THE AFIB REPORT

Your Premier Information Resource for Lone Atrial Fibrillation!

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Editorial

In this issue we begin the reporting of the results of our third lone atrial fibrillation survey (LAFS III). This survey is designed to elicit information about physical conditions, health conditions, and lifestyles that would have an effect on episode severity or indeed on the initial development of LAF. Our database now contains 236 afibbers of which about 100 answered the questions in LAFS III. The most interesting findings in this initial report involve the low rate of smoking and the high incidence of GERD (gastroesophageal reflux disease) found among afibbers.

Also in this issue, we describe two arrhythmias, premature atrial complexes (PACs) and premature ventricular complexes (PVCs), which affect lone afibbers. We also discuss supraventricular tachycardia, an arrhythmia that, like LAF, is initiated by PACs. This, together with the discussion of atrial flutter in the April issue, completes our review of arrhythmias involving the upper (non-ventricular part) of the heart.

Yours in health and sinus rhythm,
Hans Larsen

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Findings from LAFS III – Part 1

The latest LAF questionnaire (May 2002) yielded 105 responses of which 36 were from afibbers who had not previously participated in a survey. This brings our total database to 236 respondents with lone atrial fibrillation.

The majority (42%) of these 236 respondents have the vagal variety of LAF, 30% the mixed form, and 11% the adrenergic. The remaining 17% have chronic LAF. The average (mean) age of respondents with paroxysmal LAF is 53 years (54 years for adrenergic, 56 for mixed and 51 for vagal) with a range from 19 years to 81 years. The average age of chronic afibbers is 57 years with a range of 34 to 82 years. The average age at diagnosis for paroxysmal (intermittent) afibbers is 46 years (48 years for adrenergic, 49 for mixed and 45 for vagal) with a range of 14 to 80 years. The average age at diagnosis for chronic afibbers is 48 years with a range of 8 to 72 years. Fifty per cent of respondents were between the ages of 40 and 55 years when first diagnosed and only 7% were 65 years or older. This finding clearly refutes the generally held belief that lone atrial fibrillation is an "old age" disease.

A. Episode Severity

Adrenergic afibbers had an average (median) of 4 episodes (0-68) lasting an average (median) of 12 hours (0-72) resulting in an average (median) 24 hours being spent in afib during the 6-month survey period. Corresponding numbers for mixed afibbers were 5 episodes of 3 hours duration for a total afib time of 22 hours. Vagal afibbers, on average, had the most episodes (6) lasting the longest (6 hours) resulting in an average 48 hours being spent in afib over the period. This observation can, in part, be explained by the fact that many vagal

afibbers are on the wrong drugs (beta-blockers and antiarrhythmics with beta-blocking properties) for their condition.

There was a clear association between time spent in fibrillation and both the number of episodes and the average duration of each episode (p=0.0001). This association was particularly strong for vagal afibbers with the correlation between episode duration and total time spent in fibrillation being the most pronounced (r=0.7026 p=0.0001).

The total time spent in fibrillation increased significantly with age from an average (mean) of 26 hours per 6 months at age 30 years to 80 hours at age 60 years. This age effect was particularly strong for vagal afibbers who spent an average 109 hours in afib at age 60 years.

The total time in fibrillation also increased with the number of years since diagnosis. A recently diagnosed afibber spent an average of 53 hours in afib while someone diagnosed 10 years ago spent an average of 81 hours in afib over the 6-month period.

There was a trend for afibbers who reported bowel problems to have fewer episodes and mixed afibbers with bowel problems also spent less time in fibrillation. This finding could, however, be confounded by the fact that women, who do spend less time in fibrillation, also are more likely to have bowel problems.

B. Effect of Gender

The majority (81%) of respondents are male. Whether this actually reflects the distribution of LAF in the general population or is an indication of the relative use of the Internet among men and women is not clear. Only 11% of female respondents had the vagal variety while 30% had the mixed type. Adrenergic and chronic each had a 20% female component. The median age at diagnosis was 52 years (range of 22 to 70) for women and 47 years for men. This difference is statistically significant.

Women, particularly with the mixed variety, tended to have significantly shorter episodes than did men and spent less time in fibrillation. Women were more likely to have a bowel disorder and were less likely to have engaged in prolonged strenuous physical activity than were men. Women were also more likely to be suffering from an autoimmune disorder.

C. Summary of Questionnaire Responses

10. Have you ever been told that you have an enlarged left atrium?

Twenty-four or 23% of respondents had been told that they had an enlarged left atrium. This diagnosis was most common among adrenergic afibbers (46%) and chronic afibbers (33%) and less common among mixed (14%) and vagal (17%) afibbers. The size range for an enlarged atrium was 42 to 50 mm. An enlarged left atrium was associated with advanced age and the use of vitamin E and beta-carotene. However, the use of vitamin E is also strongly associated with advanced age so this no doubt confounds this finding. The finding regarding beta-carotene is puzzling but fairly robust (r=0.37 p=0.0001). An enlarged atrium was not associated with increased episode severity and physical activity did not correlate with an enlarged atrium.

12. Were you a smoker when you experienced your first episode?

Only 13 or 13% of respondents answered yes to this question (20% among vagal afibbers, 8% among adrenergic, 7% among mixed, and 6% among chronic). Smokers were more likely to have had a dysfunctional childhood (p=0.01). Smoking at the time of the first episode was not associated with an increased episode severity.

13. Are you now a smoker?

Only 5 or 5% of respondents answered yes to this question (4% among vagal afibbers, 8% among adrenergic, 7% among mixed, and 0% among chronic). Almost 80% of vagal afibbers had thus given up smoking after experiencing their first episode. With only 5 smokers in a population of 103 respondents it is not possible to draw statistically valid conclusions regarding possible associations. However, there was a trend for current smokers to be younger, to have been on Valium, and to have been brought up in a dysfunctional home. Considering that about 30% of the male American adult population are smokers the proportion of smokers

among afibbers (5%) is clearly exceptionally low. Is this part of the generally healthy lifestyle of afibbers or could smoking actually be protective? Smoking is primarily a nicotine delivery system. Nicotine is known to increase circulating levels of cortisol, DHEA, norepinephrine (noradrenaline) and epinephrine (adrenaline)[1-3]. While this would be bad news for adrenergic afibbers and possibly for mixed afibbers it could be helpful for vagal afibbers. Norepinephrine and epinephrine increase sympathetic nervous activity. Vagal afibbers have an excess of parasympathetic activity so increasing the sympathetic activity (through smoking) would tend to balance their autonomic nervous system and may help to prevent vagally-mediated afib episodes.

A nicotine patch, chewing nicotine gum or, for fast action, using a nicotine nasal spray are all efficient ways of delivering nicotine to the circulation[4]. It is conceivable that a vagal afibber could cut down on the number of episodes or avoid them altogether by using a slow, continuous delivery system such as a 2 mg nicotine patch or nicotine gum. Using a nasal spray after dinner might prevent post-prandial episodes. I want to emphasize that this is all pure speculation on my part. I have no scientific evidence to support it. However, if I had the vagal kind of LAF (I am adrenergic) I would definitely give it a try. Although not without long-term side effects, the nicotine approach would probably still be safer than amiodarone and some of the other antiarrhythmics and, if it works, could pave the way for the development of a novel class of drugs to treat vagal LAF.

14. Have you ever been diagnosed with a bowel disorder?

Twenty-five or 25% of respondents answered yes to this question (32% vagal, 25% mixed, 17% adrenergic, and 11% chronic). Women were more likely to have bowel problems and there was also a significant association between taking beta-carotene and having bowel problems. Afibbers with a bowel disorder were also significantly more likely to have an autoimmune disorder and a dysfunctional childhood. Sixteen (67%) of the 24 respondents who specified their disease had irritable bowel syndrome (IBS), 7 (29%) had diverticulitis, and 1 (4%) had Crohn's disease. IBS was particularly widespread among vagal afibbers. Seventeen respondents specified how long they had had the disorder; 13 years was the average with a range from 1 to 40 years.

The estimated prevalence of IBS in the general population is 15 to 20%. The confirmed incidence among our 102 respondents is 16%; thus it would not appear that the IBS incidence among afibbers is abnormally high. The confirmed incidence of diverticulitis is 7%, which again is not out of line with the prevalence in the general population. So overall our survey data does not support the contention that bowel disorders are more common among afibbers than in the general population.

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

Thirty-eight or 37% of respondents answered yes to this question (40% vagal, 38% mixed, 33% adrenergic, and 28% chronic). Respondents who had answered yes were also more likely to report a correlation between an afib episode and a flare-up of their condition as well as an association between diet and episode severity. Eighteen (51%) of the 35 respondents who specified their disease had GERD, 29% reported acid reflux, 11% a hernia, and the remaining 9% described their condition as heartburn. Thirty respondents specified how long they had had the disorder; 11 years was the average with a range from 1 to 30 years. Heartburn and acid reflux are common symptoms of GERD so it is reasonable to conclude that 34 of the 104 respondents could be classified as having GERD. This gives an incidence of 33%. GERD, heartburn or acid reflux is not a steady condition, which makes it difficult to estimate the prevalence in the general population. The overall prevalence may be around 7 to 10%, but up to 50% of Americans report at least one episode of heartburn per month[5]. In retrospect it is clear that the questionnaire should have been more specific in regards to frequency of symptoms. Nevertheless, GERD could be an important afib factor. We will look at the GERD connection in a future issue of The AFIB Report.

This concludes the first instalment of the evaluation of the LAFS III survey results. In the next issue we will take a look at the answers to the remaining questions.

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PACs, PVCs and Other Arrhythmias

An arrhythmia is any deviation from a normal heart beat. Arrhythmias can be divided into two major groupings - those affecting the ventricles (ventricular arrhythmias) and those affecting the atria (atrial or supraventricular arrhythmias). Arrhythmias may also be divided into two groups depending on whether the heart rate is excessively low (bradycardia) or excessively high (tachycardia). Fibrillation (atrial or ventricular) involves a rapid and chaotic beating of the heart. Atrial fibrillation results in rapid and irregular heart and pulse rates while ventricular fibrillation results in death (cardiac arrest). Atrial flutter is similar to atrial fibrillation except that the rhythm is orderly. Both atrial fibrillation and flutter can be intermittent (paroxysmal) or chronic. Premature atrial complexes (PACs), premature ventricular complexes (PVCs), and supraventricular tachycardias are other common arrhythmias.

Premature Atrial Complexes (PACs)
PACs, also known as atrial extra systoles or atrial premature beats, are extremely common and can be found on 24-hour Holter monitoring in over 60% of normal adults[1]. They are usually entirely benign and do not require treatment unless they are very frequent or results in uncomfortable palpitations. PACs originate from foci of “rogue” heart cells that decide to take on a beat of their own. Depending on when the PAC “fires” it may not be transmitted to the ventricles at all, but in some cases it may cause a pause in the normal heart beat rhythm, which may or may not be followed by a more forceful ventricular contraction[2].

PACs can be precipitated by stress, fatigue, fever, thyrotoxicosis, tobacco, caffeine, and certain other stimulants and drugs including cold medications and weight-loss preparations. PACs may also be a sign of underlying heart disease such as ischemia (angina pectoris), heart failure or myopericarditis. If underlying heart disease is present it should be treated irrespective of whether the PACs cause palpitations or not[1-4]. PACs can initiate atrial fibrillation, atrial flutter or supraventricular tachycardia. Some fairly recent research has shown that these arrhythmias originate from the same focal points that generate PACs[5]. PACs can be distinguished fairly easily on an electrocardiogram; they are characterized by a smaller and earlier than expected P wave. The P wave originates in the sino-atrial node and is the electrical impulse that initiates the heart beat.

The obvious way to avoid PACs is to avoid the triggers. In more severe cases minor tranquillizers or beta-blockers may prove helpful[1]. There is also growing evidence that a magnesium deficiency can increase PACs and that magnesium supplementation can reduce them. Researchers at the US Department of Agriculture recently reported that magnesium deficiency is associated with an increase in premature beats, both supraventricular (PACs) and ventricular (PVCs). Their clinical trial involved 22 postmenopausal women who were maintained in a metabolic ward for 6 months. None of the women had been diagnosed with atrial fibrillation. All the women ate a diet that provided less than half of the recommended daily intake of magnesium for the first 81 days of the trial. They were then randomly assigned to receive either a placebo or magnesium gluconate capsules with each meal for the next 81 days. The daily magnesium intake in the placebo group was 130 mg versus 411 mg in the supplement group. The current Recommended Daily Allowance (RDA) is 320 mg for women and 400 mg for men. The researchers noted that the women in the placebo group had significantly more premature beats (11/hour average) than the women in the supplemented group (6.5/hour average) when evaluated using 21-hour Holter monitoring. They conclude that a magnesium deficiency can lead to an increase in premature beats and caution that people who use diuretics, live in areas where the drinking water is very soft or are predisposed to premature beats or magnesium loss may require more magnesium than normal. They also point out that recent surveys show that many Americans are deficient in magnesium and have a dietary intake of only 200 mg/day or less. Although the trial did not target people with atrial fibrillation there is no reason

why its conclusions should not be applicable to afibbers. I believe the results clearly support the idea that magnesium, preferably as maleate, citrate, gluconate or orotate, is a very important supplement for afibbers[6].

Premature Ventricular Complexes (PVCs)

PVCs are also extremely common and can be found on 24-hour Holter monitoring in over 60% of adult males[1]. PVCs are only of concern if heart disease, especially ventricular dysfunction, is also present. They do not originate in the atria, but rather in the ventricular muscle itself or at least somewhere below the atrioventricular node and bundle of His (see the February 2001 issue of The AFIB Report for further explanation). The PVC results in a longer than normal contraction of the ventricles and is followed by a longer than normal pause between beats. I am not aware of any way that an individual can distinguish between a PAC and PVC just by being aware of the heart beat and pulse; however, the difference is easily discernible on an electrocardiogram. A PVC produces an odd-shaped and much broader (longer lasting) QRS complex than normally seen. The QRS complex is a record of the electrical impulses that initiate the contraction (depolarization) of the ventricular part of the heart; it follows the P wave on the electrocardiogram[1,4,7,8].

The triggers for PVCs are quite similar to those for PACs and include caffeine, cocaine, alcohol, and other stimulants. An electrolyte imbalance, particularly if it involves potassium or magnesium, can trigger PVCs, as can an overly acidic condition (acidosis) and a low blood level of oxygen (hypoxemia)[4]. Many drugs including anti-psychotics, tricyclic antidepressants, asthma and emphysema medication (theophylline) and several heart drugs (digoxin, flecainide, sotalol and dofetilide) can also trigger PVCs[4]. They tend to become more prominent with age and their frequency may also increase premenstrually and during pregnancy[7].

PVCs are particularly common after a heart attack and, if they are frequent enough, can initiate a fatal ventricular arrhythmia. They are also a common feature in angina, heart failure, valvular heart disease, and mitral valve prolapse. The risk of PVCs initiating ventricular arrhythmias depends on the seriousness of the underlying heart problem. The risk is very low in the case of mitral valve prolapse, but high in the case of congestive heart failure[7]. PVCs, in the absence of heart disease, do not require treatment unless they are very frequent or result in fatigue or uncomfortable palpitations. PVCs related to mitral valve prolapse also generally do not require treatment other than reassurance that “all is well”[1,4,7].

The number of PVCs experienced can be markedly reduced by correcting electrolyte imbalances and avoiding known triggers. If this is not adequate then mild tranquillizers or beta-blockers may do the trick. Beta-blockers are particularly indicated if the PVCs occur primarily during the day or under stressful conditions[1]. Stronger antiarrhythmic drugs such as disopyramide (Norpace), flecainide (Tambocor), propafenone (Rythmol), and especially, amiodarone (Cordarone) are usually not indicated for PVCs without underlying heart disease and should be used with extreme caution if heart disease is present[7]. Radio frequency ablation of an ectopic focus may also be applicable in some cases of troublesome PVCs[4]. As is the case with PACs, magnesium supplementation may be useful in reducing the frequency of PVCs[6].

Supraventricular Tachycardia

Supraventricular tachycardia (SVT) is a common arrhythmia. It can occur even in otherwise healthy young people and as a complication after childbirth. There are several different varieties of SVT, but the two most common are atrioventricular (AV) nodal re-entrant tachycardia and atrioventricular re-entrant tachycardia. SVT can occur in people with underlying heart disease as well as in those without. It is usually abrupt in onset and termination and may be paroxysmal (intermittent) with episodes lasting from minutes to hours, persistent, with episodes lasting several weeks, or chronic. The paroxysmal form is by far the most common and involves heart rates of 150 to 250 beats per minute sometimes, particularly in children and young people, rising to 300 bpm. The heart rate is usually regular as opposed to atrial fibrillation where it is highly irregular. Japanese researchers have observed that SVT episodes tend to occur more frequently during the day than at night and that night time episodes are slower and shorter than daytime ones. They believe that autonomic nervous system balance plays an important role in the initiation of paroxysmal SVT episodes[9].

For some SVT patients palpitation or chest pain is the only indication that they are experiencing an episode. Others experience lightheadedness, shortness of breath, dizziness, weakness, and frequent urination (polyuria). The frequent urination usually occurs within a few minutes of the onset of the episode and is caused by the

release of atrial natriuretic peptide (ANP) induced by stretching of the atria. Many afibbers have observed a similar phenomenon[1-4,8,10,11].

AV nodal re-entrant tachycardia is caused by a fault in the conduction pattern around the AV (atrioventricular) node that passes the electrical impulses from the atria to the ventricles. Normally, there is only one path for the signals to enter the bundle of His and from there activate the ventricles. In AV nodal re-entrant tachycardia there are to paths – a fast one and a slow one. The signals normally use the fast path; however, if a premature atrial complex (PAC) happens to arrive at the AV node when the fast path is closed (following depolarization), then the PAC may use the slow path and initiate a re-entry phenomenon which will result in tachycardia. In other words, two conditions are needed to initiate SVT – a fault in the conduction pattern in the AV node and a suitably timed premature atrial beat.

AV re-entrant tachycardia accounts for about 30-40% of supraventricular tachycardia while AV nodal re-entrant tachycardia accounts for about 50-60% of cases. AV re-entrant tachycardia is caused by the presence of an alternate pathway between the atria and ventricles that completely bypasses the AV node. The alternate pathway, in the vast majority of cases, is the so-called bundle of Kent, a cardinal feature of Wolff-Parkinson-White Syndrome (WPWS). WPWS is caused by the incomplete separation of atria and ventricles during fetal development, but its presence may not be felt until adulthood. As in AV nodal re-entrant tachycardia an AV re-entrant tachycardia episode is caused by a premature atrial beat (PAC) entering the alternate pathway at an inopportune moment and setting up a re-entry circuit initiating the arrhythmia. The symptoms are similar to those of AV nodal re-entrant tachycardia and neither condition is life-threatening in the absence of underlying heart disease.

Since SVTs are initiated by premature atrial complexes it stands to reason that avoiding the triggers causing PACs is also effective in preventing SVTs. The most common trigger factors are emotional stress, fatigue, fever, thyrotoxicosis, alcohol, tobacco, caffeine, cold medications, and weight-loss preparations. Digoxin intoxication is also a common trigger for SVTs. Based on the information obtained from our LAF surveys, MSG, aspartame and cold drinks should be added to this list.

Many SVT episodes can be promptly terminated by manoeuvres aimed at stimulating the parasympathetic (vagal) nervous system. These so-called Valsalva manoeuvres include stretching the arms and body, squatting, coughing, holding one's breath and straining as if blowing up a balloon, lowering the head between the knees, or plunging the face into a basin of ice-cold water[2,10]. Carotid sinus pressure is also very effective in stopping SVT, but should only be performed by a doctor or at least after thorough training by a doctor as there are cases where it should not be used. In some cases of mild SVT, particularly if initiated by emotional stress, taking 5-10 mg of diazepam (Valium) or 30 mg of flurazepam (Dalmane, Somnol) may ease symptoms considerably[2].

If Valsalva manoeuvres and carotid sinus massage fail to stop the SVT several rapidly acting drugs will terminate it in 90% of cases. Intravenous adenosine is the agent of choice, but the calcium channel blockers verapamil and diltiazem are also highly effective. A team of German and Italian researchers recently reported that oral doses of approximately 200 mg (3 mg/kg) or flecainide can be safely and effectively used at home to stop episodes of paroxysmal supraventricular tachycardia. They observed an 80% success rate within 2 hours, but emphasize that the flecainide tablet should be taken in crushed form within 5 minutes of the start of the episode[12]. There is emerging evidence that this approach may also work well for vagal afibbers[13].

In severe cases, such as when the patient has chest pain or faints, electrocardioversion may be used to stop SVTs. Medication with beta-blockers or calcium channel blockers can be used to prevent or at least reduce the number and duration of SVT episodes. These drugs work specifically by altering conduction velocities in the AV nodal pathways and are therefore not effective in preventing atrial fibrillation. In more severe cases propafenone or flecainide may be used for prevention. The best long-term solution to SVT prevention is radio frequency ablation of the alternative pathways around the AV node and elimination of the foci initiating the PACs. The success rate for this procedure is now approaching 95%[4].

Researchers at the University of New Mexico have just completed a study aimed at evaluating the cost effectiveness and quality-of-life outcomes for drug and ablation therapies. Patients who underwent ablation therapy scored considerably higher on all quality-of-life measures both 1 and 5 years after ablation. Specifically,

ablation patients had far fewer episodes of dizziness and palpitations than did the patients treated with drugs. During the 5-year follow-up period, ablation patients averaged 1.2 clinic visits per patient and had no emergency room visits or hospitalisations. The drug-treated patients averaged 3.7 clinic visits, 17 emergency room visits, and 2 hospitalisations per patient. The total cost of therapy for the 5 years was $7500 for the ablation patients and $6200 for the ones treated with drugs. The researchers conclude that radiofrequency catheter ablation "is associated with a sustained improvement in quality of life and a reduction in disease-specific symptoms, particularly in women and in patients aged younger than 50 years"[14]. A Swiss study found that 96% of 94 patients who had undergone radiofrequency ablation for SVT were "strongly satisfied" or "satisfied" with the procedure and their relief of symptoms[15].

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