

Your Premier Information Resource for Lone Atrial Fibrillation
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<http://www.afibbers.org>

VIRTUAL LAF CONFERENCE

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SUBJECT: Post-procedure PACs

Given the general mechanism of action, ablative techniques should surely stop not only AF but PACs too (or at least lead to a significant reduction in them).

[Unless, of course, many of the PACs experienced both BEFORE and AFTER the successful ablation originate elsewhere other than the PVs. But that said, surely the vast majority of LAFrs have triggering PACs originating in the PVs as opposed to elsewhere in the heart.]

I have, however, noticed many successful ablatees reporting no reduction in PACs or even an albiet often gradual INCREASE in PACs AFTER a successful ablation..... Anecdotal evidence anyone??

More particularly, has anyone noticed INCREASED PACs after ablation though the ablation largely or completely resolved their AF? If so, WHY WHY WHY would this be the case?

Any and all discussion would be of great interest to me and no doubt others also.

Mike F.

Hi Mike,

Here is just a thought:

All ectopic foci do not result in AF. If you get rid of those that results in AF perhaps you could still have some left that will generate some PACs. A PVI just ablate at a specific area in the heart and how do you distinguish between PACs and PVCs?

Gunnar

Mike,

By far the majority of PACs originates in the pulmonary veins. A pulmonary vein isolation procedure isolates the pulmonary veins from the left atrium thereby preventing the PACs from propagating impulses which could initiate atrial fibrillation. However the ablation does nothing to stop the "production" of PACs in the PVs themselves. As a matter of fact, it would not be surprising if PACs increased in frequency after an ablation due to post-procedure inflammation.

In my opinion, the best and perhaps the only way of reducing PACs is through supplementation with potassium,

magnesium and taurine.

Hans

Mike F is falling prey to the concept that ablation is a "cure", in that it somehow restores the heart to a healthy state. Or, alternatively, that the heart was completely healthy except for one defect, which caused a-fib, and ablation repairs that one defect.

A-fib appeared because of heart deterioration, probably to some considerable extent a general deterioration. An ablation further damages the heart, but the additional damage is specifically designed to block a-fib, by interfering with its ability to sustain itself.

After the ablation: (1) the original deteriorated heart condition remains unchanged; (2) the additional damage deliberately created to block a-fib is a fact; and (3) the final symptoms observed are a complex result of (1) and (2).

Hopefully, the final result (3) includes an absence of a-fib plus other symptoms which are hopefully not too bothersome.

Wil

As understand it, an ectopic in the PVs post ablation shouldn't result in a PAC i.e. it wouldn't cause the atria to contract (and shouldn't produce a P wave on a chest ECG because the number of cells actually firing is relatively low) So IMHO it's likely that any PAC recorded post ablation is either originating away from the PVs or is a result of 'electrical leakage' if they originate in the PV.

There is a suggestion that AF is not quite as chaotic as was once thought and that the PVs actually drive AF. If you were to examine the heart with a high enough resolution ECG it would look like the PVs were in atrial flutter (or maybe micro reentrant AF) but by the time this regular activity propagates into the atria it degrades into AF.

My vote would be that most ectopics recorded post ablation are ectopics occurring in the atria. The reason why some folk may see an increase is that none of them are managing to kick start AF (because ectopics external to the PVs don't get inside the PVs to 'get the AF motor running').

Just another one of my speculative guesses :)

--

James D

Hi Wil,

You said,

"A-fib appeared because of heart deterioration, probably to some considerable extent a general deterioration."

AF due to a defect within the heart could be due to a deterioration, or one could be born with a valve defect, etc. I would like to know where you found information saying LAF is due to heart deterioration or even a general deterioration, or is that just your opinion?

Thanks,

Jim

Mike

Since my ablations I get lots of ectopics but no Afib.

My first ablation was 'the whole works' ie PVI plus all the possible lines because I had been in chronic Afib for 18

months. Nevertheless it recurred after 3 days.

Pr. Haissaguerre did my second ablation immediately, and afterwards he said that he had found an extremely toxic focus in the coronary sinus which was very difficult to find and ablate, and that he wasn't sure that he had been able to get all of it.

I have always assumed that this is why I get ectopic beats but the ablation lines have prevented Afib so far so I do consider it a success. And if Afib should happen I would hope that the lines would mean that it was impossible for it to sustain itself.

Gill

James,

I guess I should have been a bit more precise in my reply. What I meant to say is that I think the PACs originate in the area around the pulmonary veins that was encircled by the ablation lesions during the PVI procedure. The myocardial sleeves, which extend quite far into the pulmonary veins, might be a prime generator of these ectopics. However, because the effect of the ectopic beats cannot propagate beyond the lesion circles there will be no afib.

Hans

Hi Hans, thanks for the clarification but I'd still go further and add to you last sentence with"

"However, because the effect of the ectopic beats can not propagate beyond the lesion circles there will be no afib and no PAC'

Since the PV activity doesn't propagate beyond the ablation line the Sinus Node is not 'beaten to the firing gun' and no atrial contraction occurs due to the ectopic in the PV. I doubt whether a PV ectopic would be felt or produces more than smallest of spikes on an ECG (and the PV ectopic shouldn't interfere with where the normal 'P' wave starts)

This link was posted a little while back

<http://www.edwards.com/Europe/Products/AtrialcoxPVideo.htm>

If you've not already seen John Boineau's talk he explains what I was trying to get at in my earlier post in much clearer detail. Sadly, if my memory is working, the link requires Internet Explorer to get at the videos (something I wouldn't normally recommend running)

So I'd still say that actual PACs (ectopic beats that result in the early contraction of the whole atria) post ablation are either a result of the ectopic occurring in the atria or a faulty ablation line not blocking an ectopic that starts in a PV.

I guess what I'm really trying to say is we should avoid calling a PV ectopic a PAC in the case where it doesn't trigger the whole atria to contract :)

--

James D

Hi Hans and Mike,

Thank you both for creating and deciding on such a titillating topic.

I thought the following articles might shed some light on the subject:

Trigger Activity More Than Three Years After Left Atrial Linear Ablation Without Pulmonary Vein Isolation In Patients With Atrial Fibrillation.

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=16022965&dopt=Abstract (thank you Marian) "trigger activity remains relatively frequent but short and does not induce AF episodes in most patients."

This suggests to me that the successful triggering of AF may be more dependent on some ectopic/reentrant foci than on others. Effective ablation might require elimination of the DF site (?toxic focus) - the one with the dominant frequency - and not just any old abnormal foci. Indeed PVI gained popularity because focal ablation was too tedious, especially when these ectopic foci seemed to be emanating from within the PVs. So, instead of individual targeting, isolation became the favored approach.

For some reason these 'toxic foci' inside or outside the PVs can elicit vagal reflexes (bradycardia, asystole or hypotension) when stimulated. Pappone has shown that elimination of these vagal reflex sites increases the success rate.

"Atrial fibrillation ablation"

http://www.italheartj.org/pdf_files/20050045.pdf

"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 denervation."

Vagal reflex sites probably represent the DF.

Natale et al. has shown that ectopic foci within successfully isolated PVs continue to fire and that these foci behave like nodal cells when anti-arrhythmics are given to such patients. Such foci of course are contained and cannot trigger AF or impact in any way atrial rhythm.

"Response to pharmacological challenge of dissociated pulmonary vein rhythm"

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=15720448&dopt=Abstract

Creation of new ectopic foci also seems possible, depending on atrial pressures and stretch and concomitant electrolyte imbalance. Over 19 months post ablation my ectopics have dropped dramatically. I'm sure part of the reason for this is my decreased vagal tone (vagal reflex site was identified and ablated) as evidenced by my persistently elevated resting HR (about 40 to 45 preablation and 55 to 60 postablation). The other part is the successful to date PVI by Prof Haissaguerre.

PC

My acceptance of a heart deterioration/a-fib connection is the result of inductive reasoning. Some inputs have been:

- (1) All parts of our body deteriorate as we age, mostly irreversibly (hair turns gray; skin becomes less elastic; memory becomes less functional; hearing deteriorates; eyesight deteriorates; etc.) It seems likely the heart too would deteriorate as we age. One probable indication of this is the steady decrease in max heart rate with age.
- (2) A few years back, when ablation was less successful and less taken for granted, there were numerous discussions about the nature of the various forms of heart deterioration noted during ablations, and how each form of deterioration related to reduced ablation success.
- (3) The fact that the young rarely experience a-fib. A-fib is primarily a problem of older folks. This suggests a heart deterioration component.
- (4) The fact that those who abuse their hearts, marathoners being the prime example, suffer much higher rates of a-fib. This too suggests heart damage/deterioration as a component of a-fib.
- (5) This web site is largely populated by obsessively athletic folks. While they probably have excellent cardiovascular health, their a-fib suggests that deterioration of the electrical health of their heart was an unintended consequence of their improved cardiovascular condition.
- (6) Those a-fibbers with additional non-a-fib related serious heart problems have lower ablation success rates. This suggests that, consistent with the presence of their other heart problems, there would be increased levels of general heart deterioration, which could explain their lesser ablation success prognosis.
- (7) Its late, and my youthful 18 year old brain, encased in this 70 year old body, refuses to retrieve any more thoughts tonight, but it could if it wanted to.

Wil

Hi Wil, I was 28 when I developed AF. As far as I can tell the only abnormal thing about my heart is that I have unusually large pulmonary veins. So, in my case, I'm not sure whether I'd call it deterioration or just unusual anatomy. I've tried the idea of promoting a new diagnosis of 'anatomical AF' to cater for this situation but it's not caught on yet:)

If the PVs are responsible for holding the 'mother rotor' during AF that actually drives AF it doesn't matter if an initiating ectopic originates in the PVs or the atria. If you electrically isolate the mother rotor/toxic foci from the rest of the heart AF won't maintain itself.

For many years I've been a fan of AF being a self-sustaining arrhythmia and am not keen to completely dismiss this idea for some AFers. Perhaps it's what distinguishes successful and unsuccessful ablations? If a person's AF can sustain itself without the PVs they'll be still susceptible to AF post-ablation. For these folk maybe it is fibrosis or some other anatomical structure which allows a mother rotor to exist or maybe AF can indeed be self-sustaining in some individuals whose hearts show no sign of deterioration?

As has been said many times AF is a symptom of multiple diseases, if we can get better at diagnosing which one we have we might get better at tailoring a solution to the individual. I've no doubt deterioration plays a large part in a lot of people with AF - I'm just not sure how much it plays a part in Lone/idiopathic AFers

--

James D

Actually I'd agree with both Wil and James, depending on your preferred vantage point.

Relation of age and sex to atrial electrophysiological properties in patients with no history of atrial fibrillation
"The authors conclude that the mechanism triggering atrial fibrillation may be different between older and younger patients with atrial fibrillation, because younger patients who have no marked substrate for atrial fibrillation may need many trigger beats to induce atrial fibrillation."

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12765452&dopt=Abstract

This author is presumably talking about pathologic AF and LAF respectively.

Those with LAF are more likely 'trigger fibrillators', while those with pathologic AF are more likely 'substrate fibrillators'. In fact the driver or rotor theory of AF (DF or dominant frequency driven) speaks to the former, and the multiple reentrant wavelet theory (of Moe) speaks to the latter.

Mechanisms of atrial fibrillation

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=15005213&dopt=Abstract

Clearly substrate deterioration is what primarily drives 'substrate fibrillation'. The \$64 question is what drives 'trigger fibrillation'? Is it deterioration? Clearly AF episodes themselves hasten deterioration of both trigger substrate and maintenance substrate. But there had to be a trigger before the first episode and for most LAFers this trigger is essentially of congenital origin, potentiated by lifestyle to some extent IMHO. Such a lifestyle does not appear to trigger LAF in most normals, those not so predisposed. Is this process deterioration or biochemical individuality or?

PC

Aloha PC,

"Is this process deterioration or biochemical individuality or?"

What about From the Afib Report, March 2004 (and your post in CR35

<http://www.afibbers.org/conference/session35.pdf>):

Conduction cells in pulmonary veins.

Researchers at the Cleveland Clinic report that autopsies of the myocardial sleeves of pulmonary veins in five atrial fibrillation patients revealed the presence of P cells, Purkinje cells, and transitional cells similar to those found in the

heart's conductive (nodal) tissue. Conductive cells were not found in five control patients without atrial fibrillation. The researchers point out that whether or not these conductive cells are involved in AF still needs to be determined. Journal of Cardiovascular Electrophysiology, Vol. 14, August 2003, pp.803-09

<http://www.blackwell-synergy.com/doi/abs/10.1046/j.1540-8167.2003.03075.x?cookieSet=1&journalCode=jce>

Since most normals don't have these P cells, that would seem to be a difference.

George

Hans,

Thanks for putting my post in the CR. Its something that's always bothered/fascinated me. I guess this indirectly ties in with something else that's always intrigued me: from all my readings here over the years, I've concluded that some folks get lots of ectopy but little or no AF, whilst others need only the occasional PAC to trigger an AF episode.

James,

Thanks for clarifying what I was trying to get at wrt PACs in the PVs surely NOT being able to initiate a PAC POST (successful) PV isolation.

George,

I'm with you that some younger folks (like James) without ANY of Wil's cardiac degeneration per se get lots of PACs and ultimately LAF as a result of 'simply' having nodal tissue where it shouldn't be.

PC,

As we have discussed previously elsewhere - and following on from George's contribution - I class myself as an individual with a lot of trigger activity (likely in the PVs, but who knows without an EP exam) but thankfully not much in the way of marked substrate. Hence my frequent ectopy (over the last 20 years) and relatively infrequent LAF (6 episodes over the last 8 years).

You also state: "For some reason these 'toxic foci' inside or outside the PVs can elicit vagal reflexes (bradycardia, asystole or hypotension) when stimulated. Pappone has shown that elimination of these vagal reflex sites increases the success rate." Whether or not my own ectopy as aforementioned is driven to some extent by the activity of atoxic foci/vagal reflex site, I don't know. But I do find it intriguing that much of my (frequent) ectopy takes the form of slower arrhythmias frequently including 1.5 to 2 second pauses (sometimes mid-run-of-ectopy but particularly at the end of a few second run of ectopics).

(My own (UK) EP told me recently that if he needed an ablation that he'd go to Pappone.) I'm particularly intrigued with the driver and rotor theories of AF. I would regard myself as more of a driver-type/trigger fibrillator: forgive the perhaps naive gait of this question, but does this mean I can actually have an AF episode WITHOUT any Moe multiple re-entry wavelet action going on? If so, how?

Wil,

Can you see the sense in what George and PC are saying wrt to LAFrs likely being trigger fibrillators and AFrs likely being rotor fibrillators? It makes sense to me that some younger folks here (such as James) really can and do have LAF as a result of anatomical deviations from the norm as opposed to the cardiac degeneration as typically seen in older individuals.

Cheers guys,

Mike F.

Correction:

When addressing Wil in my above post, I meant to say:

"Can you see the sense in what George and PC are saying wrt to LAFrs likely being trigger fibrillators and AFrs likely being substrate fibrillators?"

(Hans, I know (and am glad that) you are enjoying a full LAF/AF-free life, but how about an edit facility?? (-;)

Mike F.

Spectral Analysis Identifies Sites of High-Frequency Activity Maintaining Atrial Fibrillation in Humans
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=16061740&dopt=Abstract
"paroxysmal AF patients were more likely to harbor the DF site within the pulmonary vein, whereas in permanent AF, atrial DF sites were more prevalent."

George,

This is a nice article by the Bordeaux Group (actually it was published within two weeks of my ablation there) that gets at the P cell thing. I've communicated with Prof Haissaguerre on this point. He doesn't believe in P cells in the PVs, while Natale obviously does. I think the answer lies in the fact that veno-atrial stretch upregulates at least one of the channels that essentially defines nodal cells, i.e., the funny current (a Na channel). Perhaps these abnormally placed P cells are not congenital in nature but created by long standing veno-atrial stretch. The difference in the location of DF sites between paroxysmal and persistent AF supports this interpretation.

Mike,

I don't pretend to be an expert on the intricacies of cardiac electrophysiology, but my simple minded understanding is that the cycle length of the DF is quite short (well over 300 cycles per minute) and overwhelms the atria, making NSR impossible. These sites are not firing on the basis of automaticity, but on the basis of reentry. There's interplay between foci in the atria and foci in the PVs. When intra-atrial and PV pressures go up, so does the automaticity of these 'P cells', esp. those within PVs. When AERP and PVERP shorten sufficiently, i.e., enhanced vagal tone, conditions are ripe for reentry and the DF jumps into action and voila - AF. I believe the DF sites represent a subset of these 'P cells' with significant automaticity that happen to be very close to vagal ganglionic plexi (the part of the nerve that includes the nucleus not just a conductive fiber). It seems rather coincidental that these 'toxic foci' just happen to be located where all the vagal ganglionated plexi are usually found - PVs, coronary sinus, roof of the left atrium, etc. Prof Haissaguerre ablated at least three such sites in my heart, including the first three sites listed. Anecdotally I continue to experience occasional very brief episodes of tachycardia out of the blue (always just half a dozen or so beats), but never AF. This is presumably because the good Prof 'took out' my DFs, so no reentry now.

So, in LAF it may be a case of one or more rotors but not six or more reentrant wavelets, which I can more easily envision wandering around the microfibrotic atrial landscape in pathologic AF.

Just my opinion FWIW.

P.S. Look up the Bezold Jarisch Reflex.

"Bezold-Jarisch-like reflex during radiofrequency ablation of the pulmonary vein tissues in patients with paroxysmal focal atrial fibrillation"

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=retrieve&db=pubmed&list_uids=9930906&dopt=Abstract
"RF catheter ablation of the pulmonary vein tissues could evoke a variety of profound bradycardia-hypotension responses. The Bezold-Jarisch-like reflex might be the underlying mechanism."

The observation that stimulation of DF sites often results in a strong vagal response (hypotension, bradycardia, asystole) at least suggests proximity of the DF site to a ganglionated vagal plexus. If true, this could easily translate to maximal vagally mediated shortening of the ERP at the DF site => reentry.

Hope no one got a brain cramp. Apologies in advance.

PC

I am 10 months post PVI ablation and am experiencing increased PACs, at times. I experience some mild afib too that is triggered by the PACs. I thought I was cured a few times, until they showed up again. I didn't know what they were

until I wore a Holter monitor for a month and consulted my EP.

My afib was unique before the ablation - a mild 80-90 bpm, but going on for 5 days straight generally. It seems the PACs have replaced that pattern, going on for days and sometimes they are more uncomfortable. I feel like the ablation was only a partial success.

Ray H.

The engineer in me thinks of any newly manufactured device, if it conforms to design specifications, as being perfect, because it does not exhibit behavior which is outside of its design goal, expressed as design limits.

In our case, a newly manufactured heart, if it conforms to design specifications, will not exhibit a-fib, because such behavior is outside of its design limits.

All manufactured devices, as they age, will eventually begin to exhibit behavior which is outside of the design limits. To an engineer, the appearance of such (mis)behavior is thought of as resulting from deterioration of the device from its original perfect state.

Such deterioration takes many forms:

(1) It may involve a device flaw which is present at manufacture, but which other portion(s) of the device blocked, neutralized, or hid. With time, because of the deterioration of those portion(s), their ability to block, neutralize, or hide the original flaw decreases, and device misbehavior caused by the originally present fault suddenly appears. This is the kind of history observed when P cells in the pulmonary veins, present when the heart is new, are eventually able to cause a-fib.

(2) It may involve a portion of the device which is composed of material which is not stable with time. As the material undergoes slow change, device performance is affected, but for a long time the change is unnoticed because device performance remains within design limits. But, eventually, the material changes enough to cause device performance to go outside of the design limits. This the kind of history observed when fibrosis of the atrial wall slowly progresses to the point where the degree and form of fibrosis creates the possibility of a reentry wave which initiates a-fib.

(3) It may involve a portion of the device which is susceptible to damage caused by some unexpected environmental condition. The damage could be temporary or permanent, but the environmental condition causes deterioration of one or more portions of the device, which causes device performance to go outside of design limits. This is the kind of history observed when a virus infects the heart, which can cause deterioration of one or more portions of the heart, resulting in the appearance of a-fib.

(4) It may involve the device being used in ways not anticipated in the original design study which set the device specifications and limits. For instance, the device could be operated at too high a temperature, or with too high a duty cycle, or with too little maintenance, any or all of which could cause unexpected forms and/or degrees of device deterioration. This resulting deterioration could cause device performance to go outside of design limits. This is the kind of history observed when the heart endures obsessive athletic excesses, resulting in deterioration of the heart, and the appearance of a-fib.

(5) It may be caused by an unanticipated cosmic event. This is the kind of history observed when someone who does not understand the definition of the word deterioration gets hit smartly with a rolled up web site, which could cause a-fib to suddenly appear.

Wil

Wil,

Device 1 is manufactured WITH a design flaw and quite early on in its projected lifetime fails to perform within normal parameters.

Device 2 is manufactured with NO such design flaw - or any other design flaws - which well into its projected lifetime goes on to fail to perform within normal parameters.

OK; perhaps both devices HAVE from a strict point of view (as per the definition of the word deterioration) failed as a result of deterioration. Surely, however, there are many different kinds of deterioration and the application of the word to both Devices 1 and 2 really does fail to reflect the overall situation in anything LIKE a satisfactory and illustrative manner.

Mike F.

I don't have any problem characterizing the misbehavior of any device as being caused by a group of nested hierarchical conditions and behaviors.

Such a nested hierarchy is formalized by some form of the usual upside down tree beginning with the "perfect" new device at the top. As the device progresses through its life it would follow numerous paths downward through the matrix of nested hierarchies corresponding to the multiple changes in the device's condition and the associated changes in its performance.

From a language standpoint we need to assign words so we can intelligently discuss this nested hierarchical matrix and its components. There should be a word assigned to reference the overall matrix, additional words assigned to reference each major component of the matrix, and further words used to describe subcomponents, etc.

You are objecting to my use of the word deterioration to describe the overall process formalized by the matrix.

If you don't like using the word deterioration to describe the overall process of changes in the device which cause it to misbehave during its lifetime then you are free to suggest some other word which we can use to represent the overall process.

You may trying to argue that your form of heart deterioration should not be viewed as deterioration because it didn't appear until the one third point in the heart's lifespan and was partially caused by a component present when the heart was new. To the engineer in me, your heart problem is characterized by deterioration of the type described in (1) above.

Wil

Had an ablation in Oct 06 -got rid of the afib, but plagued by PACs - used to get them before the ablation too. Situation complicated by myocarditis last year with increased arrhythmias. To cut a long story short - tried everything - mg, k, taurine, fish oils coq10 etc etc - all to no avail. Enter the humble local GP who was convinced the cause was anxiety - especially after 6 emergency visits to A&E last year. I thought he was wrong and told him so. In desperation I tried one of the 1 mg lorazepam tablets he prescribed last year (but which I never took). Hey presto the PACs have disappeared completely (at least for the last 48 hours). Before this I was getting hundreds and possibly thousands a day. I hope it works - and I feel more relaxed too. I'm not a pill popper - but if these work they get my vote. He told me they can only be a temporary solution because of addiction - but there are other medications. I'm going to post something about this on the BB too - it might help someone else.

Martin

Mike and all - Glad this topic is being examined more thoroughly. The question still in my mind about what or why the initial signal (PAC) is initiated in the first place. We seem to know the sequence of events and that ablation only stops the ectopics from becoming AF but what is the true source of the aberrant signal?

Is it a lack of enzyme or a mineral or some intracellular defect in a particular cell or neuron that is 'broken' like in an unravelled telomere (following the genetic link) that creates improper firing?

I don't pretend to be conversant in a discussion of telomere unravelling but I've heard numerous references and comments to that and about 'broken' genes to wonder if there may not be a link to AF. Some say some repair or even

prevention is possible; other not.

In my mind, the focus of why PACs occur at all has to go farther back to other origins...and that would seem to be an area of deep research that will not be of much current help to us....should it ever be researched at all.

Before my ablation, I had afib daily or every other day for almost a year while on the anti-arrhythmic, Flecainide. Then I did my heroics program attempting to cure myself while I waited six months for ablation. I managed to control the ectopics/afib to the extent I was totally AF free for a few months before ablation and virtually totally free of PACs after ablation - even off the post-procedure drugs. Then, I had the two AF breakthroughs.....so clearly, there are other origins not addressed by ablating the active drivers or potentials... or perhaps the ablation burns regenerate in time to the point that allow PACs to proceed through the whole process and bring about an AF event. (two separate issues)

Because afib is occurring in a much younger population - at least many posting on our BB are often much younger than what we saw formerly - I have to think there is some other connection which I why I think the focus needs to go back to both a gene becoming flawed and some biochemical deficiency that supports the a 'malfunction' of sorts.

My recent breakthrough of afib after three years post ablation certainly has me thinking about origins.

Jackie

Hi Jackie,

You might find the below interesting, given your recent developments.

The different mechanisms between late and very late recurrences of atrial fibrillation in patients undergoing a repeated catheter ablation.

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=16643390&dopt=Abstract

"The right atrial foci played an important role in the very late recurrence of AF, whereas the left atrial foci (the majority were PVs) were the major origin of the late recurrence of AF after the catheter ablation of paroxysmal AF."

Late recurrence post ablation was defined as about 3 months and very late was about 2 years.

Furthermore, the above is even more noteworthy, given your gender.

Predictors of non-pulmonary vein ectopic beats initiating paroxysmal atrial fibrillation implication for catheter ablation.

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=16168291&query_hl=1

"In a univariate analysis, only female gender was related to the presence of non-PV ($p = 0.016$) and SVC ectopic beats ($p = 0.012$)."

Regarding why PACs (increased in LAF) are initiated in the first place, it as appears that the culprit is mechanical stress, specifically intra-atrial pressure. AT1s, a measure of mechanical stress, are increased in LAF but not in normals. Why is that? They are not hypertensive. They generally have good ejection fraction. No detectable heart disease that might compromise contraction. But it's there. I think it's predominantly mild MR over time. The stretching does two things. 1) it upregulates the funny current, thereby increasing automaticity => more ectopics; 2) more natriuretic peptide production => urinary Mg wasting => more PACs.

Just my opinion.

PC

Jackie: As I suggested earlier, I think the presence of only two events, so widely spaced over three years, suggests that the outcome of the original ablation was ever so slightly marginal, and if you are careless about your health, a barely self sustaining a-fib can begin which is easy to squelch.

We all have trouble retaining our awareness of how fragile our lives have become. You acknowledged that you had been careless prior to the last episode. Were you careless prior to the first episode?

Wil

Wil - yes - both times I had not regularly taken as much magnesium or potassium as I had during the 'heroic' days... each time either too busy to be mindful or just negligent and both times, I had elevated stress levels. In retrospect, a bad combination.

I haven't reverted to old triggers or eating habits and in fact have refined even further a diet that is termed 'clean eating'.... whole foods, eliminated gluten/gliaden, mostly organic, cooked from scratch, no packaged food, no regular intake of alcohol... only an occasional glass of wine - one glass in six months.

It well could be that the first ablation was marginally successful and I'm hanging by a fragile thread. If that's the case, then I'll probably know sooner or later if a touch up is in order and hopefully, that will address any formerly undetected foci.

Time will tell. I'm not losing any sleep over the prospects. What will be will be.

Meanwhile, my heart remains calm again; no regular ectopy and only an occasional run of sinus tachy.

Jackie

Jackie,

"... both times I had not regularly taken as much magnesium or potassium as I had ..."

Your negligence leading to breakthroughs as well as Peggy's have served as a strong motivator for me. There have been a number of times when I've been already for sleep & remembered that I've forgotten to take my Mg, K & taurine. I lie there & think, "do I really need to get out of bed and go take those supplements." Then I think of breakthroughs and haul myself out & go do it. My last two breakthroughs also occurred during a period when I was negligent about replacing my stock of taurine.

George

George, with me it seems that i not only have to have reduced my intake of one supplement or another over a several day time period, but in order to get an afib episode out of that, i need to have ingested something severely nonpaleo. The Colonel's deep-fried delectables in one case, and a whole palette of christmas "goodies" in the other, starting with precooked spiral ham and continuing thru all kinds of wheat flour and sugar xmas treats, culminating with sugar-free cookies sweetened with i-don't-know-what artificial sweetener. The realization that i am comfortable in bed and have not taken my "meds" happens with some frequency, and i just drift off while thinking i really should go do that, and usually i get no trouble from this occasional lapse. Add some plastic food to the equation, though, and here comes trouble.

PeggyM

Jackie: Two widely spaced short episodes of easily squelched a-fib for a truly tiny percent of the total time since your ablation wouldn't seem like "I'm hanging by a fragile thread" to most a-fibbers.

Now stop goofing off and just do what needs to be done first ... keeping a-fib at bay is always the highest priority ... always ... then there will be time to do everything else ...

I don't want to be seen as being condescending. I know it isn't always easy. But, someone apparently has to give you a smack on the side of the head occasionally to help keep you on the straight and narrow path.

Wil

George - Yes - I've done that with nattokinase as well. I typically take the magnesium, potassium, taurine and NK at bedtime when I'm downstairs; but sometimes I'm already upstairs and forget. Then the subconscious kicks in and I, too, haul myself out of bed. I now have a stash both up and downstairs so I have no excuse - unless I'm totally brain-dead by the time I go to bed. ;)

Will - I meant the fragile thread was this balance of optimal electrolytes.... apparently, I can come depleted fairly rapidly tipping the balance and I vow to not let complacency set in again. It simply is not worth it.

(PS. No one who expects to remain breathing should ever consider smacking me on the head or elsewhere!) ;)

Peggy - I guess we all know our Achilles by now. Resisting temptation is the key.... started with that Apple ;)

Thanks all.

Jackie

Jackie: Just a parity check which may be redundant since you know yourself and afib so well but I'm wondering if you really had actual afib or a series of intense ectopics. A lot of people, me included, confused ectopics for fib and I may still.

As I remember, you send they were sending an event recorder to you but you've had nothing since. How long can you keep it? Would it be worthwhile to back off your supplements while you have the recorder if you don't get symptoms otherwise, just to check and see what's really going on in your chest?

Gordon

Hi Gordon - thanks for the input. No, I didn't receive an event recorder. No need yet. They usually send it for 2 - 3 months - depending on how many are available. They charge alot for the service so I would think the monitors pay for themselves with a small amount of 'lease' time for each patient.

I have no doubt that I was in afib. Had that too long to not recognize the beat, symptoms, etc.

But, if I have more activity, then I'll certainly request some type of monitoring system.

Meanwhile, my heart is calm and steady.

Jackie

Jackie

A comment from my very inexpert view...

1 In Bordeaux, after my recurrence and second ablation, Pr. H told me that he thought the AF might well have stopped by itself, but because I was from overseas he did the second one right away.

2 An EP in London once told me that ablation might not prevent AF from starting, but the lines should stop it from sustaining itself.

These two things suggest to me that an occasional breakthrough doesn't mean that the ablation has failed, but that the lines are doing their job.

Gill

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