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

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SUBJECT: LAF & ANTIDEPRESSANTS

An afibber recently reported to me that he could prevent atrial flutter from turning into atrial fibrillation by taking two 530 mg capsules of the herb valerian. Another has reported that the use of Effexor XR (venlafaxine hydrochloride) has kept him afib-free for 6 months. Prior to taking Effexor he experienced about 3 episodes per month each lasting about 4 hours. This afibber is 40 years old and was diagnosed with LAF (vagal type) at age 37. His daily dosage is 150 mg of Effexor XR.

Venlafaxine works by preventing the reuptake of both serotonin and norepinephrine (noradrenaline). It has no effect on acetylcholine or histamine receptors. Thus it would tend to increase the level of circulating norepinephrine which could be a good thing for vagal afibbers.

My own experience with antidepressants involved 7 months on Paxil (paroxetine). In the beginning it worked very well and kept me afib-free for up to 2 months at a time (I have the adrenergic type). However, it eventually gave me serious night time bradycardia so I gave it up.

Nevertheless, I feel the subject of antidepressants and perhaps tranquilizers and afib would be an interesting one to discuss in the Conference Room so this is the opening salvo. Has anyone had experience (good or bad) with antidepressants? Does anyone have any theoretical musing to offer as to why they might work to prevent afib episodes?

Hans

I definitely think that antidepressives of the selective serotonin reuptake inhibitor type, do work to prevent LAF. For several years between my first episodes of LAF (vagal type) and recent episodes, I was taking Prozac (fluoxetine) at low dosages (2.5 to 10 mg per day). At these dosages I had no undesirable side effects of Prozac excepting for a sensitivity to caffeine. Prozac and caffeine use some of the same metabolic breakdown pathways and so caffeine and related stimulants have a significantly longer half-life when Prozac is also taken. I had no episodes of LAF during that time.

I stopped taking Prozac in the fall because the generalized anxiety I had been experiencing most of my life had, after much therapy, disappeared. The Prozac was no longer necessary for anxiety, but then, during a stressful period of time, LAF again occurred. I have, since then, treated the LAF with the vitamin and mineral supplements suggested here and I have had no episodes in

four-and-a-half months and the palpitations I have experienced for most of my life have also disappeared.

Michael

Hello Hans,

I find the effect of anti-depressants quite interesting, in their relation to serotonin and AF. It seems, from your post, that inhibiting serotonin works to stop AF for one, whereas, some have gotten AF because of it, or at least they attribute the cause, to the drug, and one of the side effects listed, is arrhythmia. So it seems to me that serotonin is playing a key role in AF, maybe because one is lacking in, has too much of, or the neurotransmitters dopamine and serotonin are out of balance. In my case, my amino acid panel revealed that Phe. was normal, but tryptophan was to the low side, therefore leading me to speculate, that there was more dopamine, and not enough serotonin, at the synaptic cleft, whereas, it could be the opposite for someone else. I feel, now, that I could be a mixed AFer, tending to be more predominantly vagal. So, do adrenergics tend to have too much serotonin, and vagals not enough, and do mixed have an imbalance of both serotonin and dopamine? Or could it be, that the pineal gland is not operating properly, in its breakdown of serotonin to melatonin, and too much or too little of melatonin is the problem. I also find the circadian rhythm quite perplexing and interesting, in regards to you, as your rhythm of incidences and timing of AF, seem to follow a distinct pattern. I did notice in your post on the BB, that you had been AF free for, I think it was, 30 days and counting. Did you change something, or was it possibly the sunshine on your vacation, that stimulated your pineal gland. In my reading, I found that the pineal gland operates by the light in the eyes and that most of Vit D, is absorbed through the eyes. Think about animals that have heavy fur coats, and probably do absorb most of the rays of the sun through their eyes, whereas we wear clothing. So, how many of you wear sunglasses? I know that I did. Another question, is how many of you wear normal eyeglasses all day, that could possibly magnify the effects of the sun in the eyes, whereby over stimulate the pineal? I know this is a stretch, but feel we should not leave any stone unturned. Anyway, that's my two cents worth for the moment and I only wish that everyone could get an amino acid panel done, to possibly tell the real story of exactly what is in, or out of balance, with each of us. Thank you, Hans, for opening up this topic, for discussion.

Richard

Richard,

Most SSRIs (selective serotonin reuptake inhibitors) block both serotonin and norepinephrine reuptake to varying degrees. I believe it is the inhibition of norepinephrine reuptake that may benefit vagal afibbers by achieving a better balance between acetylcholine (vagal neurotransmitter) and norepinephrine (adrenergic neurotransmitter). Reboxetine (Vestra) is probably the most effective norepinephrine reuptake inhibitor among the SSRIs although some tricyclic antidepressants are also effective. Citaprolam (Celexa) would be the most effective serotonin reuptake inhibitor.

Venlafaxine (Effexor) is fairly balanced as far as serotonin and norepinephrine reuptake inhibition is concerned. Some of the SSRIs prevent actylcholine reuptake at the muscarinic receptors. Paroxetine (Paxil) is the most effective acetylcholine reuptake inhibitor among the SSRIs, but some tricyclic antidepressants are even more effective.

Panic disorder is best treated with potent serotonin reuptake inhibitors. Panic disorder probably has some resemblance to the triggering phase of adrenergic and possibly mixed LAF so citaprolam might be the best choice while reboxetine would probably work best for vagal afibbers. Please note that this is pure speculation on my part. I have no data to back it up.

I believe most vitamin D is synthezised in the skin. Sunscreens and clothing block this synthesis. I did develop a nice tan so I am sure my vitamin D stores are well stocked now.

Hans

Hans--Your discussion of the differential effects on neurotransmitter reuptake of the various SSRIs is very interesting. Is there a simple source for this information?--I have never run across it.

These differential effects would explain very well, for example, why most people find Prozac to be the most stimulating of the SSRIs.

Michael

Michael,

There was an excellent article on the pharmacology of antidepressants published in the Mayo Clinic Proceedings. That is the reference for my statements. The full reference is

Richelson, Elliott, Pharmacology of Antidepressants, Mayo Clin Proc. 2001;76:511-527.

I can highly recommend it for anyone interested in the finer details of SSRIs and tricyclic antidepressants.

Hans

Hans,

Sounds like us VMAFers should stay away from Paxil in particular and possibly try Reboxetine (Vestra). The bananas have really helped my sleep so perhaps SSRIs deserves more exploration.

Wish I could contribute to this topic, but I'm not in the habit of prescribing medications for my patients (ha-ha).

PC

Hello Hans,

Thank you for your reply. In what I have read, however, it is my understanding that SSRIs don't block serotonin, they increase it and keep it at the synaptic cleft longer. I'll continue to read. If you read the following article, you'll find that serotonin is not one of the players in the orchestra of neurotransmitters, but rather the bandleader.

http://www.nasw.org/users/twoharts/serotonin.htm#chap2

For example, Prozac, Zoloft and Paxil are "serotonin reuptake inhibitors," also referred to as SRIs or SSRIs. Normally, serotonin is pumped back into the nerve cell that released it after it is used once. These drugs interrupt that process so that more serotonin is available to reach the target

receptors. Most of the serotonin-active drugs act to increase the availability or activity of serotonin in the brain.

Richard

This is what I have understood about SSRIs and I quote from Kathleen deMaisons book, *Potatoes not Prozac*. Before I do, I have to say that I am not promoting this book for anyone. There is a lot in it that makes sense and helped me to get back to normal. But there are things (such as the potato I do not eat - but bananas would work as well). The trick is to eat a lot of protein at every meal, evenly spaced out each day. Never go hungry and discover food that hold you between meals. Eat no refined carbs, white flour, sugar etc.

"Drugs like Prozac, Paxil, Effexor and Zoloft turn off the reuptake pumps so second hand serotonin stays between cells and continues to hit the serotonin receptors. In effect, your brain is getting more use out of the serotonin you have....

As with the chocolate solution however there are significant problems with taking antidepressants. First, as we saw in Barbara's case, your brain will close down some receptors after a while in response to the increase in serotonin caused by the antidepressant and you will have to increase your dose or change the type of medication. Second, many of these drugs are very expensive and must be taken under the supervision of a doctor, and your treatment may not be covered by insurance. And third, even these newer antidepressants have unpleasant side effects. You may experience nausea, jitteriness, weird dreams or problems with your sleep. You may find that you have no sexual drive, are less sensitive to sexual stimulation and cannot achieve orgasm. While antidepressants can be life saving if you have a serious depression that does not respond to anything else, the side effects may be a high price to pay for this relief.

There are other options. Certain ways of eating can significantly alter your serotonin levels. What and when you eat can be a wonderful ally in your seven-step process. If you eat a baked potato (with the skin) as a snack before you go to bed, you will put the biochemistry in motion to get tryptophan into your brain to make serotonin"

And from my own perspective, and alluded to in the book, although the drug companies say that antidepressants are not addictive. They are as addictive as heroin. Why you ask. Heroin works in the very same way on endorphin receptors in the brain as SSRIs do. You take heroin/serotonin (even if it is recycled serotonin) then the endorphin/serotonin receptors down regulate. You then need more and more to get the same hit. When you stop taking them most of the receptors are turned off - your body has been adjusting itself to get what it thinks is a normal equilibrium.

So your body has adjusted to large amounts of the recycled serotonin or synthetic endorphin (heorin) and when you stop - what is coming in naturally is not enough. It takes a long time for the receptors to up-regulate and whilst you are waiting for this to happen you experience severe withdrawal or depression.

Add to this, another side effect of heart arrhythmia (and even lengthening of the QT wave) another known side effect of SSRIs and tricyclic antidepressants then there is another time bomb waiting to go off. I think that in some circumstances it may help as a stopgap. But brings with it a new set of problems. And balance I think is what an affiber is looking for.

I have had experience with valium, and Tegretol - related to tricyclic antidepressants. I honestly believe now after reading up on Tegretol that it caused my problems with AF and near death experiences especially when I read about the effects on glutamate metabolism and enzymes in the liver. My post I know what caused my AF explains it.

Fran

Richard,

I absolutely agree with you. SSRIs block the reuptake of serotonin and/or norepinephrine thus making more available because the recycling (reuptake) is prevented. I guess my previous message was not clear.

Fran,

I certainly agree with you that SSRIs can have very serious side effects and should not be entered into lightly, but then neither should flecainide, amiodarone or propafenone. I guess for some afibbers SSRIs may actually turn out to be the lesser evil. Any way, if certain SSRIs were found to work in preventing afib it may be possible to find a natural equivalent that would do the same.

Hans

Hans,

If SSRIs increase serotonin at the synaptic cleft, then wouldn't just increasing serotonin in the system do the same, if not a better job, without the side effects? Do you believe that the amino acids could be out of balance, as are mine, due to less-than-perfect dietary habits over the years, and if the aminos were balanced, then one could gain good health again? I still feel strongly, that somehow balancing the aminos, and changing the diet completely around would ultimately fix our problem. I think in the beginning we would have to supplement until all our stores had reached an equilibrium and the body had healed, and then just maintain a very good diet from that point on. I guess I'll be the guinea pig, as I will be meeting with Dr. Gersten on Thurs. and I'll be having every molecular test I can get. From that point, as discussed at his site, I'll have an amino acid powder made specifically for my needs, along with recommended supps. and we'll see what happens. By the way, Hans, do you think that being in the sun could have helped your serotonin/melatonin, being that we are both in the NW where we don't get that much sun? Thank you, Hans.

Fran,

Thank you for your info. I do find this subject interesting, esp. in that serotonin and melatonin also play a role with glutamate and GABA. Did you ever find a facility to do your amino tests?

Richard

Richard:

What Hans means is that SSRIs block the reuptake of serotonin, not that they block the production of serotonin. Hope that helps...

Jerry

Thank you Hans and Jerry,

I did at first think he meant that it blocked serotonin, but he clarified that. I feel like I serotonined

everyone to death on the BB, so I don't have much else to give here, and haven't had much time. On my return, I'll post as to what Dr. Gersten had to say. I do strongly feel, however, that everyone keeps speaking of drugs helping in some way, when in fact, I believe that the amino acids could do the very same thing, but that the electrolytes must have balance, as well. I still maintain, that if a drug works, then find out what the drug is doing, and then you may find the answer to your problem, as such was your case Jerry. Cipro used and depleted stores of Mg, therefore, Mg. was your problem. If one can't sleep, then melatonin is in short supply. If one is depressed or anxious, then serotonin is the problem. If one can't get an erection, then nitric oxide is the problem, which is derived from arginine. If a sodium channel blocker works well, then one has too much sodium, etc.....

Remember, how many years Dr. Gersten searched for answers, and he ultimately found amino acids to be the answer and he healed himself. That's why after reading his website, I felt that I had finally found a doctor that made any sense about our ailments. Hans, I wish you could be tested in this manner. It is my understanding that you could order the test yourself and have it done.

Have a wonderful Memorial weekend,

Richard

Richard,

We are all looking forward to hearing your amino acid test results. I know I certainly am, and if something is found amiss in your results, I will look into having my own profile done.

Hans

I have experience mainly with the SSRI citalopram, which can according to some literature cause bradycardia - not good for VMAFrs:

http://216.239.37.100/search?q=cache:8jGCVlQkyY0J:alertpubs.com/Issues/2002/Sda042002.PDF+Citalopram-induced+bradycardia+and+presyncope.&hl=en&ie=UTF-8

As regards use of this drug correlating - if at all - with my last three episodes of AF:
I had a 2hr episode in Oct99 - problem is, I'm afraid that I cannot remember whether I was on cital. or not before/at the time of the episode. In May02 I had a 20hr episode some 4 months after having STOPPED cital. In Nov02 I had a 4hr episode after having taken 20mg/day of cital since May02. I have since then slowly reduced the cital to the point where I now take just 5mg per day. It was 187 days between my last two episodes, and it is as I write (and knock on wood) 194 days since my last. So to conclude from my experience with cital?? No conclusion of any sort I'm afraid. I suspect that the overall picture is in my own case (and many others) just too complex as regards my biochemical and psychological state. I intend to 'kick' the cital soon. As I frequently bang-on about, I am a VERY firm advocate of paleo diet with LOADS of quality protein and veg plus some nuts and berries..... and less and less supplements as time goes by. Plus some therapy!

Best wishes all,

Mike F.

Mike

Noticed in your key search words you also looked for presyncope. Did you ever pass out?

Fran

Fran,

No, but a few years ago I had a couple of runs of palps (few seconds duration) which made me feel unpleasantly and increasingly thick/hot/light-headed as they progressed. It felt like I would have passed out had the run continued for more than a few seconds... felt as a fast fluttery feeling in the neck... really GOT MY ATTENTION when it happened! Had me gasping and I suppose, on reflection, well on the way to a panic attack (I also had a couple bad PAs around the same time). No more such palps for the last 5/6 yrs thankfully (they were before my 3 diagnosed AF episodes).

Mike F.

Noone, I believe, has addressed the other part of Hans' original post, which mentioned tranquilizers. I believe Hans hypothesized in his book that certain tranquilizers i.e. Ativan might reduce vagal tone and might prevent an episode in a vagal afibber. Anyone have experience or thoughts on this. Oftentimes I have a decent amount of time between the pre-afib period and the onset of an episode. I am considering trying Ativan or perhaps other tranquilizers to see if they might ward off the inevitable.

Kerry

Kerry

I think I partly acknowledged this in my reference to valium. I was given valium and temazapam (sleeping tablet) prior to onset of AF in conjunction with passing out and convulsing. When I had my first baby I tried to stop taking these. No one told me the problems with withdrawal to these drugs. In reading up about the diazepam group drugs I see that withdrawal from valium can produce seizures and heart rhythm disorders. This is only supposed to be short term though. Mine carried on. Interestingly diazepam drugs of which ativan is one works on the GABA receptors. It also makes it difficult to produce GABA once you have stopped the drug and makes you very sensitive to free glutamate as the balance goes awry. Free glutamate is an excitory neurotransmitter that can be eaten direct from food and it’s known to cause heart rhythm disorders and seizures amongst about 90 other problems.

I was then given Tegretol (related to the tricyclic antidepressants) to stop seizures. I was also given a 10mg valium after every fit. In reading up about tegretol, they too affect electrical circuit of the heart, not to mention certain enzymes in the liver and over excitability of the glutamate receptors. They never did stop me passing out and seizing and my AF went from short term bouts to long term bouts. I diagnosed myself with panic attacks as Dr's thought I was just being anxious and I knew it was much worse than that.

About 10 years after onset of AF and seizures I was given digoxin after them catching AF in full swing. I took these faithfully and my AF went from paroxysmal to chronic. Also my body changed. I felt dehydrated and itchy all the time. I got fibromyalgia and chronic fatigue, I got chest pains, my seizures changed in nature and I now had near death experiences. Had a year on antiarrhythmics and went from worse to near dead.

I now take nothing. I am scared stiff of any tablets. I feel they are to blame for my AF. I try not to eat any free glutamate and a paleo diet. I have not had a seizure since I first went on sotalol in March 2000. I do not get AF either.

I reckon that in some cases tranquillisers may help as a stopgap for AF, if the reason for the AF is related to glutamate, but the long term problems may in some cases outweigh any benefits if a balance in the ANS is what is being sought. However, each of us has to weigh up the pros and cons for themselves and make educated judgement on whether it may help or not.

In my case I can't help but wonder if it brought it all on.

Fran

Thinking of SSRIs and tranquillisers, I was wondering if there is a sub-group of a-fibbers here who, like myself, broadly conform to the following (somewhat crude) criteria:

1. A history (5-20 yrs) as an adult of diagnosed anxiety/GAD, bouts of/a tendency towards mild depression, OCD etc. AND/OR

2. Post Traumatic Stress Disorder - whether the event was as a child OR adult (in my case as a child), and whether the stressor was acute or chronic (in my case chronic),

3. Quite a long (and likely associated with the manifestations of 1. AND/OR 2. above) history of ectopic activity,

4. Relatively few episodes of AF - say one or so per year - despite 3. above.

5. A history of GERD - likely associated with 1. and 2. above.

I appreciate that it is a whole interwoven 'cocktail' of subtle biochemical imbalances which predispose one to/result in the precipitation of AF, but I nevertheless wonder about the existence of subsets of AFers, and the above is my proposal (admittedly largely based on myself!) for one such subset..... a subset whose precipitating/predisposing factors are perhaps largely psychologically driven/manifested..... and a subset who are - in the context of this CR topic - most likely to have used SSRIs and/or tranquillisers. I look forward to hearing from others of you out there who seem to fulfill the above criteria. (After all, there certainly seems to me to be a contrasting (to that I am proposing) subset of individuals who invariably after a 6-12month 'apprenticeship', seem to quickly settle into a pattern of frequent and regular episodes of AF on a weekly or fortnightly basis, with episodes furthermore appearing almost inevitably once ectopy occurs.) Again musing to myself, I do wonder if most individuals out there who might well fit into 'my' proposed subset of AFers will not be attending this conference room since they have only had relatively few AF episodes... On the other hand, if they are - as is likely - highly anxious like myself, then they will be at least looking in to learn all that they can! Looking forward to any and all responses/comments/views which are forthcoming.

Mike F.

Yes, Mike, I am very much of this group, with the exception of post-traumatic-stress and OCD, my symptoms fit well.

I used an SSRI on and off for perhaps ten years; the longest continuous period of SSRI use was perhaps 18 months (at a minimal-effective dosage so that there were few, if any, significant side-

effects).

I also spent a lot of time in various kind of therapy and was finally free of excessive anxiety only after understanding very thoroughly the psychological dynamics of my family. And then putting my understandings to work in the way that I have been dealing with my family (and people in general).

I wouldn't hesitate, by the way, to resume SSRI use, if and when generalized anxiety becomes a problem again. I think this is always a possibility.

I make frequent use of anxiety-reducing techniques, such as progressive relaxation and exercise; I make sure I get enough sleep; I eat well; I avoid more than very minimal use of alcoholic beverages and caffeine.

I also spend as much time as possible doing good things with the people I care about.

Michael

My brief history of LAF and ectopy:

Since age of 18 (forty years ago): Palpitations (ectopy); I cannot remember whether there were any particular precipitating events; the experiences were not uncommon; I do not think I noticed them daily though.

1995 and 1996: Perhaps a dozen episodes of LAF of very short duration: a few seconds to perhaps two minutes. Would occur from time to time, sometimes weeks apart, while lying in bed at night. I associated them with alcohol use during the evening. During this period I also noticed brief (a couple of seconds) runs of tachycardia (which I now associate with being in certain kinds of positions, often sitting in a car, in which my vagus nerve was affected). I stopped virtually all use of alcohol and caffeine. Drank tea on occasion.

July 1997: A relatively long episode of LAF, lasting two to three hours. I had had a pint of beer with dinner. Occurred at about 2 am. When the episode lasted more than a few minutes and relaxation techniques did not stop it (I thought the LAF might have been due to anxiety since this was a very stressful time in my life--a relationship break-up and a move to a new town, among other things), I called the 24-hour advice nurse at my HMO who said to go to the ER if the episode lasted more than 20 minutes. I went to the ER where AF was diagnosed with an EKG and the LAF remitted after a couple of hours without any treatment.

Following this I looked for advice from my physician and did as much research as I could. I knew that I suffered from anxiety so I sought help from therapists and psychiatrists at my HMO who introduced me to behavioral techniques for dealing with anxiety. These techniques were very useful. Meanwhile I used a Holter monitor which showed only PVCs or PACs averaging every 5 minutes. My research suggested that theophylline, the caffeine-like stimulant in tea, could cause arrhythmia so I stopped drinking tea (I eliminated alcohol completely from my diet because of its clear role in precipitating arrhythmia). I also started taking magnesium supplements, which caused an interesting problem when, after a few weeks, I stopped taking the supplements and found that my palpitations increased drastically for a few days. Since I was not clear about the role of magnesium in my arrhythmia, I stopped taking it.

A month after this episode of LAF, I still noticed that I could induce tachycardia (which usually preceded my LAF episodes) when I lay on my left side in bed at night. The tachycardia would stop if I immediately got up out of bed. I was very careful during this period not to sleep on my left side.

My main focus for the next six to nine months was in dealing with my anxiety. I found an anxiety group on the Internet and did a lot of reading about anxiety. I met several members of the group who lived nearby (in the Seattle region) and learned a great deal about anxiety disorders. In the spring of 1998 I started taking Prozac again (I had taken it a couple of years before for seasonal affective disorder) to help deal with the anxiety I continued to suffer from as well as periods of major depression. The Prozac was very useful at 5 to 10 mg per day.

1998 to 2002: I remained relatively anxiety-free and free from LAF until the fall of 2002 when I again started to experience brief (a few seconds to a minute or two) spells of LAF, in the middle of the night, while lying in bed.

This was also a period of stress. My mother had died in the spring of 2002. A couple of months later my father had open-heart surgery. My sister was very ill. I moved from one city to another. I was still taking Prozac for anxiety, but gradually reducing my dose, ending up at 2.5 mg per day. I was free from generalized (unrelated to a specific source) anxiety and resulting episodes of depression so I felt (with my psychiatrist) that I could reduce or stop the use of the Prozac.

In October of 2002 I had another relatively long attack of LAF, lasting about 3 hours. I went to the ER at my HMO and was given diltiazem, a calcium-channel blocker, which stopped the episode (I think the episode might have resolved anyway). The ER physician said that he thought my LAF was related to something called "holiday heart" and recommended that I avoid alcohol which is the culprit in "holiday heart." I had had two glasses of wine with a late dinner at a restaurant before the LAF attack (which again started in the middle of the night while I was lying in bed).

I had a couple of other brief attacks of LAF since the October 2002 episode. These were in November and December of 2002. In November after attending an evening class, I returned home and ate a large sandwich about 10 pm. I sat in my recliner to read and an episode of LAF started which stopped after perhaps twenty minutes. In December after a late meal at a restaurant (and no alcohol--during this period I was not using any alcohol) ending at about 9:30 pm, I again experienced an episode of LAF after going to bed. This episode resolved after a half hour or so during which I concentrated on relaxation techniques while sitting.

During all of this period I experienced ectopic beats regularly, perhaps one event per hour. I understood that they were not uncommon and were probably unrelated to LAF.

I had various tests at my HMO including Holter monitor, EKG, echocardiogram and treadmill stress, all suggesting that my heart, aside from expected wear-and-tear from aging, was quite normal and functioning well.

At this point I discovered this wonderful web site and started thinking seriously again about vitamin and mineral supplements as a way of dealing with LAF. I had also at this point stopped Prozac completely in consultation with my psychiatrist as I was in general feeling good and was completely free from anxiety.

I started taking magnesium and potassium supplements and found that any symptoms I related to LAF, such as brief spells of tachycardia, had disappeared. I also noticed fewer and less strong ectopics. Convinced that I was well on my way to recovery, I started drinking the occasional glass of wine with dinner. I was also convinced that caffeine was probably a contributor to arrhythmia so I stopped drinking any coffee at all. When I stopped drinking coffee (I had been drinking very moderate amounts, one or two espressos a day; espresso is strong in taste but significantly lower in caffeine than drip or percolator coffee).

Then, on New Year's Day of 2003, I had another episode of LAF after hosting a New Year's Eve party. During the party I consumed perhaps a couple of glasses of wine. Since I was the host, and busily preparing and serving food and drink to my guests, I really did not have much time for

drinking much at all. At any rate, the LAF started up while I was lying in bed. Since the LAF did not remit after twenty minutes or so I went back to the ER at my HMO and was again treated with diltiazem. The LAF remitted after three hours or so and I went home.

Since then I have taken my vitamin and mineral supplement regime very seriously and have had no further episodes of LAF. I have resumed very moderate drinking of tea and coffee and the occasional glass of wine or bottle of beer with dinner. I have had very few experiences of ectopics, perhaps once or twice a day, if at all. Ectopics, which were once a regular feature of my life, have become unusual events. I have had one episode of tachycardia, starting while lying in bed at night. This followed a week in which I went out to dinner or went to parties each night for perhaps five nights in a row. At all these events I drank a couple of glasses of wine or the equivalent (12 oz of ale).

I have had a couple of episodes of tachycardia which were precipitated by lying in bed on my left side. These episodes also occurred during the period in which I increased my alcohol intake.

It is clear to me that my LAF is vagally-mediated. That it results from mineral imbalances precipitated by ingestion of more than very modest amounts of alcohol and tea and coffee, all of which are mineral and vitamin-wasting. An important additional factor in my LAF is stress. Under stress, one's hormones and enzyme systems are very demanding of certain nutrients and during these stressful times, nutrient-wasting drugs such as alcohol and caffeine must be avoided. I am also convinced that Prozac in very low doses is protective regarding the effects of stress. If I were not very committed to maintaining a daily program of exercise and stress-reduction, I would remain on Prozac as a method of dealing with anxiety and LAF.

Michael

Michael,

Thanks very much for your latest post: I enjoyed reading it. Your own ectopy and LAF experiences do indeed closely parallel my own. Whilst still myself taking 5mg/day of the SSRI citalopram, I intend to phase this drug out completely in the near future, and I genuinely believe that optimal diet and therapy/relaxation techniques will continue to greatly help us both in ensuring good health for the future. Do be sure to keep in touch via this excellent website bulletin board to let me and all the other guys and gals here know how you are getting on.

Cheers,

Mike F.

Interesting article on Cardiovascular Effects of Selective Serotonin Reuptake Inhibitors and Other Novel Antidepressants at

http://www.medscape.com/viewarticle/453618

PC

Very interesting PC. It confirms again that my problems with AF were brought about in the first place by this type of medication. And it is good to see that knowledge of how they work has been taken a step further to MAO production.

Of course all our reasons for AF may be different.

Fran

I found this article on SSRIs of interest. I'll have to read your link, PC.

Here's an excerpt on effects of SSRIs:

Blockade of fast sodium channels Slow repolarization, delay intracardiac conduction, reduce some arrhythmias at low concentrations, cause arrhythmias, seizures at high concentrations

http://www.preskorn.com/books/ssri_s3.html

Richard

PC,

Your link told me that serotonin and dopamine need to be in balance. I know that cigarettes keep dopamine at the syn. cleft and SSRI's keep serotonin at the syn. cleft, and smokers had less incidence of heart attacks with SSRI's. As stated before, my Phe. levels were normal and tryptophan levels were low and Dr. Gersten pointed out specifically that my tryp. needed to be corrected, along with copper. Very interesting, PC.

Richard

Richard,

Sorry to be so late in answering. Seems reasonable that increasing serotonin in the diet would be just as effective as preventing its reuptake with SSRIs. I will certainly be most interested in your results with the amino acid powder and yes, I do believe the constant sun exposure in HI really got my vitamin D stores replenished and was beneficial. Unfortunately I am now back to my 12-14 day interval between episodes. Anyway, it was very, very nice to be afib-free while it lasted.

Hans
