



The AFIB Mystery

By Andrew Auerbach Ph.D.

I was diagnosed with lone paroxysmal atrial fibrillation in January of 2001 on a routine physical, which included an EKG. This term was totally unfamiliar to me and I diligently went about exploring the Internet and medical textbooks to find out what was the nature of this beast. On the day of the physical I had experienced a short chemical exposure and was unsure as to whether this contributed to the problem. There was some evidence on the Internet for AF being chemically induced in some instances (e.g. alcohol or solvents). The AF incident did go away in a day and I was guardedly optimistic that this was the end of my troubles. My family physician was sceptical about a chemical cause and put me on Toprol XL (25 mg), an extended action beta-blocker. Shortly after I did have a recurrence and was forced to search for a more fundamental cause. I noted in the articles I captured on the web that AFIB was initiated by the firing of a series of nerve cells near or in the pulmonary veins that caused a disruption in the normal sinus rhythm (NSR). E. A. Butler's book on Atrial Fibrillation started my journey on the use of natural supplements for the treatment of AFIB such as magnesium, vitamin C, vitamin E, fish oil, niacin, folic acid, hawthorn, CoQ10, garlic oil and a regular one-a-day vitamin tablet. None of these supplements seemed capable of arresting my AFIB incidents, which were now appearing with frightening regularity of one to two times a week and lasting about 36 hours. I noticed that I was entering a manic (no AFIB)/depressive (AFIB) state and was very anxious if I was engaging in activities that could lead to an incident. These activities included exercise, stress, drinking coffee or any other caffeine-containing compound, or drinking alcohol. Typically the incident would happen after dinner when I was subject to a vagal type episode. I started reading The AFIB Report on line and found this to be a tremendous source of information and advice. Thank you Hans!

The Physician's Solution

My initial experience with cardiologists was not a pleasant one and these experiences shaped my later actions. Toprol XL (50 mg) was not merely ineffective it was lowering my pulse rate and intensity to dangerous levels. I was frequently afraid of passing out at work. My system has always been very sensitive to stimulants (e.g. caffeine) and this sensitivity carried over to these pharmaceutical treatments. My condition deteriorated into atrial flutter, which is much more persistent and harder to induce back into NSR. I was given Betapace™ (sotalol) in the hospital to help bring me back to normal rhythm. While this was effective, Betapace proved to have an unexpected and very unpleasant side effect; it would temporarily stop my heartbeat and I was required to cough to start it up again. Needless to say, this had a very traumatic effect on my outlook on drug therapy. This was followed by use of quinidine, which made me extremely dizzy and subject to passing out. After that incident I tried to stay with a mild beta-blocker (propranolol) at low levels (10 mg) and just relied on supplements and lifestyle changes. This was not effective and I was typically in AFIB at least once a week. This situation deteriorated when I went into atrial flutter again for a prolonged period of time.

I went to an electrophysiologist (ep1) who ascribed my drug sensitivity to a pre-sick sinus syndrome and pronounced that a pacemaker and an ablation for the flutter were in the cards for me. Drug therapy would then be used to control the condition after these procedures. Use of surgical intervention for AFIB was considered too experimental by ep1, but he believed that it would be ready for general use in 5-10 years. This was not the news that I had been hoping for and I was put off by his very aggressive form of treatment. Electrophysiologist 2 disagreed with the treatment plan of ep1 and suggested that I go on Coumadin prior to getting a cardiac conversion from the atrial flutter. This would be followed by treatment with flecainide/propranolol to control my condition. To my surprise the conversion worked without a hitch and the flecainide/propranolol combination has left me AFIB and Aflutter free. While the drugs do cause a general lowering of my energy level I have tried to back off on the dosage levels so as to be able to tolerate these drugs better. I felt a tremendous sense of relief that I could resume my normal exercise levels and not check my pulse every ten minutes. I have gone from being a reclusive hermit to a functioning member of society again. While this drug is not a cure it has arrested my incidents of AFIB for over 4 months. I am removing Coumadin from my life and substituting aspirin.

The Mystery Continues

I am still left with nagging questions on AFIB. Are there natural substitutes for flecainide, which is a dangerous drug? Will the continual use of this drug lead to other problems? Can some natural substitute be used to desensitize the nerve cells to stimulation? Will surgery some day provide a safe, effective cure for AFIB? Are dental amalgams causing some of this problem?

I was distressed that my journey with the medical establishment took so many twists and turns. Why can't one recommended medication be prescribed in the beginning without subjecting the patient to countless ups and downs and threatening situations?

Only by sharing our successes through publications such as The AFIB Report can we share our collective experiences and find an individual solution to this problem. Lone AFIB is a different beast than fibrillation caused by an underlying cardiac condition and the medical establishment has to be made more sensitive to the use of more selective treatments for lone AFIB.

I feel blessed that I have been able to come back from the abyss, but I fear the dark shadows that sometimes creep into my consciousness. I pray the battle is won for now and look to the future for a real cure to this disorder.

California, December 2001

Postscript – October 2002

Over the past year I have experienced 2 afib episodes. They both required cardioversion because of the persistence of my atrial flutter. Both incidents were vagal in nature and I was able to relate them to specific triggers (low medication, late night meal out). Working together we can, hopefully, get the answers we need in a reasonable time frame.

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