

VIRTUAL LAF CONFERENCE

Proceedings of 44th Session
September 22, 2005 –

SUBJECT: Predicting LAF in Children

Aloha fibbers and former fibbers,

Hans has been good enough to allow me to pick your brains here in the Conference Room on a topic that you might find titillating. Actually your advice is sought in helping tweak the questions in what may become lafsurvey – 10. The primary purpose of this survey is to explore the possibility of predicting AF many years in advance, i.e., the likelihood of its future development in our children. Do our offspring have greater vagal tone than their peers? Is their baroreflex more vigorous? Does this put them at greater risk for developing LAF?

Autonomic tone plays a central role in LAF. Heart rate variability (HRV) is a measure of autonomic tone. Respiratory sinus arrhythmia (RSA) is another measure of autonomic tone. Unfortunately in the absence of special equipment HRV and RSA are difficult to measure. But what about PACs?

The basic premise of this project is that PACs are a direct measure of vagal tone and the goal of the survey is to evaluate vagal tone (baroreceptor mediated) using change in number of ectopics with and without vagal maneuvers as proxies. Age matched controls will be sought with which to compare your responses. At this point in time we're concentrating on the pediatric population, but may expand that target depending upon your response and the range of ages of your children/grandchildren. And don't feel neglected if you have adrenergic type of LAF. The baroreflex is triggered not only by increased pressure but also increased pulse pressure (difference between systolic and diastolic pressures). Perhaps the increase in either triggered by stress also acutely increases vagal tone thereby precipitating an episode. Accordingly, your own input is vital as well.

Part of the survey is historical and that part will be the easiest for you to complete. The other portion is much more difficult and unfortunately much more critical in determining any correlations. It is imperative, when you take these ectopic beat and HR measurements, that your child/grandchild be as quiet and inactive as possible. Always repeat the per minute HR/ectopic beats measurements twice and average your results. In some cases this may mean repeating the physical activity/vagal maneuver and recounting the PACs. This may all seem quite tedious, but the validity of the results depends on your being very meticulous. Since you all have AF, your appreciation of ectopics should be well exceed the norm. Unfortunately it is impossible to differentiate an atrial ectopic from a ventricular ectopic. However, PACs are much more numerous than PVCs, and PVCs shouldn't be triggered by a vagal maneuver, since the ventricles have no vagal innervation.

In order to keep the survey manageable, I have divided it into two parts – one covering your own situation, and one aimed at gathering data about your children/grandchildren. You may complete either or both of these section. I would also be interested in any thoughts you may have on how to evaluate the effect of potassium supplementation on PAC frequency amongst LAFers. Achieving the requisite uniformity and objectivity in this to pass muster with mainstream medicine seems difficult, at least to me.

If my hunch is correct, that increased PACs indicate increased future risk of LAF, then perhaps certain steps may be taken to forestall its development, e.g., no endurance sports, better dietary choices, hypertension prophylaxis, etc. Awareness in and of itself brings benefits.

A word on ectopic beats may be in order. A vagal maneuver should elicit PACs and not PVCs, since the ventricles have no vagal innervation. Furthermore, most PACs are followed by a beat of normal strength whereas for PVCs the following beat is definitely stronger. The vast majority of PACs reset the SA node. PVCs rarely do. These are less difficult to detect if your child is breathing somewhat deeply and slowly. A 10 second cycle (inhalation and exhalation) may be optimal for detecting these and their timing in the cycle.

You may find the following definitions helpful in completing the questionnaire:

- *Hypertension: Systolic pressure > 140 mmHg or diastolic pressure > 90 mmHg.*
- *Prehypertension: Systolic pressure > 120 mmHg or diastolic pressure > 80 mmHg.*
- *Symptoms of reactive hypoglycemia: hunger, nervousness, perspiration, shakiness, dizziness, light-headedness, sleepiness, confusion, difficulty speaking, and feeling anxious or weak, within 4 hours after a meal.*
- *Vagal maneuvers: sitting down, lying down, bending over, Valsalva maneuver (bearing down with closed glottis), drinking something cold, carotid massage*
- *Aerobic conditioning: running, basketball, cycling, swimming, tennis, etc.*
- *Endurance sports: aerobic conditioning to extreme, e.g., marathons, triathlons (frequent workouts well in excess of an hour).*

Here then are the questions I propose to ask in the survey.

Questions relating to respondent

- ***Age at time of diagnosis and age at first episode***
- ***Gender, height, weight, race, handedness (left or right)***
- ***Type of afib at diagnosis***
- ***Involvement in aerobic activities and endurance sports prior to diagnosis***
- ***Presence of other disorders such as:***
 - mitral valve prolapse
 - GERD (gastroesophageal reflux disease)
 - hyperthyroidism
 - hypothyroidism
 - prehypertensive
 - hypertensive
 - diabetes
 - reactive hypoglycemia
 - chronic lung disease
 - pulmonary embolus
 - congenital heart disease
 - pericarditis
- ***Family history of afib. List all relatives with the condition. If known, specify age at diagnosis and whether afib was lone or involved underlying heart disease***
- ***Heart rate at time of diagnosis (an estimate is OK)***

- **Blood pressure at time of diagnosis (indicate if hypertensive medication used)**
- **Presence of extra and/or skipped beats (ectopics) before treatment (if any). Specify condition during which extra/skipped beats occurred such as:**
 - during or after a vagal maneuver
 - after a large and/or late meal
 - after skipping a meal
 - during stress
 - during exercise
 - after exercise
 - other
 - unsure
- **Association of extra and/or skipped beats with initiation of afib episode (no association, always, most of the time, etc)**
- **Heart rate at time of diagnosis (an estimate is OK)**
- **Blood pressure at time of diagnosis (indicate if hypertensive medication used)**

Questions relating to respondent's children/grandchildren

- **Number of children and grandchildren**
- **Age, gender, height, and weight of each child**
- **Presence of heart disease or other disorders.**
- **Extent of participation in sports**

Specific measurements

- **Average heart (pulse) rate and number of extra and/or skipped beats upon arising and before bed**
- **Average heart (pulse) rate and number of extra and/or skipped beats while reclining quietly**
- **Heart rate and number of extra and/or skipped beats while lying on the left and right side**
- **Heart rate and number of extra and/or skipped beats while reclining after exercise of sufficient intensity to elicit a pulse rate over 100 bpm**

Please let me have your comments and input on the proposed survey questions listed above. Any suggestions you may have for additional questions would also be most welcome.

PC

PC,

"However, PACs are much more numerous than PVCs..."

In my case this is not true. The report from the 24 hour Holter after my first AF episode showed an average of 2 PAC's

and 24 PVC's/hour. My subsequent measurements with my Polar S810 has confirmed this general ratio. Also here is a quote from Hans,

"Author: Hans Larsen (---.gv.shawcable.net)

Date: 06-01-05 11:24

George,

My last Holter prior to my first ablation recorded 2668 PVC's and 259 PACs over a 24 hour period. It was also noted that my PVCs increased very substantially in the 2 hours preceding the start of an afib episode. - I have no explanation for that :-)

Hans"

"If my hunch is correct, that increased PACs indicate increased future risk of LAF"

I had several ECG's in the years prior to having AF. These showed no ectopics on the samples I've seen.

In this article "Taurine Role in Cardiology and Cardiac Arrhythmias" by George Eby

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

George talks about having 15,000 PAC's/day, but no mention of AF. The gist of the article is how he keeps the PAC's at bay with 12 grams of Taurine (3 grams, 4x/day).

This reinforced my own hunch that the lack of Taurine was at least partially responsible for my two most recent AF breakthroughs, 5 1/2 months ago. I had run out and not bothered to get more, then I had two, 1/2 hour AF episodes several weeks apart.

I have not been able to correlate my levels of ectopics with much of anything. I do know that they decrease after taking my supplement regimen. However there is much variability - day to day and hour to hour. For example, if I take a two hour HR sample & divide it into 24 - five minute bins, there will be much variability between bins, with many bins at 0.

I can say that overall my supplement program keeps the ectopic level relatively low and AF away, but fine tuning the data more than this gross level is very difficult.

It is good to see your post. The one upside of this affliction is the mental stimulation of your posts.

Best regards,

George

Hi George,

Thanks much for your expeditious post. Perhaps many have more PVCs than PACs. I'll wait to see what others post on this.

The critical feature of this hypothesis is that we're not just looking at the frequency of PACs. We're looking at their frequency in the three minutes immediately after a vagal maneuver while lying on ones back. Such a pinpoint time frame should make background PVCs unlikely. Even at 3,000 PVCs per day this translates to only 2 per minute. Hopefully we can get a baseline on the number of background PVCs prior to initiation of the vagal maneuver. It may not be the number of PACs that is critical to the triggering of AF. It may be a hyper responsive baroreflex.

But that's the purpose of the post - to address the holes in the approach.

PC

PC

It might be good if you add a couple of things to the list of pre existing disorders. Any kind of digestive dysfunction IBS, celiac, etc and candida (sugar cravings) and poor immune system.

I truly believe that this is crucial from an early age what nutrients your body is getting even if you are eating good food which most children now don't have and some of us may not have had growing up whether it is what we were fed or what we disliked or wouldn't eat to get the goodness we needed. Any kind of bowel or undiagnosed stomach complaint from an early age will cause lack of nutrients needed to grow healthy.

My son has been diagnosed under the autistic spectrum. I bought a book called children with starving brains. It's all about how these children come on in leaps and bounds on a gluten and milk free diet and supplementing extra nutrients (2 of them being B6 and mag). Sam has Aspergers - he is very intelligent but lacks social skills.

I am about to send of a urine sample to test for opioids in his urine for gluten and milk to confirm that he is suffering from these so called healthy foods

I know this isn't related to AF but it is very important in our child's health. I have already cut out gluten (very difficult) and it has been noticed by myself and in school what a difference it has made - more focused and more eye contact.

What I'm trying to say is from an early age the foods that we are eating could stop the nutrients we need from getting through.

So many afibbers including myself have improved changing diet and supplementing. There must be something in it.

Thanks for listening.

Tonigirl xx

Aloha Tonigirl,

Hope you're controlling your sweet tooth better than I am.

Your concerns about the dietary shortfall and digestive dysfunctions of our children and its impact on the subsequent development of AF are noteworthy. They certainly deserve to be evaluated in a survey.

However, the one I'm floating is fairly narrow in its target. Although there are some general historical information requested of LAFers, the data targeted from our children/grandchildren is specifically limited to PACs while lying down immediately after some physical activity that elevates HR over 100 bpm. I would feel lost trying to evaluate any survey containing diet data.

PC

No problems PC. Thought it was worth a mention though.

As for my sugar cravings, after introducing beans and pulses in my diet they have really helped aduki, canellini, kidney etc. I make a three-bean salad with salmon or tuna and you will be full for hours.

Still eating some chocolate though, don't think I will ever get away from it

Tonigirl

"It is imperative, when you take these ectopic beat and HR measurements, that your child/grandchild be as quiet and inactive as possible"

Ummmm, PC, I don't think i understand how to do these measurements, supposing i can get my grand daughters to cooperate. Are we supposed to be taking these measurements with some kind of equipment? What kind?

PeggyM

Hi Peggy,

Thank you for asking that particular question.

No, the only equipment you should need is your expertise as an LAFer. I'm hoping that the LAF experience is enough to create an excellent palpater of skipped or dropped beats.

But that is the purpose of this discussion. Do you and others feel that attempting to discern such things is asking too much?

PC

So we are supposed to be taking the kid's pulse? And if it isn't perfectly regular, we are not supposed to freak out and snatch the kid to the doctor, but to analyze the irregularities as afib or ectopics, is that it? Pardon the dumb questions, something about this proposal makes me feel real ignorant.

PeggyM

Yes, that's correct. What is it about the proposal that makes you feel ignorant? If your child does exhibit AF (HIGHLY unlikely), then better to know this earlier than later. If your child exhibits increased ectopics relative to an age matched control group, then this might possibly indicate a predisposition to LAF. It's also better to know this earlier than later.

The weak link in all this is the possible suspect nature to the data due to failure to appreciate ectopics.

PC

Aloha PC,

Peggy may be correct, not everyone may be in the habit of taking their own pulse. Here are some instructions. Your MD training may allow you to give better instruction than what I've come up with here.

What you do while feeling the pulse is also feeling for quick or slow beats. While there is some variance in the length of time between pulses, it is usually not detectable to someone taking a pulse, unless it is different enough to be an ectopic beat (PVC or PAC).

SECTION 1: HOW TO FEEL FOR PULSE

You can locate a pulse on yourself by doing the following:

1. Place the fingertips of the first two fingers of one hand over any relatively large superficial artery, such as the ones presented in this exercise.
2. Compress the artery firmly over the underlying hard tissues and then immediately ease up on the pressure.

3. Maintain slight pressure over the artery until you can feel blood pulsing through the artery.
4. Note the regularity and strength of the pulse. What happens when you compress harder? How about when you barely compress the artery?

NOTE: It takes much practice to find the right "touch" when palpating pulses, so don't get discouraged. You will be able to find all of your pulses with some practice.

From:

http://www.gen.umn.edu/courses/1135/lab/blood_pressure_pulse/bp_pulse_lab.html

Also see:

<http://user.gru.net/clawrence/vccl/chpt4/PULSE.htm>

George

Thanks for that George.

One of the questions I'd included in the survey is self evaluation of each LAFers ability to detect ectopics. If one is not capable of palpating ones own pulse without the basic instruction outlined above, then perhaps I will have to reevaluate the reliability of such an undertaking as represented by this proposed survey.

Thoughts from others on this would be greatly appreciated.

Perhaps the survey should target a smaller more experienced group of LAFers.

PC

PC,

I think people can handle this -- they can certainly practice on themselves first. I think clarity is always good whenever you are expecting someone to do something for you -- assume nothing.

I rarely take a radial pulse on my wrist - I use a pulse point just in front of my ear. This is a poor choice for taking someone else's pulse, however.

George

George,

Your comments and suggestions are always much appreciated. I'm sure that many will others echo these sentiments in general whenever you respond to their posts. You're chasing Jackie for real superstar status. You both may have missed your calling as physicians.

PC

P.S. Sure wouldn't want to make an as_ out of u and me (assume)

Aloha PC,

Thanks for your kind words. I have great respect for the medical profession. My difficulties in figuring out my own

condition have only served to increase this respect and the difficulties involved in figuring out what is causal in many illnesses and treatments.

One of my weaknesses is assuming that I am best at figuring something out, even if I have little or no expertise in that area. I am slowly disciplining myself to seek out experts with much more knowledge than I do for consultation and assistance. This board, with its wealth of knowledge, has taught me much on many levels. I am very appreciative of you and the others who regularly contribute their wisdom here.

George

PC,

You have probably already seen this study & perhaps posted it on the board somewhere, anyway, I thought it interesting.

George

JAMA 2004 Jun 16;291(23):2851-5 (ISSN: 1538-3598)

Fox CS; Parise H; D'Agostino RB; Lloyd-Jones DM; Vasan RS; Wang TJ; Levy D; Wolf PA; Benjamin EJ
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CONTEXT: Atrial fibrillation (AF) is the most common cardiac dysrhythmia in the United States. Whereas rare cases of familial AF have been reported, it is unknown if AF among unselected individuals is a heritable condition. OBJECTIVE: To determine whether parental AF increases the risk for the development of offspring AF. DESIGN, SETTING, AND PARTICIPANTS: Prospective cohort study (1983-2002) within the Framingham Heart Study, a population-based epidemiologic study. Participants were 2243 offspring (1165 women, 1078 men) at least 30 years of age and free of AF whose parents had both been evaluated in the original cohort. MAIN OUTCOME MEASURES: Development of new-onset AF in the offspring was prospectively examined in association with previously documented parental AF. RESULTS: Among 2243 offspring participants, 681 (30%) had at least 1 parent with documented AF; 70 offspring participants (23 women; mean age, 62 [range, 40-81] years) developed AF in follow-up. Compared with no parental AF, AF in at least 1 parent increased the risk of offspring AF (multivariable-adjusted odds ratio [OR], 1.85; 95% confidence interval [CI], 1.12-3.06; P = .02). These results were stronger when age was limited to younger than 75 years in both parents and offspring (multivariable-adjusted OR, 3.23; 95% CI, 1.87-5.58; P < .001) and when the sample was further limited to those without antecedent myocardial infarction, heart failure, or valve disease (multivariable-adjusted OR, 3.17; 95% CI, 1.71-5.86; P < .001). CONCLUSIONS: Parental AF increases the future risk for offspring AF, an observation supporting a genetic susceptibility to developing this dysrhythmia. Further research into the genetic factors predisposing to AF is warranted.

Aloha PC,

As a follow on to the last study, here are two others that examine risk of AF with obesity. Perhaps some of the "genetic" predisposition might be due to size, both height & BMI. Since both are correlated with atrial size (as is activity level).

George

JAMA 2004 Nov 24;292(20):2471-7 (ISSN: 1538-3598)

Wang TJ; Parise H; Levy D; D'Agostino RB; Wolf PA; Vasan RS; Benjamin EJ
Framingham Heart Study, Framingham, Mass 01702-5827, USA.

CONTEXT: Obesity is associated with atrial enlargement and ventricular diastolic dysfunction, both known predictors of atrial fibrillation (AF). However, it is unclear whether obesity is a risk factor for AF. OBJECTIVE: To examine the association between body mass index (BMI) and the risk of developing AF. DESIGN, SETTING, AND PARTICIPANTS: Prospective, community-based observational cohort in Framingham, Mass. We studied 5282 participants (mean age, 57 [SD, 13] years; 2898 women [55%]) without baseline AF (electrocardiographic AF or arterial flutter). Body mass index (calculated as weight in kilograms divided by square of height in meters) was evaluated as both a continuous and a categorical variable (normal defined as <25.0; overweight, 25.0 to <30.0; and obese, > or =30.0). In addition to adjusting for clinical confounders by multivariable techniques, we also examined models including echocardiographic left atrial diameter to examine whether the influence of obesity was mediated by changes in left atrial dimensions. MAIN OUTCOME MEASURE: Association between BMI or BMI category and risk of developing new-onset AF. RESULTS: During a mean follow-up of 13.7 years, 526 participants (234 women) developed AF. Age-adjusted incidence rates for AF increased across the 3 BMI categories in men (9.7, 10.7, and 14.3 per 1000 person-years) and women (5.1, 8.6, and 9.9 per 1000 person-years). In multivariable models adjusted for cardiovascular risk factors and interim myocardial infarction or heart failure, a 4% increase in AF risk per 1-unit increase in BMI was observed in men (95% confidence interval [CI], 1%-7%; P = .02) and in women (95% CI, 1%-7%; P = .009). Adjusted hazard ratios for AF associated with obesity were 1.52 (95% CI, 1.09-2.13; P = .02) and 1.46 (95% CI, 1.03-2.07; P = .03) for men and women, respectively, compared with individuals with normal BMI. After adjustment for echocardiographic left atrial diameter in addition to clinical risk factors, BMI was no longer associated with AF risk (adjusted hazard ratios per 1-unit increase in BMI, 1.00 [95% CI, 0.97-1.04], P = .84 in men; 0.99 [95% CI, 0.96-1.02], P = .56 in women). CONCLUSIONS: Obesity is an important, potentially modifiable risk factor for AF. The excess risk of AF associated with obesity appears to be mediated by left atrial dilatation. These prospective data raise the possibility that interventions to promote normal weight may reduce the population burden of AF. Comment In: Comment In: RefSource:JAMA. 2004 Nov 24; 292(20):2519-20/PMID:15562134

Author(s): Frost L ; Hune LJ ; Vestergaard P
Affiliation: Department of Cardiology, Aarhus University Hospital, DK 8000 Aarhus C, Denmark. lars.frost@as.aaa.dk
Title: **Overweight and obesity as risk factors for atrial fibrillation or flutter: the Danish Diet, Cancer, and Health Study.**

Source: **Am J Med (The American journal of medicine.) 2005 May; 118(5): 489-95**

Additional Info: United States

Standard No: ISSN: 0002-9343; NLM Unique Journal Identifier: 0267200

Language: English

Abstract: PURPOSE: We examined the association between the body mass index analyzed as a continuous variable and by categorization according to World Health Organization criteria (normal weight, overweight and obesity) and the risk of a hospital (inpatient as well as outpatient) diagnosis of atrial fibrillation or flutter. METHODS: Population-based prospective cohort study conducted from December 1993 to December 2001 among 47589 participants (22482 men and 25107 women) without preexisting cardiovascular or endocrine disease and with a mean age at baseline of 56 years (range 50-64 years) in the Danish Diet, Cancer, and Health Study. Subjects were followed up in the Danish National Registry of Patients and in the Danish Civil Registration System. RESULTS: During follow-up (mean, 5.7 years) atrial fibrillation or flutter developed in 553 subjects (372 men and 181 women). The adjusted hazard ratio for atrial fibrillation or flutter per unit of increase in the body mass index was 1.08 (95% confidence interval [CI]: 1.05 to 1.11) in men and 1.06 (95% CI: 1.03 to 1.09) in women. When using normal weight as a reference, the adjusted hazard ratio for atrial fibrillation or flutter by overweight was 1.75 (95% CI: 1.35 to 2.27) in men and 1.39 (95% CI: 0.99 to 1.94) in women. The adjusted hazard ratio by obesity was 2.35 (95% CI: 1.70 to 3.25) in men and 1.99 (95% CI: 1.31 to 3.02) in women. CONCLUSION: Overweight and obesity are associated with an increased risk of a diagnosis of atrial fibrillation or flutter.

Hi George,

Thanks much for the abstracts. I'd seen some but not all that you posted. I'm certainly not surprised by any of the conclusions.

However, these articles are all addressing AF and not specifically LAF.

We all know that typical AF arises in structurally abnormal hearts - infarcts, heart failure, etc. And of course, much of this is genetically determined. Diabetes is very much in play as a genetic disease, although we can be our own worst enemies in making it worse. I believe that the reason an elevated BMI is associated with AF (? LAF) is because obesity leads to diabetes, which leads to CV disease. I personally think that hypertension should be included to some extent in causing a structurally abnormal heart. It all depends on how closely you look.

In view of the recent discussions regarding LAF and its association with height v. weight v. BMI your posted abstracts bring up an interesting point.

If one excludes LAFers with associated hypertension from LAFers as a group, can one discern a pattern of decreasing BMI amongst LAFers (v. increasing BMI amongst AFers)? Could the opposite correlation amongst LAFers be hidden in the data, overwhelmed by the number of non lone AFers?

This is not to detract in any way from the notion that LAF as well as AF are genetically determined to some degree. Why has LAF been relatively frequently described in BB players (Bill Bradley, Akeem Olajuwon) but not in any football players of which I'm aware. Clearly football players have higher BMIs than BB players.

$BMI = (\text{weight in kg})/(\text{height in meters})^2$

I believe that LAF is being given short shrift in all this, partly because it's hard to accumulate LAFers in sufficient numbers for a statistically viable study. But Hans has a very valuable resource in this BB and CR that might enable just that. The trick is to study them in a manner that does not require technology or unattainable sophistication. Otherwise they would all have to visit some research center.

So far, the response to this proposed survey is not very encouraging. I'm not sure whether LAFers are just uninterested in such things or that they are overwhelmed by what may appear to be a sophisticated study. It certainly requires some physical effort but the intellectual demand is minimal.

PC

PC, when i think about doing these examinations i am overwhelmed with negativity. The biggest thing is, what if i do detect some irregularity in pulse? I don't think i am competent to tell ectopics from afib in anybody but myself. My face would immediately show that i found something other than perfect regularity, and i would be panicking out my grand daughters over nothing. And also their mother and myself, i might add.

Another thing, what exactly is meant by a vagal manoeuvre? What should i be trying to get these girls to do, and then i should take their pulses immediately, or after some time, or what? I check my own pulse, but only so as to reassure myself that it is regular. Are you talking about a more formal pulse taking like nurses do, with timing it and counting the beats in thirty seconds, and multiplying by 2 to get the beats per minute? If it isn't perfectly regular, how do i record that? That will require a watch with a second hand, or a digital one. I could get one, i guess, but i don't really feel very enthusiastic about this, as you can see. I don't think i know what it is that you need recorded anyway.

PC, it could easily be the old-fuddy duddy factor. Likely other respondents are a lot more competent in these matters than myself. Have you gotten much response from others on this, other than George and Toni and myself, i mean, maybe by private communication?

PeggyM [the old fuddy duddy]

Hi there Peggy,

As to the clock - double click the time in the lower right hand part of the screen on your computer & it will bring one up, with a second hand. Pulse rate is in beats per minute. The other thing you are looking for is ectopics (PVC's & PVA's) per minute (or hour hopefully).

From your post, I assume you just feel your pulse to assure yourself that it is regular.

I don't see that PC has stated a sampling time (i.e. the number of ectopics in 5 minutes), so you would have to pick a consistent time period. I will say that there is a lot of variability and randomness in ectopic rates. For example, this morning I sampled my pulse with my heart rate monitor for 40 minutes while meditating. If I break the 40 minutes into 5 minute bins, here are the results:

PVC PAC

1 0
0 0
1 0
0 0
1 0
2 0
0 0
0 0

PVC PAC

Totals 5 0

I doubt if many people would have the patience to take a 40 minute sample by feeling their pulse on their wrist. Even fewer could get kids to do so. The question is - what is a reasonable sample time? I remember reading that the EP's consider less than 10 PVC's/hour a non-event, this would be 1 in six minutes. On the other hand, in a study referenced in Hans's 1st book, ectopics went from .9/minute to 4.2/minute before AF. This would be 54/hour to 252/hour. 4.2/minute would be 21 in a five-minute sample period.

So what is a significant number of ectopics per minute (or hour), and given normal variability and randomness, how long do you need to sample to ascertain whether what you are detecting is significant? Perhaps PC will weigh in here.

My suggestion, Peggy would be to practice on yourself, especially in recognizing ectopics. Then when you feel comfortable, you might try it on the kids -- but make it a game and fun - not a serious thing. That would help, I think.

Here is PC's definition of vagal maneuvers: sitting down, lying down, bending over, Valsalva maneuver (bearing down with closed glottis), drinking something cold, carotid massage.

Cheers,
George

Aloha Peggy,

George is exactly right. Try to make it fun. And practice on yourself to sharpen your ability to detect ectopics.

Part of the challenge of this approach is that it's never really been done before - not in LAFers and not in normals and certainly not in the offspring of LAFers.

Vagal maneuvers are as George described. However, I'm proposing a specific vagal maneuver. The individual to be tested gets his/her HR well above 100 bpm via any means desired. After this has been achieved he/she lies flat (supine or horizontal on back). Immediately thereafter the pulse is checked for ectopics and the results tabulated for each of the ensuing three minutes. That's it. This is a powerful vagal maneuver and should be a good proxy for vagal tone, at least that stimulated via the carotid sinus (baroreceptor in the neck). Then I try to compare these results in an age matched group without LAFer parents/grandparents.

What I'm trying to get at is not what triggers an episode but rather "Is there a background of increased vagal tone that over time (stress, poor electrolyte balance, ...) leads to LAF.

The rest of the survey is concerned with historical type questions. There may be some interesting correlations there as well.

PC

P.S. You, George and Toni have been the only input to date, unfortunately.

Aloha PC,

"...but not in any football players of which I'm aware."

Well, I'm an ex-college defensive lineman (and a light one at that). My BMI in college was the same as it is now -- 27. There was a time AM and BC (after marriage and before children) when I worked out harder and longer than now, and my BMI was 25 with 11% body fat. Maybe someday I'll get back to that state, but I come from a long line of high BMI people - my paternal grandfather had a BMI of 37.

I'm sure my issue is chronic fitness and electrolyte issues. There have been times that I was much fitter and more vagal than today. The difference being the oxidative damage that has occurred over time and low electrolytes. I'm not sure why the latter happened. Plus the extra pacing cells that we AF'ers have.

George

For English units:

$BMI = (\text{Weight in Pounds} / (\text{Height in inches})^2) \times 703$

George,

That's very interesting about your past and present BMI. I presume you have always been normotensive.

It would be interesting to know whether LAFers with a slightly higher BMI (relative to other LAFers) have had slightly more success in controlling their LAF with close attention to electrolyte balance. Perhaps those on the lower end of the BMI scale have had the least success with such measures.

Many LAFers have complained that weight loss aggravates LAF frequency and duration. This might be related to hypoglycemia, but who knows.

Could the issue be more vagal tone related than BMI, although higher vagal tone is usually associated with lower BMI? Perhaps you're some kind of weird hybrid, one of those outlier types.

It is interesting to speculate about such matters, but until the pertinent data is evaluated through a survey, it will remain nothing more than pure speculation.

PC

Hi PC,

So it is a beautiful Saturday morning & I decided to try your test on myself. I went for an hour bike ride with 150 BPM max HR, average about 130. Then I lay down immediately after stopping. I checked for ectopics both manually and with my Polar. The result - zero PVC's and PAC's in the 3 minute period. By the way, I had not taken any supplements in the morning prior to the ride.

I suppose vagal AF'ers who're prone to going out of rhythm might want to avoid the test themselves, so as to not end up out of rhythm.

Cheers,
George

PC

I'm fairly new to this website but thanks to it was recently able to self-diagnose as having VMAF, thanks to a post by Victor Thuronyi--"Vagally Mediated Atrial Fibrillation - A Patient's View".

So being a newbie I still have a lot of research and sleuthing to do to get anywhere near the superior medical/scientific knowledge that some participants display on the BB and Conference Room but would like to contribute if I it is relevant and useful, so I'm apologising in advance if I'm out of line and should not be here.

I notice you discredit Toni's suggestion about bowel dysfunction and diet shortfall as being outside the "narrow" survey target, but I would have thought that these issues were relevant when taking the specific measurements required in your survey, e.g. what food/drink if any the child had ingested prior to the testing (couldn't a big meal/Chinese takeaway/sport-sugar drink etc possibly influence and give misleading results?).

Peggy had some issues with difficulty of testing young children. Why not canvas some of your children's hospitals and get details of children who have presented with AF, LAF, PVC'S, PAC'S etc and survey these children as they already have some of the issues of interest, find out about their parents, follow thru on their status as adults.

Why not go back one step and look at babies. I'm sure your leading maternity hospitals would have records on prenatal, postnatal babies with heart rhythm disturbances and what the outcomes were, their status as children, teenagers, adults, find out about their parents etc.

Or go forward one step and concentrate on young teenagers where you will get a lot more cooperation and communication. Put ads in the papers seeking people with unexplained, transient heart problems -these people may never seek medical advice, might never look at a website like this one until provoked. I think one needs to look outside the circle so you can get more participants to make the survey meaningful.

Finally I could understand reluctance to participate in the conference room discussion especially from us newbies and laypersons as sometimes the discussion can be quite technical and intense, with a constant need to refer to the glossary and some terms don't even appear there eg. baroreflex, respiratory sinus arrhythmia, hypertension prophylaxis.

Thanks,
John
