Your Premier Information Resource for Lone Atrial Fibrillation
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VIRTUAL LAF CONFERENCE

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SUBJECT: Ectopy and AF

Just thought I'd pop something in here since I saw the opening and am keen to show willing! And its a favourite of mine! Apols in advance for this topic not having been as thoroughly and studiously researched and referenced as per PC and Jackie.... I just kinda threw it together off the cuff in 15 minutes or so.

I get approximately 100 single ectopics and one-to-several few-second-long runs of ectopy (both regular and irregular) per day EVERY day. However, I've had no AF for 2 years. (I have previously had 5 few-hr-long self-converting nocturnal episodes between Oct99 and Nov 03 - no meds). I also have a long history of GERD (a couple of erosions on last endoscopy a couple of years ago) for which I currently take omeprazole 20mg late evenings which helps enormously.

I am intrigued with the relationship - if any - between frequent ectopy (both PACs and PVCs, both single and in short runs, and both regular and irregular) and AF - chiefly given the following:

1. Many, many individuals over on the MedHelp Heart Forum get frequent ectopy on a daily basis - some with up to 20,000 single ectopics per day (both PACs and PVCs), and many with short runs of all varieties of associated arrhythmias including SVTs. Remarkably few of these individuals, however, get AF.

2. Most folks on Hans' Forum comment that once they experience more than 4 ectopics in a minute then they 'know' that they are in for an episode of AF.

3. I have many, many times during the last 2 years (indeed the last 20 yrs) experienced up to 10 ectopics in a minute or even a couple of few-second-long runs of ectopy in a minute and never had AF.

4. I do not seem to be alone in this regard here on Hans' Forum given that another forumee Bill has experienced a lot of ectopy over the last 30 years but has 'only' had 6 episodes of AF during that time. It is likely that most folks out there who only get similarly infrequent episodes of F do not ever make it here to Hans' Forum.

5. A significant body of research exists showing that most if not all members of the wider population experience ectopy and arrythymia. They are (blissfully!) not aware of it, and neither does it lead to AF. Please, for example note the following extract - it is old but nonetheless valid because of that.

"TI - *The rhythm of the normal human heart*.
AU - Clarke JM; Hamer J; Shelton JR; Taylor S; Venning GR
SO - **Lancet 1976 Sep 4;1(7984):508-12**.

The 24-hour cardiac rhythm was studied in 86 subjects (41 male, 45 female) aged 16-65 years, after exclusion of 15 additional volunteers with suspected abnormalities. The electrocardiogram was recorded continuously for two 24-hour periods. In this apparently normal population, 10 subjects (12%) had disturbances of rhythm which are widely believed to be of serious prognostic significance; they included frequent ventricular ectopic beats, R-on-T and multifocal ventricular ectopic beats, bigeminy, and ventricular tachycardia. Supraventricular tachycardia, infrequent ventricular

ectopic beats, junctional rhythm, and second-degree heart block were also observed, and if these are included most of the subjects showed some disturbance of rhythm. Bradyarrhythmias and tachyarrhythmias were equally common in waking hours and during sleep. These disturbances were not confined to the older age-groups. Heart-rate but not the number of arrhythmias was significantly higher in smokers."

I guess what I really find myself accordingly wondering about is..... Just how important is ectopy to AF?? OK, I appreciate that ectopic activity is implicitly involved in AF. But given that one ectopic can cause AF in some individuals whilst 20,000 ectopics do not in other individuals, then ectopy is surely NOT therefore a significant factor in the nature of the condition of AF itself?? I am accordingly putting forward the idea that other factors such as fibrosis, oxidative inflammation, other inflammatory stressors such as GERD, and ANS imbalances are far more at the heart of AF than is ectopy as of and in itself. If this is the case, then why - and flying somewhat in the face of my aforementioned points - do current ablation techniques focus upon preventing ectopic focii from precipitating AF?? Given my points as aforementioned, would it not be far better to concentrate on dealing with other ROOT causes of AF such as fibrosis/inflammation?? Would other forumees agree or disagree and why??

Mike F.

Oops, from the last paragraph of the above post:

"If this is the case, then why - and flying somewhat in the face of my aforementioned points - do current ablation techniques focus upon preventing ectopic focii from precipitating AF??"

That question was obviously more than a little ill-considered..... given that that ablation techniques CAN ONLY focus upon preventing ectopic focii from sending AF-initiating signals to the atria. (I did state that I had rushed the posting a little - apols again, but I'm extremely busy work-wise at the moment.) What I was/am driving at is - given the context of my post above:

1. Why do corrective therapies for AF focus upon ECTOPY so much? Could we not instead look into therapies other than surgery to reduce, for example, fibrosis and inflammation?
2. Why does the remaining (after ablation) ectopy - often frequent/significant - experienced by many successful ablatees NOT (thankfully) result in AF episodes?

Mike F.

Mike,

Thank you for starting this very interesting topic. You are absolutely correct. Ectopy (PACs and PVCs) are indeed very common - probably experienced by about 60% of the population. Atrial fibrillation, on the other hand is relatively rare - probably experienced by somewhere around 1% of the population. So clearly, as you state, ectopy is not the cause of afib, but it is the precipitating factor for an episode in afibbers who are otherwise pre-conditioned to afib.

I quite agree that it would make more sense to find the root cause of afib and eliminate that rather than focus on preventing ectopy from starting an episode (the purpose of ablations). Unfortunately, finding root causes of anything is not a hallmark of the medical industry - covering up symptoms with, preferably, life-time medication use is what the game is all about.

In answer to your question 2 in your second posting. The scar tissue created during the ablation, particularly around the pulmonary veins, prevent a run of ectopics from spreading to the atrium and create the chaotic contractions characteristic of afib.

Hans

Hans,

Thanks for your encouraging words. From your above post:

"In answer to your question 2 in your second posting. The scar tissue created during the ablation, particularly around the pulmonary veins, prevent a run of ectopics from spreading to the atrium and create the chaotic contractions characteristic of afib."

Noted, but given that successful ablatees actually FEEL ectopics AFTER their ablations. I'm accordingly assuming that the errant signal/s emanating from the focus/focii in the lower PVs ARE making it down past the scar tissue to the atria in order for the ectopic to be felt?? My point therefore being that the ectopic activity is STILL occurring even after an ablation. As such it may be that those experiencing AF AFTER an ablation are those (obviously in addition to those whose focii were elsewhere in the atria i.e. NOT only in the PVs) for whom one or two ectopics were/are enough to precipitate AF. Other individuals for whom ablations were/are successful (in eliminating AF episodes) but who still experience ectopy comprised those individuals who would NOT (before their ablation/s) have had an episode of AF further to just the odd ectopic.

Mike F.

Hi Mike,

I agree wholeheartedly with Hans. An excellent topic for the CR. In fact I've done quite a bit of thinking about the PAC AF connection, but from a little different angle – why do LAFers have so PACs (mine) v. why others with just as many or more don't get AF (yours). An upcoming survey may shed some light on this. Your prescient question and suggestions are partially addressed in a 2004 article entitled "Impact of Premature Atrial Contractions in Atrial Fibrillation" at
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=15078396&dopt=Abstract

In order to get a better handle on your question I think it would be quite helpful to look into the matter of incidence PACs in the "normal" population. This is highly dependent on the length of time during which the ectopics are monitored. George has been gracious enough to provide a couple of good references on the topic. In one 24 hour Holter study of 50 healthy male medical students 56% exhibited PACs. In another 24 hour Holter study of 50 young women without apparent heart disease 64% exhibited PACs. Numerous long term studies on such individuals reveal no increase in sudden death or decrease in longevity. In one study PACs developed in 4%-16% of subjects during maximal stress testing. However, there is no prognostic data for such individuals of which I am aware. Interestingly 2% of the healthy male medical students also exhibited supraventricular tachycardia during the 24 Holter monitoring. So, clearly the vast majority, if not everyone, experiences PACs.

This then leads to the question of their frequency. We know PAC/PVC frequency increases with age. According to the medical screening protocol for pilots in the USAF, the following is their approach to PACs/PVCs: PACs/PVCs comprising < 1 % of total beats is classified as rare or occasional; PACs/PVCs comprising 1% to 10% of total beats is classified as frequent; PACs/PVCs comprising > 10 % of total beats is classified as very frequent. Those with coupling of ectopics are also carefully investigated.

PACs and PVCs are both triggered by automaticity, reentry, or triggered activity (early or late afterdepolarizations). In the atria electrolyte abnormality, ischemia and catecholamines are the primary agents responsible for automaticity. Triggered activity in the atria (v. the ventricles) is less well understood, at least by me. So, that leaves reentry.

We've all experienced increased PACs during vagal maneuvers. The following 2005 article entitled "Heart Rate Turbulence After Atrial Premature Beats Before Spontaneous Onset Of Atrial Fibrillation" at
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=15653028&dopt=Abstract
may shed some light on this.
It suggests that PACs increase vagal tone and, as has been stated in the BB post at
http://www.afibbers.com/forum/read.php?f=6&i=18571&t=18571, increased vagal tone enhances reentry. And, of course, the diurnal nadir (low) of blood potassium at midnight only serves to enhance this likelihood of reentry.

So, as Mike has suggested, it appears that the substrate is more critical than the PACs. And these substrate changes appear to be mediated by vagal tone. This is borne out by the fact that LAF is increased at least 10 times in one study "Lone Atrial Fibrillation In Vigorously Exercising Middle Aged Men: Case-Control Study" at

http://bmj.bmjjournals.com/cgi/content/full/316/7147/1784. This finding is reinforced by another entitled “Long-Lasting Sport Practice And Lone Atrial Fibrillation” at http://eurheartj.oxfordjournals.org/cgi/content/abstract/23/6/477

This conclusion is further underscored by the findings in “Role Of Atrial Electrophysiology And Autonomic Nervous System In Patients With Supraventricular Tachycardia And Paroxysmal Atrial Fibrillation” at http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=9741520&query_hl=20

This interesting article looked at a group of 50 individuals with paroxysmal supraventricular tachycardia (PSVT) with (23) and without (27) paroxysmal atrial fibrillation (PAF). They found significant differences in atrial size and AERP dispersion (both increased) in patients with PAF than in those without PAF. Baroreflex sensitivity (BRS) was also higher in patients with PAF than in those without PAF. This again plays to the theme of enhanced vagal tone causing the defective substrate.

However, it also appears that additional ectopic foci can be created by episodes of AF. The Bordeaux group has determined that the number of non PV foci is directly related to the length of episodes. Forty-eight hour episodes seem to be a threshold of sorts for this phenomenon. I believe that this is mediated by electrolyte imbalance perhaps in a select subgroup so predisposed. Angiotensin II and reactive oxygen species are both increased during exercise, especially in the context of any dehydration, as is often the case with endurance athletes. They are both potent agents for damaging cell membranes. (http://ndt.oxfordjournals.org/cgi/content/full/14/11/2585). This is where the inflammation connection may arise, since inflammatory markers are also increased by endurance sports.

That’s my long answer Mike. My short one is that you may have increased ectopics but your substrate is good. Congratulations.

PC

1. Mike, when you had your ectopics without afib, did you have an EKG to verify that you didn't have afib?

2. Can the sinoatrial or atrioventricular nodes be the source of an ectopic by emitting electrical signals at the wrong time?

3. From PC's message, "It suggests that PACs increase vagal tone and, ...
increased vagal tone enhances reentry."

More generally, increased vagal tone slows the heart rate and gives the ectopic focii more time to fire and thus increases PACs. If PACs increase vagal tone, then there is a positive feedback that maintains afib, i.e. PACs increase vagal tone which increases PACs which increases vagal tone, etc.

Bob K.

PC & Mike

Here is a link to the canine study abstracts I recently posted in a response to Mike. In the first they perpetuate the AF w/vagal stimulation. In the second, AF is not sustainable when vagal nerve fibers are ablated.
http://www.afibbers.com/forum/read.php?f=6&i=18749&t=18749#reply_18749

In this month’s Afib Report, Hans reports on a study showing HR increases after ablation. This is obviously related as vagal nerve tissue is damaged during ablations and this damage may be partially responsible for the ablation's success.

George Eby eliminated his PAC's with high doses of taurine:
http://www.coldcure.com/html/taurine.html
However, he was another one who had large quantities of PAC's (15,000/day-720/hour-12/minute) without AF.

"PACs/PVCs comprising < 1 % of total beats is classified as rare or occasional; PACs/PVCs comprising 1% to 10% of total beats is classified as frequent; PACs/PVCs comprising > 10 % of total beats is classified as very frequent."

At 60 BPM, these would be:
1% - 36 PAC'S/HR or 864 PAC'S/day
10% - 360 PAC'S/HR or 8640 PAC'S/day

The often quoted study of PAC increase prior to AF, showed the PAC count increasing to 4.2 PAC'S/min, which is 252 PAC's/hr and 6049 PAC's/day.

Clearly George Eby is in the very frequent range, and more than double the PAC rate needed to initiate AF in the study, but he did not have a substrate that would propagate the AF.

In my own case, K/Mg/taurine supplementation conspires to keep me in NSR. Why? As per PC's reference above to http://www.afibbers.com/forum/read.php?f=6&i=18571&t=18571 increasing serum K lengthens AERP and decreases Purkinje fiber automaticity. I notice the latter in the form of fewer PVC's.

As I've previously commented to PC, I think that at least some of the athletes propensity towards AF is due to the tendency of exercise to deplete electrolytes However, going along with this is an unanswered question - why do some people, like Mike, react to supplementation with increased ectopy?

I also believe that there are a group of LAF'ers who do not respond to supplementation (or respond sufficiently) prior to ablation, but for whom supplementation is necessary post ablation to control ectopy and avoid relapse into AF.

Mike, maybe if you took up endurance exercise, you could create a substrate that would sustain AF (just kidding).

As I write this, it occurs to me that, as has been shown, many have PAC's. Perhaps those who have AF have somehow created a substrate that facilitate reentrant circuits. Exercise can do this by increasing vagal tone and oxidation, and tends to deplete electrolytes. But then what about the non-athletes?

Good topic, Mike!

George

Aloha George and Mike,

Great topic and glad you two keep beating on it.

Regarding my statement that vagal tone does not trigger PACs, at least not directly, let me try to further clarify by asking a question.

By what mechanism can acutely increased vagal tone trigger a PAC?

In the above post I neglected to mention that coupling of PACs, i.e., consecutive PACs with a very short R-R interval, are quite significant in triggering AF. I think this speaks to reentry as the primary contribution of vagal tone to initiating AF, along with shortening of the AERP and conduction velocity (allowing reentry). But what is actually triggering the PAC (that reenters its own circuit with consequent coupling)?

If one were to eliminate the PACs, increased vagal tone should theoretically be impotent wrt initiating AF. Does everyone with increased vagal tone experience increased PACs? I think not, given the fact that bradycardia (slow heart rate) alone appears not to induce AF.

As previously speculated, I think the trigger is either local stretch or ectopic P/Purkinje cells, or both. A vagal maneuver could certainly provide the local stretch without involvement of the vagus.

Just my thoughts on the matter. Discussion and rebuttals encouraged.

PC

Here is something else from the forum that arguably belongs here - firstly two abstracts posted by Geirge and secondly my (modified) response thereto:

"Here are two abstracts which might shed some light. Notice in the first they perpetuate the AF w/vagal stimulation. In the second, AF is not sustainable when vagal nerve fibers are ablated.

Author(s): Elvan A ; Pride HP ; Eble JN ; Zipes DP
Affiliation: Krannert Institute of Cardiology, Department of Medicine, Indiana University School of Medicine, Indianapolis 46202-4800, USA.
Title*: Radiofrequency catheter ablation of the atria reduces inducibility and duration of atrial fibrillation in dogs*.
Source: **Circulation (Circulation.) 1995 Apr 15; 91(8): 2235-44**
Additional Info: UNITED STATES
Standard No: ISSN: 0009-7322; NLM Unique Journal Identifier: 0147763
Language: English
Abstract: BACKGROUND: The purpose of this study was to prevent induction of sustained atrial fibrillation (AF) by radiofrequency catheter ablation (RFCA) of the atria in an open-chest canine model. METHODS AND RESULTS: In dogs randomized to acute studies, RFCA of the atria was performed after reproducible induction of sustained AF (lasting > 30 minutes) with burst stimulation or premature atrial pacing and perpetuation by low level cervical vagal stimulation or IV infusion of methacholine. Additionally, in four dogs, the long-term effectiveness of RFCA was assessed 7 to 21 days after ablation. Continuous discrete transmural lesions were produced with radiofrequency energy pulses (20 to 40 W for 60 seconds) delivered to five atrial epicardial sites and endovascularly to the coronary sinus wall. RFCA electrically isolated regions of the atria that became dissociated from the nonisolated parts. Atrial RFCA markedly attenuated vagally induced shortening of effective refractory period (ERP) at both isolated and nonisolated test sites located in the left and right atria ($P < .001$, $n = 5$). RFCA rendered noninducible sustained AF maintained by cervical vagal stimulation. The dose-response curve relating the dose of methacholine required to maintain AF was shifted down and to the right. AF was only inducible with high doses of methacholine. Atrial RFCA reduced the maximal sinus rate and prolonged the corrected sinus-node recovery time ($P < .001$, $n = 6$). However, RFCA did not affect atrial contractile function, AV-nodal ERP, or AV-nodal or His-Purkinje conduction times. In dogs in the chronic group, normal sinus rhythm and normal AV conduction were preserved and AF was only inducible with a high dose of methacholine. No atrial perforations resulted. CONCLUSIONS: RFCA in open-chest dogs produces partial vagal denervation and reduces the inducibility of AF.

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Author(s): Chiou CW ; Eble JN ; Zipes DP
Affiliation: Krannert Institute of Cardiology, Indiana University School of Medicine, Indianapolis 46202-4800, USA.
Title: *Efferent vagal innervation of the canine atria and sinus and atrioventricular nodes. The third fat pad.*
Source: **Circulation (Circulation.) 1997 Jun 3; 95(11): 2573-84**
Additional Info: UNITED STATES
Standard No: ISSN: 0009-7322; NLM Unique Journal Identifier: 0147763
Language: English
Abstract: BACKGROUND: The purpose of this study was to investigate the functional pathways of efferent vagal innervation to the atrial myocardium and sinus and atrioventricular (AV) nodes. METHODS AND RESULTS: Using vagally induced atrial effective refractory period shortening, slowing of spontaneous sinus rate, and prolongation of AV nodal conduction time as end points of vagal effects, we determined the actions of phenol and epicardial radiofrequency catheter ablation (RFCA) applied to different sites at or near the atrial myocardium to inhibit these responses. We found that efferent vagal fibers to the atria are located both subepicardially and intramurally or subendocardially. Most efferent vagal fibers to the atria appear to travel through a newly described fat pad located between the medial superior vena cava and aortic root (SVC-Ao fat pad), superior to the right pulmonary artery, and then project onto two previously noted fat pads at the inferior vena cava-left atrial junction (IVC-LA fat pad) and the right pulmonary vein-atrial junction (RPV fat pad) and to both atria. A few vagal fibers may bypass the SVC-Ao fat pad and go directly to the IVC-LA or RPV fat pad and then innervate the atrial myocardium. Vagal fibers to the sinus and AV nodes also converge at the SVC-Ao fat pad (a few fibers to the sinus node go directly to the RPV fat pad) before

projecting to the RPV and IVC-LA fat pads. Long-term vagal denervation of the atria and sinus and AV nodes can be produced by RFCA of these fat pads and results in vagal denervation supersensitivity. Vagal denervation prevents induction of atrial fibrillation in this model. CONCLUSIONS: The newly described SVC-Ao fat pad receives most of the vagal fibers to the atria and sinus and AV nodes. Elimination of the fat pads with RFCA selectively vagally denervated the atria and sinus and AV nodes.

Cheers,
George

George,

Interesting about the denervation of vagal pathways to the atria as a means of preventing AF - as per PC's comment above ["I think this speaks to reentry as the primary contribution of vagal tone to initiating AF, along with shortening of the AERP and conduction velocity (allowing reentry)"].

The second abstract cited above is over 8 years old..... I wonder if anyone else has taken these ideas forward since? RFCA of the SVC-Ao fat pad would seem to - at least on the face of it - offer considerable promise for vagal LAFrs."

Mike F.

Hi PC,

So would GERD or other digestive issues be considered like vagal maneuver?

George

This is a timely topic for me as lately I have begun to wonder if I am ONLY experiencing ectopics and not AFIB.

Prior to my two ablations at NYU (Nov03 and May04), ectopics were always followed, in short order, by AFIB. But since the ablations, ectopics are just about never followed by AFIB. And now I'm contemplating the possibility it is NEVER, and not just PERHAPS NEVER.

I guess I don't believe ectopics cause or provoke AFIB. I used to focus on the rise in resting heart rate that always preceded the ectopics. But then I started to believe that even the rise in resting heart rate was just an effect of something else – probably a rise in adrenalin that sometimes was due to stress and sometimes was due to nothing in particular. Whatever caused the adrenalin rise led the waterfall that cascaded down to elevated resting heart rate, ectopics and finally afib.

The ablations (and low meds) have certainly prevented the cycle from completing itself. Nevertheless, the ectopics are not enjoyable, either, though not nearly so debilitating as AFIB and probably pretty harmless. The main issue of the ectopics is the anxiety that they MAY lead to AFIB. I think if I knew for certain that AFIB was not longer possible, the ectopics would be easier to ignore.

Currently I am taking 6.75 mg of atenolol every 8 hours. Recently, I lowered the flecainide dose to 12.5 mg every six hours or so. My HR is typically in the 55-58 range, and my BP is usually about 110/65. I am 50, exercise just about every day and am 5-11 and 168 lbs. I supplement with mag glycinate (400-600 mg), Potassium (two glasses of Low Sodium V8 and a banana) and 60 mg of CoQ10. I also avoid most simple carbs and eat very little gluten.

I am thinking again about dropping the flecainide altogether and seeing what happens. I tried this a few times in the first five months after the second ablation without success. But I have not tried for almost a year. Funny. After nearly perfect NSR on low meds for 15 months or so, I dread the thought of even a day or two of fib. And yet, prior to the ablations, I had at least several hours of fib every day for four years. And in the days leading up to the ablations, I was in fib 70 percent of the time. It's amazing what people can learn to live with!

I have always wondered if I was adrenergic or mixed (I sometimes had fib after exercising, and still get ectopics when I

lay down, though they subside after a few minutes). I think I am at least predominantly adrenergic. This is why I have some hope that, post ablations, I may be able to stay out of fib with a beta blocker alone. I would even be willing to take more beta blocker if it allowed me to stop the flec. Especially if it keeps the ectopics to a minimum.

It's probably about time I at least gave it another try. It would certainly help me decide about having a third ablation.

Hopefully I have stayed on topic here. It seems I darted in and out of it a bit.

BillB

Bill,

Do you have any data to show whether your ectopics are mostly PVC's or PAC's. A Holter test would be the most certain. PVC's usually feel like a "skipped" beat followed by a "hard" beat. PVC's do not initiate AF, so shouldn't be a concern.

George

Hi George,

Your question about GERD is a good one. Previously I always thought that the mechanism for AF episodes associated with GERD was as follows: GE junction irritation stimulates vagal afferent (sensory) nerves that terminated in the brainstem, causing stimulation of vagal efferent (motor) nerves to the GE junction. This activity in the brainstem would also stimulate immediately adjacent motor fibers to the SA/AV nodes and both atria. The consequent increase in cardiac vagal tone would then lead to AF.

However, during my hospitalization in Bordeaux I experienced very mild but distinct upper abdominal cramping for one or two days after the ablation. I never mentioned it to Prof. Haissaguerre, because it was so mild, and I thought some kind of referred pain, although it definitely extended across the midline. After returning home I just happened to come across an article entitled "Acute Pyloric Spasm And Gastric Hypomotility: An Extracardiac Adverse Effect Of Percutaneous Radiofrequency Ablation For Atrial Fibrillation."

The conclusion of that article stated that "thermal injury during endocardial LA RF energy delivery may extend into the mediastinum and rarely may involve the periesophageal nerves, resulting in a syndrome of acute delayed gastric emptying." So, while the theory that the signals causing increased cardiac vagal tone travel from the GE junction to the brainstem and then to heart is still valid, I think a direct link between the GE junction and the LA causing AF is definitely plausible.

I still think there is something else at work triggering the PACs.

Hi Bill,

Thank you for posting your thoughts.

There are many articles that describe the onset of AF to be a result of increased sympathetic tone abruptly followed by an increase in vagal tone. In other words autonomic transition seems to be significant in its genesis. Could it be that the increased automaticity of the former is responsible for the PACs? This sympathetic stimulation could range from a dream to a sudden loud sound or stress, etc. Perhaps the sudden increase in cardiac output stimulates the vagus via the baroreflex, which is supposed to be more sensitive in LAFers, at least according to one of my above referenced articles.

PC

It is difficult to find a reference describing the cause of PACs. Here is some of what I've come up with.

From: http://www.heartinfo.org/main.asp?page=askdoc&answer=6

In addition to the SA node, any muscle cell in the heart can send out an impulse, causing an “extra” or early heart beat. This serves as a protective mechanism for the heart – in case anything happens to the SA node, some other part of the heart can keep it going. The early beats don’t happen often because the natural rate of the SA node is faster than the natural rate of any of the muscle cells. Occasionally however, one of these cells will send out an impulse before the SA node. When the muscle cell which sends out the impulse is in one of the top two chambers of the heart (called the atria), the extra or early beat is called a “PAC” (for premature atrial contraction) or “APC” (atrial premature contraction). When the muscle cell which sends out the impulse is in one of the bottom two chambers of the heart (called the ventricles), the extra or early beat is called a “PVC” (for premature ventricular contraction) or “VPC” (ventricular premature contraction).
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http://www.nlm.nih.gov/medlineplus/ency/article/001100.htm

Ectopic heartbeat is an irregularity of the heart rate and heart rhythm involving extra or skipped heartbeats.

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Ectopic heartbeats are an arrhythmia involving small variations in an otherwise normal heartbeat. In many cases, they may occur without obvious cause and be benign.

Other times, however, they are associated with electrolyte abnormalities in the blood which should be corrected. They can also be associated with ischemia, or local reduction in blood supply to the heart. In addition, ectopic beats may be caused or aggravated by excessive smoking, alcohol consumption, caffeine, certain medications, and some illicit drugs.

Ectopic beats are rare in children other than those with congenital heart disease. The majority of extra heartbeats in children are PACs (premature atrial contractions), which are almost always benign.

In adults, ectopic beats can occur more commonly, and underlying reversible reasons should be investigated even if it turns out that no treatment is ultimately needed.

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http://www.fpnotebook.com/CV14.htm

Types: Premature Atrial Contractions

1. Causes
1. Usually idiopathic and benign
2. Catecholamine excess
3. Hypoxia
4. Myocardial Ischemia
5. Congestive Heart Failure
6. Acid-base disturbance
7. Electrolyte abnormality

George

George,

I think they're PACs. I believe when I had my Holter (Jul04) after the May04 ablation, Dr. Chinitz at NYU mentioned PACs, not ectopics, and no Fib. At that point, he suggested I drop all meds. But when I did, the fib and ectopics came

back. Of course all that was about 16 months ago.

About 60 days after the second ablation (Aug04), I went into a period of about eight months without any fib or ectopics. But each time I tried to get off the meds, things got dicey again. At that point I just assumed the ablation was not totally successful, and stopped trying to wean off.

In the Spring of 2005, ectopics started popping up. I increased the atenolol a little and they went away for a couple months. Now they pop up every once in a while. Most of the time I have none. Some days I have a few. And once in a while I have quite a few over a period of hours. In addition, there are periods when I feel ectopics in my chest, but they never reach my heart.

To describe the sensation, its feels like my heart is about to beat, then does not. Then that beat feels skipped altogether, there is a pause, then a regular one occurs.

Can flec actually cause ectopics? It would seem, being pro-arrhythmic, that it could. It would be nice if when I went off the flec this time the ectopics went away. ... But I could not be that lucky, could I?

BillB

Bill,

Some day when I have time, I should hook up some monitors and simultaneously listen to my heart with a stethoscope so I can accurately describe what PAC's & PVC's sound like. Now I just see them on a monitor.

George

PC may have answered this question but I probably missed it. I guess some of this discussion is a bit over my head. For me, PACs always preceded (if not "caused,") fib. Prior to the ablations, I mean. Now, PACs are seldom if ever followed by fib.

My question is if the ablation is meant to prevent the ectopic beats originating in the pulmonary veins from reaching the atria, and a completely successful ablation stops FIB, why doesn't it also stop the ectopics from reaching the atria (provided they are coming from the PVs)? And it ectopics are still negotiating their way through the ablation wall, then why is there no fib? Particularly among those of us who were predisposed to fib in the first place.

Up until now I have been giving the flecainide a lot of credit for my post-ablation NSR. I have assumed there were a few holes in the ablation wall that could allow the ectopics to get through. If 500 ectopics/hour were flung at the wall, some would pass through the holes. But because of the flecainide, there were not 500, but more like 5 or 10. And most of the time, none of them found the holes. Thus, no fib.

I can understand it if ALL the ectopics were blocked by a successful ablation. Then perfect, drug-free, NSR would be easy to follow. I can also understand a few holes in the line, and reduced ectopics due to meds. But presumably all "successful ablatees" have blocked beats. And many still experience PACs. Some a lot of PACs. Unless burns were being done on the atria side of the wall, it just seems illogical that these PACs would not lead to AFIB.

BillB

Bill,

Are you sure it is not PVCs you are experiencing? A successful ablation would have no effect on PVCs as they originate in or close to the ventricles ie. well below the ablation "rings". Potassium supplementation is very successful in keeping PVCs at bay.

Hans

Bill,

If I had to guess, from what you describe, I'd say you're having mostly PVCs & not PACs. But without a monitor it is hard to say.

You bring up a good question - does the ablation stop a PAC from happening, or just keep it from repeating - becoming reentrant. Maybe PC can answer this question.

You might try upping your K intake through foods (www.fitday.com) & supplements so you are getting 4-5 grams/day. Also 3 grams/day taurine might be beneficial. See http://www.coldcure.com/html/taurine.html and search for Jackie's posts on taurine.

George

Bill & George,

A good question indeed! As I said in my second posting to this CR session above in reply to Hans:

"Hans,

Thanks for your encouraging words. From your above post:

"In answer to your question 2 in your second posting. The scar tissue created during the ablation, particularly around the pulmonary veins, prevent a run of ectopics from spreading to the atrium and create the chaotic contractions characteristic of afib."

Noted, but given that successful ablatees actually FEEL ectopics AFTER their ablations. I'm accordingly assuming that the errant signal/s emanating from the focus/focii in the lower PVs ARE making it down past the scar tissue to the atria in order for the ectopic to be felt?? My point therefore being that the ectopic activity is STILL occurring even after an ablation. As such it may be that those experiencing AF AFTER an ablation are those (obviously in addition to those whose focii were elsewhere in the atria i.e. NOT only in the PVs) for whom one or two ectopics were/are enough to precipitate AF. Other individuals for whom ablations were/are successful (in eliminating AF episodes) but who still experience ectopy comprised those individuals who would NOT (before their ablation/s) have had an episode of AF further to just the odd ectopic."

I guess successful ablations EITHER: prevent PACs from occurring (with any ectopy being felt comprising PVCs) OR successful ablations do NOT prevent PACs from occurring but they DO stop PACs becoming re-entrant.

Has anyone had PACs noted during monitoring after a successful ablation?

Further views guys?

Mike F.

Aloha Bill,

An excellent question, as previously acknowledged.

You've been given excellent responses as well. I'd agree with others that they are probably PVCs and K+ should help that.

However, one should remember that a significant number of PACs originate outside the PVs, as underscored in several of the CC symposium presentations kindly furnished by Jackie on the BB. The Bordeaux experts and others don't stop at the PVI but routinely go looking elsewhere for PAC activity. It would seem quite plausible that some of

these might be missed. And perhaps you really are experiencing PACs.

This then brings up the obvious question of why no AF in some of these individuals. Is it the critical mass thing? Perhaps Moe (1957) was/is right in proposing that six or more wavelets are required to trigger AF. Perhaps AF is prevented by ablation if and only if these extra PV sites of PAC activity drop below some threshold value.

Whatever ablation does to isolate ectopic activity, we must remember that it also denervates some of the cardiac vagal nerve fibers. Nonetheless, inflammation can induce AF in the recuperative period. As the nerve fibers regenerate, the soil can presumably became more fertile for sustaining AF. So, personally I'd watch my electrolyte balance and limit my workouts somewhat.

It is also worth noting that Natale et al. have demonstrated that in at least some successfully ablated patients ectopic activity continues in the PVs without spreading to the left atrium. One can only wonder what this must feel like. However, it would not manifest as an abnormality in HR or rhythm.

Mike,

I should soon be able to respond to your last question.

PC

If in fact I am experiencing PVCs, not PACs, would flecainide reduce their number? And would the atenolol have any effect one way or the other?

I guess I will know better in the next few days what I am experiencing. I intend to skip the flecainide for 48 hours after tomorrow morning's 12.5 mg dose (I'm only taking 50 mg/day, so I don't think any more weaning is needed). From what I gather here, if I start to experience fib, (a) they are probably PACs and (b) I should start to serious think about ablation number three.

BillB

From a recent forum posting by Jackie over on the Forum:

"Titled **ATRIAL FIBRILLATION AND THE VAGUS: STIMULATION OR DENERVATION**?

Seil Oh, MD, PhD."

*Author(s): Chiou CW ; Eble JN ; Zipes DP
Affiliation: Krannert Institute of Cardiology, Indiana University School of Medicine, Indianapolis 46202-4800, USA.
Title: Efferent vagal innervation of the canine atria and sinus and atrioventricular nodes. The third fat pad.
Source: *Circulation (Circulation.) 1997 Jun 3; 95(11): 2573-84*
Additional Info: UNITED STATES
Standard No: ISSN: 0009-7322; NLM Unique Journal Identifier: 0147763
Language: English
Abstract: BACKGROUND: The purpose of this study was to investigate the functional pathways of efferent vagal innervation to the atrial myocardium and sinus and atrioventricular (AV) nodes. METHODS AND RESULTS: Using vagally induced atrial effective refractory period shortening, slowing of spontaneous sinus rate, and prolongation of AV nodal conduction time as end points of vagal effects, we determined the actions of phenol and epicardial radiofrequency catheter ablation (RFCA) applied to different sites at or near the atrial myocardium to inhibit these responses. We found that efferent vagal fibers to the atria are located both subepicardially and intramurally or subendocardially. Most efferent vagal fibers to the atria appear to travel through a newly described fat pad located between the medial superior vena cava and aortic root (SVC-Ao fat pad), superior to the right pulmonary artery, and then project onto two previously noted fat pads at the inferior vena cava-left atrial junction (IVC-LA fat pad) and the right pulmonary vein-atrial junction (RPV fat pad) and to both atria. A few vagal fibers may bypass the SVC-Ao fat pad and go directly to the IVC-LA or RPV fat pad and then innervate the atrial myocardium. Vagal fibers to the sinus and

AV nodes also converge at the SVC-Ao fat pad (a few fibers to the sinus node go directly to the RPV fat pad) before projecting to the RPV and IVC-LA fat pads. Long-term vagal denervation of the atria and sinus and AV nodes can be produced by RFCA of these fat pads and results in vagal denervation supersensitivity. Vagal denervation prevents induction of atrial fibrillation in this model. CONCLUSIONS: The newly described SVC-Ao fat pad receives most of the vagal fibers to the atria and sinus and AV nodes. Elimination of the fat pads with RFCA selectively vagally denervated the atria and sinus and AV nodes.*

My guess is that the young fellow is 'on the money' with this area of research - at least for vagal AFrs, but likely for AMAFrs and VMAFrs alike. As pointed out by PC above when he states "we must remember that it (ablation of the PVs)also denervates some of the cardiac vagal nerve fibers", I am personally intrigued with the idea of ablations perhaps evolving to the point where they include the SELECTIVE (as opposed to accidental/incidental as is the case currently with PVA) denervation of the atria.

Mike F.

A couple of related thoughts WRT ectopy and the ANS.

We have here predominantly been talking in terms of ectopy and AF caused by excessive vagal tone. This makes particular sense for those folks who have all their lives undertaken endurance sports - as stated many times in research, endurance athletes have a six to ten-fold higher chance of developing AF than average. Why? three reasons in simplistic terms - 1) larger atria 2) more oxidative stress and 3) excessive vagal tone. But what about other (non-endurance sport types) vagal AFrs?? I'm remembering here a comment by a fellow called Richard who used to post a great deal to the Forum about amino acids: he once said he believed that the problem with his own ANS wasn't excessive vagal tone but LOW SYMPATHETIC tone. This interests me since I have often wondered whether or not chronic stress as a child (and adult) could kind of exhaust the sympathetic side of the ANS by having ran it at full throttle for too long. If this is the case for some of us here, what can we do about it? What would be a definitive way of discovering whether or not the sympathetic side of our ANS were sub-normal??

Any ideas?

Mike F.

A totally new direction for this conference topic.

What effect does gastric dysrhythmia’s have on have on increased vagal tone and hence pac’s and ultimately afib?

The gastric dysrhymia’s I am talking about are tachygastria, bradygastria and stomach fibrillation (af of the stomach). My afib and pac history very closely matches Mike F with the biggest component and influence being a history of gastric troubles and a great suspicion that our pac troubles originate in the digestive system, in particular the esophagus and stomach. Upwards of eighty percent of afibbers have reported some sort of digestive troubles.

I have come to the conclusion that, in my case, any run of pac’s and afib was preceded by a gastric dysrhymia of some sort. This gastric dysrhymia greatly increased vagal tone and created a fertile field for ectopics and afib.

My reasoning behind gastric dysrhymia’s ultimately causing afib goes back to PC’s post on 04-28-05 discussing the P cells and their origin in the neural crest in the developing foetus. Basically, PC’s post states that some P cells are left behind and lodge in the PV,s in their race to reach to reach and settle in the nodes of the heart and this could happen through a faulty gene turning off too soon or whatever (hope I got this right PC).

This set me thinking. What if this “fault” was also ALSO through the electrical systems of the stomach, esophagus etc? In other words not only is the electrical systems of our hearts faulty but also the electrical systems of our stomach and digestive systems? A double whammy. If both the stomach and heart electrical systems are faulty does this predispose us to eventual afib?

Tachygastria, bradygastria and stomach fibrillation can last from several seconds to hours and days just like afib. It is

almost a parallel of arrhythmias of the heart.

This is an interesting post about gastric dysrhymia's and will give you some idea of what I am getting at:

"Department of Medicine, Cedars-Sinai Medical Center, CSMC Burns and Allen Research Center, Los Angeles, California 90048, USA.

Using the technique known as electrogastrography, we studied the postprandial response of gastric myoelectrial activity in subjects with type II diabetes. Seventy-one subjects with type II diabetes underwent 1 hr of fasting electrogastrography recording. HbA1c and fasting serum glucose levels were obtained. Subjects then underwent an additional 2 hr of electrogastrography recording in the post prandial state. Sixty of the 71 patients (85%) had gastric rhythm abnormalities in the fasting state. Forty-six of 71 subjects (65%) responded to the test meal by improving their electrogastrography tracings (responders) while 35% did not respond (nonresponders). The time spent in bradygastria during the fasting state by responders was 26.3+/-12.8% vs 10.9+/-8.5% for nonresponders ($P < 0.0001$). The percent tachygastria during the fasting state in responders was 19.8+/-13.0%, which was less than nonresponders (38.3+/-29.7%) ($P < 0.001$). Fasting plasma glucose and HbA1c could not be used to predict the gastric myoelectrical response to meal. In conclusion, gastric rhythm disturbances are common in type II diabetes; there was no correlation between HbA1c levels, age, duration of diabetes, or fasting serum glucose and gastric dysrhythmia in response to meal; two groups of subjects emerged: those who became less dysrhythmic in the post pradial state (responders) and those who did not (non-responders); and fasting bradygastria was associated with responders and fasting tachygastria was associated with nonresponders."

As you can see, gastric dysrhymia's are very common in diabetics. As hypoglycemia is over represented in afibbers could gastric dysrhymia's be very common amongst us too? I would bet a good deal of money that they are!

Has any afibber had an electrogastrogram (EGG) and an ECG together to investigate if ectopics and afib start with an electrical hiccup of the stomach? I bet the answer is NOT A SINGLE ONE OF US.

Mike F, as you started this topic maybe you can be the guinea pig and have an EGG and ECG combined? You would be ideal with your gastric problems.

What are your thoughts on the above, particularly your thoughts PC?

Dean

Dean,

Great original thoughts!

George

Interesting ideas Dean - I remember you proposing and discussing these ideas previously. Regrettably, I severely doubt that the NHS in the UK would be up for monitoring me as you suggest - they really only do tests that THEY consider to be appropriate usage of limited funds.

You state: "to investigate if ectopics and afib start with an electrical hiccup of the stomach?" This I like a lot, especially given my propensity to experience an ectopic some 50% of the time that I belch - especially when I've succeeded - after some straining/trying - in getting some wind up to alleviate some GERD discomfort. I'll be interested to see what PC and the other folks here have to say on this topic. Maybe this subject area merits discussion here in this CR in its own right - and your posting above could kick it off? I'm sure that I remember you citing other additional pieces of research pertaining to other gastric dysrhythmias back when you raised this area of discussion previously??

Mike F.

Aloha Dean and Mike,

Couldn't agree with you more. However, I would suggest that there may be no need to invoke a developmental abnormality to explain it.

The body has all kinds of reactions that underscore the sometimes unexpected reflexes triggered by vagal stimulation. For example, splashing cold water onto one's face or gulping an ice cold drink (thereby stimulating the vagus) causes the heart rate to slow (along with a decrease in AERP and an increase in dispersion).

I think gastric dysrhythmia of whatever etiology could also result in a vagal reflex triggering AF. The question is whether this reflex goes through the brainstem or operates locally via direct contact with the immediately adjacent parasympathetic ganglia (see above post on 11/2).

PC

Hi Mike and PC,

Maybe Hans could put gastric dysrhymia's as a separate topic for some future discussion - it sure sounds interesting. When I was researching electrogastroscopy a year ago it was a new and very experimental field. I have since lost all the references to the websites I visited by I do remember that when researchers were doing EGG's they noted on several occasions there were "electrical spikes of unknown origin" in their graphs - sounds intriguing with regard to afib.

I suppose all of this is but another piece of the afib jigsaw to put together.

Dean
