. I know Prog Haissaguerre ablated a few of these foci as well, because I remember his urgent request during the procedure for me to cough several times to raise my HR. He'd obviously discovered a vagal reflex focus that caused bradycardia.

Bill, your barrage of strange beats could represent transient tachycardia as well as ectopics and bigeminy. Flecainide, a vagolytic, might alleviate these, because its action decreases substrate fertility, i.e., reentry is less likely and reentry can only occur if the refractory period is sufficiently short. In other words post ablation you may still have sufficient vagal tone to enable reentry but not enough to sustain AF.

Wavelength = Refractory period x Conduction velocity (the shorter the WL the more wavelets that can exist and the more easily the AF critical mass of six or more can be attained)

But more importantly the atenolol probably suppresses PACs via beta receptor blockade. So, any automaticity caused by sympathetic stimulation is suppressed. This obviously should also lower your HR.

Just my opinion.

PC

Bill,

Though I'm not in the ablation club, I'd thought I'd describe my experience with flec. I looked back at the rhythm strip (Polar S810) of my last AF episode, nearly a year ago. I took an on-demand dose of flec to convert. One thing that I noticed is that my ectopic count dropped significantly for several days, so the half life of flec seems to be fairly long.

If I were in your shoes, I might slowly reduce my meds to see "how low I could go" & still have the quieting effect of the flec.

I'd also try adding taurine. My last two AF episodes occurred when I was only taking Mg & K, without taurine. Since I've added the 4 grams/day of taurine in, I've been AF free.

George

PC,

An interesting topic! I have never been satisfied with the AF explanation (simple imbalance of either symp. or para-symp.) as PVI would seem to favor the vagal side of the AF equation. My mostly vagal episodes had turned mixed and then persistent by the time I had my ablation. (Natale, 2 years ago) Contrary to others here, my PACs did not reappear and have not been a problem so far. But in addition to the PVI, Natale also noted activity near the superior vena cava and ablated that also. More to the subject, signs of my sinus dysfunction (suspect SSS) disappeared and my HR rose from the high 40's to mid 50's, all of which point to either blockage or destruction of vagal nerves. Like you, I'm very happy with that result. Your ganglia ablation fits that just nicely.

But then I have read in the past that nerve fibers to the heart can regenerate but commonly do not. Perhaps regeneration is more common than I've thought? And the location of the ganglia is shown above the muscular sleeve(s) of the PV's although the author(s) in your reference believe that they are ablated, which if in the location shown would risk severe stenosis. But the drawing shown is from Zipes, who studied on dogs. And there are many anatomical differences in human patients, making PVI's a more difficult business, and ganglia location inexact. All in all, the "all LAF is predominantly vagal" theory makes sense to me.

But something in the ablation has decreased my PACs, and perhaps part of the answer is my never having known gastric problems. The authors (Pachon M et al) of your reference offer up a new AF scenario in another article – by studying the spectrum of atrial potentials, they have seen 2 distinct types of atrial muscle: the compact (CM) and the fibrillar (FM) myocardium. In AF these FM are clustered in "AF nests" and ablation of these nests have had a 94% success rate (34 patients). Perhaps some additional clusters are located in non-ablated portions of the atria and will cause PACs rather than AF (making them "PAC nests"). Very interesting work down in Brazil!

Food for thought? Thanks for the mental exercise.

Regards,
Anton

George,

I have tried taurine and magnesium taurate. Both brought on intestinal distress. I intend to try again, though. As I said above, though I believe in the procedure, I'd rather avoid another ablation.

Regarding flec, I'm kind of split on that. In my case, it did seem to persist in my bloodstream for awhile. For a week or so, I had very few PACs and no bigeminy. But as the days went by, things got worse and worse. By the end, I was either in bigeminy or feeling like I was about to go into bigeminy most of the time. Yet, I have also found taking flec every eight hours instead of every 12 hours yields better results. Go figure.

I know I can get away with less flec. I have lived with 50 mg per day, and even 37.5. But I get runs of ectopics and bigeminy. At my current levels, I get practically zero of both.

My regular EP suggested I try 50 mg of atenolol and no flec. This may work. But my HR is usually around 52-55 bpm at 25 mg/day. I don't want to get slower than that.

BillB

Hello everyone,

I think this an excellent idea PC. There are a number of people on this board that contribute a great deal, and you are certainly one of them. I had my ablation done in Bordeaux last Sept. by Prof. Haissaguerre. Towards the end of my procedure, I experienced a tamponade, which in my case, I refer to as an accidental puncture of the atrium wall. I am grateful for the fact that I was in such good hands, as the problem was rectified very quickly.

I don't want to drag this out by going through my experience again, but perhaps it may be important to someone. Typically when you are treated in Bordeaux, you are released from the hospital on the Fri. after your ablation, monitored for the weekend, return on the Mon. and if all is well you are good to go. Because of what I experienced set be back three days, makes me wonder if I should have stayed longer, for a possible touch up.

Because of the tamponade I experienced, I was kept in the hospital three more days than normal. During the week after my ablation, I had no afib, but at times felt extremely fatigued. I must add that I did have a terrible time sleeping during my time in France, and this quite likely contributed to my fatigue. We where scheduled to leave for home on the following Fri. morning. Which did not leave enough time to make a rational decision with regards to possible touch-up. Two days before we where to leave for home, I really had some scary things happen. Over the next two days I experienced a number of short runs of afib. I returned to the hospital, and they did everything possible to induce afib, but nothing happened. I know we probably should have stayed longer, but all we wanted to do was go home.
I would suggest anyone going to Bordeaux, plan on a three-week stay just in case.

After six weeks of experiencing a few very short runs of afib and missed beats, I definitely felt the healing start to take place. I have been afib free since last Nov. 1st. I do still feel the occasional missed beat. It may be that I will have to live with this, but its not a big deal compared to afib. At this time I am medication free, which is wonderful. My daily intake includes, one 81 mg of coated aspirin, Magnesium, taurine, and potassium, which I get through L.S. V8, and bananas.

This has been a very busy month for my family. Four birthdays, 40th anniversary, etc. I have probably raised to many wine glasses recently which have contributed to my missed beats. It is to easy to forget the obvious triggers, when you feel you have conquered the beast, But I honestly think you must remain cautious, because it is still there just waiting to strike again. I would love to be free of the missed beats, as it is a constant reminder of what it used to bring on.

Thanks to everyone,
Lou

I am considering an ablation and curious about these PAC's. I have a few questions:
- Are they un-comfortable?
- How long do they last?
- How often do they occur? Every day.. couple times a week?
- Does one lead to others? in other words, you have days where nothing happens and then you have days where you have several?
- Do you think you will have them less with time?
- Can they re-map (like AF) and, in fact, happen even more in the future?
- Had you known you would have these afterwards, would you still have had the ablation? (Gordon indicated he would have but what about other folks?)

and...
- Do you think having surgical ablation (mini-maze or other) would make a difference in this? Do people experience PAC's after surgical ablations?

Your posts are most interesting to read... you guys provide interesting insight. I thank you for your efforts.

-Ruth

Ruth,

With a minimal amount of meds, I have pretty much no ectopics at all. Without them, I would say the number of blips I have seem to be far worse than most. This leads me to believe there may be a single spot creating bigeminy that could be zapped.

Small runs of PACs are not all that noticeable and not comfortable. When I had days with lots of PACs, I was aware of

them, but they were not particularly uncomfortable. The bigeminy is very noticeable, and rather uncomfortable

I can say without hesitation that I would still have had the ablation if I'd known I'd be left with PACs. Even the worst day of bigeminy is better than the best day of fib (Sounds like a bumper sticker!). Also, PACs/bigeminy are basically benign. That cannot be said for fib.

I would not let concern over PACs stand in your way of an ablation. Two years ago, I was in AFIB about 65 percent of the time, with or without meds. The rest of the time, I was just kind of waiting around for the next episode to begin. Now I am fib free, and with minimal meds, PAC-free. I lead a normal life. And I still haven't given up hope I can again be PAC-free and med-free.

BillB

Ruth,

The ablation doesn't create the PAC's. It is just that pre-ablation, for many AF'ers, PAC's lead to AF. Post ablation, the PAC's can still be there.

George

Anton,

Those are great comments. Thank you for your input. I think it is noteworthy that in the referenced article detailing these 21 patients there were 13 with sinus node dysfunction. Of these 13 there were 11 with sinus bradycardia with paroxysmal AF. The author even references an article by Haissaguerre et al. that alludes to the above viewpoint.

Regarding PACs originating outside the PVs, my opinion is that some sort of ectopic nodal tissue has to be involved. See Session 35 in the CR. These P cells have been described in the PVs of AFers but not in controls (small Natale et al. study) and cells morphologically very similar to P cells have been described in several EP studies from Taiwan. Additionally they have been described in areas of atrial stretch (mechanical stress).

The reverse remodeling ("destretching") that occurs during the period of nerve fiber regeneration may result in fewer PACs postablation for some. Others may have "agglomerates of rogue cells", as Hans calls them, left behind in the atria. These foci are probably what are identified by the increase in AF cycle length that occurs during ablation of certain foci. I'm sure you've read post ablation reports by the EP describing 10-15 ms of cycle lengthening was achieved after ablation a site in the coronary sinus ostium or the posterior wall of the left atrium or ... Of course, for most the vast majority of the AF cycle lengthening occurs after isolation of the PVs.

Bill,

Perhaps George is correct in that it's not the atenolol it's the flecainide that decreases your ectopic activity. Decreased vagal tone caused by the flec would make an ectopic P cell or its equivalent less susceptible to sympathetic stimulation (increased automaticity), because the background HR (automaticity) is higher.

More food for thought.

PC

Hi Ruth,

PACs are primarily a product of hypokalemia, atrial stretch and sympathetic stimulation, i.e., stress, ?GERD, and ?.

Therefore, because there is a reduced cardiovascular response to stress with advancing age, there should be fewer PACs over time, all else being equal. But if you begin to develop high blood pressure or decrease intake (or increase loss) of potassium, the equation changes.

Regarding your other questions, there is ABSOLUTELY no question that my PACs have decreased post ablation. It's just that they seem to occasionally increase sufficiently to appear on my "radar". They're not particularly bothersome. As noted by Lou, preablation they were harbingers of an episode and no one needs such reminders.

PC

PC: One quick note that I had "forgotten" regarding nerve fibers vs ganglia ablation-- In a significant number of PVI's, continued electrical activity can be seen in the PV's which have been successfully isolated. This activity suggests that the blockade is working but perhaps the (vagal) ganglia are still active, at least in those significant cases studied. I have a reference somewhere if needed...didn't mean to come back as "devil's advocate" !

After my ablation I suspected something like that as I would experience some "butterflies" that often preceded AF, yet no AF would appear. After I read about the continued electrical activity I wondered if this could be the source of my "butterflies". I kept very watchful during the first year after ablation, with HR monitor, B/P monitor, and three Holter tests. During that time the "butterflies" disappeared and I conveniently forgot about them. Hope to get another Holter monitor test soon, but my others were more PVC's than PAC's and I never could tell the difference without Holter results.

Regards,
Anton

Hi Anton,

You're absolutely right. Natale and others have demonstrated reentrant arrhythmias in the successfully isolated PVs that don't communicate with the atria. I believe our James D experienced that.

Who knows what that would feel like? It would more likely be due to sympathetic stimulation of ?P cells in the PVs. Successfully ablated vagal ganglia would certainly make the left atrial soil less receptive to initiation of AF.

PC

My experience, FWIW....

Female, aged 68. Paroxysmal AF for many years, gradually increasing in frequency and duration until it became permanent in 2001. Ablation in Bordeaux Jan 2003, touch up 3 days later because of recurrence. One 2-hour episode of AF 10 days later, NSR since then. Annual 24 hour holter found no AF but some PACs and occasional PVCs..

After the ablation I had fairly frequent ectopics which Pr. Haissaguerre said I should forget about because everyone gets them. This I did, but in the last 9 months they started to become more frequent, and I started to get atrial bigeminy. These progressed rather like the AF had done, ie more frequent and longer duration. Thought about Hans's PAC tamer but it has too many calories - I have lost 84 pounds since the ablation through diet and exercise and I want to stay slim.

I sent my 'annual report' to Bordeaux in January and asked about the ectopics and bigeminy. They replied that no medication was required for ectopics even in bigeminy, but suggested that I might try taking magnesium tablets.

January 2006 - started with chelated magnesium, gradually increased to 900mg per day then reduced back to 800mg because of bowel intolerance. No change in ectopics or bigeminy.

March 6th. Started Taurine, gradually increased to 3000mg per day. Ectopics and bigeminy slightly reduced.

April 4th. Started potassium chloride, ½ teaspoon a day. Ectopics dramatically reduced. Feels as though someone has turned the volume down, and the few I now get are very gentle compared with before supplementation when it felt as though my body was being shaken by them.

I now get whole days when I hardly feel a single ectopic and have no bigeminy at all. For example, I feel my pulse in bed at night and used to have only 2 to 10 normal beats between ectopics. Last night it was 203, then I gave up counting.

This is still an experiment in progress. I intend to stay on present doses of supplements to get a longer term picture, then will try adjusting the quantities one at a time until I get the perfect cocktail.

The only drawback is the heap of pills I have to take - hard for someone who has never taken pills for anything in the past, but worth it to get rid of the ectopics and bigeminy.

Gill

Gill, as you may have guessed, i would very much like your excellent post on getting rid of pac's and pvc's to be available to be accessed via The List. This account is just exactly the kind of thing people need to be able to read when looking for a solution to ectopics, whether before or after ablation. I cannot just mark it for The List as i would if it were in the general bulletin board, because the search function does not, to my sorrow, search the Conference Room sessions. Please will you post this to the bulletin board? You have given such a careful, exact account, i would really like for people to be able to find it via the search function in the regular bb.

PeggyM

Gill: Great news for those of us who have residual PAC/PVC's after our ablations. Thanks for sharing.

Considering the quantity of taurine and maybe others you are taking you might consider taking them in powder form instead of horse pills. If iherb through Hans doesn't have it, try www.beyond-a-century.com. I believe Peggy has a potassium powder she gets through iherb although it sounds like you are buying that as a powder already.

Gordon

I did not mention this earlier in this thread, but I also have had what I call "blocked beats." With these, I have that butterfly feeling in my chest, but when I feel my pulse, it is unaffected.

I assume these are ectopic beats coming from the PVs that get stopped in their tracks by the ablation wall. I always felt the more ectopics being generated, the more likely some would find a hole in that wall, possibly causing a full ectopic.

As long as I stay on the flec and atenolol, I do not even get these. So it appears the meds reduce/prevent the ectopics at the source - the PVs. My guess is that, for some, the magnesium/potassium/taurine combination does the same thing. For some, this works even before an ablation. For others, it works only after the ablation. And for still others, it doesn't work too well at all.

The whole idea of ectopics after an ablation was a bit muddled to me. I mean the ablation was designed to stop the ectopics that lead to fib. But even though the fib is gone, the ectopics are still there! Dr. Chinitz explained this well to me. But I don't understand it well enough to explain it here. It's just another step beyond the PVI that changes the nature of the heart tissue.

BillB

Another thought occurred to me after my post. Doe the magnesium/potassium/taurine combination do the same thing as the flecainide does? Slow down the QT interval? And if so, is it any less healthy to do this with flecainide than high amounts of magnesium, etc.? Also, is the main problem with flecainide the intended effect, slowing the QT interval, or whatever additional things it does that are unintended? Or both?

I do not ask this to stoke the perennial natural vs pharmaceutical battle. But I assume flecainide does come from a natural substance of some sort, and is just super concentrated. And by ingesting large amounts of minerals we are

creating the same kind of super concentration. And excessive amounts of minerals cause potentially dangerous side effects, too. intestinal problems, kidney problems, bacterial formation, etc.

Maybe I should just take the flec and not worry about it?

BillB

Bill,

I would say the K/Mg/Taurine is better & much lower level of potential side effects. That being said - whatever works for you is best. The supps work for me and I've not had to use any flec for a year - this is great. My use of flec was limited to on-demand. I found that the supps helped the flec. That is, my conversion time went from 20 hours during the initial phases of my supplement program to 20 minutes after 4 or 5 months. After that the supps were enough to keep the AF at bay, so I didn't need the flec at all.

My own feeling is that the supps are correcting an electrolyte imbalance. The flec also works on the electrochemistry, but is a more draconian solution.

Again though - if the flec works & the supps don't - take the flec. Our systems are all different.

George

BillB:

Post Ablation Activity:
You claim not to understand it but your posted words say you do; at least to the degree I do. The ablation is mainly a PVI where Isolation = blockade. The ablation gets as close to the offending source as is safe and then a blockade is set up. In some, all electrical activity from the PV's seems to halt and in others continued electrical activity which is stopped by the blockade has been measured. (The caged bird still sings!)

Actually the action is far from being fully understood, so join the (muddled) theorists with your best guess. My "butterflies" that once heralded an onset of my AF, no longer were predictors after ablation. And they seemed to have gone away after almost a year for no known reason. I have no way of knowing if this feeling was PV electrical activity or not, but just know I remained in NSR. But a few ectopics still come calling-- my Holter showed they were mostly PVC's and very little PAC's -- I could never distinguish between them when they happened. Since they both are very few in number I consider myself luckier than those of you with problems. The various combinations of what works highlights how different we can be towards this stuff!

Best of luck with your choices,
Anton

PC: In the above message I didn't insert the results of my Holter monitor 90 days after my ablation. The summary over a 24-hour period as relates to ectopics is:

Underlying atrial rhythm was sinus at rates from 62 to 145 per minute averaging 78 per minute. Slowest HR between 1300 and 1400; fastest in the 0800 hour.

79 PAC's, more during waking than sleeping hours; 2 pairs during the 0700 - 0900 time frame.

2130 isolated PVD's, most during the 0300 - 0900 hours

21 pairs of PVC's, must during the 0600 - 0900 time frame

Six runs of PVC's all occurring in the 0700 - 0800 hours; with up to 6 beats in a row corresponding to rates as fast as 193 per minute.

No long pauses. No AV block. No conduction disturbances.

The above would seem to correlate with your "all vagal" theory since all happened at night when the vagus nerve is relaxed.

A codicil to this is that I am still a little hyperthyroid from past Amiodarone use and therefore still on Atenolol, Coumadin and Tapazole. Also, at that time I was supplementing with taurine at 1 gm/day; now 3 gm/day.

Any correlation to your situation?

Gordon

Gordon,

Sorry I didn't answer your question before.

I do take taurine but not arginine and only a minimal dose of taurine at that (500 mg). I mainly concentrate on potassium.

Regarding your Holter results, I have more PACs per day (about 150 or so, unless I encounter the very infrequent above described episodes). But I have far fewer PVCs (about 1-2 per hour), but, like you, they occur at night during sleep with almost 50% between midnight and 2AM. Upon comparison with the number of PVCs recorded on a 24 hour Holter several years before my ablation they have decreased by 95%. This can only be due to better potassium balance and decreased vagal tone. I think physiologic beats tend to occur at night during high vagal tone, when they are really escape beats. On my Holter recordings these PVCs are narrower and only the absence of a P wave helps to identify them. Otherwise PVCs have a much wider QRS complex.

Like you, my PACs are spread throughout the 24 hours. However, I think there may be a slight correlation with meals. PAC count goes from a background of a couple per hour to perhaps a dozen or two that crop up after meals. Unfortunately I haven't kept records of when I eat or more specifically when I finish eating. Further exploration in this area is planned. I'm sure only a minority of LAFers have this problem, but only those with GERD seem to know about the correlation. A dozen or two PACs in an hour doesn't really appear on my radar.

Wil Schuemann was good enough to post on the BB some time back his Holter results with and without potassium supplementation and I've included his data below. It certainly mirrors my experience.

06/22, successful ablation by Dr. Natale.
06/28, 18 total ventricular ectopic beats, 603 total atrial ectopic beats. 06/29, 456 total ventricular ectopic beats, 468 total atrial ectopic beats. 06/30, 209 total ventricular ectopic beats, 2179 total atrial ectopic beats. 07/03, 41 total ventricular ectopic beats, 108 total atrial ectopic beats. 07/07, 15 total ventricular ectopic beats, 99 total atrial ectopic beats. 07/10, 5 total ventricular ectopic beats, 108 total atrial ectopic beats. 07/14, 57 total ventricular ectopic beats, 62 total atrial ectopic beats. 07/15, 8 total ventricular ectopic beats, 67 total atrial ectopic beats.
07/18, 54 total ventricular ectopic beats, 132 total atrial ectopic beats. 07/22, 5 total ventricular ectopic beats, 85 total atrial ectopic beats.
07/25, 19 total ventricular ectopic beats, 64 total atrial ectopic beats. 07/29, 4 total ventricular ectopic beats, 88 total atrial ectopic beats.
08/02, 37 total ventricular ectopic beats, 100 total atrial ectopic beats. Holter data with 4 gms of KCl supplementation (24 hours of data):
08/06, 0 total ventricular ectopic beats, 61 total atrial ectopic beats.

PC

PC - you said:

"However, I think there may be a slight correlation with meals. PAC count goes from a background of a couple per hour to perhaps a dozen or two that crop up after meals."

This rings of a food-related symptom to me - as in leaky gut or a food allergy/sensitivity reaction. Responses of elevated pulse and even the ectopy leaves me suspect of the previous meal or an accumulation of a burden that becomes overloaded by the insult of the next meal.

Are you aware that you eat the same foods day in and day out; as - breakfast is always "ham and eggs" or milk and cereal? etc.

Foods repeated at every meal, every day can and do have adverse effects especially if leaky gut syndrome is in place. One example that comes to mind is based on a person I know who has an obvious wheat allergy. He eats pasta and bread every day and not long after every meal, he goes into a fit of sneezing that can only be linked to allergy. He's a dentist with some has biochemistry knowledge but refuses to acknowledge the connection. Co-incidentally, he suffers greatly with degenerative arthritis and has had knee replacements and is looking to hip replacement. The holistic circles link this degeneration to food allergy from LGS. I am just expanding this to possibly why PACs might occur after meals in some people and based on your comment above.

I know that when I had candida overgrowth, after each meal, I'd get PACs that frequently led to AF. Once I killed off the candida, that syndrome stopped - and then I progressed to bedtime AF. :)

You could consider requesting the Intestinal Permeability Assay from Great Smokies.

Jackie

PC: I see that Wil takes 4 gm/day of potassium (citrate?) and since he says it is a supplement it must be in addition to his dietary potassium.

You don't say how much you take and I'd be interested to know.

I keep reading that one can OD on potassium supplements and I seem to remember reading that 15 gm/day for total intake is the upper limit and that is for people without kidney disease or other potassium related issues.

I also remember reading that the normal intake through foods is around 5 gm/day. I now get about an extra gram daily through LS V-8 juice and magnesium/potassium glycinate pills, but I take Fosinpril, a potassium sparing ACE inhibitor so I may be getting 6 gm/day in total. Probably not enough, eh?

I have a regular appointment with my cardiologist Thursday morning so I'd appreciate any and all data and references to take with me to discuss with him. He likes pills with double blind tests and not supplements.

Thanks, as always, for your prompt and cogent responses.

Gordon

aloha Jackie,

Thanks for your comments.

Who knows? It certainly is possible. Kind of hard to separate the exact cause, e.g., GE junction irritation or allergy.

The one recent episode of increased PACs followed a meal at Genki Sushi. I ate nothing unusual. In fact I ate exactly what I usually do and that has never been correlated with increased atrial ectopy, at least to my knowledge.

But I have to look at all of this more closely. It's more of a curiosity that an irritant.

PC

Hi Gordon,

I don't take anywhere near as much K supplements as you do - maybe only a gram or so. But I eat a ton of fruits and vegetables.

The 15 gm figure as the upper limit for K intake is mentioned in Michael Lam's book (www.lammd.com). That's a helluva lot of K.

Please ask your cardiologist what he thinks of all those PVCs, especially the couplets and runs. Rather curious in the face of so much K. Runs of monomorphic PVCs have certainly been described in normals, but it would be good to explore that further. No need to win the AF battle only to lose the CV health war.

PC

PC: I take less than one gm/day of supplemental K, and that's considering the 500 mg from the V-8 as supplemental. The other 5 gm supposedly what the normal person gets from dietary intake. I was quoting your email on Wil taking the 4 gm/day of supplementary K.

It sounds to me like I'm getting about the same total K as you are, maybe less, as I consider myself a normal amount of veggie consumer.

Gordon

PC - What about this?

Did you eat sushi? raw fish? mercury content? Can you be sure it wasn't mercury or PCB etc? Each fish has to be different in toxicity or lack thereof.

Leave no stone unturned.

Also - in the next post, you say.... I eat tons of veggies and fruit... is it weighted more toward fruit than veggies? If so, I'm reminded of your excellent CR post on potassium depletion with increased insulin requirements. Fruit, while certainly full of nutrients, is so loaded with fruit sugars that in order to manage my insulin sensitivity issues, I am very limited with fruit intake. You could be depleting more K++ than you're taking in.... would that be possible? Or at least not getting to the optimal IC saturation.

At the end of the day, I still think the answer lies in potassium or lack thereof.

But I still have another hypothesis that I'll try to assemble and present eventually for more knowledgeable minds than mine to consider.

Be well,
Jackie

Hi Jackie (sorry about the lower case aloha previously for you certainly deserve the upper case),

Thank you so much for your concern and always-useful information.

Although I have to agree with you that more often than not potassium is at the root of most PACs, I don't believe this is always the case. Plenty on the BB have not found as much utility in potassium supplementation as we have. Furthermore, in this instance potassium supplementation didn't do anything for me v. the usual situation in which they disappear within 5-10 minutes.

Why the increase in PACs on this particular occasion, who knows, but I plan to investigate it further with the Holter.

Thanks
PC

P.S. I'm top heavy with broccoli, avocado and bean sprouts and lesser amounts of fruits (oranges, apples and bananas mostly).

Aloha Gordon,

If I had that many PVCs I'd try some sustained release potassium, like K-Dur (800 mg of K as KCl). Some are gastrosensitive to K as KCl, but I'm not. It's only available by prescription and you might ask your cardiologist about it. Blood K has its diurnal nadir around midnight and could explain at least some of those PVCs. It would be a nice experiment, if you were on a Holter.

Let us know what he says about them.

PC

Hi PC,
I'm a lone voice in the wilderness here but sounds like a gastric disturbance after eating your meal leading to tachygastria –rapid abnormal gastric electrical activity. The link below is based on babies and children but would equally apply to adults.

"The presence of a motility disturbance of the stomach has been reported in GER, but is generally felt to be less common. Abnormalities in gastric motility have been reported both using nuclear medicine looking at emptying times and by measuring antral motor activity with motility catheters.

It is felt that the majority of these gastric emptying disturbances are related to abnormalities in gastric electrical activity. This abnormal activity may consist of rapid electrical activity (tachygastria) or slowed activity (bradygastria). The authors were able to confirm an increased prevalence of abnormal electrical activity in children with GER.2

Another study showed that the epithelial permeability in the stomach to hydrogen ions differs between healthy subjects and patients with active GER.3 All, these studies show, is simply that there is impaired function. Chiropractic has been shown to affect stomach acidity and gastric motility by having a direct stimulating affect on the thoracic sympathetic nerves and on the vagus nerve."

http://www.icpa4kids.org/research/articles/pregnancy/Ultrasound.htm

Dean

Gordon, about that K intake, have you ever kept a food diary for a few days or a couple weeks, just to make sure you are really getting as much K in your food as you think? That 5 grams is the ideal, and it is not what the average person consumes at all. "Normal" [that slippery word!] potassium intake is a lot lower than that.

PeggyM

Peggy: I haven't done that yet. I'm still trying to figure out what would be a safe level of potassium that might reduce or eliminate the PVC's. I know that is below 15 gm/day and suspect it is above the 5 gm that we get from food if we eat right.

My present thought is to continue my present eating pattern, which is pretty consistent because I am on Coumadin, and gradually increase the K to see when the ectopics go away.

However, I don't feel them and felt very few when I was on the Holter so I don't know of any way to tell if they're reducing other than another Holter day. I have a Medisana Cardiocheck on the way to me but it isn't here yet. Any ideas?

Someone posted a while back that if we could feel our heartbeat we probably weren't getting enough magnesium. Dr. Lam's recommendation of 500 mg maximum has to be translated into the effective amount of magnesium from the amount of the supplement. For example, I take magnesium orotate, 500 mg/day; how much of that is magnesium absorbed? Dr. Lam says there is no maximum for taurine. I presently take about 2 gm/day.

I need a lot of research yet, the first part of which is to see what my cardiologist will go along with in my well baby checkup tomorrow. Ill also ask him about arginine but I suspect I know the answer to that already. Of course, physicians give ADVICE, not orders and we patients have to ultimately decide for ourselves what to do.

Gordon

"Someone posted a while back that if we could feel our heartbeat we probably weren't getting enough magnesium."

Gordon, that was potassium, not magnesium. Check with Jackie about that, she was the one who taught it to me.

PeggyM

Peggy,

You are correct - I believe it was a chiropractor of Jackie's that told her that & PC later referenced it, too.

Gordon,

As I recall, the 15 grams max is per dose at one time, not per day. I agree with Peggy that most do not get anywhere near 5 grams/day from food. I've personally taken 3 grams/day for 18 months with no deleterious consequences. I don't think that any of us, from what I've seen on this board are skirting with maximums. Most of us are struggling to keep our serum K above 4 mmol/l. There are a couple of cases whose serum K was 5 or 5.5 mmol/l & they should closely monitor their K intake, so this serum K does not increase more. If anyone has kidney issues then that is a different story.

Paleo data indicate a K/Na ratio of 20:1, with most today, the ratio is 1:5, a 100 fold difference. There is some data that blood pressure normalizes with a ratio above 5:1. My geologist friends say that their understanding of rocks, soils and mineral concentrations supports the 20:1 ratio.

George

Gordon - a quote from our friend, Michael Murray, and from my original potassium post a long time ago:

Michael Murray, N.D. says: "It is critical to maintain potassium levels within the body. This can best be done by consuming foods rich in potassium and avoiding foods high in sodium. The daily intake of potassium should be at least 3 to 5 grams a day."

"Most Americans have a potassium-to-sodium (K:Na) ratio of less than 1:2. This 1:2 ratio indicates people ingest twice as much sodium as potassium. Researchers recommend a ratio of 5:1 to maintain health….or 10 times higher than the average intake.

Some of the **potassium containing foods**:

Asparagus ½ cup 165 mg. potassium
Avocado ½ 680

Carrot, raw 1 225
Corn ½ cup 136
Lima beans, cooked ½ cup - 581
Spinach, cooked ½ cup 292
Tomato, raw 1 med. 444

Apple 1 med 182
Apricots, dried ¼ cup 318
Banana 1 med 440
Cantaloupe ¼ melon 341
Peach 1 med 263
Strawberries ½ cup 122

Unprocessed meat
Chicken 3 oz. 350
Lamb, leg 3 oz 241
Roast beef 3 oz 224
Pork 3 oz 219

Cod 3 oz 345
Flounder 3 oz 498
Haddock 3 oz 297
Salmon 3 oz 378
Tuna, drained 3 oz 225

Signs of Potassium Deficiency and Symptoms
Muscle weakness
Fatigue
Mental confusion
Irritability
Weakness
Heart disturbances,
Nerve conduction problems
Problems with muscle contraction
-often seen in the elderly

Dietary deficiency is typically the cause – too much sodium; low potassium. However, dietary deficiency is less common than that among people who regularly exercise and have higher potassium needs.

POTASSIUM LOSS FROM EXERCISE
The amount of potassium lost in sweat is quite significant, especially with prolonged exercise in a warm environment. Athletes or people who regularly exercise have higher potassium needs. Because up to 3 grams of potassium can be lost in one day by sweating, a daily intake of at least 4 grams of potassium is recommended for these individuals.

REPLACING POTASSIUM
Over 95% of the body's potassium is in the cells. A potassium shortage results in lower levels of stored glycogen. Because exercising muscles uses up glycogen for energy, a potassium deficiency produces great fatigue and muscle weakness, the first signs of potassium deficiency.

Potassium supplements are available in forms of either potassium salts (chloride and bicarbonate) potassium bound to various mineral chelates (aspartate -a no-no for afibbers-, citrate, etc.) and food-based potassium sources. Supplements are restricted to only 99 mg. per dose because of problems associated with high-dosage potassium salts; however, popular so-called salt substitutes such as NoSalt and Nu-Salt are potassium chloride and provide 530 mg of potassium in 1/6 of a teaspoon. Potassium supplements are also available by prescription in flavored formulas but can produce nausea, vomiting, diarrhea and ulcers when given at high-doses.

Dr. Murray recommends only food sources or food-based supplements.

The estimated safe and adequate daily dietary intake of potassium set by the Committee on Recommended Daily Allowances is 1.9 grams to 5.6 grams. If diet does not meet body potassium requirements, supplementation is essential to good health. This statement is particularly true for the elderly, athletes, and people with high blood pressure.

SAFETY ISSUES
Most people can handle excess of potassium. The exception is people with kidney disease and they may experience heart disturbances and other consequences of potassium toxicity. Individuals with kidney disorders usually need to restrict potassium intake and follow the dietary recommendations of their physician. Supplements are contraindicated when using a number of prescription medications, including digitalis, potassium-sparing diuretics and the angiotensin-converting enzyme inhibitor class of blood pressure lowering drugs.

Jackie

Yes - it was my chiropractor who quoted from a nutritional pioneer, Royal Lee, that if you can feel your heart beating at night when you lie down, it's a potassium deficiency. PC did chime in and acknowledge observation as well.

Jackie

George -

" Paleo data indicate a K/Na ratio of 20:1, with most today, the ratio is 1:5, a 100 fold difference. There is some data that blood pressure normalizes with a ratio above 5:1. My geologist friends say that their understanding of rocks, soils and mineral concentrations supports the 20:1 ratio."

I believe the information conveyed here going back to paleo diets and eras indicates that the intake was mostly from plants with an occasional kill of meat that would last for some time....as compared to the modern day diet, and of course, the animals consumed had a natural balance of sodium to potassium resulting from their own internal homeostasis.

Salt (as a preservative or condiment) in paleo times was only available if in that particular region. In later years, it became a highly sought and a precious commodity - was brought in to communities via caravan/ships etc. from those places harvesting it but in the early paleo times - salt (added) didn't unbalance the potassium content at all. We've evolved to the point where sodium chloride permeates everything that isn't harvested and eaten in the natural form.

It would be difficult to OD on potassium from natural foods.

Jackie

From Krispin's URL:

Formula for daily potassium goal:
Your Ideal Body Weight in pounds times 40 mg. =_____ mg. per day.

For me, at 190 pounds, I would need 7.6 gm/day of potassium. Unless I can learn to exist on baked potatoes and tomato juice, I ain't gonna make it without supplements. Using her food chart I got about half that yesterday.

Thanks for the correction on potassium vs. magnesium sufficiency for feeling your heart beat in bed.

Gordon

Hi Dean,

Thanks for your input.

I'm coming around to your way of thinking. Years ago, I suspected that it was some kind of reflex similar to the swallowing reflex and AF or cold fluid ingestion and AF, and then I began to consider GE junction irritation of nearby cardiac vagal ganglia. Given the temporal qualities of the described episode of increased PACs, I'm with you on some kind of direct electrical stimulation/connection between the GE junction and those cardiac vagal ganglia.

Theoretically, there should be a postprandial increase in PACs beginning after about 30 minutes after eating no matter what I eat. The actual numbers may vary from day to day but the temporal correlation should still be discernible.

PC

PC
You state:
“Theoretically, there should be a postprandial increase in PACs beginning after about 30 minutes after eating no matter what I eat. The actual numbers may vary from day to day but the temporal correlation should still be discernible.”

I’ll run this theory by you to explain the above statement and see what you think. A while back you posted your theory about the origin of the rouge pole cells in the neural crest in the developing foetus and how this was behind the “faulty” electrics in afibbers. I always thought your theory was pretty spot on but didn’t go far enough. Why would this only affect the heart? Nature is not that accurate. It is known that afibbers have significantly increased gastric problems compared to the rest of the community, so based on this, I’m of the opinion the electrics of our digestive systems are faulty too through the same process you describe with the neural crest and pole cells.

In other words, the same process that gives us afib and ectopics in the heart is occurring in the digestive system, the esophagus and stomach. I base this on the comprehensive gastro tests I had when my GP, gastro and EP all came to the conclusion that my afib and ectopics was originating from my esophagus and stomach.

I had a Barium meal test in 2002. I was having mild ectopics during the procedure and when given the heavy barium meal it lodged in the lower esophagus and was exceptionally slow to enter the stomach. The doctor supervising said it was unusual and indicated a motility problem in the lower esophagus. The same thing happened when I swallowed the marshmellow he gave me. The doctor actually rotated the screen to show me the barium lodged in the lower esophagus. I then visited the gastro and he said the barium meal test was “wrong” and sent me for a esophageal motility test (suppose to be far more accurate than the barium meal) and pH study. When I had the esophageal motility test I never had ectopics before or during the procedure so interestingly the test showed NORMAL esophagus motility.

With the barium meal test, had I witnessed with my own eyes the actual mechanism of my af, the INTERMITTANT functioning of the motility of lower esophagus causing a vagus electrical storm and ectopics?

Is a similar thing responsible for your (and many others) occasional intermittent ectopics after a meal? I don't think you have had a motility test?

I know of very few afibbers who have had the esophagus motility test or the pH study (both tests are physically disgusting!) Furthermore, I know of no one who has had an EGG (electrogastrogram). Maybe a combined ECG, EGG and pH study, will reveal more? I know this has been done and reported on PubMed while the person was going in and out of afib but I can’t locate it.

A penny for your thoughts, PC.

Dean

Hi Dean,

I've got plenty of thoughts on that topic and posted some of them in CR Session 33. Look in the section on swallowing and AF. Swallowing induced AF and esophageal motility abnormalities are closely related. Although I suspect it's more

prevalent than a "rare disorder", it's not the grand unifying theory for LAF either.

In addition to the references listed there are others as well:

Swallow syncope associated with paroxysmal atrial fibrillation.
Gordon J, Saleem SM, Ngaage DL, Thorpe JA.
Department of Cardio Thoracic Surgery, Yorkshire Heart Centre, Jubilee Wing, D-Floor, The General Infirmary, Leeds, UK.

Swallow or deglutition syncope is a very unusual potentially lethal but treatable disorder. We report the case of a 26-year-old woman, who presented with a history of recurrent, multiple fainting episodes precipitated by swallowing. Twenty-four-hour manometry and pH recording together with continuous 24-h ECG monitoring revealed multiple episodes of symptomatic and asymptomatic paroxysmal atrial fibrillation, and significant gastro-oesophageal reflux associated with swallowing. Oesophageal function tests and continuous electrocardiographic evaluation is important in the diagnosis of this rare condition.
Eur J Cardiothorac Surg. 2002 Mar;21(3):587-90.

Radiofrequency catheter ablation therapy of swallowing-induced atrioventricular nodal reentrant tachycardia: report of two cases.
Satish OS, Yeh SJ, Yeh KH, Wen MS, Wang CC, Chou CC, Wu D.
Nizam's Institute of Medical Sciences, Hyderabad, India.

We describe two patients who presented with a history of recurrent palpitations on swallowing of solid food. The event-recorder and Holter monitoring documented episodic supraventricular tachycardia (SVT) initiated by atrial premature contractions (APCs). During electrophysiological study (EPS), swallowing of solid food consistently induced APCs and their activation sequence, morphology of P wave were suggestive of their right atrial origin in them. Drug challenge did not affect the APC onset during the swallowing. During EPS, slow-fast variety of atrioventricular nodal reentrant tachycardia (AVNRT) was induced and successful radiofrequency (RF) catheter ablation of slow pathway resulted in total relief of their symptoms. (PACE 2005; 28:594-597).
Pacing Clin Electrophysiol. 2005 Jun;28(6):594-7.
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=15955197&dopt=Abstract

Dynamics of Swallowing-Induced Cardiac Chronotropic Responses in Healthy Subjects
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Abstract
Simultaneous recording of ECG and swallowing movements in healthy humans (n=23, age 20-57 years) showed that each swallow is accompanied by transient tachycardia with initial abrupt and pronounced heart rate increase. These rapid changes in heart rate (evaluation by maximum increment of heart rate over two successive heartbeats, ?HR2bt) are typical of vagal chronotropic responses. The amplitude of tachycardia induced by a single swallow was significantly higher in the supine position (13.1±5.6 bpm) compared to the standing position (8.5±3.8 bpm; $p<0.0001$). Chronotropic responses to a series of three or more successive swallows consisted of two phases, the initial abrupt acceleration and subsequent slower growth of heart rate. In the standing position, the portion of the first rapid phase significantly decreased, while the portion of the slower phase increased compared to the supine position. The amplitude of tachycardia induced by a single swallow and parameter ?HR2bt can serve as indices of the strength of parasympathetic modulation of the heart. By contrast, further slow increase in the heart rate determined by summation of responses to a series of successive swallows can result from not only inhibition of the parasympathetic influences,

but also enhancement of sympathetic activity during swallowing.
Bulletin of Experimental Biology and Medicine
135 (4): 322-326, April 2003
http://www.kluweronline.com/article.asp?PIPS=471664&PDF=1

Successful treatment of deglutition syncope with oral beta-adrenergic blockade.
Marshall TM, Mizgala HF, Yeung-Lai-Wah JA, Steinbrecher UP.
Division of Cardiology, University of British Columbia, Vancouver.

A case of deglutition syncope of 20 years' duration in a patient without cardiac or esophageal disease is presented. The therapeutic efficacy of beta-blockade is documented by symptomatic improvement, repeat esophageal balloon inflation and tilt-table testing. This suggests the Bezold-Jarisch reflex or sympathetic nervous system may be involved in the pathogenesis of deglutition syncope.
Can J Cardiol. 1993 Dec;9(10):865-8.
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=7904229&dopt=Abstract

Swallow Syncope.
SRIVATHSAN, K., et al.
Swallow syncope is a rare disorder caused by hypersensitive vagotonic reflex in response to deglutition. A 26-year-old man complained of recurrent light-headedness and near syncope on swallowing was hospitalized for monitoring and evaluation. Continuous electrocardiographic and invasive arterial pressure monitoring showed ingestion of a solid meal evoked light-headedness and complete AV block without an escape rhythm that lasted for 5.6 seconds. This patient received a Medtronic Kappa (401B) DDDR pacemaker with the rate drop feature. The patient has remained asymptomatic on follow-up for the past 2 years. (PACE 2003; 26:781-782)
http://www.blackwellpublishing.com/abstract.asp?ref=0147-8389&vid=26&iid=3&aid=22&s=&site=1

PC

PC: Just returned from my regular quarterly app't with my cardiologist and discussed the couplets and runs. He said they are a worry if there is any underlying structural damage to the heart or arteries. Since I don't have that and was in the hyperthyroid condition at the time of the Holter, he feels it is something only to watch at this time.

We also discussed K-DUR supplementation. On my last blood test a couple of weeks ago my potassium level was 4.82 which is toward the high end of normal so he doesn't want to prescribe that for me.

My EKG today was clean so I requested to get off Coumadin. His answer was that many times there is silent afib after ablations and some think that ex-fibbers should stay on Coumadin for the rest of their lives if they can tolerate it. He did agree under duress to live with the recommendations of the Natale team since they're more specialized in that than he is so I am getting an appointment to see Dr. Natale when he is in Marin on May 11 who will evaluate and send a report to my conservative cardiologist.

Salwa says that a big part of the whole goal of the ablation procedure is to get patients off drugs so she is sure that Natale/Hao/Hongo will apply that goal to me upon satisfactory examination results.

Thanks for all you help and inputs.

Gordon

Gordon,

One thought re: Coumadin & silent afib. To me this issue is one of duration. Certainly there is silent afib. However, how long does it last - 2 seconds, 2 minutes, 2 days or 2 weeks? I don't think that short runs of afib are really dangerous from a clotting standpoint, especially if you don't have other reasons for a clot to form (high BP & etc).

If you checked your pulse daily either manually, with a stethoscope or with a measuring device (Polar S810, Freezeframer), you could easily verify that you did not have silent afib and therefore would have not afib related stroke risk.

It seems to me that it would be healthier to check for afib this way than to stay on a drug like Coumadin forever. I wonder why the cardios never think this way -- it is because they don't think we are capable of recognizing afib on our own. I would venture to say that most of us here that are experienced with afib can easily recognize it.

George

Hi Jackie! Glad you reran your post about potassium. When I was in AF and after ablation I was always very attuned to my heart rate and there were other locations other than wrist and carotid artery that the pulse was evident. Ear, temple, and even knee come to mind. And even in bed with the knees flexed upwards against covers I can feel a pulse rate-- would you classify that as lack of potassium? I also have a slight tinnitus so when still I can "hear" the beat at least sometimes.....

Hope you're doing well and perhaps getting a little spring golfing.

Anton

George: Thanks and I think you're right. I have a FreezeFramer and a Medisana Cardiocheck so I should be able to tell if something's wrong.

Also, one cardiologist told me that the home blood pressure machines won't record a pulse if I am in afib or flutter but I'm not sure if they would distinguish between ectopic beats and fib.

Anyway, Salwa, the head EP nurse at Marin said she would teach me in a few minutes how to tell what's going on.

What's your opinion on potassium supplements if my blood test comes back showing my level in my blood being normal?

Gordon

Gordon,

As to K - if your serum readings are consistently in the 5 mmol/l area, then I wouldn't push more. Especially on an early morning, fasting test, when the serum levels are going to be at their low point. If you were lower normal - 4 mmol/l or less then I'd think otherwise.

Your said, "On my last blood test a couple of weeks ago my potassium level was 4.82 which is toward the high end of normal" - I would agree with your doc about not taking supplemental potassium. Working on food would be OK.

Afib shows up very obviously on the Freeze Framer. I have version 2.0 & on my software, I'd uncheck "Enable Artifact Detection" and "Enable HRT filter". To do this I go to Edit - Options. With these settings off, you should be able to "see" PVC's & PAC's. PVC's will be one beat downward (slow) spikes about 1/2 the rate of the beat before & after. PAC's will be one beat upward (fast) spikes or a fast followed by a slow. You must keep the hand with the sensor on it quite still while reading to avoid artifacts in the data.

After sampling, set the time axis to 30 and then scroll back through. You should be able to easily see the PVC's & PAC's then. Afib will look chaotic.

I don't have a FF example, but here is a rhythm strip of afib (first 10 minutes) from a Polar S810j. Later in the strip are examples of PVC's & PAC's:

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

Conversion is at about 10 minutes.

This is a zoom of some of the AF section:

http://home.att.net/~g.e.newman/af2.jpg

The FF rhythm strip will look similar. It presents a beat rate average every 1/2 second, instead of the actual beat rates, so there will be a slightly different character, but still the overall appearance will be the same.

George

Gordon,

Another thought re: supps (& I haven't carefully reread your prior posts). I would work on magnesium & taurine as they do help the cells utilize the K better.

George

George: Thanks. I do have a FreezeFramer and I'll work with it a bit. I also just got the Medisana Cardiocheck so I may get more data than I know how to handle.

However, with a sure fire way to test for fib I would test several times daily if it met I could get off Coumadin.

I may try George Eby's arginine addition to taurine and magnesium if Dr. Natale says I can get off the Coumadin, although I am having minimal ectopics felt these days with 2 gm taurine, 1 gm magnesium and 12 oz of LS V-8 per day; so if it ain't broke any more why try a different fix now?

Gordon

Gordon,

When I was having afib regularly, I could tell within 4 beats of a pulse point, just in front of my ear. When I had an episode, I'd usually be in denial and want to check again, then I'd go check with an electronic device, which would confirm my initial diagnosis that I was in afib.

From the description I've read on the internet of the cardiocheck device, I'd probably use the FreezeFramer to sample PVC's & PAC's (unless I were having them every couple of seconds). This is because they stand out really well on a rhythm strip.

I've found I can also use the manual pulse sampling for ectopics. I have to pay more attention than I do with the electronics, but I can focus on my pulse and count ectopics per minute (or hopefully - minutes per ectopic). I use this when I'm in the back country, camping and away from the electronics.

In any case, it sounds like you are well provisioned to determine whether you are in afib & it shouldn't take more than a few seconds, however frequently you feel you need to sample, to make sure you're not in afib.

Good luck!

George

I've been plagued with these PACS for over a year, usually before a bowel movement, and ceasing afterward. Meals seem to exacerbate the PAcS, likely due to peristalsis action on eating. These can last 2 or 3 hours, and cease right

after a bowel. This suggests irritable bowel disease or candidiasis intestinal permeability in my case. The cure is not easy though in theory simple. Kill off candida and heal the gut wall.

Wee

Hi Wee,

In one of the above referenced articles by Browning and Mendelowitz on "Musings on the Wanderer" it states "Further studies have revealed that stimulation of vagal afferents at different levels of the gastrointestinal tract elicits other vagally mediated gastrointestinal reflexes, including inhibition of food intake (45), inhibition of gastric emptying (40), motility (23), acid secretion (30), and pancreatic secretion (27). Vago-vagal reflexes are not, however, restricted to the gastrointestinal system. For example, activation of cardiac vagal afferent neurons elicits the Bezold-Jarish reflex comprised of an increase in cardioinhibitory parasympathetic activity and a decrease in sympathetic activity resulting in bradycardia and hypotension (4). Similarly, activation of respiratory vagal afferents evokes bronchoconstrictions (26)."

"The ability of nodose neurons to influence the activity of unstimulated neighbors may be a mechanism behind some examples of pathological disturbances in one organ affecting another organ. It is well known, for example, that a clear relationship exists between patients with airway hyperresponsiveness and gastroesophageal reflux disease (24), or irritable or inflammatory bowel disease (41). The ability of nodose neurons to communicate with each other may suggest that some of these effects may occur at the level of the vagal afferent neurons themselves."

So, is "Candida" the problem or is it excessive cross stimulation?

PC

In 2004 Hans and I discussed possible explanations for something we'd both observed on the Freeze Framer. A single swallow reflexively triggered an immediate but transient increase in HR. Hans even suggested we could start a research project to compare percentage increase in heart rate upon swallowing among afibbers and non-afibbers. While sitting, swallowing induced a 16% increase in HR for both Hans and me. The increase for the wonderful Mrs. Larsen was only 4%. For my equally wonderful wife it was 7%.

Googling the topic revealed Russian research done the year before that appeared in the Bulletin of Experimental Biology and Medicine.

The baroreflex appears to be integral in this phenomenon. Swallowing is accompanied by transient tachycardia with initial abrupt and pronounced heart rate increase. The amplitude of this tachycardia is greater in the supine v. the standing position and is due to a transient decrease in cardiac vagal tone (1-2 secs). In fact the increase in HR generated by a single swallow can serve as an index of the strength of parasympathetic modulation of the heart. A series of swallows can enhance sympathetic activity (6-10 secs) with additional increase in HR, underscoring the participation of both arms of the autonomic nervous system in the baroreflex.

There have been numerous electrophysiologic studies demonstrating PAC production triggered by swallowing. Infrequently this has been shown to induce supraventricular tachycardia and/or atrial fibrillation.

Prior to my successful ablation in mid July 2005 upon reclining for the evening I often experienced a dramatic increase in PACs (often followed by AF). Since then these PACs have completely disappeared. Furthermore, swallowing induced tachycardia is now only 7%, indicating that cardiac vagal tone has decreased post ablation. This would appear to support the view that PVI works because it destroys nearby vagal ganglia and not because it destroys their fibers (which regenerate).

For some HR postablation eventually returns to preablation levels, suggesting that vagal ganglia were not eliminated by the PVI. I often wonder what the long term success rate for PVI is in such individuals.

Given Hans' strong cardiac vagal tone, perhaps his stress induced
"adrenergic" lone AF is really just another manifestation of VMAF. Preablation my posture related PACs always

required at least 6-10 secs before appearing, indicating their sympathetic origin. But vagal tone paves the way.

Could lower esophageal motility in some predisposed individuals (?GERD) contribute in a similar fashion via the baroreflex to PACs and the onset of LAF?

What do you think Dean?

PC

Aloha PC,

Your % increase in HR seems so precise. How would you interpret this:
http://home.att.net/~g.e.newman/swallow.jpg

At each point marked, I took several swallows from a water bottle. There is an increase, however the baseline is a bit harder to compute because of the magnitude & sinusoidal nature of the variation in normal HR (not counting my lovely PVC's - time for my bedtime supps).

BTW when I hit Go To Top, you can see the tree structure of the responses.

George

George,

You are only supposed to take a single swallow to get a measure of vagal tone. Otherwise you begin to include sympathetic input after six seconds. Also, if you compress the data over a number of minutes, the baseline will become flatter, making measurement more accurate. I'll email you my tracing from May 2002 and the abstract of the referenced article, and all will become clear.

PC

PC,

"Could lower esophageal motility in some predisposed individuals (?GERD) contribute in a similar fashion via the baroreflex to PACs and the onset of LAF? "

Yes, I'm sure Dean WILL be most interested in this latest discussion and especially in your closing question from it as above.

As for myself, I'm heading for an endo exam this Friday owing to my most uncomfortable once-every-month-or-so esophageal spasms/cramps (as in low esophageal motility). These can wake me in the middle of the night or come on half-way through an evening meal. These occurrences typically take the form of an hour or two of intermittent cramping (makes your eyes water) with the cramping lasting 10-20 seconds followed by 30 seconds respite and so on until the whole thing gradually diminishes. To start the ball rolling, as it were, my doc arranged the endo as a first step with the gastro chap then maybe considering motility testing as a follow-up. I've always known FOR SURE that, for me at least, my upper digestive tract woes have always been closely intertwined with my frequent PACs and occasional AF. My upper DT problems started in 87/88 and the PAC/PVC issues eventually followed thereafter. Even now, an increase in heartburn/GERD/esophageal cramping/bloatedness will inevitably result in a flare-up of ectopic activity. Incidentally, are you aware of any meds which can increase esophageal motility? Or is it simply a case of eating very small means more often rather than only two big meals per day (yes, I know which is the most sensible regardless of upper DT issues!)?

Thanks for your latest thoughts - much appreciated as always.

Mike F.

Hi Mike,

Thanks for your post.

If you visit

http://ajpgi.physiology.org/cgi/content/full/284/1/G8#B36

and read "Musings on the Wanderer: What's New in Our Understanding of Vago-Vagal Reflexes?", you might find additional (more than anecdotal) evidence supporting our mutual view that cross stimulation of baroreceptor receptors during esophageal motility/swallowing can initiate LAF in the predisposed.

FYI the "wanderer" is the vagus, which you probably already knew.

Lending further support to the baroreflex connection with LAF is another article detailing the role of glutamate. Glutamate is a well known neurotransmitter substance within the CNS, but dietary glutamate does not readily cross the blood brain barrier. However, it now appears that outside the CNS it is not only associated with hypoglycemia via beta receptors in the pancreatic islets but also with the baroreflex via metabotropic glutamate receptors on nodose ganglia.

PC

Hi PC,

Thanks for the article and scan. I do now understand. I decided to try again, but recording with the Polar, as it actually reports beat rates, rather than on 1/2 second intervals which the Freeze Framer does. I took a swallow of water every minute, on the minute for 5 minutes. Here are the results from the Polar software. The first rate reported is at the minute, then the next 3 rates. The scan is here:

http://home.att.net/~g.e.newman/swallow2.jpg

On the scan, the downward spikes after 5 min 30 seconds are artifacts.

1 min
73 76 77 76
2 min
70 72 75 75
3 min
73 72 74 37 (PVC)
4 min
73 73 74 75
5 min
70 70 72 73

You can see your swallows even if they weren't labeled. Mine just seem part of the random beat variation. My HR was higher than yours, although when meditating this morning for 45 minutes, the average was 49 bpm, so I'm pretty vagal overall.

Here is this morning's scan - pretty quiet, only 3 PVC's during the 45 minutes.
http://home.att.net/~g.e.newman/meditate.jpg

I guess this is a good thing for me, I don't know.

Cheers,

George

PC:

Part of the problem with understanding the CNS activity is that one can't tell vagal and sympathetic balance. i.e. For example-- How much of each is reflected in one's resting heart rate?

Some early reports (1999?) suggested HR increase after PV ablation was transient and returned to normal in about 1 month. The trend now seems that there are permanent changes upward to HR and vagal denervation is the likely culprit. In one sense this represents ANS dysfunction as the vagal-sympathetic balance has been altered. For those with tachycardia issues this could be a big problem, but for you (and I) the increased HR is a blessing.

One other indicator of vagal-sympathetic balance is HRV. My Holter monitor results are not a good example as they include a lot of exercise in the days monitored. Using freeze-framer are both your and Hans's HRV results show evidence of a drop in vagal tone? When I awaken at night and take my pulse it "seems" faster than before ablation so this "suggests" a decrease in my vagal tone. Since the vagal ganglia were pictured quite a distance from the PV ostia, it's hard to convince me that they were ablated too. Perhaps I need anatomical help. :>)

Anton

Hi Anton,

Thank you for your continuing contributions.

Measuring total parasympathetic tone to the heart is a most difficult undertaking. As I see it, the primary problem is measuring the contribution of that part (the dorsal motor nucleus of X=10th cranial nerve=vagus n. or DMNX) that controls our HRs during sleep (diurnal). Measuring the more dynamic contribution from the nucleus ambiguus (NA) is less difficult. The referenced article seems to handle that quite nicely in stating "the amplitude of tachycardia induced by a single swallow and parameter ÄHR2bt can serve as indices of the strength of parasympathetic modulation of the heart". But this appears to be the branch of the vagus that causes AF.

I'm with you on the drop in vagal tone causing the increase in HR. Perhaps, as we grow older, the vagal nerve endings that are damaged by PVI are less likely to regenerate, at least in some individuals. This would get around the requirement for destruction of vagal ganglia to explain this drop in vagal tone.

The absence of PACs when I lie down for the evening is striking. Preablation their appearance was as dependable as the sun rising in the AM. In my mind the baroreflex connection to PACs is undeniable. When going supine, the increase in vagal tone paved the way for PACs due to the reflexive increase in sympathetic tone and associated increase in automaticity.

As I previously reported, my cardiac vagal tone (dynamic not diurnal) has decreased postablation. Not only is my baseline HR faster but also the per cent increase in HR with a single swallow is less than before. Also, my high frequency peak (vagal tone) on power spectrum analysis is less than before. This can be tricky to measure on the Freeze Framer because it can change depending on when this is measured. For instance, at the same HR I found that I could modulate the swallow induced tachycardia, if I performed this test a half hour after working out. The vagal rebound state at that time appears to accentuate the response.

PC

Hi PC,

Thanks for your detailed reply a couple of days back re the swallow syncope and esophagus motility. I noticed you started a separated discussion with experiments on this subject. Esophagus motility and GERD is my “thing” so hopefully I can contribute here as I have had all the gastro tests.

“There have been numerous electrophysiologic studies demonstrating PAC production triggered by swallowing. Infrequently this has been shown to induce supraventricular tachycardia and/or atrial fibrillation.”

-SVT was one of my main problems, but only after meals and not because of swallowing. Proved latter to be connected to reflux.

“Prior to my successful ablation in mid July 2005 upon reclining for the evening I often experienced a dramatic increase in PACs (often followed by AF)”

-When I had the 24hr pH study, I was watching the monitor strapped to my waist and when standing the pH was around 5 to 8 but on reclining the pH started to drop. At one stage I fell asleep on the couch for 10mins and was woken by ectopics. I looked at the monitor and it was reading 2. When the gastro showed me the 24hr pH graph he asked me when I had ectopics. The ectopics coincided with the sharp peaks on the graph ie. strong acid reflux. The graph was, unfortunately, quickly snatched by my EP and disappeared! So I have no record of it to show you.

“Could lower esophageal motility in some predisposed individuals (?GERD) contribute in a similar fashion via the baroreflex to PACs and the onset of LAF?"

-Again, I had 2 tests for this.
The first was the barium meal test - I was having MILD ECTOPICS throughout this test as it involved supine position (always ectopic inducing) on the examination couch and body being turned through 360 degrees sideways and length ways. When I drank the heavy barium it sat stubbornly in my lower esophagus and refused to enter the stomach. Attending doctor said this was unusual and I viewed the monitor distinctly showing the barium sitting in the lower esophagus. The same thing happened when I ate the marshmallow (light weight). It just sat in the lower esophagus. If I remember correctly the attending doctor wrote “very slow clearing of the distal esophagus”.

The second test is suppose to be the more accurate and involved sticking a tube in my nose and down my throat to the LES. It was then slowly pulled out an inch at a time. At each stage I drank a glass of water and readings taken. This proceeded until tube was out – disgusting!! I panicked before and during this test thinking it would put me into afib but there were NILL ECTOPICS throughout the test. The test showed NORMAL motility of the esophagus.

My gastro said the barium test was “wrong” and the motility test was correct……hmmmm.
My own conclusion is that with the barium test, faulty esophagus motility was causing ectopics OR……. ectopics were causing faulty esophagus motility (chicken and egg).
The more accurate electronic motility test recorded a normal reading because there were NO ectopics during this test.

Getting sidetracked - Another thing with the barium test was that it showed I have a “wandering stomach” ie. Stomach is not held in place by the other body organs consequently shifts its position and location. A small percentage of the population has this feature (is it common in afibbers who report afib originating in stomach area?) I have mentioned this before on the board but no explanation forthcoming re effects of afib. Could this be causing atrial/esophageal stretch? Any comments PC?

“Could lower esophagus motility in some predisposed individuals (?GERD) contribute in a similar fashion via the baroreflex to PACs and the onset of LAF?”
-In my experience an emphatic YES but it needs to be proven by more research.

I note Mike F is going to have a stomach scope on Friday. After the results of this maybe he can talk his gastro into having a combined 24 hr pH study and 24hr Holter monitor together at the same time? Now that should reveal something.

Dean

Hi Mike,
I notice your going to have a stomach endo on Friday. After the results of this and if the gastro sends you for more follow up tests maybe you can talk the gastro into having a combined 24 hr pH study and 24hr Holter monitor together at the same time? (if the UK health system will pay for it!).
Now that should reveal something.
Dean

Hi Dean,

Clearly there is a connection between the cardiac vagus and the GI tract vagus. The only real question is "Does this involve a reflex (and the brainstem) or is there some kind of direct cross stimulation?"

Probably both.

Just as clearly there is a connection between a vagal maneuver (bending over, lying down, etc.) or some activity that triggers the baroreflex and PACs. Presently there are several threads on the BB related to this.

Presently I'm experimenting with my Holter on this latter issue. I've carefully documented on myself the percentage of PACs that appear to be due to potassium imbalance. Once those are eliminated, how many of the others are due to vagal maneuvers? The Holter has an event recorder and I'm trying to correlate the myriad PACs that occur throughout the day (minimum 2.5gm K+ supplementation) with a vagal maneuver. This involves about 100-150 PACs over a 24 hour period. This about doubles if I skip the K+.

Postablation I no longer feel any bedtime PACs (unless K+ supplementation is absent). This is in distinct contrast to the situation preablation (K+ was not particularly helpful). One preablation Holter counted over a 1000 PACs over 24 hours.

Perhaps there is a correlation between the strength of the deglutition tachyarrhythmia (DT) response (AKA increased HR with swallowing) and baroreflex mediated PACs. But not everyone has a Freeze Framer for the former or a Holter for the latter.

As George has suggested, perhaps his absence of a strong DT response means that baroreflex mediated PACs are minimal and those PACs that he does measure are due to a transient shortfall in blood K+.

The below article
Tachyarrhythmias triggered by swallowing and belching
at
http://heart.bmjjournals.com/cgi/content/full/81/3/313
talks a lot about the esophageal connection.

Indeed one of the references is an article entitled
Tachycardia upon swallowing. Evidence for a left atrial automatic focus
at
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=retrieve&db=pubmed&list_uids=1245814&dopt=Abstract
that implicates a focus in the left atrium in the etiology of the heightened DT response.

Perhaps this is one of the foci (vagal reflex focus) that Prof Haissaguerre ablated, thereby eliminating an important source of PACs for me.

Always cogitating.

PC

P.S. George, better watch those DTs.

Hi Dean, PC and all.

Belching often induces an immediate ectopic for me. But then again, so does stumbling/tripping when out walking (startle response there I suppose).

Had the gastroscopy/endoscopy yesterday. No sedation, just the throat spray. The nurses were surprised I didn't want the sedative..... After 5 minutes of intermittent retching and gagging with my eyes streaming, I knew why! But a tough

guy like ME CANNOT have a sedative surely! Anyways, I can tell you that as the camera was threaded in/swallowed down through my esophagus, boy was there some ectopic activity or what! For that first half a minute or so, I was figuring AF was gonna chime in, but it thankfully (owing to my good substrate eh PC) did not. Anyway, the exam revealed nothing whatsoever of note with everything in the pink as it were.

Owing to the fact that I have complained of my esophagus cramping issues, the gastro who did the exam said I would get an appointment with the specialist who might well do motility and ph testing. If I can swing it, I'll push for getting the 24hr Holter at the same time if I possibly can.

Cheers,

Mike F.

Mike:

I'm due an endoscopy soon - I had a similar experience to you first time around, so I'll take the sedation, though I sort of expect to be triggered into AF.
I just posted on the main bulletin board on my urine-test for PH and episodes. So far I had one really noticeable spike in acidity levels (last night and this morning) and I had a 3-hour episode this morning.

Also PC, thanks for the really useful links on belching and tachys. I was astonished at the BMJ article - no previous reports? Do these people EVER go on boards like this? For me belching is at least 95% a trigger, but I wonder if it's the combo of belching and high acid levels in the lower esoph. I also get little runs if I'm reclining or scrunched up when eating- I often feel as though food is trapped in the hiatus hernia that I have and that it presses on the heart.

I'm increasingly drawn to your solution PC - I'd probably still suffer with GERD post PVI but it looks as though vagal tone isn't affected so much!!

David

Well, Strike That Last Post!
I had a mercifully brief episode this morning, while I was in AF I took my urine PH reading: 7.5 It was the same last night - normal. I also triggered not through my usual belching, but just scrunching up in bed.

Another two theories bite the dust!

The only thing that makes any sense is this concept of vagal tone, but I'm damned if I know what it is, or what causes it!!

David

This CR topic on PACs has touched on the role of potassium. Although I'll be the first to proclaim the absolute importance of adequate potassium in avoiding PACs and LAF, I don't believe that potassium is the whole story. Preablation I experienced numerous PACs upon reclining. Post successful ablation they have completely disappeared. Clearly vagal tone is a big player but understanding its role in PAC production and its correlation to the body shape of the typical LAFer (taller with less abdominal fat) seems elusive.

Session 48 of the CR abounds with speculation about the possible connection between height and insulin sensitivity => lower blood glucose => lower blood potassium => LAF. However, height may also be implicated in the etiology of PACs and LAF via the baroreflex. Philippe Coumel's seminal case of VMAF involved an individual who could trigger AF by lying down and terminate it by then standing up. This is a rather dramatic example of the contribution of the baroreflex to the initiation of LAF.

Pertinent particulars (references available):

The baroreflex (=baroreceptor reflex) is the body's rapid response system for dealing with changes in blood pressure. When the baroreceptors (carotid and aortic arch) sense an increase in hydrostatic pressure, a signal is sent to the brainstem, which then sends another signal to the heart, decreasing HR and contractility, and similarly for a decrease.

Stimulation of the aortic or carotid baroreceptors, e.g., lying down, results first and foremost in a change in vagal tone (1-2 seconds) followed by a change in sympathetic tone (6-10 seconds).

Baroreflex sensitivity (BRS) is directly proportional to height in normotensive adolescents.

Reduced BRS is seen in men with increased total body and abdominal fat and appears to be linked to their higher level of abdominal visceral fat, i.e., LAFers have greater BRS than normals.

Reduced BRS is seen in the metabolic syndrome and in insulin resistance. This translates to increased BRS in the insulin sensitive, e.g., LAFers, esp. those with a low waist to hip ratio (WHR).

Venous pooling and volume depletion are believed to be the main causes of orthostatic intolerance (=fainting after application of lower body negative pressure (LBNP)).

Although orthostatic intolerance is one manifestation of the baroreflex, physical factors such as height and plasma volume are dominant, i.e., the tall and the dehydrated are more likely to experience orthostatic intolerance.

Those with greater BRS experience more pronounced autonomic adjustments to orthostatic challenge.

Therefore, in the tall compensating for enhanced venous return (v. the short) during postural changes, e.g., standing to sitting or sitting to supine, should require enhanced baroreceptor mediated cardiac vagal tone (v. the short). It would seem that greater height would require a greater response from the baroreceptors, given equal orthostatic challenge and BRS. This seems perfectly rational, since leg length, which is directly proportional to height, should also be directly proportional to venous pooling during postural changes, e.g., supine to sitting, sitting to standing. Longer legs contain more blood.

Combine increased BRS due to male gender, physical fitness, less abdominal fat, youth, etc., with increased height and the recipe for LAF is greatly enhanced.

In summary, the tall may be at greater risk for LAF not only on the basis of the baroreflex but also on the basis of glucose/potassium homeostasis (Session 48 of the CR). Predominance of the latter is more likely to manifest as adrenergic LAF and of the former as vagally mediated AF. BRS may be just another feature, along with insulin sensitivity/resistance, parasympathetic/sympathetic balance and probably also hs-CRP level (inflammation), differentiating physiologic LAF from pathologic AF.

PC

Baroreflex -- Just imagine the plight of the poor giraffe!

I'm just short of 6 feet and thin so my baroreflex would theoretically be more susceptible. Before ablation I noticed differences, dizziness upon rising, bending over, AF conversion etc. that did suggest baro problems. I even devised a makeshift tilt test to see if I could convert to NSR. The first time I tried it I did convert but failed after that. Now after my ablation the symptoms are all but gone. Any relation to the drop in vagal tone?

Potassium-- Recently read high vagal tone activates potassium channels but the details are vague at best. But would potassium tend to increase vagal tone?

Anton

The question I posed about potassium increasing vagal tone highlights my confusion on this subject. Perhaps I need to go back to basics! Any response would be welcome.

i.e. I believe (or my first premise is) that potassium stimulates the parasympathetic nervous system. So if I believe that my AFib or PACs is due to heightened vagal tone (2nd premise), why would I try potassium?

The same sort of question is for magnesium. Under the premise that magnesium inhibits the sympathetic system and AFib/PACs is due to increased vagal tone, why would I try magnesium?

Of course if my premise on the action of K and Mg were wrong OR that AFib is vagal is wrong, then I might try both K and Mg. Perhaps I'm simplifying too much or I've misunderstood the basics. Help?

Thanks,
Anton

Well, when I'm going through a bad run of episodes (like now) I can trigger by reclining - and I'm 5'5"! I'm not overweight either. I don't supplement with Potassium, though I eat well (greens, bananas, etc) I'm wondering if I should.

David

Hi David,

Thanks much for your response.

If you can trigger AF by reclining, then you have high BRS (baroreflex sensitivity), i.e., high vagal tone. Presumably you pursue some aerobic activity on a regular basis.

One article on deglutition tachyarrhythmia speculated that based on their experimental findings, it would appear that DT is due to a vagal reflex but ACh is not the neurotransmitter.

Frankly it can't be sympathetic because the reflex occurs within a second.

Another unrelated recent article has documented the presence of glutamate receptors on nodose ganglia. This latter is involved in the baroreflex and lies outside the blood brain barrier. Could this be the explanation for glutamate sensitivity in triggering AF for some?

PC

Folks,

I note from my most recent blood works (a month or two back) that my serum K was quite high at 4.9. Can anyone illuminate me as to whether this means that my intracellular levels are ALSO HIGH? Or by implication LOWER since more K is outside the cells? Any broad relationship here that anyone knows of? Am I right in recalling that Hans' K levels were very low just prior to an episode?

TIA,

Mike F.

Hi Mike,

First of all, I hope TIA means "thanks in advance" and not "transient ischemic attack".

Your recollection of Hans situation is correct. But there is no real solid correlation between intra and extracellular K+,

although I suspect that low intracellular levels are not going to generally accompany high extracellular levels. Furthermore, intracellular K+ levels vary greatly unlike Mg++.

There is also a diurnal variation to blood K+ levels, and K+ is preferentially excreted by the kidneys, when the blood pH is upper range of normal or frankly alkalotic. So, extracellular K+ pressures are multifactorial.

PC

PC,

Thanks for your input which is noted. I'll choose to take some degree of reassurance from your phrase "although I suspect that low intracellular levels are not going to generally accompany high extracellular levels"

And my use of the acronym TIA was, I'm pleased to report, referring to 'Thanks in advance'!

Cheers,

Mike F.

The initial post on this topic of PACs speculated about the possibility that actual isolation of the PVs might be less critical in terminating AF than actual elimination of vagal reflexes and their associated vagal ganglia (focal ablation). Pachon et al. (2005) have described the particulars behind this speculation.
http://europace.oxfordjournals.org/cgi/content/full/7/1/1

The fact that these vagal reflexes happen to be located in the same anatomical areas identified by Haissaguerre et al. as triggering foci for AF is rather provocative.

Anton and I continue to experience increased basal HRs and lower HRVs many months after our respective ablations, which included elimination of vagal reflexes after completion of the PVI.

In a recent article evaluating ablation efficacy Pappone et al state, “Patients free of recurrent AF were characterized by marked and prolonged heart rate variability changes consistent with vagal withdrawal which were more pronounced in those in whom vagal reflexes were elicited and abolished. In this study also we provided, for the first time, maps localizing parasympathetic innervation around and outside PV areas: the roof junction of the LSPV as well as the postero-inferior junction of the left and the right inferior PVs are the optimal sites for eliciting and eliminating vagal reflexes.”
“*At 12-month follow-up, 85% of patients without vagal reflexes were free of symptomatic AF, compared with 99% of patients with vagal reflexes and complete vagal denervation*.“
http://www.italheartj.org/pdf_files/20050045.pdf

Pachon et al. have introduced additional data underscoring Dr. Pappone et al.’s findings. They call these vagal reflex foci “AF nests” and found them in 34/34 AF patients and only in 1/6 controls (only in this one case it was possible to induce AF despite an absence of AF history).
They go on to describe these AF nests as the “real AF substrate”.
http://europace.oxfordjournals.org/cgi/content/abstract/6/6/590?ijkey=649f413c1265c0f5fb8d961446c35e4f6a3a5ab6&keytype2=tf_ipsecsha

I’ve always struggled in understanding exactly how vagal tone actually causes PACs. Vagal tone decreases automaticity and therefore, should suppress them. However, if vagal tone is high, then anything that causes withdrawal of vagal tone could trigger PACs.

My previous post on a transient but immediate increase in HR induced by swallowing is undoubtedly an example of this. It occurs within a second or so, thereby eliminating any sympathetic component to this reflex, which would otherwise require 6-10 seconds. Clearly there is vagal withdrawal caused by the swallowing. This measure of cardiac vagal tone was increased in Hans and myself but not in either of our wives.

During vagal withdrawal muscarinic receptor sites work in reverse and cause an increase in cyclic AMP and with it the activity of the funny current. This is the ion channel that enables nodal cells to fire spontaneously. When this is switched on, a cell can become the source of ectopics (PACs). In fact over time atrial stretch causes upregulation of this particular ion channel and may be yet another reason why “AF begets AF”. Low blood K+ also potentiates the funny current. It’s called funny because through it hyperpolarization causes activation => automaticity. This ion channel distinguishes pacemaker cells from other cardiac cells.

The recent LAF survey (LAFS-11) underscored a significant correlation with height. When a tall person sits down or lies down there is a relatively greater increase in venous return from all the blood in those long legs. These vagal reflex sites may be easily triggered by such vagal maneuvers. Physical fitness would only serve to further enhance vagal tone. Swallowing, especially of anything cold, could easily trigger subsequent ectopic activity and perhaps AF.

PACs in “normal” individuals often occur during low intensity exercise. The very first physiologic reaction during exercise is vagal withdrawal followed by an increase in sympathetic activity.

For those LAFers with GERD and an esophageal connection to their episodes other reflexes and sources of vagal withdrawal may be at work.

Such a view also incorporates hypoglycemia (=> low blood K+) and glutamate into the picture, since glutamate receptors have recently been described in vagal reflex ganglia outside the CNS. Just more food for thought.

PC

At first glance the topic of vagal reflexes may not seem all that pertinent to "Pre and Post Ablation PACs".

For me in the preablation timeframe with or without K+ supplementation numerous PACs would be triggered upon reclining for the evening. Postablation this never happens with or without K+ supplementation.

Therefore, it seems logical to me to incriminate vagal reflex foci (AF nests as Pachon et al. call them) in the initiation of these ectopics. Vagal reflexes are defined as bradycardia, hypotension, AV block, .. Clearly these foci are sensory and are communicating via reflex arc with the SA node (bradycardia) and the AV node. The former is innervated by the left vagus and the latter by the right vagus. During my ablation I remember Prof Haissaguerre instructing me to cough after stimulation of one of these vagal reflex sites caused immediate bradycardia.

The existence of atrial baroreceptors has been known for some time, but they have only been described around the PVs and the SVC. The real question is whether these extrapulmonary foci cause AF or whether they develop as a result of AF.

Clearly environmental factors play a role in their expression, e.g., endurance sports. Just as clearly anthropometric (genetic) factors are also integral - see LAFS11. The fact that 34/34 with AF and one of 5 without AF had these "AF nests" suggests to me that they precede AF. This is certainly consistent with the positive family history for many LAFers.

I know the vast majority of you are not at all interested in the why of LAF, but it is only through such machinations that we can all move forward.

BTW I repeated my 24 Holter test with and without K+ supplementation. The results were even more striking this time. During the first I avoided all supplements, starting 12 hours prior to the recording. This resulted in 219 PACs and 75 PVCs over 24 hours. The second recording was taken while only supplementing with K+ (3 grams in divided doses throughout the day and prior to bedtime). PACs went to 11 (95% reduction) and PVCs went to 26 (about 2/3 reduction).

It's truly wonderful to be in NSR day in and day out.

PC

PC: Very interesting. You did all this without the universal supplement, magnesium? Do you take fish oil and/or COQ10 regularly?

Also, I have read your writings about blood K+ levels and intracellular levels and wonder if you monitored these during your experiments described above. And, if you did, did the blood level of K exceed the normal?

Which K+ supplement are you using?

You make us think, which is a lot more work than just listening to our cardiologists, but also a lot more productive and useful.

Indeed NSR all the time is a wonderful thing and maybe someday the fixation with what our hearts are doing will cease to be top of mind as they are acting just like normal people's, whatever that is. Maybe even better than normal people's hearts since we've learned to be nice to our hearts.

I meet with Dr. Natale next week and the only issue I have with him is getting off Coumadin now that my thyroid is back into the normal range. Any suggestions for other issues to discuss?

Thanks as always,
Gordon

Hi Gordon,

Thank you for your positive comments.

I stopped taking Mg++ supplements several months ago, because they didn't seem to help. After an entire year of ingesting about 800mg per day of elemental Mg++ in its hydrated form (the most absorbable and bioavailable) my intracellular levels went from 34.3 to 34.6. But those tests are expensive and I don't think they're really contributory in my experiments. The proof of the pudding is in the eating and if PACs decrease while on K+ supplementation, what do I care wrt intracellular levels.

Although I regularly take both CoQ10 and fish oils they don't seem to impact the PACs that much. Previous 24 hour Holter recordings, while on supplements including these two and just 600mg K+, reveal PACs running 100 - 225 and PVCs running 75 - 150. Adding 2.5 gm K+ lowered these to well under 100 and 50 respectively. It appears to me that part of the problem with K+ supplementation is that it must be done frequently, e.g. 400mg q3h. This is especially critical around meals, when the dose should be upped a bit. I take potassium gluconate but the chelated form is not as critical as with magnesium. Sometimes I'll sprinkle KCl on my food, although I never sprinkled salt on food at anytime in my life.

You've certainly hit the nail on the head in saying we've learned to be nicer to our hearts. Attention to health in general has been greatly enhanced by LAF.

When you see Dr. Natale next week, ask him if he thinks P cells are in any way related to vagal reflex foci. He'll probably look at you like you were from outer space.

PC

PC,

I'm glad that having had an ablation, you can experiment. Viewing myself as a biochemistry experiment is fun however, I'm a bit reluctant to mess with success. In this, I mean that I'm loathe to completely refrain from taking a supplement just to sample my ectopics & prove its efficacy.

As a thought, would a timed release formulation of K+ work well for you? I believe Prof. H. prescribed such a med for

Hans after his ablation.

Also, even though your Mg intake doesn't seem to help your ectopics, might it be beneficial in some other fashion (perhaps not even related to the heart)?

George N.

Hi George,

Can't disagree with your thinking on experimentation.

I'm just about to try another experiment. Eat a predetermined amount of nori (dried seaweed) with and then perhaps without K+ supplementation to see what happens on the PAC front over time (since ingestion).

As you know, I've always believed the two are separate and distinct in their effect on PACs. Hopefully I can put this to rest, at least for me, once and for all.

Regarding time release K+, I occasionally used K-Dur (800 mg elemental K+). But I'd get ectopics upon reclining and AF while asleep nonetheless.

Not only did I have AF, but I continue to experience muscle twitching (fasciculations) and night time leg cramps on occasion. This is no doubt related to my inability to retain Mg++.

PC

Late to this party again. I don't get online every day so it's hard to keep up. Then, PC, when I read your post of May 3 I had to go back and rethink and restudy vagal response! I was still thinking in the old sense of slow vagal and fast adrenergic when the opposite is true.

The vagal withdrawal is interesting, creating a source of ectopics. Isn't this a quick transient response prior to the slower sympathetic activity? What happens to the muscarinic sites once the sympathetic activity takes hold? Sorry for the questioning but trying to understand.

And just yesterday I took my vitals and for the first time in months, my HR was below 50! But it's back to the low to mid 50's today so my pre-ablation vagal reflexes still seem to be blunted. Looking back at my Holter to see if my RR histogram told me anything about HRV I can see a shift "left" indicating higher HR -- latest data lost!

Still studying,
Anton

Hi PC,

"During the first I avoided all supplements, starting 12 hours prior to the recording. This resulted in 219 PACs and 75 PVCs over 24 hours. The second recording was taken while only supplementing with K+ (3 grams in divided doses throughout the day and prior to bedtime). PACs went to 11 (95% reduction) and PVCs went to 26 (about 2/3 reduction)."

"Adding 2.5 gm K+ lowered these to well under 100 and 50 respectively. It appears to me that part of the problem with K+ supplementation is that it must be done frequently, e.g. 400mg q3h."

Why do you make the statement, "frequently, e.g. 400mg q3h.", when "3 grams in divided doses throughout the day and prior to bedtime" seemed to provide a better result?

I actually thought (& got approval from my very cooperative & patient GP) about an experiment where I'd refrain from K+ supplementation & then test my serum K levels hourly for 24 hours (I'm aware of the studies you've previously

referenced regarding variability of serum K with time of day). Then I'd try various levels of K+ supplementation at varying frequencies & see what happened to serum K levels. From this, an optimum level & frequency of K could be determined. However, my practical, thrifty side won out & I decided it wasn't worth either the time or money – that's a lot of blood tests!

In a minor version of the above, a year ago, I had a fasting level of 4.1 mmol/l after a 14 hour fast (& no supps) at 8:30 AM. On a week or so later, I had a 1 PM test after eating breakfast @ 7:30 AM & taking my morning 1.5 grams of K+ (& 0.4 grams Mg & 2 grams taurine). This test came back at 4.8 mmol/l & convinced me that my 1.5 gram morning & evening doses were good enough. In addition, the 4.1 contrasts with a fasting 3.4 mmol/l two years ago. This was about two months before my 1st (known) afib episode. It also contrasts with a level of 3.2 mmol/l the morning of my first afib episode.

I concluded 1) that my supplementation program had done a decent job raising my fasting serum K level and 2) that a twice a day dosing regimen was frequent enough for me.

George N.

Hi George,

I guess I wasn't very clear on that. My 3gm K+ experiment was q3h as well. What 'm trying to get at is that blood K+ is fairly dynamic. Not only is there a diurnal variation but also meals have a definite impact. I think there is a postprandial decline that is significant and may play a greater role than vagal tone, at least in those without obvious GERD or esophageal connection to their AF. Skipping meals also seems to set the table for AF.

PC

Hi PC,

"I think there is a postprandial decline that is significant and may play a greater role than vagal tone"

From Jackie's post in this thread:
http://www.afibbers.net/forum/read.php?f=4&i=8160&t=8129#reply_8160

"Remember also that creating insulin demands on the body - like eating starchy carbs that turn to sugar, or sugary desserts, sodas, etc... will also deplete potassium"

Is there a correlation between what is consumed at meals and this postprandial decline?

BTW, being in NSR has certainly allowed your subtle sense of humor come to the surface more frequently, I'm sure this in an indicator of your overall mood. Keep it up!

George N.

PC: I did see Dr. Natale today and he said he indeed thinks that there's a relationship between P cells and vagal reflex foci. He didn't expand other than to say there are 3 different kinds of P cells. I think he sensed my knowledge limitation on that about which he was speaking.

However, FWIW, he also said that after 6 months of being afib free it is very unlikely to return. He thinks I should have a Holter every 3 months for the first year just to be sure.

Interestingly enough, to me anyway, waiting to see him along with me were two physicians in their 70's, both of whom had had PVI's in the last few years; one at UCSD and the other at Bordeaux. I don't know how long ago but both said their hearts are structurally healthy. Both now have it again and both have opted to have the procedure again. but only by Dr. Natale.

Meanwhile, I'm off the Coumadin so I can play a little more with supplements without more frequent blood tests.

Gordon

Hi PC,

At one point, there was a reference to a magnesium oil, for topical application. The referenced website showed intracellular data for Mg that went from say 34 to 40 or 41. I don't have time right now to locate the reference, but it might be something that you'd be interested in. If you can't find the reference, let me know & I'll look.

Cheers,

George

Hi PC,

I didn't find the reference, but recalled that the "magnesium oil" was really magnesium chloride. I think you could find food grade mag chloride & try soaking in it.

Non-food grade is used as a de-icer on our highways.

George

George,

I found a reference which may support your recollection that "magnesium oil" is really mag chloride:

http://www.worldwidehealthcenter.net/articles-358.html

There are many forms of oral magnesium and perhaps one is more easily utilized then the other with magnesium chloride likely to come out on top. It is what one would expect from the most common form of magnesium that comes from the sea. Oral Magnesium chloride is very well tolerated and gets absorbed very quickly and is inexpensive. It can even be spread on your skin in a liquid and possibly can be purchased in bulk.

Other references to magnesium oil:

http://www.techcheminc.com/Magnesium_products.htm

Magnesium Oil

A concentrated essence of pure sea water. Magnesium Oil does wonders as a foot soak or as a bath additive. Use anywhere from a minimum of 2 ounces up to 8 ounces daily for 1-2 months to accomplish the first stage of rejuvenation; raising DHEA & intercellular magnesium.

http://www.newtreatments.org/Hypomagnesia/ga/603/Magic%20Oil%20(Magnesium%20Oil)%20for%20excellent%20absorption

Oral magnesium is potentially laxative and it would require about a year to raise levels significantly. Intravenous magnesium is the most rapid. For most people, ten IV’s given over a two-week period is adequate. Then there is follow-up, but most people don’t like needles, and it may be difficult to find a physician to give them. The simple, painless, no risk method is absorption of magnesium through the skin. The right amount of "Magic Oil" or "Dollop of Love" (DOL) can normalize intercellular magnesium and DHEA.

It looks as if Fran was right on target when she recommended transdermal Mg via Epsom salts!

Marian

Hi George,

Sorry about the tardiness, but I've been working on something that you might find interesting. I'll eventually post it.

Regarding the postprandial decline in blood K+, I'd say that the movement follows the blood glucose. Eat less sugar and/or more protein and the drop should be minimized.

Regarding MgCl2, you might recall that I experimented briefly sometime back with IM Mg++ injections. First I tried MgSO4 and then MgCl2. Both either worsened AF or had no discernible effect. The latter is more painful to administer, because a greater volume is required to attain an identical portion of Mg++.

PC

About the transdermal Mg, try the article referenced in this thread:

http://www.afibbers.net/forum/read.php?f=4&i=4679&t=4679#reply_4679

Very interesting article that bears on the problem of getting enough Mg absorbed via the oral route.

"Regarding the postprandial decline in blood K+, I'd say that the movement follows the blood glucose. Eat less sugar and/or more protein and the drop should be minimized."

PC, is this another way of saying that a carbohydrate meal produces an insulin surge, and the more insulin the body produces, the more potassium is used up by that insulin? Or have i misunderstood?

PeggyM

Aloha Gordon,

Thanks so much for asking Dr. Natale that question. A little over a year ago I sent an email to the lead author of an article from the Cleveland Clinic demonstrating the presence of P cells in PVs, inquiring about future efforts to demonstrate P cells in other areas of the left atrium.

It turned out that the lead author was a research fellow from Argentina no longer with the CC. This I found out from Dr. Natale, who responded by email quite quickly. Such research is fairly expensive and unfortunately they had no such future plans.

As I indicated to George, I'm working on a post that you might find titillating.

Mahalo
PC