THE AFIB REPORT

Your Premier Information Resource for Lone Atrial Fibrillation!

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Welcome to our autumn issue. The role of inflammation in atrial fibrillation continues to be hotly debated. In 1997 Dr. Andrea Frustaci in Rome found evidence of inflammation in the heart tissue of lone afibbers and demonstrated that treatment with the anti-inflammatory drug prednisone could help prevent recurrence. In 2002 American and Greek researchers independently arrived at the conclusion that AF patients, including those with lone AF, had higher blood levels of the inflammatory marker C-reactive protein (CRP) than did controls without AF. They also observed that persistent afibbers had higher CRP values than did paroxysmal afibbers, and that a high CRP level reduced the chance of a successful cardioversion.

In 2006 researchers at the Massachusetts General Hospital found no difference in CRP levels between healthy controls and lone afibbers (without hypertension). They concluded that AF on its own (without underlying heart disease, hypertension, or obesity) is not associated with evidence of systemic inflammation (elevated CRP levels). To further confuse the issue, electrophysiologists at the Hopital Cardiologique du Haute Leveque (Bordeaux) reported that a successful ablation significantly reduces the level of CRP, while an unsuccessful ablation does not. This would support the idea that being out of sinus rhythm causes inflammation (high CRP levels) rather than the other way around. This would also explain why CRP levels increase from paroxysmal to persistent to permanent AF.

In this issue, Swedish researchers report that lone atrial fibrillation is not associated with increased levels of CRP or interleukin-6 (IL-6), Greek researchers confirm that elevated CRP levels are associated with a poorer chance of a successful cardioversion, and University of California researchers report an association between an increase in CRP levels following an ablation and early recurrence of AF. So the cause/effect relationship between inflammation and AF is still not entirely clear. However, as a systemic inflammation is a bad actor all around and has been implicated in atherosclerosis, angina, heart attack, diabetes, depression, and most common cancers, it is clearly a good idea to keep CRP levels under control; if necessary, by supplementing with such natural anti-inflammatories as fish oils, Zyflamend, beta-sitosterol, bromelain, curcumin, boswellia, and Moducare.

Also in this issue we report on the perils of marathon running, that 8 million Chinese join the ranks of afibbers, age is no detriment to a successful PVI, statin drugs are dangerous, known risk factors for lone AF are many and varied, and much more to read.

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 lots of NSR,

Hans

Highlights

Risk factors for lone atrial fibrillation

UTRECHT, THE NETHERLANDS. The majority (80-90%) of all patients with atrial fibrillation (AF) have some type of cardiovascular disease or abnormality that can explain the cause of their AF. Thus heart disease-related AF involves an arrhythmogenic substrate, that is, an atria which is diseased, has been stretched, or contains a substantial amount of fibrotic tissue likely formed by long-term inflammation.

On the other hand, ***lone*** atrial fibrillation, does not involve diseased or otherwise compromised atria, but is rather an "electrical" problem where certain triggers initiate afib episodes through their action on especially sensitive foci mostly located in and around the entrances of the pulmonary veins into the left atrium. This explains why lone afibbers have normal life expectancy, a low stroke risk, and why paroxysmal (intermittent) afib rarely progresses to persistent or permanent. However, for AF patients with diseased atria (arrhythmogenic substrate) the situation is quite different. These patients have a substantially greater stroke risk, higher mortality, and are much more likely to progress from paroxysmal to permanent AF.

While heart disease is, by definition, not a cause of lone AF, there are other known causes. First among these are electrolyte disturbances (particularly deficiencies in magnesium and potassium) and autonomic nervous system imbalances. However, alcohol consumption, especially binge drinking, and cocaine and certain pharmaceutical drugs can also increase the risk of lone afib. Thyroid disorders can be an underlying cause, as can hypoglycemia and an excessive consumption of tyramine-containing foods. It is also clear that chromosomal abnormalities can increase the risk of lone afib. A recent LAF survey revealed that 43% of 100 respondents had a close relative with cardiac arrhythmia – so the "genetic connection" is by no means uncommon. Finally, there is also evidence that the rare disease pheochromocytoma can cause AF to develop. Once all these possible causes have been ruled out, ***lone*** atrial fibrillation becomes "idiopathic" which really is the proper designation for the type of afib most "members" of www.afibbers.org experience.

Lately, several additional initiators of lone AF have been discovered. In a recent review by Dutch researchers it is pointed out that obesity, obstructive sleep apnea (OSA), increased pulse pressure (the difference between systolic [pumping] and diastolic [filling] blood pressure), systemic inflammation, and long-term participation in vigorous endurance sports have all been associated with an increased risk of developing AF.

Obese people (BMI greater than 30) have been found to have twice the risk of developing lone afib when compared to those with normal body weight (BMI between 18.5 and 25 kg/sq.m). Similarly, the presence of OSA increases afib risk by a factor of 2. Several studies have found a clear association between systemic inflammation (high levels of hs C-reactive protein and interleukins) and the presence of lone AF, but it is not clear whether inflammation causes afib or afib results in inflammation.
Schoonderwoerd, BA, et al. New risk factors for atrial fibrillation: causes of 'not-so-lone atrial fibrillation'. **Europace**, *Vol. 10, 2008, pp. 668-73*

Editor's comment: Although it is of great interest to discover additional possible causes of afib, the great majority of lone afibbers do not have any of these conditions, so the most important causes of the ***lone*** afib epidemic are still to be discovered.

Platelet activation in acute atrial fibrillation

MAYWOOD, ILLINOIS. It is not known whether atrial fibrillation as such results in a hypercoagulable state that could increase the risk of ischemic stroke. A group of cardiovascular researchers at Loyola University Medical Center now provides an intriguing insight into this question.

Their study involved 22 patients with paroxysmal afib who were scheduled to undergo radiofrequency catheter ablation. The patients did not have left ventricular dysfunction, rheumatic valve disease, mitral valve prolapse, or any significant valvular regurgitation; however, about 30% had hypertension and about 14% had diabetes. The study participants were divided into two groups of 14 (Group A) and 8 patients (Group B) respectively. The only statistically significant difference between the two groups was a greater preponderance of men (93%) in Group A than in Group B (50%).

All patients were in sinus rhythm when the study began and had sheaths (tubes) inserted in the femoral veins and coronary sinus (via the right internal jugular vein) for collection of blood samples. Atrial fibrillation was induced for 15 minutes by burst pacing (330 bpm or a cycle length of 180 ms) in Group A resulting in a ventricular rate (heart beat) of 121 bpm. In Group B atrial pacing to achieve a ventricular rate at 120 bpm without going into afib was applied for 15 minutes. Analysis of blood samples taken at the coronary sinus in Group A revealed increased platelet activation and thrombin generation as well as reduced nitrogen oxide production when compared with Group B. The blood sample (systemic) taken from the femoral vein did not change with pacing indicating that the effect is localized to the heart – at least for the first 15 minutes.

The researchers conclude that their findings may help explain why short episodes of atrial fibrillation predispose to stroke, especially in patients with underlying vascular disease such as diabetes and hypertension.

Akar, JG, et al. Acute onset human atrial fibrillation is associated with local cardiac platelet activation and endothelial dysfunction. **Journal of the American College of Cardiology**, *Vol. 51, May 6, 2008, pp. 1790-93*

Editor's comment: Obviously, the question is "Do these findings apply to lone afibbers without hypertension and diabetes?" Unfortunately, the researchers did not separate out the effects due to hypertensive/diabetic subjects vs. those with no stroke risk factors, so it is impossible to say and, in all fairness, the study population really was not large enough to allow such a separation. However, the results of this study would certainly support the supplementation with natural antiplatelet agents such as vitamins C, E, B3 and B6, and fish oils, some of which would also have an inhibitory effect on the formation of prothrombin. Nattokinase would not have any effect on platelet activation or prothrombin or thrombin formation, but would be effective in increasing fibrinolytic actively and thereby prevent blood clots from forming.

The two faces of marathon running

ESSEN, GERMANY. It is well established that regular physical exercise reduces the risk of cardiovascular disease, but it is equally well established that vigorous exercise increases the short-term risk of coronary events (heart attack and stroke), especially among elderly people not accustomed to exercise. A group of German researchers have now taken a look at the cardiovascular risk profile of 108 apparently healthy male marathon (42 km) runners aged 50 years or older and with at least 5 marathons under their belt. Not surprisingly, they found that the marathon runners' Framingham risk score (7%) was significantly lower than the score in a group of healthy age-matched controls (11%). Marathon runners experienced a 52% higher HDL cholesterol level, an 18% lower LDL cholesterol level, a 15% lower body mass index, and a 12% lower systolic blood pressure as well as a significantly lower resting heart rate (65 bpm vs 76 bpm). Coronary artery calcification (CAC) score was similar in marathon runners and age-matched men; however, when CAC scores were compared for marathon runners and age-matched men with a similar low Framingham risk score, then 36% of marathon runners were found to have a CAC score of 100 or higher as compared to only 22% in the control group.

MRI studies of the marathon runners indicated that 12% had late gadolinium enhancement (LGE), a marker of cardiovascular damage. Overall, LGE was associated with high CAC scores and an increased number of completed marathons. During a mean follow-up of 21 months, 4 coronary events occurred among the runners, 2 of which required resuscitation.

The researchers conclude that marathon running does not seem to protect against atherosclerosis as indicated by a high CAC score and may, in fact, exacerbate the problem due to excessive vascular

oxidative stress, and frequent bursts of inflammatory cytokines experienced during long-distance running. The authors conclude that, *"Regular marathon running has a beneficial effect on the cardiovascular risk factor profile but the extent of calcified coronary plaque is underestimated from that risk factor profile, with 36% of marathon runners aged 50 years or greater having a CAC score of 100 or greater and 9% of these requiring coronary revascularization during two years of follow-up. Advanced CAC scores seem to contribute to increased myocardial damage and appear to impair outcome. Frequent marathon running may not protect these athletes from the risk of coronary events*."

Mohlenkamp, S, et al. Running: the risk of coronary events. **European Heart Journal**, *Vol. 29, 2008, pp. 1903-10*

Schmermund, A, et al. The risk of marathon runners – live it up, run fast, die young? **European Heart Journal**, *Vol. 29, 2008, pp. 1800-02 (editorial)*

Editor's comment: This study adds to the evidence that exercise in moderation is good, while vigorous exercise taken to extremes may produce unhealthy stress on the cardiovascular system.

Is there an age limit for PVI ablation?

PALO ALTO, CALIFORNIA. The prevalence of atrial fibrillation among individuals 75 years or older is about 15%. Clearly it is important to answer the question, "Is PVI ablation safe and effective for this age group?" A team of electrophysiologists from the Cleveland Clinic, Marin General Hospital, and the Umberto I Hospital in Italy have pooled their outcome results for a total of 174 patients over 75 years of age who underwent pulmonary vein antrum isolation and isolation of the superior vena cava guided by mapping of electrically active areas and intracardiac echocardiography.

The patients were followed for an average of 20 months by serial event recorder transmissions. The immediate complete success rate (no afib, no antiarrhythmics) was 73%. An additional 16 patients (out of 20) were afib-free after a second procedure (repeat rate of 16%) bringing total success rate to 82%. An additional 18 patients maintained sinus rhythm with the use of previously ineffective antiarrhythmics resulting in a partial success rate of 10%. During the 194 procedures, 1 patient suffered a stroke, 3 experienced groin hematomas, and 1 suffered a hemothorax secondary to right internal jugular vein catheterization. No tamponade, atrial-esophageal fistula, or pulmonary vein stenosis were reported.

During the first 3 months, 3 patients experienced a thromboembolic event despite being on warfarin. However, in 2 cases their INR was below 2.0. In the third case, the patients had the stroke 2 days after undergoing cardioversion and it is suggested that the atrial thrombus (blood clot) was secondary to the atrial stunning that follows direct current cardioversion.

The researchers point out that their common strategy is to discontinue warfarin after 3 months in all patients whose left atrial mechanical function is normal provided they have not experienced recurrence of AF. This would seem to be safe and appropriate since no thromboembolic events were observed over a 16-month period in the group that discontinued warfarin. The conclusion drawn from the study is that, "*PVI appears to be a safe and effective treatment strategy for the eradication of AF in septuagenarians. Medium- to long-term success can be achieved in most patients, and the overall rate of complications is low."*

Corrado, A, et al. Efficacy, safety, and outcome of atrial fibrillation ablation in septuagenarians. **Journal of Cardiovascular Electrophysiology,** *Vol. 19, August 2008, pp. 807-11*

Hurwitz, JL. Atrial fibrillation treatment in the elderly. **Journal of Cardiovascular Electrophysiology**, *Vol. 19, August 2008, pp. 812-14 (editorial comment)*

Editor's comment: The finding that pulmonary vein ablation is safe and effective even for patients over the age of 75 years is certainly welcome news. Although the participants in the study did not have lone afib (68% had structural heart disease and 24% coronary artery disease) and 65% had a CHADS score of 2 or higher, there is no reason to believe that the researchers' conclusion would not be applicable to lone afibbers who could perhaps expect an even better outcome.

Eight million Chinese join the ranks with AF

BEIJING, CHINA. It is estimated that 2.5 million people in the USA and 4.3 million individuals in the EU suffer from atrial fibrillation (AF). Up until now, it has not been clear whether AF is a disease related to Western civilization and lifestyle, or whether it is a worldwide phenomenon. Chinese researchers now provide a partial answer. A large-scale epidemiological study involving more than 29,000 individuals from 13 provinces and 14 ethnic groups in China found an age-adjusted AF rate of 0.61%, suggesting that approximately 8 million adults in China have AF. This prevalence is very similar to that observed in the USA and Europe, thus pretty well laying to rest the idea that AF is somehow related to Western lifestyle and diet.

The Chinese researchers confirmed that the risk of AF increases with age and is more prevalent among men. The percentages of nonvalvular AF, valvular AF, and lone AF were 65%, 13%, and 22% respectively. Hypertension and heart failure were the most common underlying comorbidities in hospitalized AF patients – the majority (76%) of whom had nonvalvular AF with the remaining 24% having valvular AF. Thus, it would appear that lone afibbers are not hospitalized in China.

The incidence of ischemic stroke was high among non-hospitalized individuals with AF at a rate of 12.1% a year vs. 2.1% a year in the general population. Among hospitalized patients with nonvalvular AF, the rate was even higher and dramatically dependent on age. Thus, the average incidence was 4.3% in patients younger than 40 years, but almost 33% in those 80 years of age or older. The main risk factors for stroke in hospitalized patients were found to be hypertension, high systolic blood pressure, diabetes, left atrial thrombi, and age at or above 75 years.

The epidemiological study found that only 2.7% of AF patients were on warfarin and none had their INR monitored on a regular basis. A study involving 828 patients with nonvalvular AF found that warfarin therapy was twice as effective as aspirin therapy (150 – 160 mg/day) in preventing ischemic stroke.

Hu, D and Sun, Y. Epidemiology, risk factors for stroke, and management of atrial fibrillation in China. **Journal of the American College of Cardiology**, *Vol. 52, No. 10, September 2, 2008, pp. 865-68*

Statin drugs are dangerous

WASHINGTON, DC. Well over a thousand cases of rhabdomyolysis (an often fatal muscle disease) caused by the ingestion of statin drugs (atorvastatin [Lipitor], simvastatin [Zocor], lovastatin [Mevacor], pravastatin [Pravachol]) have been reported. Now the FDA has issued a warning not to take more than 20 mg/day of simvastatin if also taking amiodarone since doing so will markedly increase the risk of rhabdomyolysis. The warning also applies to the combination of simvastatin and ezetimibe (Vytorin) and the combination of simvastatin and extended-release niacin (Simcor). The FDA press release states that:

- The risk of rhabdomyolysis is increased when higher doses of simvastatin are administered with amiodarone. The precise mechanism is unknown, but is related to the fact that amiodarone inhibits the cytochrome P450 3A4 (CYP3A4) enzyme. This is the same enzyme that metabolizes simvastatin. Prescribers should consider use of another statin for patients taking amiodarone, or initiating amiodarone therapy, who require simvastatin doses greater than 20 mg daily to meet their lipid goals.

- Rhabdomyolysis has been reported with all statins. Predisposing risk factors for rhabdomyolysis include advanced age (greater than 65 years), uncontrolled hypothyroidism, and renal impairment.

- The FDA does not have data on how varying the dose of amiodarone in patients taking simvastatin affects the risk of developing rhabdomyolysis.

http://www.fda.gov/cder/drug/InfoSheets/HCP/simvastatin_amiodaroneHCP.htm

Editor's comment: If a combination of amiodarone and 20 mg/day or more of simvastatin is dangerous enough to require a warning, how dangerous is

amiodarone plus 10 mg/day? Who knows? The FDA does not seem to. What is perhaps more disturbing is that the FDA has no data regarding the effect of amiodarone dosage on rhabdomyolysis incidence when combined with simvastatin. Could simvastatin combined with a loading dose of amiodarone (800 – 1600 mg/day) be vastly more dangerous than the combination of simvastatin and an amiodarone maintenance dose of 200 – 400 mg/day? Nobody knows, so afibbers on or contemplating amiodarone should be very careful about also taking statin drugs, particularly simvastatin.

Predicting AF occurrence following ablation

BOSTON, MASSACHUSETTS. Success rates for pulmonary vein isolation (PVI) vary widely and depend primarily on the skill and experience of the electrophysiologist (EP) performing the ablation. In order to ensure optimum results it is common practice to measure the electrical potential between the pulmonary veins and the left atrium outside of the ablation rings after completing the procedure. If potentials are still present, thus indicating incomplete isolation, the ablation is continued until no potentials are evident. At this point, some EPs consider the procedure complete, while others do a "final check" by trying to induce afib by rapid burst pacing of the atrium often accompanied by infusion of isoproterenol, a drug capable of inducing atrial fibrillation. If AF can be induced, then further ablation is carried out, if necessary on the left atrium wall and roof, the superior vena cava, etc. Only when AF can no longer be induced is the procedure deemed complete. Unfortunately, the lack of inducibility does not ensure long-term freedom from afib recurrence; thus, the search continues for a method to predict long-term success before the ablation procedure is terminated.

A group of researchers from Harvard Medical School and McGill University now report that trying to induce AF by administering an external shock (as done in electrocardioversion) to the ablatee after burst pacing and isoproterenol infusion may help in determining the need for further ablation, or the continued use of antiarrhythmics, in order to achieve long-term success. The study included 116 patients who underwent PVIs guided by electroanatomical mapping (CARTO, Pappone method). For 17 of the patients it was their second procedure. Following PV isolation, AF could be induced in 19 patients (16%) with burst pacing with or without isoproterenol. Nine of these patients were rendered non-inducible through further ablation. Burst pacing induced atrial tachycardia in 26 patients, 20 of whom were successfully ablated for this arrhythmia. Subsequent to the burst pacing, 81 patients in whom AF could not be induced were given a 30 J external shock timed to the peak of the R wave (most vulnerable time for initiation of AF). Among these patients, 16 went into afib, while in the remaining 65 (80%) afib could not be induced.

After an average follow-up of 16 months, 54% of ablatees in whom AF could be induced either by burst pacing or shock had experienced recurrent AF vs. only 21% among non-inducible patients. Comparing only those who were non-inducible by burst pacing and underwent subsequent shock, the recurrence rate at one year was 60% in patients who went into afib after the shock vs. only 18% in those who did not. The researchers conclude that administering a shock at the end of the procedure to ablatees who were non-inducible by burst pacing (with or without isoproterenol) may help to guide post-procedure management so as to reduce the incidence of recurrence. They also note that besides inducibility, mitral regurgitation was also associated with a poorer long-term success. Experiencing paroxysmal (intermittent) afib was, however, associated with a significantly better long-term prognosis than having persistent or permanent afib.

Wylie, JV, et al. Inducibility of atrial fibrillation with a synchronized external low energy shock post-pulmonary vein isolation predicts recurrent atrial fibrillation. **Journal of Cardiovascular Electrophysiology** *[Epub ahead of press]*

Ilkhanoff, L and Goldberger, JL. Recurrent atrial fibrillation after ablation. **Journal of Cardiovascular Electrophysiology** *[Epub ahead of press] (editorial comment)*

Editor's comment: This study clearly demonstrates that equating complete isolation of the pulmonary veins at the end of the procedure with long-term success is not realistic. Thus, results of studies using this endpoint as proof of the capability of new catheters, robot-assisted systems, etc. should be taken with a very large grain of salt indeed.

Elevated CRP = cardioversion failure

IOANNINA, GREECE. Several studies have uncovered an association between elevated levels of the inflammation marker C-reactive protein (CRP) and atrial fibrillation (AF). Inflammatory markers, mainly CRP, have been related to the risk of developing AF, the persistence of AF (paroxysmal, persistent, permanent), recurrence after cardioversion, and left atrium enlargement. Now Greek researchers weigh in with a study designed to determine the relationship between CRP level prior to cardioversion and time to first recurrent afib episode.

The study included 60 patients with persistent afib between the ages of 61 and 75 years, 60% of whom were men. The participants were free of valvular heart disease, congestive heart failure, prior heart attack, and thyroid dysfunction, so were a relatively healthy group although not classified as lone afibbers. A significant exclusion criteria was that the patients could not have been taking antioxidants or multivitamins. They had their CRP level measured prior to direct current cardioversion and were given amiodarone after the conversion (200 mg/day x 3 during first week, 200 mg/day x 2 during second week, and 200 mg/day thereafter). Patients who did not convert or who reverted back to AF within an hour were excluded from further follow-up.

The researchers found a clear correlation between CRP level and the percentage of patients who remained in sinus rhythm over the 3-year follow-up period. In the group of patients with a CRP level less than 0.43 mg/dL (4.3 mg/L), 45% were still in sinus rhythm at the end of 3 years. The corresponding figures for CRP levels between 0.43 and 0.8 mg/dL and CRP level greater than 0.8 mg/dL were 13% and 17% respectively. The researchers conclude that baseline CRP levels can be used to estimate the likelihood of persistent afibbers remaining in sinus rhythm after undergoing a successful electrical cardioversion.

Korantzopoulos, P, et al. Long-term prognostic value of baseline C-reactive protein in predicting recurrence of atrial fibrillation after electrical cardioversion. **PACE**, *Vol. 31, October 2008, pp. 1272-76*

Editor's comment: This study adds to the evidence of a close association between inflammation, as measured by CRP level, and the risk and persistence of AF. Although it is not entirely clear whether inflammation causes AF or AF causes inflammation, it would seem prudent for afibbers to maintain their CRP levels as low as possible. This can be achieved by regular supplementation with such natural anti-inflammatories as Zyflamend, beta-sitosterol, bromelain, curcumin, boswellia, Moducare, quercetin, and fish oil.

Inflammation and atrial fibrillation

LINKOPING, SWEDEN. It is well recognized that there is a significant association between the level of the inflammatory markers C-reactive protein (CRP), and interleukin-6 (IL-6) and atrial fibrillation (AF) in general. However, it is not clear whether inflammation causes AF or AF causes inflammation. There is also evidence that the observed relationship between inflammation and AF is due not to afib as such, but rather to the comorbid conditions (heart disease, hypertension) that generally accompany it. Swedish researchers now add the results of a small pilot study to the knowledge base regarding AF and inflammation.

Their study involved 28 patients scheduled for radiofrequency ablation. Ten had paroxysmal AF, while 8 had permanent AF. None had structural heart disease or inflammatory conditions. The control group consisted of 10 patients with Wolf-Parkinson-White (WPW) syndrome and no evidence of AF. After catheterization, but before ablation all patients had blood samples drawn from the femoral vein, right atrium, coronary sinus, and the left and right pulmonary veins. The level of CRP, IL-6, and IL-8 (interleukin-8) were measured in the samples.

All study participants were found to have normal levels of CRP and IL-6, thus confirming that lone AF is not associated with an increased CRP or IL-6 level. The level of IL-8, however, was elevated in participants with permanent AF, specifically in the samples from the femoral vein, right atrium, and coronary sinus. Surprisingly, the IL-8 level was not elevated in the samples from the pulmonary veins. The researchers conclude that permanent AF is associated with a systemic inflammation, perhaps caused by vascular endothelial damage or dysfunction. They also speculate that IL-8 may somehow be consumed during passage through the lungs. Their final conclusion is that, "*Taken*

together, these data seem to support the concept that the elevated levels of C-reactive protein and IL-6 in patients with AF reported in other studies were likely related to the presence of other co-morbid conditions that existed in these patient cohorts rather to AF itself."

In an accompanying editorial two researchers point out that the increase in IL-8 may be related to endothelial activation, perhaps due to local perturbations in shear stress related to the irregular and fast heart rate experienced in permanent afib.

Liuba, I, et al. Source of inflammatory markers in patients with atrial fibrillation. **Europace**, *Vol. 10, 2008, pp. 848-53*

Melenovsky V and Lip, GYH. Interleukin-8 and atrial fibrillation. **Europace**, *Vol. 10, 2008, pp. 784-85 (editorial)*

Ablation-associated inflammation

SAN FRANCISCO, CALIFORNIA. There is substantial evidence that any kind of catheterization or surgical procedure involving the heart causes an inflammatory response. There is also evidence that inflammation can cause arrhythmia. Finally, it has been observed that recurrent afib episodes are fairly common in the first 3 months following a pulmonary vein isolation (PVI) procedure and the occurrence of such episodes is not necessarily indicative of long-term failure.

A group of electrophysiologists at the University of California now report that the level of the inflammation marker CRP increased significantly in a group of ablated afibbers who experienced recurrent episodes within the first 7 weeks following the procedure. A similar increase in CRP was not seen in afibbers who did not experience any afib episodes during their first 7 weeks of recovery. The CRP level in both groups declined between the 7-week follow-up period and a second follow-up at 26 weeks.

The researchers conclude that the extent of left atrial tissue damage inherent in curative AF ablation generates a protracted inflammatory state with proarrhythmic effects.

McCabe, JM, et al. Protracted CRP elevation after atrial fibrillation ablation. **PACE**, *Vol. 31, September 2008, pp. 1146-51*

Editor's comment: I am still not sure in my own mind whether the association between elevated CRP levels and afib is due to inflammation causing afib or afib causing inflammation. However, assuming that inflammation is indeed the causative factor, then it would make sense to take steps to dampen the post-ablation inflammation by supplementing with natural anti-inflammatories such as Zyflamend, beta-sitosterol, bromelain, curcumin, boswellia, Moducare, quercetin, and fish oil.

Elimination/Reduction Protocol

Case No. 684

Female afibber – **60 years** of age with **vagal AF** of **6 years standing;** no underlying heart disease
No. of episodes in 6 months prior to starting protocol: **25**
Afib burden in 6 months prior to starting protocol: **1000 hrs**
No. of episodes in most recent 6 months after starting protocol: **0**
Afib burden in most recent 6 months after starting protocol: **0 hrs**
Time on protocol: **33 months**
Still need to avoid triggers?: **Yes**

Main components of effective protocol

Trigger avoidance: **MSG, aspartame, alcohol, caffeine, high glycemic index foods, heavy evening meals, dehydration, sleeping on left side**
Diet changes: **Eliminated gluten and wheat, modified paleo diet.**
Supplementation: **Magnesium + taurine**
Drug therapy: **None**
Stress management: **Breathing exercises, yoga**
Approaches to shorten episodes: **-**
Approaches to reduce ectopics: **Supplementation with magnesium + taurine**

Background and details of protocol

I began supplementing with magnesium and taurine almost 2 years ago. I was already supplementing with omega-3 oil, Multibionta, coenzyme Q10, vitamin E, etc., but did not notice any difference until the taurine was added. I now supplement with 4 grams a day. I adopted a modified paleo diet and after having tests for allergies gave up eating wheat and gluten products as I reacted badly to them during the tests. This has resulted in no afib at all for 33 months. Once my cardiologist took me off warfarin, I had an immediate improvement to what remained of my GERD problem. This had already been helped by the supplements. Within a month of stopping the warfarin, the GERD disappeared completely and has not returned. My dietary regimen is very strict and absolute avoidance of triggers is a must, but it continues to be worth the effort.

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