

THE AFIB REPORT

Your Premier Information Resource for Lone Atrial Fibrillation!

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2nd YEAR



Editorial

Welcome to our 2nd year of publication. The year is beginning with some great news! The Cleveland Clinic has confirmed the connection between inflammation and atrial fibrillation. They tested 131 patients with atrial fibrillation (70 with LAF) for C-reactive protein (a marker of systemic inflammation) either during an episode or within 24 hours of converting to sinus rhythm. The average level of C-reactive protein was more than twice as high among AF patients as among healthy controls. The Cleveland Clinic report concludes that elevated C-reactive protein levels “may reflect an inflammatory state that promotes the persistence of AF”. These findings support my own (reported in the September 2001 issue of *The AFIB Report*) and bring new hope that it will be possible to eliminate or at least control LAF by finding a way of eliminating or controlling the underlying inflammation – the goal of the anti-inflammatory protocol.

More great news! The AFIB Report now has its very own home www.afibbers.org. We found it increasingly cumbersome and complicated to maintain all the afib information as part of the International Health News website so decided to set up a separate one just for afibbers. It has also become obvious that the LAF Forum Bulletin Board is becoming overloaded with now over 1800 postings! So to ease congestion and make it easier to find things we have set up a completely new board at www.afibbers.org. The old board will still be available for archival purposes, but please use the new one for posting future messages.

*Yours in health – and sinus rhythm,
Hans Larsen, Editor*

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LAF and Heart Rate Variability

A steady heart beat, as all afibbers will heartily attest to, is indeed a blessing. Normally the heart beats at a rate of 60 to 80 beats per minute, but the rate can be as low as 50 (in trained athletes) or as high as 100 (during illness or in a highly emotional state) without it being cause for concern. The heart rate is a measure of the number of contractions made by the left ventricle in a one-minute period and is usually measured on the radial artery near the wrist or on the carotid artery in the neck.

So while the heart rate itself has long been known to be an indicator of the physical and psychological condition of an individual it is only recently that attention has focused on the significance of the beat to

beat variability. Research has shown that the interval between one heart beat and the next varies and that this variation is a powerful indicator of the state of the autonomic nervous system.

Measurement of heart rate variability

The variation in the heart beat interval is usually measured via a 5-minute electrocardiogram or a 24-hour Holter monitoring. The original and still commonly used measure for the variation is referred to as SDNN which is the standard deviation of the heart beat intervals, that is, the square root of the variance. Powerful and fast calculation techniques developed in the late 1960s made it possible to extract more sophisticated and valuable information from the heart rate recordings. Most scientific work on heart rate variability now uses power spectral density (PSD) analysis to relate the relatively simple measurement of beat to beat variability to the state of the autonomic nervous system (ANS). PSD analysis uses a mathematical technique (fast Fourier transform) to determine how the power (variance in heart beat interval) is distributed across different frequency bands. There is now general agreement that the power in the low frequency band (LF) from 0.04 to 0.15 Hz (cycles/second) is an indication of sympathetic (adrenergic) branch activity and that the power in the high frequency band (HF) from 0.15 to 0.40 Hz is primarily an indication of parasympathetic (vagal) activity. It follows that the ratio of LF/HF is a measure of the balance of the autonomic nervous system with a higher number indicating an excess of adrenergic activity and a lower number indicating an excess of vagal activity. The total power (TP) in the PSD analysis, that is the power obtained by considering the whole frequency spectrum, is another important measure of heart rate variability.

Analysis of heart rate variability is still in its infancy, but normal values are believed to fall within the following ranges:[1]

- Total Power (TP) – 2450 to 4500 milliseconds squared (ms^2)
- Low Frequency Power (LF) – 750 to 1600 milliseconds squared
- High Frequency Power (HF) – 770 to 1200 milliseconds squared
- LF/HF ratio – 1.5 to 2.0

Significance of heart rate variability

Not surprisingly, heart rate variability analysis was first used as a measure of heart health. In 1996 researchers involved in the Framingham Heart Study concluded that a lower than normal heart rate variability (HRV) was associated with an increased risk of angina, heart attack, and other cardiac events[2]. Other researchers have found correlations between low HRV and the risk of neuropathy in diabetics and between low HRV and depression[1,3]. There is also a very significant association between low HRV and the risk of dying after suffering a heart attack. It is postulated that decreased HRV correlates with increased sympathetic (adrenergic) or decreased vagal tone, which may predispose to ventricular fibrillation[4]. British researchers report that reduced HRV is a risk factor for atherosclerosis in both diabetic and non-diabetic patients[5]. A group of American and Dutch researchers recently concluded that low HRV not only increases the risk of heart disease especially death from a heart attack, but may indeed precede a number of different diseases. As a matter of fact, they make the profound statement that “sympathetic predominance, as reflected in low HRV and high heart rate, may be indicative of less favorable general health, with HRV being a more sensitive indicator than heart rate”. The Researchers also point out the HRV decreases with age, high insulin levels, physical inactivity, smoking, and rapid and shallow breathing[6].

It is clear that low HRV is a predictor of poor health and an increased risk for cardiac death. Does this mean that high HRV is beneficial? Not necessarily. A recent study carried out by Dutch researchers clearly shows that elderly people with a very high HRV have an even higher risk for sudden cardiac death than do elderly people with a very low HRV. So it would seem that there is an optimum range, the exact limits of which still need to be determined[7].

HRV and atrial fibrillation

HRV analysis has been used extensively in the study of atrial fibrillation. LAF episodes can be divided into two groups; those that are preceded by an increase in LF power and a decrease in HF power consistent with an increase in sympathetic (adrenergic) tone, and those that are preceded by a decrease

in LF power and an increase in HF power consistent with an increase in parasympathetic (vagal) tone. The changes in HRV are apparent at least 5 minutes before the actual episode[8,9]. It is also clear that most vagal afibbers have no underlying heart disease while adrenergic type afibbers often do. There is also considerable evidence that the number of ectopic (premature) beats increases prior to an episode in patients with lone atrial fibrillation[10].

Atrial fibrillation patients with focal ectopy originating from the pulmonary veins experience a significant increase in HF power and a decrease in LF power during the 20 minutes preceding an episode[11]. This may mean that focal AF is primarily found among vagal afibbers raising the interesting question "Is radio-frequency ablation likely to be more beneficial for vagal afibbers than for adrenergic ones?"

Other research has established that afibbers who have undergone cardioversion for atrial fibrillation are twice as likely to have another episode if their LF/HF ratio is high[12,13].

External influences on HRV

It is clear that the autonomic nervous system is dysfunctional in lone atrial fibrillation patients and that the extent of this dysfunction manifests itself in several measures of HRV. HRV is highly influenced by emotional state. Anger and anxiety increases LF power while depression reduces overall HRV[3]. Alcohol consumption increases the LF/HF ratio and the blood level of norepinephrine[14]. Exposure to air pollution (particulates) may reduce HRV[15]. Physical exercise, on the other hand, has been found to increase HRV[16,17]. The ingestion of sugar (glucose) is associated with a significant increase in the LF/HF ratio[18]. Melatonin increases vagal tone (HF power) in men when they are lying down so this may not be the greatest supplement for vagal afibbers[19].

Sleeping position can also affect HRV. Sleeping on the right side increases HF power and minimizes LF power leading to a low LF/HF ratio; norepinephrine secretion is also minimized when lying on the right side[20,21]. This could mean that adrenergic afibbers should preferably sleep on their right side while vagal afibbers may do better on their left side.

The link between exposure to electromagnetic radiation (EMF) and HRV is still controversial. Some studies have found a link while others have not. Researchers at the Midwest Research Institute in Kansas City recently concluded that changes in HRV may occur when exposure to electromagnetic fields is combined with stress[22]. Does this mean that your HRV could change if you sit in front of your computer screen while under stress? It is possible, but there is no evidence one way or another that I know of.

Russian researchers believe that abnormalities in the structure of the spine particularly around the seventh and eighth cervical or the first thoracic segments can result in irritation of the sympathetic nerve bundles passing from the spine into the inner body. This, in turn, could affect the sympathetic side of the autonomic nervous system and cause changes in HRV[23]. This phenomenon may explain why some afibbers, presumably adrenergic, have gotten relief from chiropractic manipulations.

Pharmaceutical drugs can also affect HRV. Propafenone (Rythmol) has been found to decrease HRV and LF/HF ratio and flecainide (Tambocor) decreased HRV in heart attack patients[1]. ACE inhibitors (quinapril) and beta-blockers (metoprolol) both increased HF power and decreased LF power and LF/HF ratio in heart attack patients. It is interesting that the effect of quinapril was most pronounced between 2 and 4 AM, 8 and 11 AM, and 7 and 10 PM. Metoprolol was most effective between 8 AM and 12 noon and between 7 and 10 PM[24].

Obviously there are many external factors that may affect HRV. The question then is what can be done to control HRV and prevent imbalances?

Control of HRV

Breathing oxygen-fortified air may be helpful for adrenergic afibbers, but should not be attempted except with the cooperation of a physician. Research has shown that breathing air containing 31% oxygen markedly increases HF power and reduces LF/HF ratio in congestive heart failure patients[25].

Hyperbaric oxygen therapy is also effective in increasing HF power and reducing LF power and the LF/HF ratio in both healthy subjects and in heart disease and stroke patients[26]. These effects are only temporary, but could perhaps be useful in aborting an impending episode. For longer term effects it seems that the mind itself must be engaged. Transcendental meditation has been found to reduce norepinephrine levels and is probably doing this by reducing LF (adrenergic) power[27]. The two most promising techniques for achieving HRV control would appear to be the HeartMath techniques and Thought Field Therapy (TFT).

HeartMath techniques

Rollin McCraty and his colleagues at the HeartMath Institute in California (www.heartmath.com) have found that emotions are strongly related to HRV. Both anger (negative emotion) and appreciation (positive emotion) increase HRV, but only anger increases the LF/HF ratio as well[28]. McCraty also found that sincere appreciation can significantly increase DHEA levels while decreasing cortisol levels[29]. The HeartMath techniques balance the autonomic nervous system (ANS) and achieve a state of coherence between the brain and the heart. The Freeze-Frame technique deals with immediate disturbances in the ANS while the Heart Lock-In technique is aimed at establishing coherence and balancing the ANS on a long-term basis. Both techniques are very simple.

The idea behind Freeze-Frame is to recognize the stressful feeling, shifting your focus to the heart region, and then recalling a positive feeling or experience to “crowd out” the stressful feeling[29].

The Heart Lock-In technique is meant to be practiced on a daily basis. It is quite similar to meditation and has 5 steps much like the Freeze-Frame technique. You can find the details at (www.heartmath.org/public_service_techniques.html).

The HeartMath techniques are extremely powerful. A clinical trial carried out at the Kaiser Permanente Clinic found that 60 out of 75 atrial fibrillation patients improved markedly and 14 were able to get off all antiarrhythmic medications after practicing the two HeartMath techniques[30].

The HeartMath Institute has a computer program available that will help evaluate progress in using the techniques (www.freezeframer.com). However, the program is not necessary in order to apply the techniques effectively.

Thought Field Therapy

Thought Field Therapy (TFT) was developed by Dr. Roger Callahan about 20 years ago (www.tftrx.com). It is based on the observation that many psychological and even some physical disorders have their origins in negative thought patterns. The purpose of TFT is to eliminate these thought patterns. The technique involves the stimulation of a sequence of acupuncture points by tapping on them while focusing on the emotion created by thinking about the problem. The precise sequence of tapping depends on the nature of the problem to be eliminated. Tapping on an acupuncture point delivers mechanical energy to the point which is converted to electrical energy which travels along the appropriate meridians and in doing so collapses the negative thought patterns and thereby elicits healing. TFT is now practiced by thousands of psychologists and other health practitioners around the world and, according to its proponents and healed patients, is highly effective.

Dr. Callahan and other TFT practitioners recently released a series of papers discussing the relationship between TFT and heart rate variability. Their work shows that resolution of a problem automatically brings with it a beneficial change in HRV, that is, excessively low HRVs are increased and excessively high HRVs are decreased[31,32]. Dr. Callahan presents a series of 20 cases where the correlation between a successful TFT session and a beneficial change in HRV is clearly demonstrated[31]. Drs. Pignotti and Steinberg discuss 39 cases involving various disorders such as anxiety, depression, fatigue, attention deficit hyperactivity, obsessions, and anger. All cases were successfully treated with TFT and their resolution was accompanied by a beneficial change in HRV. One of the cases involved a 60-year-old man with atrial fibrillation. Prior to treatment his HRV was excessively high. After treatment it decreased to the normal range and the overall balance of the autonomic nervous system was significantly improved[33].

A very large study of TFT was carried out at Kaiser Behavioral Medicine and Behavioral Health Services in Honolulu. A total of 714 patients were treated for 31 different types of psychological problems. Improvements were observed in all 31 categories and were accompanied by improved HRV and autonomic system balance[34]. Relief workers in Kosovo reported a very high rate of success (247 of 249 treatments) using TFT to treat various traumas resulting from the war and ethnic cleansing[35].

Although the evidence for effectiveness of TFT and its strong correlation with HRV improvement is almost overwhelming the technique is still rejected by the “psychology establishment”[36-38].

TFT has spawned other similar but simpler techniques for problem resolution through tapping on acupuncture points. The “Emotional Freedom Technique” (EFT) (www.emofree.com) was developed by Gary Craig, an electrical engineer. EFT differs from TFT in that it uses a standard tapping sequence on 12 acupuncture points irrespective of the nature of the problem to be solved. EFT is very easy to learn (from a skilled practitioner) and is purported to be highly effective. I certainly intend to look into it further as a possible treatment for LAF.

Conclusion

It is clear there is a strong association between a dysfunctional autonomic nervous system (ANS) and lone atrial fibrillation. Analysis of heart rate variability (HRV) is an excellent method for determining the degree and direction of the ANS abnormality. HRV, in turn, is affected by many external factors and, it would appear, can be beneficially changed by the use of new energy psychology techniques such as Freeze-Frame, Heart-Lock-in, TFT and EFT.

Note: HeartMath, Freeze Frame, and HeartLock-In are registered trademarks of the Institute of HeartMath. Freeze Framer is a trademark of Quantum Intech.

Personal Update on Anti-Inflammation Protocol – Part II

The road to success is indeed fraught with many and varied hurdles! As reported in the December editorial I temporarily discontinued the anti-inflammatory protocol on November 29th. This was done for three reasons:

- I wanted to see what would happen if I went off the protocol.
- I was due to have a series of blood tests on December 3rd and wanted to make sure that the results were not affected by the protocol supplements.
- I wanted to do some more research on the supplements to make sure they had no long-term adverse effects.

Well, on December 4th I got my answer in the form of a 60-hour episode! This was followed by a 25-hour one on December 10th. So I now have the answer to what would happen if I went off the protocol!

In the meantime, I have heard from several afibbers who have found MSM (methyl sulfonyl methane) helpful. MSM has strong anti-inflammatory properties and is a cholinesterase inhibitor meaning that it enhances parasympathetic activity – just the ticket for adrenergic afibbers, but possibly not so great for vagal ones. MSM also crosses the blood/brain barrier and is reputed to bind to mercury and help excrete it. Sounded like a winner, so I decided to add 1000 mg of MSM (taken with breakfast) to the 3 capsules of Moducare (taken ½ hour before main meals).

I had also come across some very interesting information on American ginseng (*Panax quinquefolius*). Apparently American ginseng not only helps keep blood sugar levels under control, it also partially blocks sodium channels. This effect is similar to that of antiarrhythmics such as flecainide (Tambocor) and propafenone (Rythmol). Seemed like a promising candidate for the protocol so I decided to take 500 mg

with breakfast. I should point out that only American ginseng has the above effects – Korean, Siberian and Chinese ginseng do not.

My personal anti-inflammatory protocol now consists of 3 Moducare capsules per day plus 1000 mg of MSM and 500 mg of American ginseng. I want to emphasize that I have the adrenergic form of LAF and MSM may not be good for the vagal kind. I began the new protocol on December 15th and within 4 days it had completely eliminated ectopic beats and produced a very satisfactory HRV graph on the FreezeFramer. I have found no evidence that any of the components of the protocol have any long-term detrimental effects.

The Holiday Season also gave me the opportunity to confirm that I do indeed have a serious reaction to wheat-containing foods. I normally avoid them, but with all the special cakes and cookies on offer during the holidays I confess I did “slip”. Invariably I would end up with numerous ectopic beats about 12 hours later. So wheat in all its many forms is definitely no good for me.

That’s all for now. I will continue to keep you posted of my progress.

The AFIB Mystery

by Andrew Auerbach, Ph.D. (daauerbach@msn.com)

First Contact

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 skeptical 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 vein 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 per 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 their 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.

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