

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

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SUBJECT: TERMINATION OF AN AFIB EPISODE

Background Information:

Some mechanism must be responsible for the spontaneous termination of a paroxysmal afib episode - without drugs or electrical cardioversion. What is this mechanism? If we knew, we might be able to take steps to achieve earlier termination.

Several possibilities have been discussed including changes in atrial natriuretic peptide (ANP) and brain natriuretic peptide (BNP) levels; changes in sodium, potassium, magnesium, and calcium levels; hydration, dehydration, exercise, acidosis, alkalosis, nitrogen oxide generation and I am sure some I have forgotten.

An obvious accompanying question is: "Are there different mechanisms involved in the termination of adrenergic vs vagal episodes?"

The purpose of this first session of the virtual LAF Conference Room is to brainstorm and debate the question: "What makes an afib episode stop?"

Hans

Responses

It just so happens that I've been giving considerable thought to this very question. So here's another gnat straining attempt. Hopefully some of you can give me feedback.

The AV node is still under autonomic control during AF and the ventricular rate will reflect the prevailing tone, higher for ALAFers and lower for VMAFers. Accordingly there may be different mechanisms involved in the termination of vagal vs adrenergic AF. But I know relatively little about ALAF.

My goal as a VMAFer is to increase sympathetic tone to wrest rate control from the ectopic atrial foci/agglomeration back to the SA node. At the start of an episode of AF ANP is released. This

inhibits both renin and aldosterone secretion and effectively lowers blood volume (hypovolemia) through the resulting diuresis. Na⁺ is secreted/excreted and K⁺ is absorbed. At some point this hypovolemia stimulates release of sufficient catecholamines to elevate the low blood pressure (I find that this happens sooner rather than later if one avoids all fluid intake after the AF is triggered). When that point is reached, the AF terminates. The accumulated K⁺ results in fewer PACs in the immediate post AF period. Thereafter increasing secretion of renin and aldosterone result in loss of K⁺ and gradual increase in PACs, unless K⁺ supplementation is undertaken.

PC, MD v54

IF we can ACCURATELY ascertain just what is going on in the heart and the rest of the body just prior to the termination of an a-fib episode, then we might not only be able to secure more rapid conversion to NSR, but may also discover some more clues as to what factors actually precipitate the a-fib episode in the first place (Another - albeit obvious - topic for this room?). Whilst PC has certainly already covered some of the mechanics of conversion to NSR better than can I, I can at least concur that I too have noticed (even though I have 'only' had 3 a-fib episodes to date) a marked reduction in PACs after a-fib episodes. Although the ANP secretion resulting from the atrial stretching during a-fib certainly and dramatically increases urination, I am unsure as to how much this urination effect actually/deliberately/specifically plays a role in conversion to NSR as opposed to 'simply' comprising a function to do no more than reduce blood pressure through fluid loss. When asked, my own cardiologist was unsure as to the role - if any - of ANP secretion in conversion to NSR.

Something absolutely DOES happen to bring about spontaneous conversion to NSR, and ANP secretion is the single most APPARENT (and known) response by the atria to a-fib that we have to work on/with at the present time. If one accepts that potassium re-balancing results in spontaneous conversion to NSR, and one accordingly makes the simplistic presumption that it is ONLY low potassium which brings about the a-fib in the first place (hugely unlikely?), then a-fib is surely quite a dramatic first indication of such a temporary deficiency! And what exactly brings about this temporary deficiency in the first place given that one's body contains enough potassium somewhere in its tissues for the re-balancing act to take place which results in conversion to NSR??!! Sorry if I am talking rubbish as a result of not quite yet having grasped all the detail of PC's detailed and excellent postings thus far..... but hey, I'm up for having a go.... I'm VERY keen to learn all that I can.

Mike F. v41

My one word answer would be "change". OK not very specific I know but I can point my finger towards a few things.

I'd like to half agree and half disagree with PC's comments. PC wrote:
"I'm a VMAFer and although I will go into AF when my rate is going too slow once I'm in AF my rate is always high. I'm not entirely convinced that the mechanism that keeps us in AF is the same that puts us there."

PC also wrote:
"I've managed to come out of AF early by upping my heart rate too. My average duration is around the 27-hour mark (mean of 125 episodes) but if I either jog up the stairs or eat a large meal at around the 18 hour mark I can sometimes terminate an episode. It happens pretty immediately so I think there's a definite cause/effect going on. I've managed to do it 4 out of my last 10 episodes (average duration for the last 10 episodes is 21 hours)."

So what changes?

As Hans points out in his book there are at least couple of conditions that must be met for AF to happen:

1. a triggering mechanism.
2. a heart in a state where it's both capable of being triggered AND
3. sustaining AF.

My guess as to what brings point 2 about is a change in refractory period (the resting period in which cells cannot be re-stimulated). Under normal conditions heart cells have sufficient refractory period so that even if triggered AF will not sustain itself. (An awful lot of people have ectopics and don't have AF and a lot of AFers have ectopics that don't always result in AF). Under abnormal conditions, when the refractory period is too short, once triggered AF is able to sustain itself by 'chasing its tail' (the refractory period is so short that cells will respond to restimulation). Once triggered AF can be self sustaining even if the initial trigger is no longer present.

I also guess that throughout my episodes the refractory period starts to lengthen again. At some key point, if I up my heart rate, I can persuade my heart to hit a 'refractory wall' - the rate is sufficiently high and the refractory period sufficiently long that AF can't sustain itself, the next beat from the SA node results in NSR. I should point out that my return to NSR is like flipping a switch, I'm not hoping in an out of AF at the end. Occasionally I do notice a run of ectopics that puts me back into NSR - very similar to the run of ectopics that puts me into AF. Given the longer refractory periods I can see why a triggering mechanism could also terminate AF (an early trigger would also hit the 'refractory wall').

What mechanism causes the refractory period to shorten to put the heart in a susceptible state?

If I only knew! I suppose there are several candidates from electrolyte imbalances to faulty ion channels to bunged up gap junctions! Perhaps my 'normal' state is to have a short refractory period?

What mechanism causes the refractory period to lengthen when I'm in AF?

I don't know! Perhaps it's simply exhaustion? Is it possible that all this rapid firing tires the cells out and lengthens the refractory period?

What's clear is that once I've returned to NSR I'm safe from AF for another 12 days or so. Has the AF managed to repair something? Does it take 12 days for my heart cells to recover from the last onslaught and once recovered is my 'natural' refractory period too short?

I've possibly oversimplified my answer, the heart is a 3 dimensional nightmare, the successful propagation of a heart beat is truly remarkable (I'm amazed AF isn't then norm!) Perhaps the termination of AF happens at some nice spot in this 3D landscape? Almost like a damn stopping the flow of water a line of refractory cells can stop the tail chasing? It's already known that some areas are good trigger points for AF, perhaps it's these points, or others, that are good place to terminate AF?

As ever, I'm better at coming up with questions than answers. Hope this triggers some good thoughts.

A big thank you to Hans for coming up with another excellent resource.

James D

I've stopped it twice by eating a pork chop (fried). First time, it stopped after the chop, started again during the salad (lettuce, green onion, garlic, fresh lemon juice & EV olive oil), stopped finally during the second pork chop.

William

Just a tidbit to add. I monitor my "heart rate" during episodes of AF with a Polar heart rate monitor, used by exercise fans to maintain aerobic fitness. The accuracy has been verified by my cardiologist listening to his stethoscope, negating a "false pulse" reading.

The point of my introduction is that I used to be able to terminate my episodes by vigorous exercise, but no longer. In fact, I have reached 200 bpm while playing tennis and waited for some random event to occur to revert to NSR.

I now take flecainide 2x daily and have had relatively few vagal episodes compared to my previous long-duration spells.

Martin

Dear Hans,

You are doing all of us A-Fibbers an immense service by writing your newsletter, by joining us together, and by dispensing information not readily available through traditional medicine.

My A-Fib began in 1996 following an aortic valve replacement for aortic stenosis at Cleveland Clinic. I could not be more complimentary of Cleveland Clinic or it's staff in helping me through this ordeal. A-Fib was controlled in hospital with cardio-version, and on discharge with Tenormin (50 mgm qd). A-Fib continued to be a problem throughout the years, occurring once or twice a month, lasting 6-18 hours, converting spontaneously.

Efforts by Cleveland Clinic to put me on coumadin met with resistance on my part, as did later efforts to have me seek radio ablation with Dr Natale (who is, in my opinion, a distinguished physician ahead of his time). Efforts to control A-Fib with Co-Enzyme Q-10 met with partial success, but it wasn't until June of 2002 that I felt pushed to the wall and in need of heroics. Heroics took the form of acupuncture three times a week for weeks, now down to once weekly, and a gradual settling on certain minerals/enzymes that have drastically reduced my A-Fib episodes. They now occur maybe once every six weeks, and are shorter and milder.

In addition to acupuncture, which works for reason that even I as a trained physician do not understand, I have evolved a group of minerals/enzymes that I think do forestall A-Fib. And I arrived at all of these by trial and error and over a period of time. The supplements I take are:

1. Co-Enzyme Q-10, 100 mgm every two hours (See Stephan Sinatra).
2. Acetyl-L-carnitine 500 mgm four times a day (see Bruce Ames dissertation on mitochondrial antioxidants in the October 2002 Discover magazine).
3. Alpha-lipoic acid 200 mgm four times a day (See Bruce Ames), and
4. Taurine 1000 mgm twice a day (See Life Extension Foundation).

I would rather have no A-Fib, but I can live comfortably at age 77 with diminishing A-Fib in incidence and duration, and I have comfort in that I have already consulted with Dr Natale at CC and can return to him if A-Fib does not stay in control.

Being a physician, although retired, has advantages, of course, in that I can think through situations and not rush to judgement about any given treatment.

Thank you again Hans for bringing us together.

Gordon

Wow! So much of this is way over my head, and I like to think I am pretty bright. I do not exactly understand the medical issues discussed, but I do have some anecdotal evidence concerning Afib. I am clearly a vagal afibber. Except on one occasion, my episodes have occurred in the evening, frequently if not always at the end of a stressful few days, and frequently associated with sitting on my butt and having several drinks at the end of my stressful time. My personal theory is that my normally slow HR slows down even more in the evenings anyway and that when I enter a "relaxed" state after a period of stress, my HR goes down, down, down and some mechanism tries to compensate by jumping it up a little. I get some PVCs and then BOOM, afib. Sometimes light exercise has terminated episodes, and my personal belief, without any reason or scientific evidence, is that if I can really get my rate up from something other than afib, the normal mechanisms take over and terminate the afib. But I actually have no clue. I am a life long very vigorous exerciser who is otherwise in extremely good cardiovascular condition. My age is 53 and my normal resting pulse is in the low to mid 40's. In afib, my HR shoots way up and my little exercise heart monitor looks very scary. BUT, I read on Hans' site about the benefits of magnesium and have been taking supplements for 7 or 8 months. Not coincidentally, I believe, I have not had an afib episode for almost 9 months. I started having them about 2 1/2 years ago and would sometimes go 3 or 4 months without an episode and then have several in a short time. I am not ready to declare myself cured, but I am sort of in remission, I think. Great idea to have this forum, Hans.

John B.

I agree with others that some of the technical jargon is a little tough to follow. In reading about some of the techniques discovered by various vagal afibbers to self convert I see some common threads and would like to add my own experience. With Vagal afib I believe most can find ways to help themselves by continuously trying to change something at the onset of an episode. Exercise immediately - jumping up and down to simulate jumping rope is the starting point. Do this for 15 minutes. It seems to shock the heart in some way and works for me about 75% of the time. If that doesn't do it then get on the bed or flood and raise your legs straight up in the air. Put your hands under your hips and push up to get your legs up and your feet back over your head and hold that position. It puts your body in an awkward position and somehow the heart in this position will convert back to normal rhythm. I just discovered this one and it has worked three straight times. As a last resort jump in the shower on warm and gradually reduce the temperature to cold making sure to cover your head and neck with cold water. Again this will put stress on the heart and it will convert. I hate this last one thus the last resort. I am happy to say that my episodes which now occur anywhere from 30 to 90 days apart are becoming less significant an issue in my life since I am in most cases converting in less than 10 minutes. Sometimes much less. I hope this is helpful. If any of you have any of the same experiences please let me know.

Terry

A great idea and great beginning topic.

I've always wondered what mechanism causes atria to go back to NSR. For example, if the source of the "rogue" electrical path is the pulmonary veins, does the rogue signal stop or at least

get weaker? In talking to the head nurse to Dr. Natale, she always sees strong electrical activity at the PV's (via the lasso catheter) before the ablation which halts this activity. If this errant pathway remains, perhaps the normal pathway regains its strength? If it hasn't been measured, I'm sure it should have.

If ANP is causing me to frequently urinate, something else must happen as sometimes frequent urination sometimes goes on with AFib for many hours! And hydrating does not seem to help, even with WWater. If it's hypovolemia as PC suggests than drinking water may even hurt!

As a mixed afibber type, I more often seem to have vagal episodes. If I'm in AF in the morning, exercise helps. Somewhat strenuous lifting, as a weight raised above my head often does the trick. Over-strenuous exercise does not.

I used to have success with bending over, and even built a crude tilt table to try to convert using the 10 min. regimens that the (Scandinavian?) doctors were experimenting with. Only worked the 1st time! I also had a couple of successes with Valsalva maneuvers (breath-hold and carotid massage) but no longer. But these maneuvers can help slow down the average HR, suggesting that the ANS still has a lot of control over overall rate.

Extending the refractory period, as James D suggests, of course is one of the goals of some meds which help terminate AF. Perhaps a closer look at the chemistry of that could help our understanding. In the end, it is liable to be several mechanisms that are at work to return us to "norm."

Unfortunately, time is a common converter and I've had several vagal episodes that occurred when lying down disappear after sleeping for a few hours.

Hope there is some contribution here.

Anton (M66)

Thank you for taking the time to read my post and commenting on it. I apologize for any unintended complexity. The topic and possible mechanisms are complex.

I think the timing of self conversion to NSR is very important. I believe that, at least for VMAFers, sympathetic tone is the primary determinant. This comes from several sources:

- 1) the growing hypovolemia (low blood volume secondary to ANP or atrial natriuretic peptide) with its concomitant drop in blood pressure, which causes epinephrine and norepinephrine release from the adrenals.
- 2) the diurnal variation in autonomic tone (higher sympathetic tone during the day and lower at night).
- 3) the physical activity at the time of conversion.

They all work in concert. That's why you VMAFers usually convert in the morning while walking up the stairs, strenuous lifting, etc. This lengthens the atrial effective refractory period (AERP) and blocks the propagating wavelets that allow perpetuation of AF.

A good test of this theory would be to see if one could shorten the duration of an episode of VMAF by taking a little salt at onset to help accelerate the ANP diuresis (remember no water). Do your usual AF busting routine upon arising in the AM.

PC, MD v54

I find extra beta blocker and 0 exertion helps to terminate my episodes. My doc says the beta blocker helps to suppress extra beats. My theory is that the extra beats trigger my afib, so reducing the number of them gives my heart a chance to get back to normal.

Trudy

For what it is worth, here is a non-technical, experiential reply (for the fun of it) ...

My afib comes only when I go from resting to activity too quickly... 90% of the time, this involves climbing stairs after having been at rest (in bed, watching TV, lying on a couch, etc.)

My afib almost never goes away while I'm sleeping or relaxing, or, indeed, when I'm strenuously exercising. It seems to go away when I'm at a normal level of alertness (e.g. having a conversation) ...

My pet theory is that there is a balance between my "erroneous" afib-inducing electrical signals (coming from my pulmonary veins) and my "normal" signals coming from my AV node. When the "normal" signals are foreground and the "erroneous" signals are background, my balance remains in normal rhythm. However, if the "erroneous" signals are given a chance to dominate, even for a short moment, they throw off the balance and an AF episode results. By the way, I am tall and have naturally low blood pressure so my "normal" signals can fall into a fairly "vulnerable" state when I'm at rest.

More specifically, I believe that when my normal signals are dormant (i.e. at rest), and then confronted with an "unexpected" blood-pressure and heartbeat increasing event, they are overshadowed by the "erroneous" signals and an event occurs.

Similarly, I think my afib goes away when my "normal signals" are dominant (e.g. mild activity) and the "erroneous" signals fall back into the "background".

I in turn wonder if all the discussion about linkages of episode termination to nutrition, etc. are false correlations since my afib comes and goes in a way which is directly correlated with activity levels (and changes therein) ...

By the way, for some afib comes during sleep - which seems to contradict my theory. However, who is to say a dream or nightmare (forgotten by the sleeper) doesn't cause the sudden elevation in heartbeat and blood pressure which causes this balance to fall out of whack.

I guess what I'm suggesting is to pay attention to activity levels, and changes therein, and their correlation to events starting and stopping. The states to consider are 1. weak, 2. medium, 3. strong, and 4. weak-to-strong. I'd be interested if a lot of people experience episodes starting in state #4 and going away in state #2 - regardless of other factors like nutrition.

Somebody on this board asked for a non-technical reply. Well, I hope this is taken in that spirit!!

Stuart

Stuart,

Your line of thinking is interesting and original, and being so is worthy of merit whether it is to any extent correct or not. I suppose the biggest problem with your assertion is that Hans and, I suspect, the vast majority of us here firmly believe that there are two distinct variations on the afib theme which are namely the vagally-mediated and adrenergically-mediated forms (with a third

form comprising a mixture of the two i.e. 'mixed' a-fib). Whilst I in some ways - and as I have said - quite like something about your line of thinking, your 'pet' theory does not seem - at least at first glance - to accommodate the two distinct types of a-fib. Furthermore, I have never noticed that a sudden increase in HR/BP precedes an arrhythmic event (usually PACs and occasionally a-fib) in my own case. Maybe this only applies to adrenergic a-fibbers (such as yourself from the tone of your post)? I do, however, quite like the idea that it is possible that the errant PV electrical signals are there all the time, but are 'out-gunned' by those from the AV node whilst one is in NSR. Assuming that one has a-fib originating in PVs, it is interesting to speculate whether the errant electrical activity is - as you say - there all the time but usually at lower levels (NSR), or whether such signals ONLY arise AT ALL during a-fib. Either way, the real question remains i.e. what is it that causes the imbalance which in turn either results in the PV signals 'out-gunning' the AV node OR which result in the PVs firing off in the first place.

Mike F.

Hi. I'll keep it simple!

I am 60 and have had VMAF for 11 years with increasing number of episodes and duration rising to an average 9 hours duration about every 10 days. Tried Sotalol and Verapamil, the latter helped rate control (no giddiness etc) but did not reduce incidence or duration. There was no way I could terminate an episode, it just had to run its course.

Four months ago I changed to Disopyramide and my cardio now tells me I no longer have AF just missed beats. These I can terminate easily by light exercise so this represents a huge improvement in my life! I think the incidence is also reducing as well as the duration (of missed beats). This would have been full AF prior to Disopyramide.

As to the mechanism - I believe that I have an endocrine problem and intend to have tests soon. Data recorded over several months showed an almost perfect sine curve (hours in AF against the 24 hour clock) with a peak at 19:00 and a minimum 12 hours earlier (copy sent to Hans). However, I also believe that this is not the whole story as there must be an additional trigger (or more than one). Stress is almost certainly one and digestive problems another (I was diagnosed as having GERD).

I think my most promising way forward is to check the endocrine thing as I don't really want to spend the rest of my life on Disopyramide despite the current benefits and very limited side effects (dry mouth).

Hope this is helpful!

Bill

Hi Bill, as Anton has already mentioned, extending the refractory period is one of the goals of some meds which help terminate AF.

A brief extract from disopyramide info have says this...

"These drugs [class Ia] decrease myocardial conduction velocity, excitability, and contractility by inhibiting the influx of sodium through "fast" channels of the myocardial cell membrane, thereby increasing the recovery period after repolarization. Disopyramide suppresses atrial flutter or fibrillation by increasing the effective refractory period and the action potential duration in the atria, ventricles, and His-Purkinje system. The effective refractory period is increased more than the action potential duration, so the myocardium remains refractory even after the resting

membrane potential has been restored. "

James D

Stuart: I think your post is valuable in that it is related to the differences in afibbers. For you at least, a very mild activity is most successful in terminating AF and for me, terminating my vagally induced AF is best done with exercise which is stronger than you advocate. Both of us are trying to achieve the "balance" that you mention which returns the atria to normal functioning. If we compare our similarities and our differences a bit more we might see why we need to tailor our exercise to the type of afibber we are.

You sound like your episodes are mostly adrenergic, yet with your low BP and your hint that some episodes occur during sleeping, it suggests vagal. Would you classify yourself as mixed? (See Hans's book for a good and understandable description of both along with the ANS which tries to achieve the body's/heart's inner "balance" or homeostasis) I'm mixed and probably have some features in common with you. (I'm M66, low BP & HR, low resting AF HR) I'll note that although you can't be sure your "erroneous" signals come from the PV's, you have accepted someone's technical opinion that over 80% of AF cases originate there. And if very mild to vigorous exercise terminates (vagal) episodes someone needs to find the underlying mechanism which could get increasingly technical. Terminating adrenergic episodes often suggests meditation, HR monitors, and other means of enhancing parasympathetic activity, but the mechanism needs explanation also because none of these "techniques" seem to have a high success rate. The "balance" you and other types of Afibbers seek may be the ANS balance, or homeostasis. But mustn't there be an added mechanism, because wouldn't we go into AF every time we exercise or every time we rest?

The next time I have a vagally mediated episode, I'll try PC's "salt" solution. But only after many such events will I be able to have an opinion on it's effectiveness. (I have a history of short term successes as do most afibbers)

Theme?-- It may be much better to know what stops AF than what starts it.

Anton

My system was so overloaded with toxins that I could not put anything on my skin, that I could not put in my mouth! I have been detoxifying my liver with homeopathics for 8 months now and it has diminished my afib episodes a great deal, it takes time, but I'm getting there.

I can terminate 4 out of 5 episodes with Nux Vomica 30C, 3 pellets under the tongue at the very onset of fib, sometimes it takes 3 more after 15 min. if it does not terminate it. At least it keeps the rate and rhythm down to where it is tolerable.

Ella (M 65 afib for 13 years)

PC, when you say the AV node is still under autonomic control during AF is of interest to me. The only way I've read about the AV node being affected by ANS is indirectly via the SA node and through the normal path. Playing devil's advocate for just a bit, if the rate control passes to the ectopic foci than the SA node is bypassed and would no longer be under autonomic control.(?) And even though it seems "logical", is it true that ventricular rate during AF is higher for ALAFers than VMAFers?

I agree with your premise that the ANS control is still at work during AF, even if inefficiently. When I am in AFib my resting rate is about 80 or slow. But exercise even mildly and I go to 150, but resting brings it back to the lower number. How do you think the ANS is still at work? i.e. Do the ectopic beats get faster or does the SA node put out more impulses or what? Since there are neural connections to the atria by the adrenergic branch, do the adrenergic "juices" (epinephrine, norepinephrine) stimulate the atrial's areas of irritability (ectopic foci) into more ectopic beats per sec? Or just stronger ectopic signal strength?

Anton

Stuart, just for the record I'm offering another "non-scientific experiential" reply - my experiences have been just as you describe. I'm most positively a vagal afibber, but my attacks at night have frequently begun when I just get up in the middle of the night from a sound sleep. They usually begin with a tachycardia which after about 10 -15 minutes degenerates into afib which I can sometimes stave off with 20 mg of a beta blocker which slows the heart rate enough so it doesn't progress any further. More than one began when I awakened from a frightening dream (like being in a burning building) or anxious about something and my heart was pounding. When it slowed down it clicked right into afib. At one time I awakened three times in one week from a dream where this happened. This was a pretty depressing discovery-hard to control triggers when they happen in a dream! I wonder how many vagal attacks may be triggered by stress or actions in a dream rather than slowed heart rate while sleeping or vagal tone.

Jamie

I think this approach from Hans to concentrate on one subject is fine. I am not prepared to deliver any scientific conclusions, but inform of my experience.

I am a man, age 67, reasonable fit with LAF of mainly vagal type. I have been in hospital a year ago with an episode >24 hrs. Converted to sinus after 10 hrs. with infusion of magnesium 30mmol/l over 8 hrs. Have since this made a log of all occurrences, which have been 33 times, lasting from 10 to 0,3 hrs. Up to 2 months ago they have all terminated by itself, while I tried to relax. I have now found that I can actively terminate the episodes by a brisk walk, or if it is too cold or rainy outside, take a run on the training bicycle. I have found that 10 minutes spinning on the bicycle make me out of breath, but also efficiently terminate the episodes. I woke up last night at 2 o'clock, and instead of laying in bed waiting for the episode to terminate, I spent 10 minutes on the bicycle, went to bed again and slept to the morning.

Odd

I have had ATF episodes, mostly brief (several seconds to a few minutes) but several of greater duration (20 minutes to about 2 hours) over the past 5 years or so. All episodes have started at night while lying in bed.

The role of posture (and increased vagal tone) in kicking off each episode is clear. Likewise, most episodes have been terminated simply by getting up out of bed and walking around or sitting upright in a chair and relaxing.

My impression is that remaining active while upright brings quicker termination than simply sitting upright and relaxing. I have tried walking around the block for perhaps 10 minutes during a longer episode; this did not terminate the ATF. I am inclined to believe that strenuous activity would have done so.

Two two-hour episodes (in recent months) were terminated in an emergency room setting with injections of a calcium-channel blocker, diltiazem.

I should say that prevention is the key and look forward to adding my two-cents' worth to a discussion of triggers and underlying causes.

Mike Ferro

Fellow fibbers,

Posting on a topic nearly a week old is like trying to sell a house that's been on the market too long and has become "stale". Nonetheless ...Please be forewarned that this is a technical post. If you plan to read it and are unfamiliar with many of the terms bandied about on this forum, please have your medical dictionaries at the ready. I decided to post in the conference room, because I wanted to more directly address my previous post on termination. I apologize in advance if some of what follows deviates from the topic at hand (AF termination).

Many of you no doubt have wondered how a biochemical approach can ever explain a condition that appears to be curable via a surgical procedure (ablation or Maze). I've wondered the same. There are many levels on which AF can be analyzed. I like the biochemical/physiological level. Many on this forum like the ambient state approach and look at the physical activity and/or food intake in the time just before and around onset and termination. Others, especially cardiologists, like to look at the phenomenon from an electrophysiological approach. Each has its advantages and offers clues that may provide insight for the other approaches. However, sometimes I get the impression that we are talking about three different diseases, not just LAF. My most recent post on Mg++ on the regular BB incorporates elements of all three. If you read it first, some of what follows may be a bit easier to digest (without precipitating an episode of LAF). I would like to attempt to explain the above mentioned biochemical/surgical paradox as well as submit another possible mechanism for termination of LAF.

A number of responses on the conference topic of termination have only confirmed my own impression that many episodes (other than those occurring during sleep or stress) begin and end because of "sudden" change in autonomic tone and hence atrial refractory period change.

- I. For a VMAFer increased vagal tone will shorten the AERP through opening of acetylcholine-dependent potassium channels. This is the first required ingredient for AF. This is aggravated by Mg++ deficiency and I suspect that taurine is instrumental in preventing this. Increased levels of intracellular Ca++ (the most significant Mg++ antagonist) are known to activate calcium dependent potassium channels and also contribute to atrial refractoriness.
- II. Inhomogeneous distribution of vagal nerve endings will increase the spatial dispersion of refractoriness. Low K+ will also potentiate spatial dispersion. Increased spatial dispersion is nothing more than fancy talk to say that the newly created wavelets (see below) will multiply (not propagate) more effectively. This is the second required ingredient.
- III. The final required ingredient is several consecutive PACs or even tachycardia to start the AF ball rolling. No one really knows why PACs occur, but some feel that they are

a manifestation of increased intracellular Ca^{++} . Radiofrequency ablation and the Maze procedure can be successful because these procedures remove PACs from the list of ingredients required for LAF. I personally think that the prime determinant of atrial dispersion is genetic. This is the “defective substrate” of LAF, not damage caused by excessive exercise. It is my belief that our (LAFers) hearts are wired autonomically in such a way that nerve impulses are more readily dispersed. This is usually not a problem until you combine nutritional deficiency with this genetic predisposition. My father and his brother died of strokes secondary to atrial fibrillation in their 70's and 80's. My mother was recently diagnosed with AF in her late 70's.

Initiation of AF is explained by the wavelet theory (Moe 1959), which has been proven electrophysiologically. Wavelets (mini waves) are not PACs and do not result in a detectable atrial contraction or a propagating wave. They are all very small and disorganized. Individual wavelets can breakup, fuse or collide with each other and wavelets disappear when they meet refractory tissue. If there is less refractory tissue, as in shortened AERP, then they are less likely to disappear. From time to time a varying number of wavelets are present in any atria (LAFer or “normal”) and the duration of each individual wavelet lasts only several hundredths of a millisecond.

Furthermore, it has been shown that the number of wavelets that fit into the atria determines the onset and perpetuation of AF. Below a critical number of wavelets there is a considerable chance for the wavelets to die out all at the same time. When more than 6 independent wavelets are present, self sustaining AF is created. Six wavelets equates to critical mass for AF. The number of wavelets that fit into the atria also depends on the atrial refractory period, conduction velocity and atrial mass. That is one of the reasons why atrial dilatation, one complication of prolonged AF, is bad news (it can accommodate more wavelets and more easily trigger an episode of AF - AF begets AF). LAF patients in NSR have decreased conduction velocity and shortened AERP (these both translate to more wavelets) vs controls without LAF.

Now let's change hats and go from electrophysiology to biochemistry/physiology. I have been thinking a bit about my previously posted suggestion to shorten the duration of an AF episode. If you'll recall I suggested that you might curtail water intake (creates hypovolemia during the ANP diuresis) to stimulate sympathetic activity and take a little salt to accelerate the action of ANP (helps increase intracellular K^+). This is not exactly what is generally recommended in patients with heart problems, especially hypertension. Accordingly I would like to forward the following additional mechanism for termination of LAF. I haven't been holding out on you, just thinking and trying to connect the dots. I believe the previously posted mechanism is very much in play. It's just hard to know the relative magnitude of each in the LAF ballet. Within 15 minutes of onset of AF intracellular Na^+ but especially Ca^{++} increase rapidly. As previously stated this further shortens AERP and allows those wavelets to multiply further. In addition AF increases O_2 demand threefold. Although atrial blood flow increases, this is insufficient to meet the rising O_2 demand and intracellular pH begins to drop. The atrial cells attempt to address this through the K/H but especially the Na/H exchangers, at least initially. There are membrane pumps (require ATP and energy), exchangers (passive and non-ATP dependent) and channels (? kind of intermediate between pumps and exchangers). In an attempt to lower intracellular Ca^{++} , the Na/Ca exchanger springs into play. So as AF progresses after the initial increase in Ca^{++} there is an additional increase in intracellular Na^+ (to lower intracellular Ca^{++} and H^+) and K^+ (to lower intracellular H^+). The ANP meanwhile is excreting extracellular Na^{++} and thereby helping to alleviate the increased intracellular Na^+ by creating a greater concentration gradient. As this Na^+ gradient increases, the K/H exchanger takes over. At some point the increasing level of intracellular K^+ is such that atrial dispersion is decreased. When this happens, the wavelets have more trouble sustaining critical mass. Functional AERP increases just before spontaneous conversion of AF. Judging from your posted comments on spontaneous conversion, this latter appears to be associated with increased sympathetic tone. I've not read about anybody terminating an episode of at least several hours duration by taking a nap and awakening in NSR.

So increasing sympathetic activity in the face of strong vagal tone appears to cause lengthening of the refractory period. This would put the final nail in the AF coffin. The increased intracellular K⁺ and decreased Ca⁺⁺ at termination would help explain the dearth of PACs in the immediate post AF period. So it would appear from this mechanism that the best tonic for shorter duration of AF would be to help (not hinder) ANP by hydrating (not dehydrating). O.J (great source of K⁺) would probably be a good candidate. WW would also be good in view of the local metabolic acidosis. So I think you ought to forget about the salt thing. I just like to think out loud as I wander through the AF maze.

PC, MD v54

To answer a question which has been posed, my episodes never come when I'm sleeping - only if I stand up too quickly and, e.g., climb stairs.

Based on the other posts I've read, my continuing observation is that there seems to be a lot of evidence that, at least for some (a lot) of people, activity levels are highly associated with events - both starting and stopping them. It also seems that events "run their course". Therefore, at least for some of us, I'm not sure I buy into nutrition as being related to termination.

I mentioned that events typically go away when I'm in an "involved", but not "exercising" state. However, I must admit I have had episodes terminate during walking and mild yard work. This links up with what several of you have reported on "mild exercise" ... Personally, I am going to pay more attention to that idea, i.e. to experiment more with finding the right kind of activity level which is associated with episode termination (I will try a brisk thirty minute walk). For me, this will be the "ah ha" I take away from this particular discussion thread.

Please understand, I am not downplaying what others are saying - e.g. about nutrition. I am just trying to sort out what on the board relates to my own personal experiences. And it seems activity levels (and changes therein) are not only the key for me, but at least a good number of other people as well.'

And, as I reported earlier, I take 4mg of Procanabid at episode onset and continue to do so every twelve hours thereafter until termination (which is twice my normal dose) - so, it may be a combination of drugs and activity levels which cause my reversion.

I will add one thing. I definitely control my personal onset by paying attention to avoiding sudden changes in activity levels. I have had this condition long enough (six years), and regularly enough (every few months) that I am almost positive I have been able to limit the occurrence of episodes by avoiding sudden increases in heart rate and blood pressure.

One other note. When an episode arises, I can sometimes shake it immediately by walking around the room or from room to room. This only works occasionally, but I mention it because it again seems to tie into activity levels and other comments on this board

Stuart

One of the distinguishing features of vagal vs adrenergic is the timing of the start of the episodes. Is it possible that the timing of termination also shows a distinctly different pattern for vagal vs adrenergic? Finding such a difference in pattern would lend weight to the argument that the termination mechanisms are different.

I am an adrenergic afibber of 13 years standing and have kept records of the start of my episodes and their duration so it is fairly easy to figure out their termination time. Here is the distribution of start and termination times for my latest 50 episodes:

START

15:00-17:00 - 34%
09:00-11:00 - 14%
01:00-03:00 - 12%
17:00-19:00 - 12%
11:00-13:00 - 0%

09:00-15:00 - 16%

TERMINATION

15:00-17:00 - 16%
09:00-11:00 - 28%
01:00-03:00 - 4%
17:00-19:00 - 6%
11:00-13:00 - 12%

09:00-15:00 - 52%

It would seem that the period 15:00-17:00 (3-5 PM) is the most likely time for an episode to start while the period 09:00-11:00 (9-11 AM) is the most likely for an episode to terminate. It is interesting that none of the 50 episodes started between 11:00 and 13:00 and that only one started between 13:00 and 15:00. No episodes terminated between 19:00 and 23:00.

Considerably more data is obviously needed before any conclusions can be drawn, but certainly the peak times for starting and terminating an episode would seem to be different. It would be interesting to see if other adrenergic afibbers have a similar pattern and if vagal and mixed afibbers have a different pattern.

Traditional Chinese medicine divides the 24 hours of the day into 12 segments each corresponding to a certain meridian. The heart meridian peaks between 11:00 and 13:00 (my least vulnerable period) while the spleen meridian peaks between 09:00 and 11:00 (best time for conversion). Does this mean anything? I have no idea!

Hans 70a

For me, although there is a very definite peak for going into AF I'd say it's less obvious to spot a good time to come out of it.

Sorry Hans - I forgot to split the times the Chinese way when I did the number crunching but here are my figures for the 85 episodes that I have good data on.

I'm vagally triggered and quite cyclical (the same 85 episodes have an average gap of 15 days with a stddev of 6)

(85 episodes)

START

00:00-02:00 - 12%

02:00-04:00 - 8%
04:00-06:00 - 7%
06:00-08:00 - 6%
08:00-10:00 - 2%
10:00-12:00 - 0%
12:00-14:00 - 1%
14:00-16:00 - 1%
16:00-18:00 - 4%
18:00-20:00 - 31%
20:00-22:00 - 20%
22:00-24:00 - 8%

TERMINATION

00:00-02:00 - 6%
02:00-04:00 - 4%
04:00-06:00 - 6%
06:00-08:00 - 4%
08:00-10:00 - 5%
10:00-12:00 - 11%
12:00-14:00 - 15%
14:00-16:00 - 9%
16:00-18:00 - 11%
18:00-20:00 - 13%
20:00-22:00 - 12%
22:00-24:00 - 6%

Clearly, I'm unlikely to go into AF between 8am and 4pm (actually more like 9am-5pm). It may turn out that this same time is also the good time for coming out of AF but I'd like to see more numbers before I'm convinced.

James D

I have been tested for magnesium levels, and am low end of normal. I have been taking supplements. However, isn't magnesium absorbed very slowly by the body? It so, I cannot see how it would trigger a termination event.

Stuart

PC,

Very interesting stuff indeed. (And not confusing at all.) At the risk of stating the obvious:

FACT: Most members of the general population experience PACs. Furthermore, a great many individuals who experience frequent PACs DO NOT get a-fib.

QUESTION: What is it that is different about the hearts of a-fibbers which enables ectopic foci to quite readily instigate a-fib as opposed to just PACs?? i.e. what predisposes a heart to a-fib wavelets? Are we back to ANS/electrolyte etc. imbalances again albeit seen from a slightly different angle?

Mike F.

Stuart,

Interesting ideas. Given that changes of activity levels do not themselves per se trigger a-fib in members of the wider population, it must surely be imbalances in the automaticity/inappropriately rapid shifts in automaticity which accompany such changes in activity levels (as you suggest) which results in a-fib (or, more specifically, the deterioration of PACs into a-fib via whatever mechanism (the holy grail?) it is whereby the atria can so readily (and unfortunately for us here) accommodate the propagation of a-fib wavelets).

I experience many PACs each day, but thankfully they have only thus far in 3 years resulted in a-fib 3 times. I can on each of the 3 occasions think of a possible change in activity levels as you suggest:

- First time Oct 99 when I awoke into a-fib from sleep - I was under great stress at the time and was having a lot of anxiety dreams. Could a dream have triggered the activity change? I certainly think so, given that I awoke out of a nightmare in the early am a few weeks ago to find my heart all over the place with all sorts of PACs/runs of PACs. Some deep breathing, and the PACs subsided and I returned to sleep. Interestingly, my psychotherapist had previously aired his view - unprompted - that my night-time a-fibs may be related to anxiety dreams/nightmares.
- Second time May 02 when submerging myself in a VERY hot late evening bath (I had also been drinking HEAVILY during the previous 48hrs, and had had a LARGE latish-evening carbo meal).
- Third time Nov 02 when I awoke with a-fib.... been dreaming again? Not sure, but I'm sure we must all forget many more dreams than we remember.

Mike F.

PC,

Based on your recent post, and combining other comments on this thread, how is this for a theory?...

The source of the "erroneous" signals is always putting out signals, they are just (under stable conditions) trumped by the "correct" signals.

However, under certain conditions (triggered by changes in activity levels), the "erroneous" signals are amplified, temporarily, more than the "correct" signals. The result is onset of AF, because the "erroneous" signals now trump the "correct" signals. The AF node is then, as your post indicates, "on its own" until it can recover. Its ability to recover, as well as the fact the "correct" signal can be trumped in the first place, depends on physiology (which might be impacted by, e.g., nutrition, heredity, mg++, etc.). However, termination may also be assisted by returning to an activity state where the "erroneous" signals no longer trump the "correct" signals.

For an analogy, I imagine a top spinning on a table. One fan is blowing up from underneath, which provides a stabilizing force. Another fan is blowing at a right angle. As long as the

strength of the upward-blowing fan is greater than the force of the right-angle fan, the top continues to spin normally. However, if the electricity to both fans is suddenly changed, and the right-angle fan reacts to that change with more of a burst of power than the upward-fan, then the top wobbles. If both fans immediately, then, return to normal mode (where the upward-fan trumps the right-angle fan), the top wavers for a while until its spin returns to vertical (i.e., the episode plays itself out, and normalcy returns). However, this is more likely to happen more quickly if the right-angle fan is blowing at its weakest strength relative to the upward-fan. That is what I call the "balance point", and that "balance point" may be different for each individual - which is why termination occurs for some at different activity levels than for others.

I know this explanation and analogy sound simplistic, however based on everything I have read and my own experience, it is a model that, to me, seems to have some possibilities.

The implication is to analyze activity levels (and changes therein) to determine their correlation with both onset and termination events - remembering that during sleep heart rate "activity levels" could change due to dreams, etc. I suppose an analysis of heart monitor data just prior to onset and just prior to termination could help settle the question. I do not know how much of that data is available, though. I, for one, have never been lucky enough to have a heart monitor on at either onset or termination.

Stuart

First PC thanks for your knowledge and effort. I've been thinking about your reply to my post and if I understand your concept, the SA node goes out of (or is knocked out of) commission, the atrials are quivering uselessly, and the remainder of the heart's components are left as ventricle pacemakers to react to the ANS inputs. (AV junction, Bundle of His, etc) Since these backup pacemakers aren't as reliable as the SA node, they produce irregular rhythm. (Do I understand it about right?)

First, one small point, if one takes the AV junction tissue (located just below the main AV node pathways and can serve as a backup pacemaker) as being separated from the AV node, then it can be said that "The AV node is the only part of the heart that cannot initiate impulses". (Guide to ECG Analysis, Catalano) Now I have also read that the SA node has a tendency to stop producing impulses when an ectopic rate is faster than the node's inherent rate. Be that as it may, I've been under the impression that the AV node is receiving these ectopics and is responsible for the ventricle's irregular behavior. So isolating areas that produce these errant signals will block their signals from arriving at the AV node receiver. (Hence maze and ablation successes)

So confused, I turned back to several sources (Catalano, Rawlings, my collection of notes) and found that two theories sort of evolved from Moe's multiple wavelet theory and from ectopic focus theory by Engelmann, and they roughly differed the way the above paragraphs differ. Now I've gone to one of the authors, a Professor/Lecturer and he generously added some interpretation. Not surprisingly, I've more questions than he had answers so I remain confused. But without quoting him (which I didn't ask) I'll just summarize most of it and let you comment:

=====
Most references nowadays offer some kind of reentrant activity in "explaining" fibrillation. The current Merck manual, for example, touches on the poetic: ". . . atria are in a seemingly chaotic rapid rhythm, produced by multiple interlacing wavelets of reentrant activity."

Simple, "circus reentry" around, say, an infarction, is an inadequate explanation for the chaos. The Merck wording nicely alludes to "multiple" areas of reentry, creating a kind of electric pandemonium because of the "interlacing." The spasms (for lack of a better word) result from the

uncoordinated depolarizations of many cells in the tissue, as if each of those cells were trying to be the pacemaker at practically the same time.

The electrical misbehavior of the atria should not cause the AV node to "go haywire," but a disease that caused the atrial fibrillation might do so.

.... Only some of the invaders can trigger the AV, so the ventricular rate is [graciously] much lower than the atrial rate during atrial fibrillation. Refractoriness of the AV cells has obvious merit in the situation.....

.... The word fibrillation refers to a peculiar twitching of fibrils, which are very small fibers of muscle. Cardiac fibrils normally contract so that the overall, coordinated effect is rhythmic contraction of the atria and ventricles. In fibrillation, the fibrils twitch unproductively; coordination is absent. The quivering atria fail to fill the ventricles as much as before, so cardiac output diminishes. The ventricular rate can be so high that inadequate filling further reduces the cardiac output.

..... It is, indeed, a complex question, and I am not satisfied that research has yet provided the fundamental answer. Exactly how a disease (or chemical disorder, etc.) can cause atrial cells to become chaotic remains in debate.

Electrical shock (synchronous cardioversion) can convert atrial fibrillation back to sinus rhythm: the fact is demonstrable, but the fundamental reason is still mysterious. We have a lot to learn . .

=====

Both theories were "proven" with demonstrations, and other authors suggest that both might be true; that is there is a wavelet AF and a focal AF type which may require different treatments! Then others re-classified AF as category I,II or III [Konings et al] which does nothing so far but add to confusion! When I visited Cleveland Clinic in August, Dr. Natale was not concerned with classification and they had abandoned the use of their fancy equipment (during his PV ablations) for searching out and measuring focal points. This would just rely on the statistics of isolating the usual focal points, which brings it to about 85% success in isolating the problem areas. (Their quoted 95% on the second try is perhaps questionable) With all this confusion I'm not surprised the source I've summarized above leaves the fundamental reason for conversion to NSR as an unknown. It almost makes me feel better! :->

PC: One last note-- Put me down as one who has terminated AF during sleep. Not often, but several times from events starting during sleep and events starting before bedtime. It's odd, and I'm not proud of it--:>)) -

Anton

Since October, 2002, when I first got this condition (Afib), I have briefly terminated my rapid heart rate and spiky afib condition for relatively short periods (one hour at a time) during my most recent biofeedback sessions. I have been practising biofeedback for one hour once a week since November 14th and can report I am making progress--the last two weeks showed much better, calmer rhythm plus a slowing down of the heart rate to the mid 70s in today's session. In fact, the last two sessions showed no afib. Of course, I relax very deeply during the sessions, but I revert back when I get home--however, the improvement seems to be gradually taking hold. I am also practising deep breathing and relaxation at home with tapes & CDs.

Also taking every possible supplement that I've read about on this website plus some standard meds (including Lanoxin, Cardizem, Vasotec, and have just switched from Coumadin to aspirin

[since I figure if there is any inflammation present, aspirin would help that as well as prevent blood clots]).

I wear a Polar watch to monitor my actual condition as I never ever feel any "episodes". It seems to be more of a persistent, chronic condition. I also track my heart rate on the computer with the software program Freeze Framer, but I have not yet mastered their mental "Freeze-Framing" technique, so I cannot report much progress there. But at least I get a chart of the heart rate in real time. I think the type of Afib that I am demonstrating is adrenergic--(although my cardiologist looks at me like he's never heard of such a word). I think it goes away at night (not sure). The heart rate calms down when I sit still and read or work on the computer and goes up quickly when I move around or exercise. The inception and prolongation of the afib was very tied up with extreme stress.

Elena

Hi Stuart,

The heart works on a 'first to fire wins' basis. Under normal circumstances its the SA node that wins this race - it's the first to fire and sets off a nice cascade. However all heart cells are capable of firing without any nerve stimulus. If for some reason the SA node doesn't fire (or is late in firing) some other cell further down stream will fire and set the cascade off from there (this is and ectopic beat). An ectopic can also happen (and probably happens most often?) when one of these cells, away from the SA node, decides to fire too soon.

So, I think your comment "The source of the "erroneous" signals is always putting out signals, they are just (under stable conditions) trumped by the "correct" signals." is true in that cells down stream of the SA node are always ready to fire if they don't receive a signal after a certain time has elapsed.

It's worth noting that the cells timers are reset each time they are fired, if a cell fails on one beat then it's an ideal candidate to produce an ectopic before the next beat.

I believe that just as rogue ectopics are capable of triggering AF they are also capable of terminating it. By firing at odd times they are acting like a very localized defibrillators, if they can persuade enough cells around them to get into time then NSR can be the happy result. (if an ectopic manages to fire enough cells then there's a period when they are not available for the meandering wavelets of AF to fire them).

Now the tricky question, is it the SA nodes fault for not firing quickly enough or the ectopics cells fault for firing too quickly or a cell not firing on one beat winning the battle the next time around?

I wouldn't be surprised if it's all 3! I'm vagally triggered and there are definitely times when my SA node is going too slow (like a 2 second pause between beats) and it's really very easy to spot these ectopics on my watch output (Polar S810) and I'm grateful for them! (This probably only happen once every few months). On most occasions it's much less obvious, it's the relative timings that are important - the SA node should always have a slightly higher frequency than other cells so it wins the race regardless of the underlying rate.

I should also point out that reasoning above is all related to the triggering mechanism that start/stop AF. The other problem of course is that our hearts so readily sustain AF when triggered. I believe that although you can induce AF in a normal healthy heart our hearts are particularly easy to induce.

Fix EITHER the triggering mechanism (the ectopics) or the substrate (the short refractory period / fast conduction rates) then AF is no longer a problem.

So easy to say - so hard to do!

All the best

--

James D

Hi Stuart,

Yes your conclusion certainly makes sense to me. Any localised disturbance/imbalance could be all that is required to alter these relative timings.

You can throw the relative times out either by shortening the ectopics cells firing time OR by lengthening the SA nodes firing time. So I think we have to be careful when pointing the finger at the ectopic site (they may well be working normally). I think it's fair to say that most often the ectopic site is too keen to fire but I'm also certain that sometimes the SA node is simply too slow.

--

James D

Hi Mike,

I think with any kind of AF it's the relative rates that count. If the SA node is wanting to go at 170 bpm but some other cells are wanting to go at 175bpm then they'll win the race and produce ectopic beats.

To extend the argument a little further... We all know that the SA node is not beating at constant intervals. Rate increases and decreases according to demand. As well as the SA node all the other cells need to still make sure their 'self fire time' remains longer than the SA nodes. It's my view that the most vulnerable times are when the heart is changing rate. (perhaps accelerating for AMAFers and decelerating for VMAFers??? but don't hold me to this :)

And just to highlight my thoughts, I've actually just gone into AF when my rate was 135bpm (but I still think it was a vagal problem)... I've been having ectopics the last few nights, which is a sure sign that AF is looming. This evening I was averaging around 60bpm and feeling like AF was about to appear, I was pretty fed up so I jumped on the exercise bike for five minutes. I was doing perfectly ok and kept my rate between 130 and 145 bpm (I'm not on any meds in NSR so my heart rates are sensible when I exercise). I made sure to do some slow pedalling towards the end of the five minutes. Less than 5 seconds after getting off the bike I popped into AF.

So what happened?

My guess is that my SA node was decelerating at a greater speed than other bits of the heart - as soon as this happens the opportunity for an ectopic is increased and (combined with the fairly short refractory periods because I was exercising) AF was not really much of a surprise. Although I'm unable to exercise because of my AF I still think it's a vagal problem. For me, AF always appears in the resting period after exercise rather than when my rate is increasing.

Is it as simple as VMAFers have too much acetylcholine at localised spots in the heart and AMAFers have too much norepinephrine at other areas? I really don't know. I do know it's a good idea for the SA node to always win the race!

The trick now is to get out of AF by finding that refractory wall, I know it's there somewhere :)

All the best

--

James D

Hi Mike,

Sorry if I implied all my episodes terminate with PACs, they don't (I'm guessing only between 10 and 15 % do). I think the crucial thing about the termination of an episode is the lengthening of the refractory period. When the refractory period is long enough - AF is no longer able to sustain itself.

I believe that PACs or an increase in rate can both terminate episodes slightly quicker than would naturally happen (by causing cells to fire quick enough or at peculiar times to put a refractory barrier in the way of AF)

--

James D (18 hours into AF and getting ready for a few experiments!)

My background rates before I go into AF are often low of the 8 AFs I've managed to capture on my watch. Two were against a background of between 70-80 bpm, 3 were against a background between 60-70bpm, 2 were against a background of below 50 bpm, and 1 (when I was exercising) was around 135bpm. The slow rates tend to have high RLX numbers (30-40+ms).

It is very common in the 5 minutes before I go into AF for my rate to increase and RLX to drop.(and I believe Hans has mentioned some research that has spotted this for vagal AFers for the 15 minutes before AF. Hans, I can't find the reference so if I'm not imagining it could you repost the ref please). There are some odd undulations as my rate yo-yos up and down for no apparent reason. Many of them (4 so far) contain sharp drops in rate immediately before I pop into AF. Once I'm in AF my rates are always high and RLX low.

Well my last one terminated through vagal activity, I've also had success in the past with exercise (increase in sympathetic tone). You can toss a coin as to which one will work (and sometimes neither work).

Surely this is a matter of timing? If the ectopic (or the SA node) beats when the cells around it are ready to fire then they will send a wave outwards that will compete with the underlying AF. I believe it's precisely this action that terminates AF early. (The AF wavelets meet refractory cells from the ectopic/SA node and have nowhere to go.)

Thanks for the refs.

<http://www.ub.rug.nl/eldoc/dis/medicine/b.j.j.m.brundel/c1.pdf> is a new one to me, I'm printing it out as I type this)

--

James D

James D,

The reference to the 15 minutes is on page 20 of The Book.

Hans

Hi PC,

I agree that it's the long AERP that is important to the termination of AF. I still believe though that anything that can introduce change in the atria when the AERP is long enough can terminate the episode (including PACs and a sympathetic jolt)

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12450601&dopt=Abstract

P.S.

Although my RLX is very often above 40ms before I go into AF as soon as I'm in it drops into the 10-15 ms range. (One of the reasons I believe a different mechanism kicks in when I go into AF). I tried the exercise bike on and off for 20 minutes but without success. I found success again in eating a large meal (came out just around the time I was starting to feel full). Duration this time was 21.5 hours (last years average was 26.8).

James D.

James,

When you say:

"I think it's fair to say that most often the ectopic site is too keen to fire but I'm also certain that sometimes the SA node is simply too slow."

Then this could explain VMAF (with typically slow resting HRs/occurring at rest), but what about AMAF?

Mike F.

James,

Thanks for the clarification. One other thing: You feel in your own case at least that a PAC may actually result in conversion of your a-fib episodes to NSR. I would have thought this unlikely for the following reasons. A-fib episodes are more refractory to spontaneous conversion the closer they are to their commencement, and given the dearth of PACs after an episode it is not unreasonable to assume that PACs DIMINISH as an episode goes on for longer and longer? Although I suppose a PAC occurring later during an episode would have a better chance of triggering conversion as the AERP is starting to lengthen? I suppose what I'm trying to say is that surely PACs are more numerous during the early stages of an episode but the episode is nevertheless highly refractory to conversion to NSR during the same early stage of an a-fib episode.

PC,

Thanks also for your further thought-provoking post. The input from yourself, James D. and others is excellent, and given that I am unable to produce anything better myself, I am accordingly reduced to trying to make a meaningful contribution by trying to critically pick at your posts!!

Whilst being very impressed with the logistical analysis of your above post, I remain a little surprised at the emphasis which you are currently placing on the importance of Mg++ as it is estimated that 85% of the US population are magnesium deficient - the vast majority of which do not get a-fib. I do not for one minute doubt the importance of Mg++ to the human body (and mind), but I'm not sure that all a-fibbers get a-fib because they are drastically deficient in Mg++, although it is, of course, entirely possible that very strict supplementation of Mg++ (as you propose for yourself) can substantially improve episode frequency, duration, and severity. I have myself been diagnosed with mild magnesium deficiency further to a retention test, but whilst my heart may wish it, I doubt that rectifying the situation will remove my predisposition towards a-fibbing. As regards calcium, I was a little disappointed to learn that my intracellular levels (RBC) were right at the low end of the normal range, since I had 'hoped' that the thousands of Ca CO₃-laden indigestion remedies I have consumed over the last 15 yrs would have resulted in this having become elevated to the point of being potentially problematic.

Mike F.

PC,

Thanks very much for the response. Notwithstanding my comments which you addressed so well, I am in overall agreement that optimising one's mag supplementation can only be beneficial as regards reducing episode frequency, duration, and symptom severity.

The dialogue between yourself and James is very interesting as regards HRV at episode onset and termination..... almost to the extent where you've got me thinking about getting a Polar S810!! However, I am in my own case and by my own admission (and diagnosed with OCD) neurotic and obsessed with my health quite enough as it is already (according to friends and loved ones). Also, with just 3 episodes in 3 years to date, I am at the present time too infrequent-a-fibber to get much benefit from a monitor? Thinking about it though, I do get lots of PACs and short runs of PACs/SVT. It is only - luckily for me - very occasionally that these deteriorate into a-fib. I wonder if some a-fibbers out there on the other hand get very little ectopic activity which however when it does present more often than not deteriorates into a-fib? Where would you place yourself on such a scale?? It would be interesting to have such info for all a-fibbers here.....

Mike F.

PC,

Don't seem to be able to e-mail you direct, so I'll post here. You asked on the conference board if I could refer you to anything interesting which I might discover pertaining to PACs and a-fib. SO here are a few abstracts for you (and anyone else interested... Jerry? Anton? James? etc.) to have a quick look at:

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12062732&dopt=Abstract

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=11797990&dopt=Abstract

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=11797990&dopt=Abstract

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=11797990&dopt=Abstract

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=11227964&dopt=Abstract

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=11139935&dopt=Abstract

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=10562264&dopt=Abstract

PC, you'll especially like:

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

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=10681806&dopt=Abstract

Although much of the conclusions of the above are reiterating what many of us already suspect to be the case, it's nice to see it in black and white. And hopefully one or two new bits in there somewhere too. Happy reading - only abstracts, so won't take long.....leaving plenty of time to get outside for a Sunday stroll (-:

Mike F.

James D.,

You say "it is the relative timings that are important" ... That is where my observations link up with your electrophysiological explanation. If activity levels, or changes therein, alter this RELATIVE timing (i.e. one frequency is impacted in a different way than the other), even if only momentarily, then a trigger has occurred and these "wavelets" (which sound to me like feedback or turbulence) bring the whole timing system down. Similarly, if a certain balance of activity levels restores proper relative timing, things can return to normal....

Does this conclusion make sense? Just trying to reach an insight before our thread runs out ...

Stuart

James,

Sorry to hear your attempt to derail a looming episode was unsuccessful. But I think many are coming to appreciate that it's the transition times when you are at risk for AF. I also think it's a transition point, perhaps something as simple as standing up, that causes us to convert to NSR.

This is much more difficult to appreciate at the very time it occurs because it is so subtle (vs onset). Your (and Stuart's) thoughts on the EP mechanics surrounding termination are very close to what's going on. I can't tell you all how great it is to see this kind of in depth discussion. I wish I could shed more light on those mechanics. I'm not a cardio but have studied the EP at some length and posted much of that on 1/6/03. But I will give Mike's question a go.

Mike,

What causes our LAF hearts to go into AF on occasion at the hands of PACs, when the "normal" heart in the population at large has many PACs as well but no AF? Factors that are necessary for AF, according to the textbooks and recent articles include:

First and foremost, a shortened AERP. Second, increased atrial dispersion (enhanced spreading of the wavelets). Third, increased PACs to trigger the episode. Conduction velocity is also in there somewhere, but I believe it's included under dispersion. (perhaps in addition to being Mg⁺⁺ deficient the LAFer heart is wired a little differently from birth in such a way that dispersion of neuromuscular impulses is increased. I know that K⁺ deficiency can also increase dispersion). As James says, I think the weak link in all this is the AERP and getting it to lengthen in some way in order to decrease the number of wavelets (if a wavelet encounters another cell that is now refractory it disappears). It is a fact that vagal tone and sympathetic tone can cause a shortening of the AERP. Judging from what most are posting on circumstances surrounding onset and termination of AF, shortening of the AERP via vagal tone appears to be the culprit responsible for onset of most episodes of AF and lengthening of AERP via sympathetic tone the culprit responsible for termination of most cases of AF. Why autonomic tone does this is certainly a mystery to me. But it appears to do this at the transition from sympathetic to vagal going into AF and from vagal to sympathetic coming out of AF. There appears to be an over-response of each at these transition points. I personally feel that this over-response on both ends is due in most cases to Mg deficiency. The neurotransmitter substance for the PNS is normally broken down by an enzyme named cholinesterase. This neurotransmitter substance normally has a half life of a minute or two. Mg⁺⁺ is required for this enzyme to do its job. If there's insufficient Mg⁺⁺, the neurotransmitter substance lasts longer and works its effect for a longer period of time, i.e., increased vagal tone. If you are doing something that causes release of norepinephrine (the neurotransmitter substance for the SNS) like running or even just walking (?having a bad dream) and then quickly reverse course to something like sitting down, lying down or even just bending over, the PNS applies the breaks more forcefully (vs if you were just standing and then sat down). This means that more of this neurotransmitter substance would be released and the vagal tone would be more prolonged and pronounced, if you were Mg deficient. This would provide lots of shortening of the AERP. Many experts think that excessive intracellular Ca⁺⁺ is what causes PACs. Could it really be excessive Ca⁺⁺ relative to Mg⁺⁺? It also turns out that the neurotransmitter substance for the SNS is removed after secretion from the nerve ending by an enzyme (its called COMT) that also requires Mg⁺⁺. So theoretically the pre AF critical shortening of the AERP could also come during transition from vagal to sympathetic tone. However, there's another enzyme called MAO that also breaks down and removes the SNS neurotransmitter substance that might fill this COMT shortfall. Perhaps the reason we all have relatively predictable durations of AF before converting to NSR is because the over-responding neurotransmitter substance continues to be released during AF. At some point this supply is exhausted and a period of time is required for the enzyme to remove this large pool of neurotransmitter substance (from the synaptic space at the end of the nerve). The time frame should be about the same during each episode. Termination would occur when the SNS tone exceeded the PNS tone, which was high at the start of AF but slowly dropped during the episode. Transition to NSR would be accompanied by a sharp drop in HRV as the tone changed from vagal to sympathetic. I've actually seen this on my Polar S810 at the end of an episode.

Erling,

Your level of expertise on the electrophysiology is such that I certainly can't answer most of your questions. But I certainly agree with your thinking as posted on the regular BB. I've attempted to explain some of this in my response to Anton above on 1/7/03 and the earlier post on 1/6/03. The atria are not richly innervated but the SA node is connected to the AV node by a neural pathway. Cardiac muscle has the innate ability to pace and propagate a wave without neural input.

<http://www.laras-lair.com/ecg.html>

Now that you are all thoroughly confused, I'll stop.

PC, MD v54

Mike,

Your point is well taken and I don't for a minute mean to imply that Mg++ is all that separates us LAFers from the rest of society in NSR, Mg++ deficient though they may be.

It takes many years before a slightly negative balance on Mg++ intake vs excretion becomes clinically evident (lots of Mg in the bone that's available and our ability to absorb deteriorates slowly). Everybody has a slightly different diet, different absorption rates, etc. They also have differing exposures to drugs, processed foods, etc that can accelerate this time frame. Our diet has only slowly deteriorated. So for the most part LAFers are going to be diagnosed from the late 40's on. Most of these are VMAFers and I believe that many of these have accelerated the process with excessive exercise. LAFers with ALAF are on the average older. The very process of aging is one of accumulated damage to all our organs, not just the heart. Where ALAF becomes just AF cannot effectively be delineated by our present approach to cardiovascular disease. Dr. Lam (www.lammd.com and his book) has a very good discussion on this. I spoke with my mother yesterday. Several months ago she'd been diagnosed with LAF. Now she has a left bundle branch block by EKG. This is not Mg++ deficiency. This is a manifestation of CV disease. Yet the initial workup didn't uncover any evidence of disease. Was it there at the time of the workup? Of course it was. Just because your doctor says there's no evidence of disease doesn't mean that you are disease free. Hence your classification as LAF is always suspect to some degree.

So my answer to your question about the rest of the population vs us LAFers and Mg++ deficiency is that:

- 1) I'm only talking about true LAF and that really means VMAF (because they're younger and would otherwise have less likelihood of significant CV disease). Autopsies on 20+ year olds during the Korean War showed many to have early signs of atherosclerosis in their coronary arteries.
- 2) There are MANY more "normals" running around out there with VMAF and have no idea whatsoever.
- 3) I think VMAF is a problem for us because so many of us were born with high vagal tone and the Mg++ has enhanced this tone and with it VMAF.
- 4) Not everyone with Mg++ deficiency expresses it as VMAF. Many others have migraines, allergies, etc. I have none of these. Do you? They may be asking the same question that you're asking except with migraines, allergies, etc.
- 5) I also think that our substrate (hearts) not only beat more slowly but also are wired a little differently and in such a way that atrial dispersion is enhanced. After all we've just had a few posts underscoring the genetic nature to AF, at least on some level.

I really don't know the answer. But I can only address that which is within my power to address and Mg++ is definitely within that realm.

PC, MD v54

James,

The only real way in my opinion to terminate an episode is by lengthening the refractory period to form a barrier as you put it. But there are no PACs. In fact there's nothing going on in the atria during AF except one big storm of wavelets. This lengthening of the AERP is precisely what terminates the episode. The medical literature states this and I've seen it happen on my Polar S810, using HRV as inversely proportional to and proxy for AERP. How and why this happens is a mystery to me. I wish I knew what happens to the HRV during termination of AF in an ALAFer. If it's the same thing, and I think it is, then Hans is probably right. Once the heart goes into AF (VMAF or ALAF), the same mechanism (lengthening of the AERP through increasing sympathetic tone) is at work to terminate the episode. It just takes awhile before the vagal tone drops to a point where the heart is susceptible to a sympathetic jolt to effect termination. It seems this has to be a jolt and not sustained sympathetic pressure. Get on your bike for a few minutes. Get off and back on, etc. And watch your RLX. If it drops below 40 when you're sitting, the AF is ripe for termination. I once terminated an episode three hours after onset by playing close attention to the RLX while sitting.

Hope you lengthen your AERP forthwith.

PC, MD v54

James,

Congratulations and thanks for the reference.

Your entry into AF is exactly the opposite of mine. My RLX (a direct measure of HRV or heart rate variability) goes up and the bpm go down. Then come the PACs and then AF. This would suggest that the heart in AF knows whether it's of vagal or adrenergic origin. Your episodes are terminated through vagal activity and mine through sympathetic.

I agree with you insofar as anything that can sufficiently lengthen AERP will terminate AF. But during AF PACs suffer the same fate as any impulse from the SA node. The originating focus is surrounded by wavelets and a veritable electrical storm of refractory tissue. Nothing can go in and nothing can come out.

<http://www.ub.rug.nl/eldoc/dis/medicine/b.j.j.m.brundel/c1.pdf> and
<http://www.escardio.org/scinfo/Guidelines/summary.pdf> (see pathophysiological mechanisms)

It sure would be nice to know the RLX and HR data for others as they go into and out of AF. Have you noticed your present pattern to be the norm or have you gone into AF when your RLX rose and your HR dropped?

Thanks.

PC, MD v54

Mike,

That's an excellent question. I think the incidence of PACs is proportional to vagal tone. I've always known from even before medical school that I had more than the usual number of PACs. If you find a study on this, please let me know.

Anton,

Your understanding in paragraph 1 is quite correct. However, after that You and James and many others I'm sure are hooked on ectopic activity during AF. Although it has been shown that one atrium can be in tachycardia and the other in AF at the same time, there is no ectopic activity in an atrium after initiation of AF. The atria are a mass of reentrant wavelets. Impulses are still coming down the pike from the brainstem ordering the SA node to generate an impulse, but the message cannot be acted upon because all atrial tissue is essentially refractory because of these wavelets. The PACs are only important insofar as they can initiate AF, as can tachycardia. The following web page explains some of this: <http://www.ipej.org/0201/anfinsen.htm>

And the following web page should answer all your pacemaker type questions:
<http://jan.ucc.nau.edu/~daa/heartlung/lectures/ekg3.html>

Discussion of the EP mechanism at the below sites are good:
<http://www.escardio.org/scinfo/Guidelines/summary.pdf> p 1235
<http://www.ub.rug.nl/eldoc/dis/medicine/b.j.j.m.brundel/c1.pdf>

The former brings out the fact that both automaticity (pacing) and reentry are required for the Moe theory (now accepted as fact). The number of wavelets that are present at any time is dependent on the refractory period, conduction velocity and mass in different parts of the atria. The formula wavelength = conduction velocity x refractory period indicates the minimal circumference (wavelength of the circle) each electrical impulse must travel to avoid reaching its origin (reentrant means this is a circle or closed circuit) before this area is again excitable. When refractoriness is short and conduction velocity is slow, e.g., excess vagal tone, wavelength is also short. In this situation more reentry circles (circuits) may exist simultaneously in the atria, since they are smaller and the atrium can accommodate more. Six reentry circles are associated with stable atrial fibrillation, while a situation with fewer reentry circles either converts to sinus rhythm, or degenerates into more reentry circles. These reentrant circuits (wavelet) can cause AF through breakup of the wavefront (the normal wave of atrial contraction before onset of AF). Individual wavelets can brake-up, fuse or collide with each other and wavelets disappear when they reach the border of the atria or meet refractory tissue. From time to time a varying number of wavelets are present in the atria and the duration of each individual wavelet lasts only several hundreds of a millisecond. And I certainly don't understand the mechanisms behind the three different variants on the Moe theory.

I would be very much interested in hearing if anyone else has terminated an episode of AF during sleep as has Anton.

This discussion on the electrophysiology of AF is all fine and dandy, but the problem is the shortened AERP and how to lengthen it.

PC, MD v54

James,

You may find the following article interesting wrt the differing modes of onset for our respective flavors of AF. I think I've had three or four episodes of AF (from well over a hundred) that arose

from what must have been from a background of dropping RLX and increasing heart. I would be most interested to know if you can terminate the latter type with a vagal manoeuvre (like eating) and can terminate the former with a sympathetic manoeuvre. You had mentioned that it was "a toss of the coin as to which one will work (and sometimes neither work)".

Pacing Clin Electrophysiol 1999 May;22(5):743-9 Analysis of heart rate variability five minutes before the onset of paroxysmal atrial fibrillation.
Fioranelli M, Piccoli M, Mileto GM, Sgreccia F, Azzolini P, Risa MP, Francardelli RL, Venturini E, Puglisi A. Division of Cardiology, Fatebenefratelli Hospital, Rome, Italy. fioramas@mbox.vol.it
BACKGROUND: Various experimental and clinical observations suggest changes in sympathetic and vagal neural regulatory mechanisms play a critical role in altering cardiac electrical properties and favor the occurrence of arrhythmic events. There is limited information about the influences of the autonomic tone on the development of episodes of paroxysmal atrial fibrillation in patients with no evidence of organic heart disease. The aim of this study was to investigate changes in sympatho-vagal balance 5 minutes before the onset of atrial fibrillation. METHODS: We evaluated 28 patients with no history of heart disease who were not undergoing pharmacological treatment and who had at least one episode of paroxysmal atrial fibrillation recorded during an 24-hour ECG Holter monitoring. We analyzed values of frequency domain heart rate variability parameters 5 minutes before the onset of atrial fibrillation (preaf period) compared to an equivalent period at least 1 hour after from atrial fibrillation (random period). RESULTS: Thirty-six episodes of atrial fibrillation were recorded and our results showed we had two types of episodes. Eighteen were classified as Type A, in which we had an increase of low frequency (LF) (79.15 +/- 10.76 in comparison with 62.64 +/- 19.55) (P = 0.004) and a decrease of high frequency (HF) (20.82 +/- 10.74 in comparison with 37.64 +/- 20.20) (P = 0.004) consistent with an increase of sympathetic tone; and 18 were classified as Type B in which there was a decrease of LF (62.82 +/- 15.38 in comparison with 85.97 +/- 8.48) (P < 0.001), and an increase of HF (36.79 +/- 14.72 compared with 14.01 +/- 8.48) (P < 0.001), consistent with an increase of parasympathetic tone. CONCLUSION: We observed abrupt changes in sympathovagal balance in the last 5 minutes preceding an episode of atrial fibrillation. This can be related to a double behavior in the Neurogenic drive: in Type A episodes there is an increase of the LF spectrum, LF:HF ratio, and a decrease of the HF spectrum consistent with an increase of neurogenic sympathetic drive; in Type B episodes there is a reduction of the LF spectrum, LF/HF ratio, and an increase of HF spectrum consistent with an enhancement of the neurogenic parasympathetic drive. In some patients, we found that the two mechanisms operate during different hours of the day and that sometimes there is an increase of sympathetic tone, and in the same instances an increase of parasympathetic tone. Heart-rate variability measures fluctuation in autonomic inputs to the heart rather than the mean level of autonomic impulse; autonomic imbalance is probably more important than the vagal or sympathetic drive alone.

Also, I would be most interested in your opinion of the following article wrt whether you've noticed any difference in duration of episodes according to HR just prior to onset.

Pacing Clin Electrophysiol 1998 Jan;21(1 Pt 2):181-5 A relationship between fluctuations in heart rate and the duration of subsequent episodes of atrial fibrillation.
Hnatkova K, Waktare JE, Sopher SM, Murgatroyd FD, Baiyan X, Camm AJ, Malik M. Department of Cardiological Sciences, St. George's Hospital Medical School, London, UK.
A relationship between autonomic tone and the onset of paroxysmal atrial fibrillation in some patients is recognised. Episodes of PAF may vary enormously in duration, however, from a few beats to many hours. Whether autonomic tone influences the duration of the episodes has been less well investigated. From a database of Holter recording taken from patients with symptomatic PAF, we identified all episodes of at least 30 seconds duration which were preceded by noise free sinus rhythm. This study examined the heart rate prior to AF onset, the change in heart rate over the final minute of sinus rhythm and the time of AF onset, and compared the data from those episodes of AF of more than 5 minutes duration to the shorter ones. Heart rate was slower before long episodes of AF, but this was found to predominantly represent data from separate

recordings. A highly significant rise in heart rate was detected prior to long AF episodes compared to shorter ones. Daytime AF episodes were slightly longer than nocturnal ones. The most important finding was that longer AF episodes were typified by a heart rate acceleration. This suggests that, regardless of underlying aetiology, an increase in sympathetic tone may be important in the sustenance of episodes of PAF.

These articles suggest, at least to me, that there are different mechanism at work during termination of AF, depending on whether the episode was adrenergic vs vagal.

PC, MD v54

I have had numerous episodes of afib stop during sleep, or deep relaxation. When my episodes occur at home, or at night, I often take half of a .125 mg triazolam (Halcion) tablet to help me relax. I listen to music with a steady slow pulse and if I sleep, I usually wake in NSR. My episodes are generally preceded by a rapid pulse (too much exercise, hot shower), and I feel they will terminate when I calm down. However, I have less fear now then I used to have, and I don't like the side effects of a tranquilizer during the day, so I often skip it and just calm myself with deep slow abdominal breathing. But, or and, I have also found that slow walking or light eating has coincided with the end of an episode. My episodes are shorter since starting the Waller water, and I feel less chaos in my chest.

Thanks to this website, I feel calmer about the whole thing, less isolated, and definitely more educated.

Thanks to all of you,
Sadja

Much to comprehend about the electrical impulses that trigger and control our heart rhythm and its regularity. Many thanks to those of you who have moved the discussion ahead exponentially.

I have a simple thought or two, and I'd like to share the ideas with you, hoping for input, regardless of depth. Item A is largely intended to address electrical issues, and Item B is intended to address chemical/ionic issues.

- A) If the heart is, as we know, an organ subject to electrical impulses, then why can't we find a way to comprehend the schema of signals coming to and from the heart, so as to comprehend how each and all affect cardiac rhythm? I know the apparent answer is: "That's what we're trying to do, Jerry, hence all this discussion." Agreed. I don't want to appear either dense or simplistic, but I'm thinking about chaotic signals coming to the heart, and also I'm thinking about where those signals come from, and how they can be organized. If we were a group of electricians developing the wiring for a complex building or mechanism, we would want to build a central electrical station of some kind, a central unit (like the CPU of a computer) into which ALL signals could be sent and their frequencies organized prior to being re-directed to their appropriate place, for the appropriate purposes. So, if the signals coming to our hearts are in any way allowed to become disorganized or depolarized, or whatever you want to call it, hence allowing for various dysrhythmias (including a-fib) to occur, why can't we find either a chemical or an electrical or a mechanical bio-CPU that would accept and ultimately regulate cardiac signals? Isn't that essentially what a pacemaker (electrical) or a maze/ablation procedure (mechanical) would do?

- B) I have long asserted on the main BB that the ANS is at the causative "heart" of our a-fib disorder, and that the chemical correction of ANS imbalances would, in large measure, take us toward a cure. So much of what has been discussed in the erudite posts within this Conference Center has dealt with Vagal and SNS/PNS tone, etc, and it seems to my layman's mind that, if we could balance/organize the ANS and the biochemical information it transmits, we might move toward a cure.

From PC's post, above: "CONCLUSION: We observed abrupt changes in sympathovagal balance in the last 5 minutes preceding an episode of atrial fibrillation. This can be related to a double behavior in the Neurogenic drive: in Type A episodes there is an increase of the LF spectrum, LF:HF ratio, and a decrease of the HF spectrum consistent with an increase of neurogenic sympathetic drive; in Type B episodes there is a reduction of the LF spectrum, LF/HF ratio, and an increase of HF spectrum consistent with an enhancement of the neurogenic parasympathetic drive. In some patients, we found that the two mechanisms operate during different hours of the day and that sometimes there is an increase of sympathetic tone, and in the same instances an increase of parasympathetic tone. Heart-rate variability measures fluctuation in autonomic inputs to the heart rather than the mean level of autonomic impulse; autonomic imbalance is probably more important than the vagal or sympathetic drive alone."

If such changes in tone can be seen prior to and after periods of AF, as the quoted study found, then why can't we discern which type of AF occurs in which type of person, then find ways to balance or correct the tone in those patients, so as to prevent or ameliorate the AF they experience? Wouldn't it be similar to giving prophylactic doses of certain meds to patients known to suffer certain disorders, thus preventing the appearance of symptoms?

There is a physician in New York City, a man named Rodriguez, I believe, whose practice focuses in large measure on the balancing of the ANS, especially in cancer patients. At first blush, it might sound suspicious, but what I read tells me there may be something to it. If there is merit to the theory that the ANS is the mechanism that can be manipulated biochemically so as to address issues such as AF, then let's have at it.

Am I being ridiculously simplistic here, or is there a way to think about this problem in more direct terms, even within the context of traditional medicine?

Jerry

PC

I think I need to start taking better logs (up until now I've only been keeping a log of how I go into AF but there's clearly a good argument for keeping logs of how AF is terminated too). One of the jobs I want to this week is to extract 10-15 minutes of data preceding AF out of the polar software and have a look at it using the HRV analysis software from Kuopio University (http://it.uku.fi/biosignal/hrv_standalone.shtml) so I can have a closer look at the LF/HF. I know the polar software does this but, frankly, I'm not very confident it does it well (it will be interesting to compare the two sets of numbers!)

I'm confident that the duration of my AF is not affected by the triggering mechanism (I'm much less confident that there is more than one triggering mechanism!). Out of the 126 episodes I've had this is the data that I'm reasonably confident about what my heart was doing before I went into AF slower rates (100bpm) average duration 25.0 hours (stdev 7.1) (17 episodes). You'll have a hard time convincing me the small discrepancy isn't explained by the sample sizes.

I'll report any interesting findings that turn up when I extract the 15 minutes run up to AF.

You also wrote:

I don't keep good enough logs but a ball park figure of between 5 and 10% of my AFs terminate when I'm asleep. I'd guess at least another 50+% terminate when I'm trying to get to sleep. Of course, it's much harder to say how many of these would have terminated earlier if I'd tried the exercise route. (24+hours of AF with little or no sleep didn't really put me in the mood for jumping on my exercise bike. Now that I know it can help I try it a lot more often!)

--

James D

Jerry,

Item A. - because the heart is so damn clever :)

The heart is simultaneously been told to slow down from one input, speed up from another input and look after itself in the absence of any inputs. Not to mention the 3d mesh of interconnectivity which relays information down (or up!) stream with it's own peculiar timing system. I think the major headache here is that the heart cells are capable of self firing without any external stimulation and once one cell fires it cascades to those around it. (so you would need one of your CPUs for each and every cell).

A pacemaker will only prevent your heart from going too slowly. So if 100% of your AFs occur when you are going too slow then a pacemaker is a good option. (I've actually been given this option but I'm still likely to go into AF once every 4-6 weeks which isn't a good enough improvement for me).

The PV ablation tries to stop the cells that self fire at the wrong time from cascading to other parts of the heart. (so if you can ablate around all the faulty cells you can fix your AF)

The maze (and some ablations) let the faulty cells fire but try to organise the way in which they cascade. They channel the cascade in such a way that although ectopics can still happen they are unlikely to degenerate into AF.

Electric cardioversion essentially turns all your cells off. If the SA node is the next thing to fire there's a good chance the heart will restart in NSR (but if the ERP is still short there's a good chance that chaos will resume)

The pharmaceutical approach tries to mess with the timing signals to maintain/regain order.

P.S. to Item A - because I can't wrap my head around a good electrical analogy I often plump for a mechanical one, so at the risk of complicating the issue....

Imagine a straight line of dominoes running from left to right. Pick any domino you like (preferably the one labelled SA node!) and cleave it in two. One half falls to the left and the other to the right, the dominoes cascade in that wonderful pattern. Until you've stood all the dominoes back up (the refractory period) you can't start the process again.

As anyone who has played with dominoes will know - if you are not careful when you are standing the dominoes back up you can knock one over and trigger an early cascade (an ectopic!). It's worth noting that this cascade will stop as soon as it meets some dominoes that have already fallen over (the refractory wall). The heart is the 3D version of this!(oh, and it also has some clever dominoes that take varying amounts of time to fall over and stand back up!)

Item B

I think it's really important to consider both the triggering mechanism AND the qualities of the heart that allow it to go into AF. The heart has to be in a state to sustain AF BEFORE the trigger happens. Given that there is an enormous number of people who have similar rates to ours and don't have AF. Also given that there are an even greater number of people who have ectopics and don't have AF I can't help thinking that we should ignore the ANS and fix the substrate (ok I know this is tantamount to heresy on this BB and I do realise the ANS plays a part in the substrate but I'm trying to stir people into thinking about the problem in other ways :) I also realise that some triggers (like multiple quick ectopics) could be capable of triggering AF in a normal heart so perhaps some AFers only have a faulty triggering mechanism and perfectly normal substrate?? (ouch!)

I'd love to know how many people they could observe these changes in that don't have AF.

Because it's not a black and white problem, how do you distinguish the bad changes in tone from the desired ones? I don't believe many (if any) of these changes in tone are considered to be outside the normal range. Fight and flight /rest and digest response in most people (and in most AFers most of the time) do not lead to AF.

Most complicated problems are solved by breaking them down into smaller, simpler ones. The annoying thing for us is that the heart is enormously complicated and the simpler breakdowns that science has provided so far have yet to contain the solution to AF. (but I think they're a lot closer than 10 years ago and an awful lot closer than 50 years ago). Perhaps the birth of Internet bulletin boards is just what science needs to fix the problem!

After writing all this I felt the tone was a little pessimistic, sorry I didn't mean it to be. If I didn't think there's a better solution out there I would have plumped for a pacemaker or ablation quite some time ago.

--

James D

Hi Mike,

Although I think its safe to say the EARP will be shorter (http://www.clevelandclinic.org/heartcenter/pub/atrial_fibrillation/AFresearch.htm#atrial_electrical) sadly it isn't the whole story,

<http://www.oucom.ohiou.edu/cvphysiology/A004.htm> describes how the SA node doesn't really have a resting potential. (green graph on the page)

I believe this slow rise (depolarisation) is called the pacemaker potential. When the pacemaker potential reaches a certain threshold the action potential is triggered.

Other myocytes are supposed to have a flat (or flatter) resting potential but I believe the gradient of this part of the curve can play an important role (along with short EARP) in initiating an ectopic.

I think it's one of these sliding scale problems. Could you have a short enough refractory period to induce an ectopic but a long enough one so AF is not sustainable? As the EARP gets even shorter there comes a point when AF is sustainable.

The other thing that helps AF is the conduction velocity. Conduction happens much quicker along the long axis of a cell (I think its to do with the greater number of gap junctions at the ends) and is

much slower perpendicular to the long axis (then dominoes don't fall at the same speed in 2D!). This difference in conduction speed (and the presence of large muscle bundles) makes it much easier for these wavelets to propagate. Add to this some myocardial discontinuities (like those that naturally occur up the pulmonary veins) or those produced by fibrosis /dilation and it really is a miracle that AF isn't the norm.

Instead of the straight line of dominoes imagine a circle of them – knock one over and the cascade starts. If you can stand the dominoes back up before the cascade reaches them again you have a wavelet.

--

James D

Brilliant, James. Absolutely brilliant. I love the dominoes image. Well done.

You took each of my issues, and you addressed them in scientific terms, even as you equated my concerns to your own considerations of AF generally, and of those particular issues specifically. Not at all pessimistic, by the way. I think it right to say that you were quite realistic in your assessments, and I'm glad. Threw a bit of needed cold water in the face of my rather simplistic optimism! Just what the world needs...a bit of balance. Wouldn't it be nice if the heart and ANS were as amenable?

Let's keep prodding ourselves, and we might just nudge the scientific threshold an inch or two closer to the cure.

As Sundance said in the movie:

"Keep thinking, Butch. That's what you're good at."

Jerry

James,

Thanks for the great post.

I've really done my best in carefully reading and trying to take on board all of the content of all the excellent posts above. Here is my take for what it is worth. Please let me know if I've got it wrong and if so where.

Let's start with a regular normal heart. Almost everyone in the general population gets the occasional ectopic now and again. An ectopic beat results when a cardiac myocyte (substrate) fires before the SA node: the EARP of that myocyte is too short/shorter than is required to delay the myocyte from firing before the SA node. I don't doubt for a second that the ANS (and/or the endocrinological system) plays a role in shortening the EARP of that myocyte which triggers a PAC: it appears that it is subtle and fast-changing imbalances between the SNS and the PNS that are involved in this regard. It is safe to say that many people get a lot of ectopic activity, but do not get a-fib: I have for some years had a LOT of ectopy (Usually 20-100 PACs and one or two short runs of the same/SVT per day on average), but have 'only' had 3 episodes of VMAF to date (during the last 3 years - although I'm sure I had a short burst of a-fib (2mins) back in 95).

The key question for me is: why does an ectopic/run of ectopics only sometimes deteriorate into a-fib?? It appears that excessive sympathetic or parasympathetic tone can result not only in the

ectopy itself, but also in the facilitation of the requisite number (6) of 'wavelets' required to initiate and sustain a-fib. It is precisely HOW ectopy can initiate these wavelets that still baffles me, although I suspect that this has a lot to do with the electrolyte balance/imbalance inside and outside cardiac myocytes, and perhaps EVEN MORE to do with how efficiently/inefficiently K⁺ and Mg⁺⁺ travel backwards and forwards across myocyte and intra-myocyte membranes.

Mike F.

James,

Thanks very much for your excellent clarification. The second web page (and links there from) which you mention is certainly educational also - if still a little on the technical side for me at my present stage of appreciation/understanding (-:

I see the logic in your suggestion:

"Could you have a short enough refractory period to induce an ectopic but a long enough one so AF is not sustainable?"

I suppose that it really is all down to myocyte EARP: a little too short = a PAC, (a little more too short = a run of PACs?) and a bit shorter still can initiate a-fib (via re-entry mechanisms).

I also like your further analogy regarding dominoes!

Mike F.

Mike and James,

Excellent posts, esp. yours James. Great questions and great answers. The only thing I can add in clarification is about the wavelets. They can only exist because of automaticity and reentry. An individual muscle fires and the impulse reenters that same cell creating a circle (closed circuit). They don't propagate, they only exist as a barrier. You theoretically could calculate the size (wavelength) of that wavelet by multiplying conduction velocity times refractory period. As you both point out, many people have a shortened AERP on occasion but no AF. I believe our innate conduction velocity (?related to dispersion) is integral in answering why we have LAF.

As you both point out, many people have slow heart rates and presumed high vagal tone without having AF. This (conduction velocity/dispersion) would appear to be the only variable. There's not been a whole lot written on it. However, everything that happens starts on a cellular level and I believe that although EP mechanisms may aid our understanding of the problem and offer interesting insight, the real solution will only come from a complete understanding of the biochemistry/physiology involved. The LAF and hormone connection I believe represents the best approach to what's happening at the cellular level. I still have no idea how a wavelet actually starts, except to say that our substrate is defective.

PC, MD v54

Would one of you gentlemen be kind enough to define "substrate" for me? I would appreciate that. I want to understand the term as well as I can.

Jerry

Jerry,

I think that a substrate is a myocyte (group of myocytes?) that in firing before the SA node precipitates a PAC. I'm sure PC and/or James will give us a more definitive answer.

Mike F.

PC,

Thanks for the additional input. As always, one or two questions. (I am slowly getting all of this straight in my head, and am very appreciative of the patience and assistance of yourself and all others here in this regard.)

I wonder just what it is exactly about a-fibbers' innate conduction velocities that differ from those of non-a-fibbers? Is it that our conduction velocities are too fast? If so, is it this excessive velocity which gives atrial myocyte tissue the ability to support the re-entry mechanisms?? What causes such increased conduction velocities.....could this in no small way be related to the health of our myocyte and intra-myocyte membranes? I always keep in mind Erling's assertion that it was sorting out the balance of his fats (fatty acids) that finally resulted in him eliminating his a-fib.

Sorry to blather on: I'm just trying to get a grasp on the discussion up to now..... before moving on to fry my brain with Hans' latest hormonal hypothesis! (-;

Mike F.

Hi PC

The view I have of wavelets is rather like the turbulent flow coming off the trailing edge of a wing. I know some wavelets can be fairly static (especially what I would consider to be the very large re-entrant circuits of Atrial Flutter) but I thought that in general the wavelets are small, short lived, meandering, combining and propagating new wavelets. They terminate through collisions with each other (hitting a refractory wall) or by hitting the natural boundaries in the atria. Is my view (and the Moe's 1962 view) wrong?

The link you posted (<http://www.ub.rug.nl/eldoc/dis/medicine/b.j.j.m.brundel/c1.pdf>) has this to say...

"...Moe put forward the 'multiple wavelet hypothesis' of AF in 1962. This concept described the propagation of reentry waves as involving multiple independent wavelets circulating around functionally refractory tissue. The maintenance of AF then depends on the probability that electrical activity can be sustained by a sufficient number of active wavelets at any time.

...AF, multiple independent wavelets activate the atria in a random reentrant way. Individual wavelets could brake-up, fuse or collide with each other and wavelets would extinct when they reached the border of the atria or met refractory tissue..... the duration of each individual wavelet lasted only several hundreds of a millisecond."

I quite agree that automaticity plays a role (and will probably be the initial trigger) but I think once the process is kick started wavelets can propagate from one and other (as well as starting through automaticity) as the re-entrant circuits meander round the atria.

I'm happy to be convinced otherwise :)

--

James D

Mike,

If we have a defective "substrate" and our conduction velocities were problematic, then they would be too slow not too fast. Remember in order to fit at least 6 wavelets in the atrium as a barrier to normal waves they have to be small. The formula that is used to determine their size is AERP (shortened equates to a small number) x conduction velocity.

Erling's words of praise for EPA and DHA (by the way I just bought some today to go with my myriad other supplements) stem from their effect on stabilizing membranes (cell, mitochondria, etc.). I don't personally think that it will adequately address our substrate shortfall. However many of its benefits coincide with those seen with Mg++ supplementation, like decrease in mental illness and asthma. So maybe there's a connection. I've been thinking about Hans post quite a bit and doing a lot of reading, as well as thinking about some of the questions you've posed re "why me" vs the rest of the population. Is it substrate, magnesium, something else or all of the above?

PC, MD v54

PC,

Thanks again for the further words. Mmmm.... too slow. I'm simplistically envisaging one myocyte passing the baton to the next and so on: if they can do this fast enough, then less wavelets = no a-fib. I like the fact that it is the conduction speed which is too slow as opposed to being too fast.....

Glad to hear you've got hold of the EPA and DHA.... I still have this 'bee in my bonnet' as regards membranes (and phospholipids) being key in all of this. Are you (as I suspect (-:) 'up to speed' as regards HOW the baton is passed.... in one sentence of layman-type speak?? (There's throwing the gauntlet down!)

Thanks again,

Mike F.