THE AFIB REPORT

Your Premier Information Resource for Lone Atrial Fibrillation!

NUMBER 78 **APRIL 2008** **8th YEAR**

In this issue, we begin a new, regular feature based on the results of LAF Survey 14. LAFS-14 received responses from 224 afibbers who had attempted to reduce or eliminate their afib burden (number of episodes x duration over a 6-month period) through means other than ablation and surgical procedures. The overall efficacy of the different protocols was summarized in the November 2007 issue of The AFIB Report. However, space limitations did not allow us to provide details of the successful protocols. Thus, the purpose of our new section is to describe the specific actions actually taken by afibbers who found ways of reducing their afib burden by at least 50% with the use of pharmaceutical drugs or such alternative approaches as trigger avoidance, supplementation, dietary changes, stress management, or elimination of underlying disease conditions such as hypoglycemia, sleep apnea, and GERD (gastroesophageal reflux disease).

Also in this issue, we report on Spanish researchers discovering important risk factors for LAF, clots in the left atrial appendage are not found in lone afibbers with normal left ventricular ejection fraction, beta-blockers and magnesium may improve cardioversion results, and the latest findings on the effect of fish oils on heart rate variability.

Finally, if you need to restock your supplements, please remember that by ordering through my on-line vitamin store you will be helping to defray the cost of maintaining the web site and bulletin board. You can find the store at http://www.afibbers.org/vitamins.htm - your continuing support is truly appreciated.

Wishing you good health with lots of NSR,

Hans

Highlights

Risk factors for lone atrial fibrillation

BARCELONA, SPAIN. While much research is being done to find new, improved methods of treating atrial fibrillation (AF), very little research is done to determine the causes of the AF epidemic. Hopefully, recent work undertaken at the University of Barcelona will help reverse the balance. The Spanish researchers determined height, physical activity level, and left atrial size in a group of 107 lone atrial fibrillation (LAF) patients and compared their results to those in a group of age- and sex-matched healthy individuals without AF. In their discussion, the researchers make the following statements of particular interest:

- "*The rise (in atrial fibrillation cases) is not due exclusively to population aging or to the higher prevalence of obesity.*"
- *"The prevalence of LAF ranges from around 2-10% in the AF population and may reach 30% in patients seeking medical attention."*
- *"LAF was defined as AF in the absence of any identifiable cause of the arrhythmia."*

- *"Atrial fibrillation was considered of 'vagal' origin when it occurred at least 80% of times during sleep and/or in post-prandial situation and 'adrenergic' when it occurred at least 80% of the time during high physical exertion and/or in situations of stress. The remaining patients were classified as suffering 'random' AF."*

The researchers measured the height, weight, and left atrial size of each study participant and also had all participants complete a validated questionnaire to determine their accumulated lifetime physical activity. Physical activity was classified into four levels – sedentary, light, moderate, and heavy. Light activities included standing and slow walking; moderate included activities that increased heart rate slightly and perhaps resulted in light perspiration, but which did not result in exhaustion; heavy activities included vigorous exercise that significantly increased heart rate. The accumulated lifetime hours of all occupational and exercise/sports activities for each level were calculated for each participant, taking into account their duration and frequency.

Forty-three percent of the LAF patients were admitted to the emergency department with a first episode of AF, while the remaining 57% had experienced previous episodes. Average age at admission was 48 years (NOTE: Our survey of 625 afibbers recorded an average age of 48 years at diagnosis). The majority (69%) of the participants were male and most (70%) had vagal AF.

The researchers observed a strong correlation between height and LAF prevalence with taller individuals (average height of 186 cm) being up to 17 times more likely to experience LAF than shorter individuals (average height of 160 cm). NOTE: Our first LAF survey in 2001 found an average height of 183 cm among male afibbers. PC's later survey (LAFS-11) found an average height of male afibbers of 181 cm as compared to a population mean of 175 cm. Thus, the correlation between height and LAF risk is well established and is likely associated with the larger atrial size accompanying tallness.

A larger left atrium (left atrial anteroposterior diameter) was associated with a 40% increased risk of LAF. The most striking finding was the association between LAF risk and accumulated moderate and heavy physical activity. Those with a lifetime accumulated moderate plus heavy physical activity of more than 9300 hours had 15 times the prevalence of LAF than did those with less than 2100 hours accumulated. More than 564 hours of accumulated heavy, vigorous physical activity was associated with a 7 times increased prevalence of LAF.

The researchers speculate that the negative effects of moderate and particularly vigorous physical activity may be related to the chronic volume and pressure overload caused by the increased activity. They conclude, "*The fact that physical activity is a risk factor for AF does not argue against exercise as a way of preventing coronary artery disease. It only offers a word of caution suggesting that the benefits obtained by physical activity, if excessively intense and over a great many hours, may be counteracted by the risk of AF.*"

Mont, L, et al. Physical activity, height, and left atrial size are independent risk factors for lone atrial fibrillation in middle-aged healthy individuals. **Europace**, *Vol. 10, 2008, pp. 15-20*

LAA thrombi rare in PVI patients

CLEVELAND, OHIO. Suffering a stroke during or after a pulmonary vein isolation (PVI) procedure is fairly rare (approximately 1.5% incidence rate), but obviously constitutes a serious complication. To avoid stroke during the procedure, patients are usually pre-screened for clots in the left atrium (LA) and left atrial appendage (LAA) using CT scanning and/or transesophageal echocardiography (TEE). In addition, heparin is used during the procedure and warfarin after to avoid post-procedural stroke. It is not known just how frequent LA or LAA clots are found in patients prior to their PVI.

Electrophysiologists at the Cleveland Clinic have just published the results of a study involving 1221 afibbers who underwent a pulmonary vein antrum isolation during the period 2000-2004. All patients underwent a pre-procedure CT scan and 60 also underwent a TEE. Nine patients were found to have a thrombus (clot) in the LAA as per the CT scan; however, when checked with TEE only three were actually clots, while the remaining 6 were smoke-like echo.

Two of the 3 patients had permanent afib with an average left ventricular ejection fraction (LVEF) of 48%, while the sole paroxysmal afibbers with a clot had an ejection fraction of only 25%. Thus, no paroxysmal afibbers with an ejection fraction of 50% or greater (normal) experienced LAA thrombi. Inasmuch as lone afibbers, by definition, have normal LVEFs (50% or greater), there were no incidences of LAA clots in paroxysmal, lone afibbers. It is likely that the two permanent afibbers had underlying heart disease (average LVEF was 48%), so it is probably safe to assume that even permanent, lone afibbers would be very unlikely to have thrombi in the LAA.

The Cleveland EPs conclude that a pre-procedure CT scan may be all that is required and that the use of TEE may not be needed in the case of paroxysmal afibbers with normal (50% or greater) LVEF.

Khan, MN, et al. Low incidence of left atrial or left atrial appendage thrombus in patients with paroxysmal atrial fibrillation and normal EF who present for pulmonary vein antrum isolation procedure. **Journal of Cardiovascular Electrophysiology**, *Vol.*

Editor's comment: This is good news indeed and confirms earlier research that lone afibbers are not prone to clot development in the LAA. As far as the CT scan or TEE is concerned, if given the choice, I would personally prefer the TEE so as to avoid the radiation inherent in CT scanning and the potential adverse effects of the contrast medium (x-ray dye) used during the scan.

Alcohol consumption linked to atrial flutter

SAN FRANCISCO, CALIFORNIA. It is well established that binge drinking and long-term alcohol abuse are risk factors for atrial fibrillation. Now researchers at the University of California report that alcohol consumption is also a significant risk factor for right atrial flutter (AFL), at least in persons at or below the age of sixty years. Their study involved 195 patients (121 with atrial fibrillation and 74 with AFL) who presented for cardioversion or ablation over a two-year period. A control group consisting of 132 patients with SVT (supraventricular tachycardia) and 54 with no known arrhythmia were also included. About 13% of participants had coronary artery disease or congestive heart failure, and 28% had hypertension.

The researchers found a strong correlation between daily alcohol consumption and the prevalence of AFL in those at or below the age of 60 years. After correcting for potential confounders (gender, race, hypertension, congestive heart failure, coronary artery disease, and body mass index) the researchers conclude that daily alcohol consumption (1-2 drinks per day) is associated with an 11 times greater prevalence of AFL. No such relationship was found among patients older than 60 years nor among those with atrial fibrillation. The California researchers also observed a strong correlation between daily alcohol intake and a shorter atrial effective refractory period (AERP) in the right atrium and speculate that a shorter AERP may facilitate the initiation of AFL by allowing propagation of a critically-timed premature atrial complex (PAC).

Marcus, GM, et al. Alcohol intake is significantly associated with atrial flutter in patients under 60 years of age and a shorter right atrial effective refractory period. **PACE**, *Vol. 31, March 2008, pp. 266-72*

Metoprolol improves cardioversion results

STOCKHOLM, SWEDEN. Electrical cardioversion is often used in an attempt to convert persistent afibbers to normal sinus rhythm (NSR). Unfortunately, the relapse rate is high and even with the use of class I or class III antiarrhythmic drugs, only about 50% of electro-cardioverted patients remain in NSR for 6 months or longer.

Researchers at the Karolinska Institute now report that pretreatment with the beta-blocker metoprolol (time-release version, Toprol XL) significantly improves the success rate for cardioversion. Their study involved 168 persistent afibbers who were randomized to receive metoprolol or a placebo starting at least a week prior to cardioversion (NOTE: only about 15% of the study participants were lone afibbers). On average, the participants were on metoprolol or placebo for 28 days prior to cardioversion and they were also prescribed warfarin (INR 2.1 – 3.0) for at least 3 weeks before and 6 weeks after cardioversion. The starting dose

of metoprolol was 50 mg/day with a 50 mg stepwise increase to a target dose of 200 mg once a day.

The participants were checked with an ECG 2 hours after cardioversion and then every week for 6 weeks, and then 3 and 6 months after cardioversion. During the first 6 weeks, 49% in the metoprolol group and 47% in the placebo group developed afib again and were given a second cardioversion. At the 6-month checkup, 46% of patients in the metoprolol group were still in NSR as compared to only 26% in the placebo group. It is also of interest to note that while 8% of placebo group members relapsed into afib within 2 hours of their first cardioversion, none of the patients in the metoprolol group did.

Nergardh, AK, et al. Maintenance of sinus rhythm with metoprolol CR initiated before cardioversion and repeated cardioversion of atrial fibrillation. **European Heart Journal**, *Vol. 28, 2007, pp. 1351-57*

Editor's comment: It is likely that the metoprolol pretreatment would be beneficial for adrenergic and perhaps mixed afibbers, but it is not at all clear that it would benefit vagal, persistent afibbers. Researchers at the Mayo Clinic have reported that a high blood level of C-reactive protein (CRP), a marker of systemic inflammation, prior to cardioversion is associated with a greater probability of afib recurrence within one month. There is also evidence that a low level of potassium is associated with poorer outcome of cardioversion. Thus, combating inflammation with *Moducare* or beta-sitosterol, and supplementing with potassium and magnesium prior to cardioversion may improve both the short- and long-term outcome of the procedure.

Fish oils and heart rate variability

BOSTON, MASSACHUSETTS. Heart rate variability (the variation in the interval between heart beats) is a powerful indicator of the state of the autonomic nervous system (ANS). The variation in the heart beat interval is usually measured via a 5-minute electrocardiogram or 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. Most scientific work on heart rate variability (HRV) 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. 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.

Other important measures derived from HRV analysis include the Poincare ratio and the short-term fractal scaling exponent (DFA1) which are related to sinoatrial node firing patterns. A low Poincare ratio and/or a high DFA1 correspond to a less erratic heart beat (more normal sinoatrial firing).

A low heart rate variability has been implicated in sudden cardiac death, ventricular fibrillation, angina, heart attack, atherosclerosis, and other heart-related problems. 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.

A team of researchers from Harvard Medical School, Washington University School of Medicine, Wake Forest University School of Medicine, and the University of Washington now reports that the consumption of oily fish and fish oils strongly influences HRV. Their study involved 4465 older men and women (average age at enrolment of 72 years) who were enrolled in 1989-1990 and then followed for 10 years. At enrolment all participants were given a resting 12-lead ECG or a 24-hour Holter monitor recording and information about their intake of fish and fish oils over the past year was obtained. The researchers observed a significant

correlation between the intake of broiled or baked fish (especially tuna) and HRV. They also observed a strong correlation between plasma levels of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) and HRV.

Study participants with a high broiled/baked fish intake experienced a greater HRV (higher SDNN) than those with a lower intake. They also showed lower LF power (lower adrenergic stimulation of the ANS) and higher HF power (increased vagal dominance of the ANS), thus resulting in a lower LF/HF ratio, again suggestive of vagal dominance. High fish consumers also had a lower Poincare ratio and a higher DFA1 indicating a more stable sinoatrial firing pattern. These correlations were also evident when comparing HRV variables with the intake of EPA + DHA. The researchers did not observe any correlation between HRV and the intake of fried (non-fatty) fish.

During the follow-up period, 542 deaths occurred related to cardiovascular causes (1.1%/person-year). The researchers found that high values of SDNN and DFA1 were associated with a 1.1% and 8.4% respectively reduced risk of cardiovascular death, while a low Poincare ratio was associated with a 5.9% risk reduction. They conclude that an increased intake of oily fish (or EPA + DHA) have significant beneficial effects on parameters influencing HRV, specifically an increase in vagal tone, modulation of adrenergic-mediated baroreceptor activity, and improved sinoatrial node function. NOTE: The average daily intake of EPA + DHA ranged from 47 mg to 927 mg.
Mozaffarian, D, et al. Dietary fish and omega-3 fatty acid consumption and heart rate variability in US adults. **Circulation,** *Vol. 117, March 4, 2008, pp. 1130-37*

Editor's comment: The confirmation that a high intake of EPA + DHA is associated with a decreased risk of cardiovascular death is indeed encouraging. However, the finding that a high oily fish/fish oil consumption is associated with vagal (parasympathetic) dominance may be less welcome news to vagal afibbers, especially since a high fish oil intake has also been associated with a lower resting heart rate.

Improved pulmonary vein isolation technique

CLEVELAND, OHIO. By far the majority of atrial fibrillation triggers are located in or around the pulmonary veins making pulmonary vein isolation (PVI) and pulmonary vein antrum isolation (PVAI) highly successful techniques for eliminating afib. Nevertheless, the procedures are not always successful with one of the reasons being that some triggers are located outside the ablation rings formed around the pulmonary veins.

EPs at the Cleveland Clinic estimate that the superior vena cava (large vein that carries the de-oxygenated blood from the upper half of the body to the right atrium) harbors about 6-8% of these extraneous (to the pulmonary veins) triggers. In a recent clinical trial they investigated the feasibility and safety of adding isolation of the superior vena cava (SVC) to the standard PVAI procedure as a means of improving overall success rate. The trial involved 407 afibbers with an average age of 55 years of which 51% had the paroxysmal variety, 39% were in permanent afib, and the remaining 10% had persistent afib.

The participants were divided into two groups – Group I consisting of 190 patients who had undergone an initial PVAI followed by a search for triggers in the SVC, and Group II who underwent a PVAI followed by empirical SVC isolation. Twenty-four patients (12%) in Group I exhibited triggers in the SVC (accompanied by triggers in the right superior pulmonary vein) that were successfully isolated leaving all 24 patients arrhythmia free for an average of 450 days post-procedure. Among the 217 Group II patients, 208 (96%) exhibited SVC potentials. These were successfully isolated by segmental ablation (approximately 50%) of the SVC circumference in 59% of patients. Complete ablation of the circumference was necessary in 19% of patients, and in 18% of patients complete isolation was not possible owing to excessive phrenic nerve stimulation.

During follow-up, 16% of all patients experienced afib recurrence. Six percent underwent successful PVAI or SVC touch-up procedures bringing the overall total success rate to 90%. The remaining 10% achieved satisfactory control of their afib through the use of previously ineffective antiarrhythmic drugs.

The Cleveland researchers conclude that the SVC harbors the majority of afib triggers outside the pulmonary veins, and that SVC isolation is feasible

and safe and should be considered as a standard adjunct to a regular PVAI. They found no evidence of SVC stenosis, but warn that SVC isolation may not be possible in all patients due to the danger of phrenic nerve injury.

Arruda, M, et al. Electrical isolation of the superior vena cava. **Journal of Cardiovascular Electrophysiology**, *Vol. 18, December 2007, pp. 1261-66*

Magnesium aids in cardioversion

HARTFORD, CONNECTICUT. Intravenous ibutilide (Corvert) is often used to chemically convert atrial fibrillation and atrial flutter to normal sinus rhythm. Unfortunately, the procedure only works in about 50% of cases and is accompanied by a 4% risk of developing Torsade de Pointes, a sometimes fatal ventricular arrhythmia. Researchers at the University of Connecticut now report that adding 4 grams of intravenous magnesium sulfate to the ibutilide protocol (2 grams before the first ibutilide dose and 2 grams over half an hour after the final ibutilide dose) will increase the odds of a successful conversion by a factor of 3.

The clinical trial included 229 patients who presented for cardioversion of atrial fibrillation (80%) or atrial flutter. Of these, 88% received ibutilide by itself, 87 patients received between 1 and 3 grams of intravenous magnesium sulfate, and the remaining 54 patients received a total of 4 grams of magnesium sulfate during the administration of ibutilide. The average age of the patients was about 66 years and most had hypertension or underlying heart disease. Only one case of Torsade de Pointes was observed during the trial and that involved a patient who was receiving ibutilide only. The beneficial effect of the addition of 1-3 grams of magnesium was not statistically significant indicating that at least 4 grams is needed.

The researchers speculate that the benefits of concomitant use of magnesium sulfate are related to magnesium's ability to increase intracellular potassium concentrations and regulate intracellular calcium concentrations.

Tercius, AJ, et al. Intravenous magnesium sulfate enhances the ability of intravenous ibutilide to successfully convert atrial fibrillation or flutter. **PACE**, *Vol. 30, November 2007, pp. 1331-35*

Editor's comment: Although there were very few lone afibbers in the study group, there is no reason to believe that the addition of magnesium sulfate to the ibutilide protocol would not benefit them as well. Also, if the beneficial effect of magnesium is related to its ability to increase intracellular potassium concentrations and regulate calcium concentrations, then it would seem logical that adding magnesium to cardioversion protocols involving other antiarrhythmics, or even electrical cardioversion, would also be beneficial. Perhaps having a warm bath with plenty of Epsom salt when using the on-demand (pill-in-the-pocket) approach with flecainide or propafenone may improve the odds of a quick conversion.

Inflammation in AF: Cause or effect?

HOUSTON, TEXAS. In 1997 Dr. Andrea Frustaci, MD and colleagues at the Catholic University of Rome made a fascinating discovery. They performed biopsies of the right atrium in 12 patients with LAF and found that 8 (67%) of them had evidence of a current or past inflammation in the heart tissue (myocarditis). They also checked 11 control subjects and found that none of their biopsy samples showed any signs of inflammation. The Italian researchers conclude that inflammation and its aftermath (fibrotic tissue) is a likely cause of LAF. The inflammation was found to be active in 3 of the 8 patients. These patients were treated with the anti-inflammatory medication prednisone. They had no further LAF episodes over a 2-year follow-up. The remaining patients were treated with propafenone, sotalol, flecainide or amiodarone and had numerous LAF episodes over the next 2 years[1].

In January 2002 two research papers were published that clearly support the inflammation connection[2,3]. Both papers, one by American researchers (Cleveland Clinic) and one by Greek researchers, report a significant association between the level of C-reactive protein (CRP), a marker of inflammation, and the presence and severity of LAF.

The Cleveland researchers found that patients with AF, with or without structural heart disease, had significantly higher blood levels of CRP than did controls (median value of 0.21 mg/dL versus 0.096 mg/dL). The average value for LAF patients was 0.21 mg/dL, which was not significantly lower than that found in AF patients with structural heart disease (0.23 mg/dL). CRP levels were generally higher if the patients were actually in atrial fibrillation or had come out of an episode within 24 hours of sampling. These patients had average CRP values of 0.30 mg/dL as compared to 0.15 mg/dL for AF patients in sinus rhythm. It was also clear that patients with persistent AF had higher CRP values than patients with paroxysmal AF (0.34 mg/dL versus 0.18 mg/dL). The researchers conclude that AF might induce or be induced by an inflammation, which in turn may promote the persistence of AF[2].

The Greek researchers tested CRP levels in 50 paroxysmal AF patients who were actually in fibrillation at the time of sampling and compared results to those obtained for 50 people in normal sinus rhythm. The AF patients had a median CRP level of 0.80 mg/dL as compared to 0.04 mg/dL for controls. The researchers observed that AF patients who could not be cardioverted had a much higher average CRP level (2.12 mg/dL) than did patients who were successfully cardioverted (0.50 mg/dL). They also noted that patients with an enlarged left atrium had considerably less success in being cardioverted. They conclude that high CRP levels are strongly associated with the presence of AF and with a lower chance of successful cardioversion[3].

In a recent review of these and other studies researchers at Baylor College of Medicine conclude that inflammation plays a significant role in the perpetuation and maintenance of atrial fibrillation. They point out that inflammation is a potent risk factor for stroke and suggest that it would be advisable to reduce it. Statin drugs are known to have anti-inflammatory properties, but their role in actually preventing AF is not clear. The use of glucocorticoids (dexamethasone, cortisone, methylprednisolone) has been found effective in reducing CRP levels and post-operative afib. There is also some evidence that pharmacological conversion with propafenone is more effective and longer lasting if methylprednisolone is added to the protocol. The addition of methylprednisolone was also found to decrease CRP levels by 80%. Finally, it is possible that ACE inhibitors and angiotensin receptor blockers may help prevent inflammation-induced atrial remodeling and thereby reduce the risk of paroxysmal afib becoming permanent.

The Baylor College researchers conclude that the preponderance of evidence supports the conclusion that inflammation is an independent risk factor for the initiation and maintenance of AF, but do caution that it is still not clear whether inflammation causes AF or AF causes inflammation[4].

In order to answer this question Turkish researchers recently carried out an experiment in which an attempt was made to induce atrial fibrillation in 39 patients undergoing an electrophysiologic study for syncope (fainting) of undetermined origin or palpitations with no documented arrhythmias. None of the participants had been diagnosed with afib, or acute or chronic inflammatory diseases. CRP levels were measured before the EP study as well as 6 and 24 hours after the study. The researchers were able to induce afib in 18 patients leaving the remaining 21 patients as a control group. CRP levels at baseline were not significantly different between the two groups (2.8 mg/L vs 4.5 mg/L or 0.28 mg/dL and 0.45 mg/dL).

However, 24 hours after the procedure CRP increased in both groups. To 3.9 mg/L (0.39 mg/dL) in the control group compared to 10.0 mg/L (1.0 mg/dL) in the group in which afib was induced (average episode duration was 4.8 hours). The Turkish researchers conclude that the induction of afib was accompanied by a significant inflammatory process and that even the EPS study by itself caused an increase in CRP (in the control group).[5]

References

1. Frustaci, Andrea, et al. Histological substrate of atrial biopsies in patients with lone atrial fibrillation. Circulation, Vol. 96, August 19, 1997, pp. 1180-84
2. Chung, Mina K., et al. C-reactive protein elevation in patients with atrial arrhythmias: inflammatory mechanisms and persistence of atrial fibrillation. Circulation, Vol. 104, December 11, 2001, pp. 2886-91
3. Dernellis, J. and Panaretou, M. C-reactive protein and paroxysmal atrial fibrillation: evidence of the implication of an inflammatory process in paroxysmal atrial fibrillation. Acta Cardiol, Vol. 56, No. 6, December 2001, pp. 375-80
4. Issac, TT, et al. Role of inflammation in initiation and perpetuation of atrial fibrillation. Journal of the American College of Cardiology, Vol. 50, November 20, 2007, pp. 2021-28
5. Pirat, B, et al. Comparison of C-reactive protein levels in patients who do and do not develop

atrial fibrillation during electrophysiologic study. American Journal of Cardiology, Vol. 100, 2007, pp. 1552-55

Editor's comment: It is obviously not clear whether atrial fibrillation is a consequence of inflammation or inflammation is a consequence of AF. However, it is known that inflammation is associated with remodeling of the atrium, which again is associated with perpetuation of the arrhythmia. Thus, it is clear that if an afibbers has signs of a systemic inflammation (CRP level above about 1.6 mg/L (0.16 mg/dL) steps should be taken to eliminate this inflammation. Apart from cutting out obvious inflammation triggers such as alcohol and caffeine, it would also be prudent to refrain from vigorous exercise and workouts until the inflammation has subsided. Supplementation with natural anti-inflammatories such as *Moducare*, curcumin, beta-sitosterol or *Zyflamend* is also an essential step in eliminating systemic inflammation and reducing CRP level.

Elimination/Reduction Protocol

In this issue we begin a new feature based on the results of LAF Survey 14. LAFS-14 received responses from 224 afibbers who had attempted to reduce or eliminate their afib burden (number of episodes x duration over a 6-month period) through means other than ablation and surgical procedures. The overall efficacy of the different protocols was summarized in the November 2007 issue of *The AFIB Report*. However, space limitations did not allow us to provide details of the successful protocols. Thus, the purpose of our new section is to describe the specific actions actually taken by afibbers who found ways of reducing their afib burden by at least 50% with the use of pharmaceutical drugs or such alternative approaches as trigger avoidance, supplementation, dietary changes, stress management, or elimination of underlying disease conditions such as hypoglycemia, sleep apnea, and GERD (gastroesophageal reflux disease).

Case No. 682

Female afibber – 56 years of age with vagal AF of 10 years standing;
no underlying heart disease
No. of episodes in 6 months prior to starting protocol: 48
Afib burden in 6 months prior to starting protocol: 192 hrs.
No. of episodes in 6 months after starting protocol: 2
Afib burden in 6 months after starting protocol: 2 hrs.
Time on protocol: 6 months
Still need to avoid triggers?: Yes, but much less so

Main components of effective protocol

Trigger avoidance: MSG, aspartame, and other excitatory food additives, caffeine, high glycemic index foods, heavy evening meals, dehydration
Diet changes: Elimination of wheat and adopted Zone diet
Supplementation: Magnesium, potassium, taurine, coenzyme Q10
Drug therapy: None
Stress management: Relaxation therapy, breathing exercises
Approaches to shorten episodes: Light exercise, hydrotherapy (ice baths, icy water on hands, hot/cold showers)
Approaches to reduce ectopics: Supplementation with magnesium, potassium, and taurine, low-sodium V8 juice

Background and details of protocol

I was having increased episodes with the use of Toprol XL which my doctor prescribed. I had taken the drug for 2 years. I began suffering with PACs and ectopics everyday as well. During that time I was faithfully avoiding every trigger I could. My situation only got worse. The first thing I did was to purchase Hans' book. I determined, with the help of his book, the web site, and the people participating on the Bulletin Board, that I was probably vagal and needed to eliminate the beta-blocker. I stopped the beta-blocker (slowly) at the same time I began experimenting with adding supplements. I experimented with dosages of magnesium to determine my bowel tolerance. I had always taken calcium and fish oil. I stopped those because my research indicated those could be counter productive. All the while I had changed my diet and continued to exercise, but not with the vengeance I used to. I eat breakfast now; I never used to. I eliminated bread and have increased my veggies. I'm not a fruit eater, but admittedly I have always had a lousy diet. I avoid triggers, watch my diet, exercise, drink lots of water and take my supplements. My biggest trigger is a large meal, especially in the evening. I try to walk or stay active at night after a meal. All this with my EPs blessing. He has given me Toprol for pill-in-the-pocket usage, but I have yet to use them. I am an avid golfer and will play in the heat of the day. I am very careful to avoid becoming dehydrated all the time, but especially when I am out golfing. I carry my own bag and walk, so I'm loaded down with water. All of these are taken after food: one 500-mg magnesium in the morning and one in the evening one 500-mg taurine in the morning and one in the evening. All of these are in the morning only - 1 multi, 500 mg Vit C, 100 mg of potassium, 150 mg CoQ10, 325 mg aspirin (doctor's continued wish, although I have only age and afib as risk factors) I know some of these doses (taurine, Vit C, mag) are low, but they are currently working. Perhaps I can increase them if things change. I started out purchasing my supplements at the corner drug store out of frustration. Once my current supply is gone, I fully intend to use the services for Hans' supplements. My education has included the need to be careful with supplements and I need to purchase the best forms of the products I take. I have yet to avoid an activity or an opportunity because of afib. Once, I played a round of golf while in afib. It did affect my putting! I think a positive attitude will go a long way in meeting this condition head on. Lastly, I'm very lucky because my condition is mild compared to many. I know from reading postings that some people suffer far more than I do. For that, I am grateful, but I have compassion for those who are not as fortunate.

THE AFIB REPORT is published 10 times a year by:

Hans R. Larsen MSc ChE, 1320 Point Street, Victoria, BC, Canada, V8S 1A5
E-mail: editor@afibbers.org World Wide Web: http://www.afibbers.org