

THE AFIB REPORT

Your Premier Information Resource for Lone Atrial Fibrillation!

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The continuing evaluation of the wealth of data accumulated during the third LAF survey brought to light at least three very interesting findings:

- There is a strong correlation between the flare-up of GERD (heartburn, acid reflux) and the beginning of an episode.
- There may be an abnormally high incidence of tonsillectomy among lone afibbers. Having had your tonsils removed would certainly affect your immune system performance, which again, might predispose to afib episodes. This is pure speculation on my part so far, but a most intriguing subject for further research.
- There may be a significant genetic connection in lone afib. Our survey data strongly supports this idea as does research carried out by a team of American and Spanish researchers. Another fascinating area for more research.

I have now experienced paroxysmal lone afib (adrenergic variety) for over 12 years. I have had a lot of experience with episodes – over a hundred of them. I have done much research on LAF and I have read and learned a lot from the almost 5000 postings on the www.afibbers.org Bulletin Board. I am becoming more and more convinced that we are unlikely to find a magic cure that applies to everyone. This, irrespective of a possible genetic connection, does not mean that there is no cure, but rather that the ultimate cure may be different for each of us. We have examples of afibbers who dramatically reduced if not eliminated their episodes by removal of their amalgam dental fillings, by correcting a magnesium deficiency, by changing their diet, by adjusting their omega-3 to omega-6 fatty acid ratio or by boosting their immune system by supplementing with Moducare.

I believe it is clear that, despite the possible genetic link, LAF is not a typical independent disease entity as such, but primarily a manifestation of some imbalance in the body. The purpose of The AFIB Report and the Bulletin Board is to bring you a “smorgasbord” of possible solutions that you can explore and hopefully find one that works for you.

Yours in health and sinus rhythm,
Hans Larsen

Table of Contents

- A. Findings from LAFS III – Part 2
- B. LAF: The Diagnosis
 - Causes of atrial fibrillation
 - Initial evaluation
 - Blood tests
 - Electrocardiogram
 - Holter monitoring
 - Treadmill testing
 - Echocardiography
 - Advanced tests
 - Need for stroke prevention

A. Findings from LAFS III – Part 2

In this issue we continue the tabulation of the answers to the questions contained in the May 2002 LAF survey.

16. Do you suffer from acid reflux or have you ever been diagnosed with GERD (gastroesophageal reflux disease)?

I actually covered the responses to this question in the August issue of The AFIB Report. However, it became clear that it was not possible to draw a

conclusion as to whether or not afibbers were more likely to have GERD than was the “normal” population because I had not asked about the frequency of GERD episodes. I have now received the responses to my supplementary questionnaire and can report that 10% of respondents have daily episodes, 14% have weekly, and 10% have monthly episodes for a total of 34%. This compares to the following values reported in the literature: for a random group of 1004 individuals – daily episodes = 11%, weekly (one or more per week, but not daily) = 12%, and monthly episodes = 15% for a total of 38%[8]. Thus it would not appear that afibbers are significantly different from the rest of the population when it comes to the incidence of GERD. I found no correlation between H. pylori eradication and GERD incidence or between childhood antibiotics exposure and GERD.

18. Have you noticed any correlation between episodes and a flare-up of GERD or a bowel disorder?

Twenty-six (41%) of the 63 respondents reporting either GERD (including heartburn and reflux) or a bowel disorder had noticed a correlation between a flare-up of their condition and worsening LAF symptoms. By far the most common correlation was between GERD and the initiation of an episode or a worsening of chronic symptoms. Eighteen of the 26 respondents (69%) reported this correlation while 3 respondents (12%) felt there was a connection with an IBS flare-up. Adding this evidence to the finding that some afibbers with GERD or reflux symptoms have found relief by taking the prescription medicine Nexium would indicate the GERD could be an important trigger for LAF and its elimination could materially improve the condition of some afibbers. More about the GERD connection in a future issue.

19. Have you ever taken tranquilizers (valium, ativan, xanax, etc.) for an extended period of time?

Eleven or 11% of respondents answered yes to this question (25% adrenergic, 17% mixed, 11% chronic, and 2% vagal). It is interesting to note the difference in tranquilizer use between adrenergic and vagal afibbers. It is likely that tranquilizers would tend to benefit adrenergic afibbers more because of their greater level of sympathetic nervous activity and accompanying anxiety. Considering that more than 60 million prescriptions are written every year for minor tranquilizers in the United States alone a use rate of 11% is probably normal and does not support the idea that past tranquilizer use could be an important cause of LAF. Respondents who had used tranquilizers were more likely to be current smokers, to have a high CRP level, and to have had a dysfunctional childhood ($r=0.38$ $p=0.0001$).

20. Do you feel more upbeat on a sunny day (high barometric pressure) than on a rainy day (low barometric pressure)?

One respondent answered yes with the comment “Doesn't everyone?” Well as a matter of fact not all afibbers feel more upbeat on a sunny day (high barometric pressure). Fifty-five (62%) of the 89 respondents who answered this question did answer yes (91% adrenergic, 70% mixed, 59% chronic, and 50% vagal). However, a sizeable proportion (38%) did not feel more upbeat on a sunny day (50% vagal, 41% chronic, 30% mixed, 9% adrenergic). Research has shown that cortisol levels are lower at high barometric pressures and that lower levels are associated with a lessening of depression[1,2]. So conceivably a person with elevated cortisol levels might feel better on a sunny day. A person who already has an excessively low level may feel worse because low cortisol levels diminish the ability to handle stress. At this point all this is pure speculation though. Obviously more research is required on a potential cortisol/LAF connection. There was tendency for women to be more likely to feel upbeat on a sunny day.

22. Have you had your tonsils removed?

Sixty or 58% of respondents had had their tonsils removed (72% chronic, 62% mixed, 53% vagal, and 42% adrenergic). Tonsils are an integral part of the immune system and serve an important function in fighting infections. During this process they can become inflamed and sometimes infected. When this happens they are often surgically removed (tonsillectomy). The rate of tonsillectomy in the United States in 1996 was 55 per 10,000 population or 0.55%[3]. Most tonsils are removed before the age of 15 years so applying the 0.55% annual removal rate over 15 years would give a total “lifetime” removal rate of 8%. This estimated rate is clearly significantly lower than that experienced among our survey participants. If it is correct and comparable then having a tonsillectomy could be an important cause of the development of LAF. If this is indeed so, then taking steps to make up for the loss of ones tonsils may be beneficial. But that, as Hans Christian Andersen would say, “Is another story” which we will explore in a future issue.

There was a significant correlation between present age (and age at diagnosis) and the likelihood of having had a tonsillectomy. Older people were more likely to have had one than were younger people indicating that the practice is becoming much less prevalent.

23. Do you consider yourself sedentary, somewhat active or highly active and in strong physical shape?

Forty (38%) of the 105 respondents considered themselves to be highly active and in strong physical shape (50% adrenergic, 43% vagal, 39% chronic, and 24% mixed). Fifty-six (53%) considered themselves somewhat active (62% mixed, 50% vagal, 50% adrenergic, and 50% chronic). Only 9 or 9% described themselves as being sedentary, again confirming that afibbers tend to lead a healthy lifestyle. Men tended to be more physically active than women. Afibbers who were highly active were also more likely to have engaged in strenuous physical activity in the past. Highly active afibbers were less likely to have an autoimmune disease.

24. Do you or did you in the past engage in strenuous physical activity for extended periods (longer than 40 minutes at a time)?

Research has shown that regularly engaging in vigorous exercise for 40 minutes or more will raise cortisol levels. Eighty-three (79%) of the respondents answered yes to this question (92% adrenergic, 87% vagal, 69% mixed, and 67% chronic) again proving that afibbers are highly active people. The level of strenuous activity did decline with age and was higher among men than among women.

25. Do you regularly supplement with vitamin E (alpha-tocopherol)?

Ninety-two (43%) of the 212 respondents who answered this question answered yes (62% chronic, 54% adrenergic, 47% mixed, and 31% vagal). The use of vitamin E increased sharply with age ($r=0.44$ $p=0.0001$) and people taking beta-carotene were also more likely to take vitamin E. There was no indication that vitamin E supplementation was either beneficial or detrimental. However, as vitamin E supplementation is very strongly associated with age a more sophisticated statistical analysis is required in order to say for certain. Vitamin E has been found beneficial in the prevention of many conditions so supplementation is a good idea especially if using a mixed tocopherol preparation rich in gamma-tocopherol. The most common daily dosage of vitamin E was 400 IU ranging from 200 to 1600 IU/day and some respondents had been supplementing for 30 years or more.

27. Do you regularly supplement with beta-carotene?

Forty (20%) of the 204 respondents answering this took beta-carotene (29% chronic, 24% adrenergic, 19% vagal, and 13% mixed). Most got the beta-carotene from a multivitamin so the most common dosage was 15,000 IU/day ranging from 7500 to 30,000 IU/day. Some users had supplemented for as long as 30 years. People who supplemented with beta-carotene (multivitamin?) were more likely to have a bowel disorder. There was no indication that beta-carotene supplementation was either beneficial or detrimental in regard to episode severity.

29. When your heart beat is irregular which pattern would best describe it?

Unfortunately, this question was not clear. What I meant to enquire about was the pattern of irregular beats during otherwise normal sinus rhythm. Many respondents, however, reported their heart beat pattern during fibrillation so the answers are not meaningful.

30. Has anyone else in your close family (parents, grandparents, siblings) been diagnosed with arrhythmias?

Forty-three or 43% of respondents had a close relative with cardiac arrhythmia (54% mixed, 44% chronic, 40% vagal, and 25% adrenergic). The most common "carriers" were the mother who accounted for 13 of the relatives (30%), siblings who accounted for 11 (26%), the father who accounted for 10 (23%), and grandparents who accounted for 3 (7%). Chronic afibbers reported the mother to be the "carrier" in 71% of cases. For adrenergic the mother was implicated in 50% of cases. The father was the predominant "carrier" among mixed afibbers (27%) and mothers, fathers and siblings shared the "honours" among vagal afibbers at 28% each.

The estimated overall prevalence of all cardiac arrhythmias in the United States is about 1% with atrial fibrillation accounting for about half of this[4,5]. Cardiac arrhythmias are generally more common among older people. With only 1% of the general population having arrhythmia is it odd that 43% of the survey respondents had a close relative with arrhythmia? This question can really only be answered definitely by comparing the rate of arrhythmia among close relatives of a group of lone afibbers with the rate in a group of age- and sex-matched controls. Too major a project for my limited resources.

Nevertheless, it is possible to get some idea about the likelihood of a genetic connection. Although we afibbers tend to be an odd bunch, it is probably safe to assume that we each had two biological parents?! This means that there were 23 cases among the 202 parents included in the survey or a rate of 11% - in other words, considerably higher than the 1% that would have been expected. This finding does not prove that LAF can be inherited, but it certainly supports the possibility.

The genetic connection is also supported by work done by Dr. Ramon Brugada and his colleagues at the Baylor College of Medicine and the University of Barcelona[6]. These researchers located three families in Spain in which 21 of 49 family members had lone atrial fibrillation. They mapped their genes and concluded that in these families a mutation in a specific chromosome region (10q22-q24) was the cause of their atrial fibrillation. Dr. Maurits Allesie, MD of the University of Maastricht in the Netherlands makes several very interesting observations concerning these findings[7]:

- If, as in the three Spanish families, lone atrial fibrillation in the general population is also caused by a genetic mutation, then Brugada's findings are of paramount importance.
- The possibility that small molecular defects in DNA can cause changes in the electrophysiologic properties of the atria that, in turn, create a substrate for chronic atrial fibrillation is not unlikely. NOTE: The term chronic in this statement does not mean permanent as opposed to paroxysmal, but rather that LAF is not an acute condition.
- The genes that encode adrenergic receptors are located at the observed mutation site on chromosome 10q. This means that the basis for familial atrial fibrillation could lie in abnormal atrial triggering mechanisms.

Dr. Allesie concludes, "The anatomical and electrophysiologic features of the atria are such that there is only a narrow margin of safety between normal sinus rhythm and chronic atrial fibrillation." Our survey findings of a possible genetic connection and the fascinating discoveries of Dr. Brugada and colleagues together with Dr. Allesie's profound observations certainly provide much food for thought and will hopefully be followed up by additional research.

This concludes our evaluation of the LAFS III results for this issue. We will complete the evaluation in the October issue.

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B. LAF - The Diagnosis

Atrial fibrillation comes in three flavours – paroxysmal, persistent, and permanent. Paroxysmal AF converts to normal sinus rhythm (NSR) on its own and episodes last less than 7 days (most less than 24 hours); persistent AF episodes usually last more than 7 days, but cardioversion is effective in conversion to NSR; permanent AF is permanent and does not respond to cardioversion. The term “chronic” refers to the fact that AF, with some exceptions, is not an acute disorder, but rather a long-term one. It is estimated that more than 2 million Americans suffer from paroxysmal or persistent AF. The incidence of AF is less than 1% in people under the age of 60 years, but increases to more than 6% in people over the age of 80 years[1,2].

Causes of atrial fibrillation

The majority (70-90%) of AF patients have an underlying heart problem, specifically one or more of the following conditions[2,3]:

- Valvular heart disease (mitral prolapse or stenosis)
- Rheumatic heart disease
- Coronary artery disease
- Angina pectoris
- Congenital heart disease
- Left ventricular dysfunction (congestive heart failure)
- Inflammatory heart disease (myocarditis)
- Tumour
- Atrial fibrosis (thickening of heart tissue due to inflammation or scarring)
- Hypertensive heart disease (narrowing of the aorta).

AF can also occur on a transient (acute) basis after heart surgery or in the aftermath of a heart attack. If none of the above conditions are present then the AF is classified as lone atrial fibrillation (LAF). It is estimated that from 12-30% of all cases of AF are of the lone variety[1].

Essential (idiopathic, primary) hypertension, electrolyte imbalances, thyrotoxicosis (hyperthyroidism), hypoglycemia, acute infections, alcohol intoxication (binge drinking), pheochromocytoma, and certain pharmaceutical drugs can also cause AF episodes[2].

Hypoglycemia (low blood sugar) may be quite common among lone afibbers. Our surveys revealed that almost 50% of respondents have medically diagnosed hypoglycemia or symptoms of hypoglycemia. The disorder is diagnosed through a 3-hour or, better yet, a 6-hour glucose tolerance test. If the fasting glucose level is below 50 mg/dL (2.8 mmol/L) or if the glucose level falls below the fasting level 4 to 6 hours after a meal then hypoglycemia is present. However, the actual blood glucose level that causes hypoglycemic conditions can vary considerably between individuals. Hypoglycemia can be controlled by religiously avoiding foods with a high glycemic index (sugar, white and whole grain bread, bananas, raisins, potatoes, rice, and wheat cereal) and by eating frequent small meals throughout the day. Alcohol should be avoided and the intake of dietary fiber increased. A daily multivitamin (and mineral) capsule is very important and a minimum intake of 200-400 micrograms/day of chromium is also recommended.

Pheochromocytoma is not likely to be the underlying cause for most afibbers as it is a rare disease. It is found in about 0.1% of people diagnosed with hypertension[4]. It involves a tumour, most often in the adrenal gland, that periodically releases large amounts of norepinephrine (noradrenaline) and epinephrine (adrenaline) into the blood stream. One of the characteristic features of pheochromocytoma is a feeling of impending doom just prior to the afib episode. Hypertension resistant to normal therapy, headaches, and excessive sweating are other common symptoms. Pheochromocytoma is treated surgically by removal of the tumour.

If no heart abnormalities or any of the above mentioned possible afib triggers are present then the condition is labelled as idiopathic (of no known cause) or primary atrial fibrillation.

Initial evaluation

Most afibbers associate their first episode with violent heart palpitations. For about 60% of afibbers palpitations or a racing heart beat is the primary symptom of an AF episode. Other common symptoms are breathlessness (13%), chest pain (8%), and dizziness (8%)[2]. Some episodes, especially very short ones, may not be accompanied by any discernible symptoms at all[2]. There have also been cases where people with no symptoms were found to be in permanent AF during a routine visit to their doctor. Nevertheless, the “afib journey” most often begins with a visit to the emergency clinic or the doctor’s office by a very frightened patient complaining of heart palpitation, breathlessness or chest pain. This visit triggers a chain of investigations and tests designed to do two things:

- 1) Determine if the patient has a heart abnormality or any other condition that explains the symptoms.
- 2) Determine if the patient is at increased risk for developing blood clots and suffering a stroke.

The first thing your doctor is likely to do at the initial visit either during or after the first episode is to enquire about any history of stroke or TIAs (transient ischemic attacks), feel your pulse, and measure your blood pressure. An irregular pulse would indicate the presence of an arrhythmia such as atrial fibrillation. A blood pressure above 140/90 mm Hg would raise the suspicion of hypertension (high blood pressure), but this measurement certainly needs to be confirmed on several other occasions before the diagnosis of hypertension is made. During this first visit you are likely to be highly nervous which could increase blood pressure significantly. Or if you are still in afib an abnormally low blood pressure may be found.

The doctor will also want to listen to your heart with a stethoscope (auscultation). By listening carefully to the noises the heart makes when it pumps they will be able to detect problems with the heart valves such as mitral valve prolapse, mitral valve stenosis, and regurgitation. By listening to the lungs pulmonary edema may be detected. Finally an eye examination is usually performed to check for signs of hypertension or atherosclerosis showing up in the small arteries feeding the eye.

Blood tests

Following this initial evaluation the doctor will no doubt send you for blood tests to check for a dysfunctional thyroid gland (hypothyroidism, hyperthyroidism, thyrotoxicosis) and for the presence of diabetes or low blood sugar (hypoglycemia). Your physician may also order a test of your electrolyte balance (sodium, potassium, calcium and magnesium); this test may not be terribly indicative, as for example, only 1% of the body’s total magnesium stores actually circulate in the blood. Nevertheless, gross deviations from normal values will no doubt be revealed. The doctor will also want to know if you are a binge drinker and may test you for pheochromocytoma, especially if your blood pressure is extremely high and resistant to treatment.

Since palpitations can also be a feature in anxiety (panic) attacks and anemia tests for these conditions may also be warranted. The next phase of the evaluation is designed to check for specific heart problems.

Electrocardiogram (ECG)

As the heart muscle contracts it generates a current that spreads into the fluids around the heart and can be measured on the surface of the body. The ECG measures the electrical activity of the heart. It involves the placing of 12 small electrodes on the chest around the heart area and on the arms and legs. It is painless, non-invasive, and takes only a couple of minutes. The ECG will pick up such heart abnormalities as bundle branch block (a common conduction fault), angina or a prior heart attack. It may also, with less accuracy, indicate the presence of a diseased atria or enlarged atria or ventricles and the possibility of significant abnormalities in potassium and calcium levels[4]. An ECG will also pick up a cardiac arrhythmia if it is present at the time of the test. At this point the afibber will probably be turned over to a cardiologist who will arrange for several more tests required to arrive at a conclusive diagnosis.

Holter monitoring

The first test will probably be an ambulatory ECG (Holter monitoring). Here the patient wears a small recorder connected to one or two electrodes taped to the chest in the heart region. The monitor is usually worn for a 24-hour period. Some models have an “event marker” button that the wearer can press if they actually feel an irregular heart rhythm. Analysis of the 24-hour recording will pick up any arrhythmias that occurred during the period and also provide a measure of the number of PACs (premature atrial complexes) and PVCs (premature

ventricular complexes) that occurred. The Holter monitor can also provide an indication that coronary heart disease is present, but confirmation of this is best done with the treadmill exercise test.

Treadmill exercise test

In this test the patient is hooked up to an electrocardiograph with the usual 12 electrodes except that they are all placed on the trunk where they are less likely to be dislodged during the exercise. The patient is also connected to a continuous blood pressure monitor. The patient walks on a treadmill until maximum heart rate and blood pressure (for the appropriate age group) is reached or until symptoms (chest pain, fatigue, arrhythmia or breathlessness) occur, which precludes further exercise. The testing protocol involves an increase in workload (speed and grade) every 3 minutes until completion. The treadmill test is most useful for detecting coronary heart disease, which manifests itself by a significant drop in blood pressure or an excessive rise in heart rate during the test. The test will also pick up exercise-induced arrhythmias. It should not be used in patients with congestive heart failure, unstable angina pectoris or severe aortic stenosis. If no abnormalities have been detected so far the cardiologist will usually conclude the testing with an echocardiogram.

Echocardiography

The obtaining of an echocardiogram is painless and non-invasive and takes only a few minutes. Echocardiography uses ultrasound to obtain a picture of the different parts of the heart and to characterize blood flow within the heart. It can produce one- or two-dimensional images of the heart and can also measure the velocity of red blood cells moving through the heart (Doppler echocardiography). The echocardiogram can clearly distinguish valve abnormalities such as mitral valve prolapse and mitral valve stenosis, it can determine the extent of regurgitation, and provide a good estimate of the size of the left atrium and the left ventricle. It can also measure the wall thickness and volume of the left ventricle and provide a value for left ventricular ejection fraction – a highly important marker of overall heart performance. A low ejection fraction is often associated with congestive heart failure. Finally, the presence of tumours can also be ascertained with an echocardiogram.

If no underlying heart abnormalities or other disease conditions have been found which can explain the atrial fibrillation then the cardiologist, at this point, should be able to make the diagnosis of lone or idiopathic atrial fibrillation. Of course, this assumes that the presence of other arrhythmias (atrial flutter, supraventricular tachycardia, ventricular arrhythmias) has already been ruled out.

Advanced (and rarely needed) tests

If any of the evaluations provide abnormal results more testing may be required, but this will usually be in order to confirm a problem rather than to further support the diagnosis of LAF.

Injection of radioactive tracers and subsequent imaging with a gamma camera (nuclear medicine techniques) can provide further information about heart structure and blood flow and is particularly useful in determining left ventricular ejection fraction. In combination with exercise testing nuclear medicine techniques are also useful for detecting coronary artery disease or for confirming that a heart attack has taken place[6].

Invasive tests such as cardiac catheterization and angiography are usually not required in diagnosing lone atrial fibrillation. An electrophysiological study may be useful in determining whether atrial flutter or paroxysmal supraventricular tachycardia is involved as predisposing arrhythmias and is also required if ablation therapy is contemplated[1].

Need for stroke prevention

The results obtained from the above evaluations determine the need for anticoagulation or other stroke prevention measures. The current Guidelines for the Management of Atrial Fibrillation spell out the indications for anticoagulation therapy (warfarin) or antiplatelet therapy (aspirin)[1]. Both warfarin and aspirin can cause bleeding so obviously should not be prescribed for patients with a tendency to internal bleeding and should be prescribed with extreme caution for patients with diverticulosis[7].

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
- Presence of 1 or more risk factors at any age* - warfarin to an INR of 2.0-3.5

* **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, heart attack or transient ischemic attack (TIA), and a persistent atrial thrombus (blood clot)[1].

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