

THE AFIB REPORT

Your Premier Information Resource for Lone Atrial Fibrillation!

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Editorial

There is more exciting news on the inflammation front! A team of Greek researchers has now confirmed the association between inflammation and atrial fibrillation. In this issue I discuss the implications of their findings and cover the details of the investigations carried out at the Cleveland Clinic. The excitement of discovering a potential major cause of AF, unfortunately, has to be tempered with the realization that not all afibbers have an inflammation – so there are still one or more variables that need to be discovered.

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We have received positive feedback on our first two “personal journey” articles, so in this issue we continue with Randy Lewis’ absorbing and insightful story. We also report on a very fascinating discovery by an international team of researchers linking reciting the rosary or a mantra to improved autonomic nervous system control. Enjoy!

*Yours in health and sinus rhythm,
Hans Larsen*

More on Inflammation and LAF

In the September 2001 issue of The AFIB Report I reported on the work of Dr. Andrea Frustaci and colleagues at the Catholic University of Rome. In 1997 Dr. Frustaci performed biopsies of the right atrium of LAF patients and found that 67% of them had signs of an inflamed heart lining (myocardium)[1,2]. I suggested that certain natural supplements might be effective in combating inflammation and outlined an anti-inflammatory protocol, which hopefully would help to reduce the frequency and/or duration of LAF episodes.

Just last month two research papers were published that clearly support the inflammation connection[3,4]. Both papers, one by American researchers (Cleveland Clinic) and one by Greek researchers, report a significant association between the level of C-reactive protein (CRP), a marker of inflammation, and the presence and severity of LAF.

American research

The Cleveland Clinic researchers tested CRP levels in patients with atrial fibrillation (AF) and compared them to the levels in control patients with no history of AF. Sixty-seven of the AF patients had lone atrial fibrillation which was defined as AF in the absence of structural heart disease; patients with hypertension and LAF, but no structural heart disease were also included

in this group. The LAF group was further divided into those with paroxysmal LAF (episodes self-terminating within 30 days) and those with persistent LAF (episodes lasting longer than 30 days, but amenable to cardioversion).

The researchers found that patients with AF, with or without structural heart disease, had significantly higher blood levels of CRP than did controls (median value of 0.21 mg/dL versus 0.096 mg/dL). The average value for LAF patients was 0.21 mg/dL, which was not significantly lower than that found in AF patients with structural heart disease (0.23 mg/dL). CRP levels were generally higher if the patients were actually in atrial fibrillation or had come out of an episode within 24 hours of sampling. These patients had average CRP values of 0.30 mg/dL as compared to 0.15 mg/dL for AF patients in sinus rhythm. It was also clear that patients with persistent AF had higher CRP values than patients with paroxysmal AF (0.34 mg/dL versus 0.18 mg/dL).

The researchers conclude that AF might induce or be induced by an inflammation, which in turn may promote the persistence of AF. They suggest that CRP levels may become useful in predicting stroke risk and need for warfarin therapy in AF patients. They also suggest that clinical trials of the use of anti-inflammatory agents in the prevention of AF may be warranted.

Greek research

The Greek researchers tested CRP levels in 50 paroxysmal AF patients who were actually in fibrillation at the time of sampling and compared results to those obtained for 50 people in normal sinus rhythm. The AF patients had a median CRP level of 0.80 mg/dL as compared to 0.04 mg/dL for controls. The researchers observed that AF patients who could not be cardioverted had a much higher average CRP level (2.12 mg/dL) than did patients who were successfully cardioverted (0.50 mg/dL). They also noted that patients with an enlarged left atrium had considerably less success in being cardioverted. They conclude that high CRP levels are strongly associated with the presence of AF and with a lower chance of successful cardioversion.

So what does it all mean?

First of all, it is clear that most patients with atrial fibrillation suffer from an inflammation as indicated by substantially higher than normal CRP levels. It would also appear that the higher the CRP levels are the more persistent and resistant to cardioversion is the AF. It is not clear from the research whether the inflammation causes the AF or whether AF causes inflammation; however, my guess would be that inflammation is a major cause of AF including LAF.

It is, perhaps unfortunately, also clear that inflammation is not the sole cause of AF. Dr. Frustaci only observed inflammation in 67% of the LAF patients who underwent biopsies. Dr. Mina Chung of the Cleveland Clinic, the lead author of their study, informed me that most, but not all AF patients involved had abnormally high CRP levels[5].

I personally do not have an inflammation. During a recent LAF episode my CRP level was 0.03 mg/dL – well within the normal range. I had been on an anti-inflammatory protocol since August 2001 so I cannot say for certain whether I ever had an inflammation, but I am convinced that I don't have one now – especially since my sedimentation rate is at the very low end of normal as well.

I would strongly suggest that all afibbers have their CRP levels tested – preferably during or shortly after an episode. If it is high then it would make sense to try to reduce it, especially since a high CRP level is also a risk factor for stroke and heart attack. I believe natural supplements such as Moducare, MSM (methyl sulfonyl methane), curcumin (with piperine), bromelain, probiotics (acidophilus and yogurt), and pancreatic enzymes may be useful in reducing inflammation. Lowering CRP levels (reducing inflammation) would be particularly important if you are planning on undergoing a cardioversion. PLEASE! If you do have a CRP test let me know your results so that I can add them to our database.

New Guidelines for Management of Atrial Fibrillation

The American College of Cardiology, the American Heart Association and the European Society of Cardiology have just issued new practice guidelines for the management of atrial fibrillation (AF). The definitions of atrial fibrillation are interesting:

- Idiopathic AF is AF of unknown origin.
- Lone AF is AF without clinical or echocardiographic evidence of cardiopulmonary disease. There is a tendency to apply this term only to people under the age of 60 years.
- Nonvalvular AF is a term used to describe AF that occurs in the absence of rheumatic mitral stenosis or the presence of a prosthetic heart valve.

The voluminous report also contains the following observations:

- The incidence of AF increases markedly with age from less than 1% under the age of 60 to more than 6% over the age of 80 years. The prevalence of lone atrial fibrillation varies between 12 and 30% of all cases of AF. In younger patients between 30 and 45% of all cases of paroxysmal (intermittent) and 20 to 25 per cent of persistent cases of AF can be classified as lone atrial fibrillation.
- There is growing evidence that AF is associated with an inflammation and tends to lead to an enlarged atrium which, in turn, can lead to more frequent episodes. It is also clear that the autonomic nervous system can trigger AF through heightened vagal or adrenergic tone. AF can be either symptomatic or asymptomatic, even in the same patient, with the most common symptoms being palpitations, chest pain, fatigue, lightheadedness, and breathing difficulties. Frequent urination, due to the release of natriuretic peptide, is also a common occurrence.
- Cardioversion is not terribly effective in maintaining sinus rhythm in patients with persistent AF. A large trial found that only 23% of converted patients remained in sinus rhythm after 1 year and only 16% after 2 years.
- Treatment with antiarrhythmic drugs is not required or recommended for patients with infrequent and well-tolerated paroxysmal AF episodes. In severely affected patients with lone atrial fibrillation of the adrenergic kind beta-blockers are usually used first followed by sotalol. Propafenone (Rythmol) and beta-blockers are not recommended for people with vagal LAF and amiodarone is the very last resort due to its toxicity. As long as sinus or AV node dysfunction is not suspected propafenone and flecainide (Tambocor) can be prescribed on an outpatient basis. Quinidine, procainamide, disopyramide and dofetilide should only be started in a hospital under close supervision.
- Ablation reduces the frequency of AF episodes in more than 60% of patients, but the risk of another episode is still 30 to 50% over the first year.
- The risk of stroke in lone atrial fibrillation patients under the age of 60 years is no greater than that found in the general population and nearly half of all AF-related strokes occur in patients over the age of 75.
- It is not clear whether patients with paroxysmal AF episodes that terminate on their own need anticoagulation. Whether to prescribe warfarin or not must be decided on an individual basis. General guidelines are:
 - Age less than 60 years and no risk factors* – no therapy or 325 mg aspirin/day
 - Age between 60 and 75 years and no risk factors* - 325 mg aspirin/day
 - Age over 75 years – warfarin to an INR of 2.0

* Risk factors are heart failure, hypertension, left ventricular ejection fraction of less than 0.35, diabetes, coronary artery disease, thyrotoxicosis, rheumatic heart disease (mitral stenosis), prosthetic heart valves, prior stroke or heart attack and prior transient ischemic attack (TIA).
Journal of the American College of Cardiology, Vol. 38, October 2001

AFIB News

Undiagnosed hyperthyroidism: a risk factor for atrial fibrillation. Austrian researchers report that people with undiagnosed (subclinical) hyperthyroidism (overactive thyroid gland) are 5 times more likely to develop atrial fibrillation than are people with normal thyroid hormone levels. The researchers studied 23,638 people and found that those with low values of serum thyrotropin (less than 0.4 mU/L) but normal values of free triiodothyronine and free thyroxine had an incidence of atrial fibrillation of 12.7%. This compared to an incidence of 2.3% among people with normal thyrotropin levels and an incidence of 13.8% in those with diagnosed hyperthyroidism.

American Heart Journal, Vol. 142, November 2001, pp. 838-42

AF episodes preceded by premature beats. German researchers have confirmed that most atrial fibrillation episodes are preceded by a series of atrial premature complexes (APCs). They analyzed 297 paroxysmal AF episodes in 33 patients and found that the APCs (ectopic beats) originated in the left atrium in 77.5% of the episodes. The frequency of APCs increased from an average of 0.8 APCs per minute to 4.1 per minute in the two minutes preceding the beginning of an episode.

American Journal of Cardiology, Vol. 88, October 15, 2001, pp. 853-57

Autonomic balance at high altitudes. Danish researchers have found that the parasympathetic branch of the autonomic nervous system takes over and becomes the dominant factor in controlling heart rate and cardiac output when "lowlanders" are exposed to high altitudes (greater than 5000 meters). This could explain why vagal afibbers often experience episodes when mountain climbing or flying in unpressurized airplanes.

Circulation, Vol. 104, October 9, 2001, pp. 1785-91

Sublingual verapamil lowers heart rate. Romanian researchers report that sublingual verapamil (Verabene, 40 mg) is quite effective in lowering the heart rate in patients with chronic atrial fibrillation. Mean heart rate declined significantly just 10 minutes after verapamil administration and decreased from an initial 113 to 91 beats/minute after 60 minutes.

Romanian Journal of Internal Medicine, Vol. 36, July 1998, pp. 219-25

Quality of life in patients with AF. Dutch researchers have just concluded a study aimed at evaluating the effect of paroxysmal (intermittent) atrial fibrillation on quality of life. The study involved 73 patients with an average age of 54 years. The researchers found that the AF patients scored significantly lower than normal subjects in terms of physical and emotional well-being, vitality and general health. A high frequency of episodes was particularly deleterious to physical well-being. A depressed vagal tone further lowered the quality of life scores, as did the presence of severe perspiration during episodes.

European Heart Journal, Vol. 22, February 2001, pp. 247-53, 196-97

American ginseng blocks sodium channels. Antiarrhythmic drugs such as flecainide (Tambocor) and propafenone (Rythmol), the so-called class I drugs, exert their effect by blocking the sodium channels in the heart tissue and thereby curbing excessively rapid heart beats. Researchers at the University of Chicago now report that American ginseng (*Panax quinquefolius*) has a similar effect at least in brain cells. Their test tube experiment showed that the sodium channel blocking effect of ginseng was comparable to that of lidocaine another class I

antiarrhythmic drug. Whether American ginseng would be effective in preventing or terminating LAF episodes is not known.
European Journal of Pharmacology, Vol. 413, February 9, 2001, pp. 47-54

Mantras and the autonomic nervous system. A team of British, Italian and Polish researchers has discovered that repeating a common mantra or reciting the rosary synchronizes and reinforces inherent cardiovascular rhythms. Their experiment involved 23 healthy adults who were instructed to either recite the rosary (in Latin) or to repeat the mantra “om-mani-padme-om” during a 6-minute test period. To their astonishment, the researchers found that the breathing rate of the participants automatically slowed to 6 cycles per minute from the rate of 14.1 observed during spontaneous breathing. This 10-second cycle (6 inhalations/exhalations per minute) is a very important internal rhythm, which synchronizes respiratory and cardiovascular rhythms as well as sympathetic and vagal outflow. Maintenance of the 10-second cycle also increases calmness and well-being. The researchers speculate that the practice of using mantras to achieve the highly beneficial breathing rate of 6 cycles/minute originated in Tibet and India and was introduced to Europe by the Crusaders where it transmuted into the rosary.
British Medical Journal, Vol. 323, December 22-29, 2001, pp. 1446-49

I tried the “mantra-breathing” myself. I repeated the mantra “om-mani-padme-om” for 5 minutes. It took me 5 seconds to say it, 2 seconds to complete the exhalation, and 3 seconds to inhale - thus making it a 10-second cycle. The effects were quite spectacular and vastly improved the coherence between my heart and brain as measured on the Freeze-Framer. I certainly intend to follow-up on this and see what it does to the heart rate variability and autonomic nervous system control if done for longer periods of time. Stay tuned!

My Story – A Work in Progress

by Randy Lewis (RANDY.LEWIS@ROCHE.COM)

It was 1990, I was 34 years old, and I was playing tennis with my manager at my local racquet club. It may not be good form to thrash your boss, but he was a good sport and my game was really on that day. I had just completed an exceptionally strong serve when I began to feel somewhat lightheaded and woozy. We were finished with our match, so I bought a round of sodas and drove my manager back to his hotel.

I was then and still am in pharmaceutical sales, and every six weeks to three months my boss would come into town to work with me for a day or two. In many ways this had been one of his typical visits, but this visit ended up being anything but typical for me. I felt great emotionally because I had just decisively beaten my boss at tennis, which I had never done before, but I also felt really lousy physically and I didn't know why. Little did I know that that day would be the start of a journey of discovery lasting many years, filled with hope, frustration and only occasionally a faint glimmer of hope.

A frightening beginning

I did not sleep well that night, and the next day I was still weak and lightheaded. I was beginning to think that I had the flu. By chance I checked my pulse and found that it was very irregular. I didn't know enough at that point to be concerned, but during the day I continued to check it and it remained irregular. I stopped at the office of a cardiologist friend, Dr. Wendell Robinson in Richland, Washington and spoke with his nurse. She took my pulse and immediately ushered me back to an exam room. Dr. Robinson came in and checked my pulse, then ordered an EKG. I thought this was a bit extreme and said as much, but he told me to humor him and to just lie still.

The EKG indicated that I was in atrial fibrillation, a term that I was not at all familiar with then. Dr. Robinson reassured me that it was not a life threatening condition, but that it was still a concern. He asked me how it had begun and how long it had been happening, and then he started an IV and gave me some verapamil. This converted me to a regular sinus rhythm very quickly, within 30 minutes. I immediately felt better; the dizziness and lightheadedness were gone, and my chest was not thumping as it had been during the atrial fib.

Dr. Robinson had an echocardiography machine in his office, something that was relatively unusual at the time, and he personally did a transthoracic (through the chest wall) echo right then. I could tell that what he found concerned him greatly, as his brow wrinkled deeper and deeper during the procedure. He told me that he saw what appeared to be an atrial mixoma, or a tumor in my left atrium. He said that if it were indeed a mixoma I would need cardiac surgery immediately to have it removed. His concern was that the tumor might fragment, as they sometimes do when they metastasize or break apart, and I could suffer a stroke. He called a cardiac surgeon in Spokane, Washington, the cardiac center of the Northwest, and told me to get up there immediately.

I left Dr. Robinson's office shaken and dazed. Yesterday I had been king of the tennis court without a care in the world, and today I was on my way to see a cardiac surgeon with a huge surgery on my horizon. I went home and broke the news to my wife of ten years. At that time we had two young daughters, ages five and one, and something like this was not what we had imagined happening at this stage of our lives.

The very next day I was in Spokane, Washington at Sacred Heart Medical Center. Before I met with the surgeon I had an appointment with another cardiologist. He would perform a transesophageal (through the esophagus) echo to confirm Dr. Robinson's diagnosis of atrial mixoma. I was sedated with Versed (a wonderful drug that is made by the company I work for, Roche Laboratories; we call it "milk of amnesia" because of the pronounced amnesic effect the drug produces.) The procedure was uncomfortable but otherwise unremarkable. Much to our surprise there was no trace of the mixoma. Apparently there had been a large enough blood clot in the atrium, where blood pools and begins to coagulate during atrial fib that it had appeared on echo as a tumor. No mixoma was a good thing, but it was also very disconcerting to contemplate the implications of a large clot breaking loose and lodging in a remote section of my brain, causing a stroke. A stroke could potentially kill or severely incapacitate me for the rest of my life. I never saw the surgeon for the open-heart procedure, but at that point I began seeing a series of cardiologists. This had the effect of producing mostly anxiety and frustration with these so-called "specialists." Not one gave me much in the way of encouragement or real direction for treating or eliminating my atrial fibrillation.

The search for clues

My experience with a-fib was to be varied; I would sometimes have episodes daily, then I would go for months without a single one. I tried to detect patterns in my behavior or environment that would possibly set it off, even to the point of tracking my episodes and how they related to the different phases of the moon. I would think I had a clue, but then my hopes would be dashed days or weeks later when another episode occurred. I continued to be very active physically, taking part in sports such as running, cycling and weight lifting. But often I would have to curtail or cease my activity because of an a-fib attack. This on-again, off-again cycle continued for years. I would alternately get very aggressive in my search for answers to my questions on treatment, then become frustrated and disheartened with the response and leave it alone. My a-fib continued with increased frequency.

In April of 2000 I had LASIK surgery on my eyes to restore my vision to 20/20. I had worn contacts or glasses since kindergarten, and was thrilled with the prospect of not having to wear either. My surgery was successful and my vision post-procedure was 20/15, or even better than normal. What was even more astounding, however, was the fact that after my surgery I went over six months without a single episode of atrial fib. I don't know why this occurred, but I have a

theory. Back in 1990, one of the little tricks that Dr. Robinson told me about that could help stop an episode of atrial fib was to apply gentle pressure to my eyeballs (with my eyelids closed.) Apparently this could stimulate an adrenergic response that might bring a restoration to sinus rhythm. At that time I knew nothing of the differences between adrenergic or vagal causes of atrial fib, and the eyeball procedure did not ever work for me. However, after my eye surgery I began to think that perhaps there was a connection with wearing contact lenses and my atrial fib. What if the pressure on my eyes from the contacts was enough to precipitate an a-fib attack? Or, could I have been allergic to the contact lens solution? Either explanation seemed as reasonable as anything I had heard or any cause I had personally experienced.

I discussed this idea with my cardiologist and he almost began laughing. He said it was a silly notion and that I would be better off putting my energy toward having a pacemaker inserted than pursuing ideas like this. Needless to say, he is no longer my cardiologist.

Cardiologists and a-fib

My theory about eye pressure precipitating my a-fib may have been silly but as all atrial fibbers know, there are things that we know CAN precipitate an attack that cardiologists don't understand. Having a cardiologist tell us we are silly for suggesting ANY possible cause is an insult to our condition and our intelligence. Most cardiologists are technicians, and they deal with a biological pump that has many, many idiosyncrasies that even they don't fully understand. And as Hans Larsen has so clearly stated, for many of us atrial fib may not even be a cardiac problem; it may indeed be a nervous system disorder that has cardiac manifestations. But cardiologists are not trained to think like this, and it is the very rare doctor that will think outside his medical training box.

Let me pause here to offer a couple of caveats to my story and explain my seemingly disrespectful attitude toward the medical community in general and toward cardiologists specifically. In my profession I get to know hundreds of physicians very, very well. I take them to dinner, I play golf with them, I listen to them discuss their frustrations with medications, HMO's, their patients, and their practices. It is an enjoyable and challenging job, but it has its drawbacks. One of the more difficult aspects of this job is finding a physician you trust enough to treat your family and yourself. As is true in most professions, most doctors are competent - not great, but not dangerous either. Then there are outliers at either end of the competency spectrum; some are brilliant, and some are idiots that you wouldn't trust to work on your dog.

Nowhere is this spectrum of competency more evident than in cardiology, and this is evidenced in particular by their approach to the treatment of a-fib. I have had cardiologists tell me I am imagining my condition, or that I should just "ignore it and learn to live with it." One told me that I should have a complete ablation and have a pacemaker installed (this was at the ripe old age of 40). Another told me that atrial fibrillation was "the hemorrhoid of cardiology"—that it wasn't life threatening, but it was a nuisance, like a hemorrhoid, and that it carried about as much interest to a cardiologist as a hemorrhoid.

The search continues

As we know, atrial fib is much more than a nuisance. Many times I have had to literally stand on the sidelines of life with tears of frustration in my eyes. I have watched my kids or friends participate in sports or other activities that I could not take an active role in because I was in atrial fib and was dizzy and light-headed. I have had business trips thwarted, special events cancelled, family events altered, all because of my condition. This condition is not a nuisance—it is a life-altering threat to the enjoyment of life, and it is very difficult to find a physician willing to listen to our frustrations and to honestly assist us in our quest for answers and, hopefully, a solution.

I have learned over the years that I have many triggers for my a-fib. Sometimes just bending over to pick something up off the floor, or sitting down in my car would trigger an episode. At other times lying down in bed would start an episode, and sometimes lying down would end one. Laughter can be a trigger, as can any extreme emotion like anger or fear. Caffeine is a definite

trigger, and sugar can also be one. At one point I thought that I had food allergies that would act as triggers, but I no longer believe this.

I have a mouthful of silver amalgam fillings, and this is an area of treatment that I am focusing on right now. I am scheduled to begin having my fillings removed in early 2002, and a physician friend who specializes in environmental medicine told me to have the filling in my #15 molar removed first. He said that this tooth is on the cardiac meridian of the nervous system, and that a filling in this tooth can potentially cause heart problems. This would be in addition to the obvious potential problem of mercury leeching into my system from the fillings; something that I firmly believe is a serious problem and may potentially be the cause of my atrial fib.

My condition has recently taken a turn for the better. This positive turn, however, came only after a particularly low and discouraging period. Since about September of 2001 the frequency of my episodes had been increasing. I sometimes had multiple episodes a day. I had a drug regimen that would restore me to sinus rhythm most of the time; I would take 200 mg of quinidine every hour for up to five hours or until my a-fib converted. However, I found that I was taking it so often that I would sometimes lose track of my doses. I was worried about toxicity from overdosing, and the drug made me very nauseous and would actually become pro-arrhythmic at subtherapeutic doses. In other words, as the amount of drug in my system would taper off, the decreasing concentration would precipitate a new attack and the cycle would begin again. I was finding myself unable to work at the level I needed, and the disruption to my personal life was profound. Something had to be done.

Change in medication

I contacted a coworker in Spokane about finding a cardiologist who specialized in electrophysiology and atrial fib. He gave me the name of Dr. Michael Kwasman, and I met with him in October to review my condition. He told me to stop the quinidine, that class 1A antiarrhythmics such as quinidine are not used anymore. He told me to try to obtain current EKG's while I was in a-fib, and to take Tambocor 200mg as needed for my atrial fib episodes. Once we had some current data to work from, we'd decide on a plan of treatment.

I went for a few weeks without the need to take the Tambocor. I am very leery of the potential side effects of this drug, and I did not want to have to use it unless I absolutely had to. Not wanting to take the Tambocor seemed to keep me from having any new a-fib episodes, at least for a while. But one morning I went into a-fib for no apparent reason and all my little "tricks" failed to put me back into a sinus rhythm. I took the Tambocor and converted back to sinus rhythm in just a couple of hours. I felt like perhaps I had found something that could at least help me to carry on a normal life when a-fib occurred. I had to take it again the next day when my a-fib reoccurred, but this time it failed to convert me and I was left frustrated and wondering what my next move should be to get back into sinus rhythm.

I had taken the recommended dose of Tambocor with no positive result. I couldn't safely take the quinidine (which at least had worked most of the time) on top of the Tambocor, so what should I do? I have since discovered that Tambocor is a poor choice, at least for me. Apparently, Tambocor is most effective at keeping someone in sinus rhythm, not in converting someone to sinus rhythm from a-fib. After 24 hours in a-fib, I became very concerned. I had been told that if I went longer than 48 hours I would have to be put on Coumadin and possibly cardioverted, two things I did NOT want to have to do. In desperation, I went to the ER of my local hospital. The cardiologist on call, Dr. Iyad Jamali, was fairly new to my area. I did not know him well but I had heard good things about him from other local cardiologists.

Atrial flutter as well?

The cardiac monitor in the ER showed that I was indeed in a-fib, and after discussing options with me Dr. Jamali suggested we try to convert with a drug called Corvert. He said it was successful about 75% of the time. If Corvert failed we would have to move on to cardioversion. I was given one milligram of Corvert over 10 minutes, and within another 10 minutes (just as they were about

to give me another milligram) I converted to sinus rhythm. I felt an immediate difference when I converted, and I called Dr. Jamali in from the nurses' station to tell him. He said he wanted me to stay in the ER for a couple of hours for observation, but that I could probably go home at that time. After about an hour in a very stable sinus rhythm, I was served some lunch by the ER staff. My wife was sitting on a stool next to the exam table where I was propped up, and we shared the lunch. Within 5 minutes after eating I became tachycardic. My pulse raced up to about 140 beats per minute. Dr. Jamali came back in and administered a beta-blocker to slow my heart rate down. It worked very well, and within another 10 minutes my pulse was back at about 70 bpm. The monitor had been on during this episode, and examination of the strips revealed some very interesting facts.

First, I had had several PAC's (premature atrial contractions) prior to the episode of tachycardia. The strips from my a-fib episode also revealed atrial flutter, something I did not know occurred. One of my cardiology textbooks states that atrial flutter "converts" to either sinus rhythm or to atrial fib. Dr. Jamali speculated that adrenaline from my meal had triggered some PAC's which in turn triggered atrial flutter, which then converted to atrial fib. Interrupting the cycle of PAC's to atrial flutter could be done easily with a beta blocker, and I was admitted to CICU for the night for observation and to be titrated and stabilized on an oral beta blocker.

A glimmer of hope

This was an extremely low point in my history with atrial fibrillation. At 45, I was in CICU and I had no idea when I would be released. If I had another episode of tachycardia I could be there for days while tests were run and different drugs were tried. I did have a glimmer of hope, however. If the cascade of events as described by Dr. Jamali was correct—adrenaline, followed by PAC's, then by atrial flutter, and finally by atrial fib—then there were things that could be done to interrupt that cascade of events and potentially halt the atrial fib. Beta-blockers are used to stem the onslaught of adrenaline by reducing the sensitivity of the cardiac physiology. My wife is a pianist who takes a beta-blocker on occasion before a performance. A beta-blocker helps to normalize the heart rate and can prevent a runaway rapid heart rate. Potentially, in my case a low dose of a beta-blocker could prevent the cascade from starting.

Also, atrial flutter can be ablated much easier than atrial fib. In my case, if the auxiliary electrical pathway that the atrial flutter was using could be destroyed then that errant electrical impulse could be prevented from deteriorating into atrial fib.

I am currently at twenty-five days post-hospitalization, and I have had a wonderful few weeks. I am taking 25 mg of metoprolol, a beta-blocker, twice a day. I have had no negative side effects from this dose but the positive effect on my a-fib has been amazing. I have been totally free from flutter, something I have since identified as the precursor to my a-fib, and I almost feel "normal." I am exercising vigorously again without any problems, and my resting heart rate is about 50 bpm. I have an appointment scheduled with my cardiologist for next month. At that time we will discuss the feasibility of ablation for the flutter. He tells me that the beta-blocker will not prevent my a-fib. From a purely physiological point of view he is correct; a beta-blocker has no benefit in pure atrial fibrillation. However, if the cascade of events I have described is correct then the beta-blocker should be very effective in preventing the atrial flutter, which deteriorates into a-fib. It is early still, but so far it seems that this theory is correct.

I have even "pushed" myself over the past few weeks to see if this theory is really working, and I have not been able to put myself into a-fib. Last weekend I ran with one of my daughters in a fun-run. It was only a mile, but it was something I would have been unable to do "a-fib free" even last month. It was great running and being able to concentrate on the race itself, rather than on my heart rhythm.

I don't know if this is the final answer to my particular problem with a-fib, but I intend to continue to pursue a solution. I will have my fillings removed regardless of what my cardiologist recommends and before I undergo any ablative procedure. The fillings may be causing errant

electrical impulses that are causing my PAC's and atrial flutter, the impulses currently controlled by the beta-blocker. Removing those fillings and then testing for current may prove as effective as undergoing an ablative procedure. And I will continue to read, hope and pray that I may one day claim victory over this elusive, dastardly enemy.

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