THE AFIB REPORT
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My Story – A Work in Progress

By Randy Lewis

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 light-headed. 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 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 humour 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 tumour 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 tumour 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 the diagnosis of atrial mixoma. 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 tumour. 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.

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 behaviour 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, HMOs, 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 – maybe not great, but not dangerous either.

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 co-worker in Spokane about finding a cardiologist who specialized in electrophysiology and atrial fib. 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? 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.

Atrial flutter as well?

The cardiac monitor in the ER showed that I was indeed in a-fib, and after discussing my options the cardiologist on call 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 the cardiologist 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. I was then 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 PACs (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. The cardiologist speculated that adrenaline from my meal had triggered some PACs which in turn triggered atrial flutter, which then converted to atrial fib. Interrupting the cycle of PACs 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 the cardiologist 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 PACs 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.

Washington, January 2002

Postscript – November 2002

Through most of 2002, until about August, I continued my cycle of on-again, off-again atrial fib and flutter. Most of my EKGs showed flutter, and it is a very uncomfortable arrhythmia. As a physician friend said, flutter has symptoms and atrial fib does not. While the statement is not entirely true in my case, it does illustrate somewhat the different physical manifestations of the two arrhythmias. The flutter would sometimes deteriorate into atrial fib, and sometimes it would convert back to a normal sinus rhythm.

During this time period I had all of my mercury amalgam fillings removed, but the net effect on my arrhythmias was nil.

Unfortunately, as the year progressed my episodes became more and more frequent, occurring on almost a daily basis. I saw my EP in September and told him I would like to have an ablation procedure for the atrial flutter. My rationale, and he agreed, was that by ablating the flutter we may be able to remove one of the probable triggers for my atrial fib. At the very least, it would stop a very uncomfortable arrhythmia and allow me to focus on management of my atrial fib. This physician does the ablation for atrial fib as well, but I made the decision to wait to have that done until the ultrasound or similar technique is perfected, rather than take the risk of stenosis and secondary pulmonary hypertension with the radiofrequency technique. Again, he agreed.

I had a successful ablation for atrial flutter at the end of October. The two weeks since the procedure have been very interesting; I have had periods of frequent PAC activity and some atrial fib, and I have had several days of calm, "normal" heart rhythm. Overall, I am pleased with the results of my procedure.

I am now focusing on dealing with my vagal afib and have been using Tambocor again to convert to sinus rhythm (on-demand approach), this time with great success.

When and if a suitable procedure is perfected to the point where they can claim a 95% or greater success rate, I will probably have a second ablative procedure for my atrial fib. Until then, I will continue to attempt to solve the puzzle that is atrial fib. Many thanks to Hans Larsen and all those who contribute to The AFIB Report and the Forum; with persistence and continued tenacity, a solution to our condition will be found, of that I am sure.

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