THE AFIB REPORT

Your Premier Information Resource for Lone Atrial Fibrillation!

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In July 2005 Italian researchers reported a convincing association between atrial fibrillation and the presence of a Helicobacter pylori infection. Researchers at the Mayo Clinic have now confirmed this association in a study of 743 patients with suspected cardiovascular problems. They found that 65% of AF patients were positive for H pylori as compared to 55% among those without AF. The correlation between afib and seropositivity to H pylori was particularly strong among younger patients (less than 50 years of age), but not significant among those over the age of 70 years. The researchers suspect that ectopy within the pulmonary veins may be aggravated by inflammation in the esophagus or stomach caused by a chronic H pylori infection. So should one embark on a H pylori eradication program? Unless you have stomach ulcers, probably not. Elimination of H pylori may increase the risk of developing GERD (gastroesophageal reflux disease) – a known promoter of afib episodes.

Most afibbers who have undergone a PVI have spent time wondering about how well the atrium performs after an ablation. Spanish researchers have investigated this and found that the ejection fraction (a measure of the atrium's pumping capacity) increased or remained constant in most patients following a successful PVI, but decreased in those whose ablation and not been successful. This is just one more reason to choose your ablation EP with care.

Other Spanish researchers have made a good stab at determining the variables affecting PVI success, while Cleveland Clinic researchers report that preexisting scarring in the left atrium lowers the chance of a successful PVI by almost 40% (from 81% to 43%).

Also in this issue we report that the bleeding risk for older patients taking warfarin probably outweighs the benefits in ischemic stroke prevention, that metabolic syndrome is a significant risk factor for AF, as is the use of alendronate (Fosamax) to prevent osteoporosis.

As many of you will know, I have an affiliate agreement with Xtend-Life, a highly respected New Zealand manufacturer of unique, well-formulated supplements not available from manufacturers in the US and Canada. Xtend-Life recently announced the release of a line of exceptionally well-priced skin care products based on natural ingredients. I am not into skin care products myself (Nivea will do just fine for me), but Judi raves about these new products, so you may wish to give them a try. Xtend-Life has a 100% guarantee of satisfaction and provides quick and inexpensive shipping worldwide. For more information go to http://www.afibbers.org/vitamins/skincare.htm

Wishing you a safe and enjoyable summer with lots of NSR,

Hans

Highlights

Afib begets afib – Or does it?

WARREN, NEW JERSEY. Animal experiments have shown that continued pacing of the heart makes paroxysmal (intermittent, self-converting) atrial fibrillation (AF) progress to persistent (episodes lasting longer than 7 days needing cardioversion) and permanent AF. Hence the expression "afib begets afib". The question is, "Do these findings apply to humans"? After all, patients with paroxysmal AF do not have their heart constantly paced and do spend often long periods in normal sinus rhythm. A group of electrophysiologists at the University of Florence, Italy, the Robert Wood Johnson School of Medicine, and Medtronic Inc. (a major manufacturer of pacemakers and implantable cardioverter/defibrillators) now provides at least a partial answer to this question.

Their study involved 330 patients with a history of paroxysmal AF and bradycardia (mean age of 70 years, 61% male). All suffered from heart failure to varying degrees and had had a pacemaker (Medtronic Model AT501) installed prior to the study. Most patients (79%) experienced some form of cardiovascular disease with 61% having hypertension; thus, the proportion of lone afibbers was insignificant and the results of the study may not apply to lone afibbers. After an average follow-up of one year and 3 months, 24% of the study participants had converted to persistent afib with the median time to conversion being 9 months (103 days). The researchers noted the following important differences between the patients who remained in paroxysmal AF (Group 1) and those who progressed to persistent AF (Group 2).

- Prior to conversion to persistent afib, patients in Group 2 were more likely to experience afib on any given day and had a higher average afib burden (no. of episodes x duration) than did those in Group 1.

- Prior to conversion to persistent afib, there was a significant linear increase in daily afib burden (mean increase of 14 sec/day) in Group 2, but no increase (on average) in Group 1.

- The transition from paroxysmal to persistent afib in Group 2 was quite abrupt and was preceded by a few days of normal sinus rhythm.

- The increase in afib burden over time was substantially higher among patients with cardiovascular disease (average 0.18 min/day) than among those with no cardiovascular disease (CVD) where the burden actually declined slightly over time (average – 0.06 min/day). Patients with CVD were also more likely to progress to persistent AF.

- Atrial premature beats (PACs) were more frequent in patients without CVD, but decreased with time in all patients.

The researchers conclude that structural remodeling (substrate modification) is critical to the transition to persistent afib. The changes to the substrate may involve fibrosis, apoptosis (cell death), and altered cellular junction proteins. In an accompanying editorial, electrophysiologists at the Tufts-New England Medical Center suggest that the renin angiotensin aldosterone system (RAAS) may play a significant role in the substrate modification and that ACE inhibitors and/or angiotensin receptor blockers may help slow down the structural remodeling. They also suggest that targeting just the pulmonary veins during an ablation (pulmonary vein isolation) is unlikely to suffice in the case of persistent or permanent AF.

Saksena, S, et al. Progression of paroxysmal atrial fibrillation to persistent atrial fibrillation in patients with bradyarrhythmias. **American Heart Journal**, *Vol. 154, No. 5, November 2007, pp.884-92*

Homoud, MK and Estes, M. Shedding new light on the pathophysiology of conversion of paroxysmal atrial fibrillation into persistent atrial fibrillation. **American Heart Journal**, *Vol. 154, No. 5, November 2007, pp.801-04*

Editor's comment: In considering the above findings it should be kept in mind that the study did

not involve a significant proportion of lone afibbers. Nevertheless, the mechanism underlying the progression from paroxysmal to persistent AF (substrate modification) is likely to be similar. If increasing afib burden is indeed a universal sign of progression, then lone afibbers who experience such an increase may wish to consider medication with an ACE inhibitor (lisinopril, enalapril, ramipril) or an angiotensin receptor blocker (losartan, valsartan, irbesartan) in order to slow down structural remodeling. Although there is no published evidence to support this, it is also possible that proteolytic enzymes (Serrapeptase, Wobenzym, nattokinase) may be useful in slowing down, or perhaps even reversing, the fibrosis component of substrate modification and thereby help prevent progression to persistent AF.

Helicobacter pylori and atrial fibrillation

ROCHESTER, MINNESOTA. In 2005 Italian researchers reported that patients with atrial fibrillation tended to have a higher level of IgG antibodies to *Helicobacter pylori* (seropositive) than did healthy volunteers. They speculated that the *H pylori* bacterium may adversely affect the NA^+/K^+-ATPase pump responsible for maintaining homeostatic balance in individual heart cells. A disturbance of this balance may trigger AF by creating abnormal automaticity or triggered activity that causes a depolarization delay, which can result in very rapid premature atrial contractions (PACs), a forerunner for a full-blown afib episode.[1]

Researchers at the Mayo Clinic have now followed up on these findings. Their study included 743 patients who were admitted to the clinic because of suspected cardiovascular disease and underwent coronary angiography or an electrophysiologic study. All patients were also tested for IgG antibodies to *H pylori.* Those who had one or more electrocardiograms showing afib during the study period (1994 to 2001) were included in the AF group (83 patients in total). The researchers found that patients with afib were more likely to seropositive for *H pylori* than were non-afibbers (65% vs. 55%). Paroxysmal afibbers (62% of group) were seropositive in 65% of cases, persistent afibbers (22%) were seropositive in 67% of cases, and permanent afibbers (67%) were seropositive in 67% of cases.

The association between seropositivity and AF varied significantly with age. In patients less than 50 years of age, the incidence of AF was 8% in the seropositive group versus 0% in the seronegative group; however, in older patients (more than 70 years of age), there was no significant difference in incidence (17.5% among seropositive versus 15.4% among seronegative patients).

There was no association between the level of the systemic inflammation marker CRP (C-reactive protein) and *H pylori* status, which is somewhat surprising since *H pylori* infection causes chronic gastric inflammation. Nevertheless, the researchers conclude that ectopy within pulmonary veins may be aggravated by inflammation in the esophagus or stomach caused by a chronic *H pylori* infection. They suggest further studies and routine assessment of *H pylori* status in younger patients with atrial fibrillation.

Bunch, TJ, et al. Frequency of Helicobacter pylori seropositivity and C-reactive protein increase in atrial fibrillation in patients undergoing coronary angiography. **American Journal of Cardiology**, *Vol. 101, 2008, pp. 848-51*

Editor's comment: There is little question that *H pylori* is a bad actor when it comes to stomach ulcers and perhaps, stomach cancer. However, whether its elimination will "cure" or prevent afib is still very much an open question, but one that further Mayo Clinic studies will, hopefully, help answer. In the meantime, routine elimination of *H pylori*, except in the case of stomach ulcers, may not be the smartest idea for an afibber. Nine years ago Dr. Martin Blaser of the Department of Veterans Affairs warned that a lack of *H pylori* may be behind the recent increase in the incidence of gastroesophageal reflux disease (GERD), Barrett's esophagus, and esophageal cancer. Dr. Blaser points out that the human stomach and *H pylori* have lived in harmony for millions of years. However, recently the incidence of *H pylori* colonization has declined in the Western world because of, among other reasons, the excessive use of antibiotics in children. This decline has been accompanied by a substantial increase in GERD and esophageal cancer. GERD is uncommon in countries where most people are colonized ("infected") by *H pylori*. Dr. Blaser believes that the most common strain of *H pylori* (cag+) is protective against GERD, Barrett's esophagus, and

esophageal cancer but can promote stomach ulcers and cancer. He believes *H pylori* exerts its effect by regulating acid secretion in different parts of the stomach.[2]

[1] Montenero, AS, et al. Helicobacter pylori and atrial fibrillation: a possible pathogenic link. Heart, Vol. 91, July 2005, pp. 960-61
[2] Blaser, Martin J. Hypothesis: The changing relationship of Helicobacter pylori and humans: implications for health and disease. Journal of Infectious Diseases, Vol. 179, June 1999, pp.1523-30

Bleeding risk with warfarin

BOSTON, MASSACHUSETTS. Clinical trials carried out in 1994 concluded that the use of warfarin in atrial fibrillation (AF) patients was relatively safe with an annual rate of major hemorrhage of 1.3%. Major hemorrhage is defined as a fatal bleeding incident, a bleeding incident requiring hospitalization with transfusion of 2 or more units of packed red blood cells, or a bleeding incident involving a critical site (intracranial, intraspinal, pericardial, intraocular, etc). The average annual reduction in ischemic stroke rate in the five 1994 trials was 1.8% for patients over the age of 75 years with no risk factors for stroke, and 6.9% for those with one or more risk factors. Thus, it was concluded that treating older patients with warfarin had a favourable benefit/risk ratio.

Elaine Hylek and colleagues at the Boston University School of Medicine now question this conclusion. Their recently completed clinical trial involved 472 AF patients with an average age of 77 years (32% were 80 years or older). Forty-seven percent of the patients were women and 91% had one or more risk factors for ischemic stroke (75% had hypertension and 35% had coronary artery disease). After being admitted with a first AF episode (59%), a recurrent episode (35%), or permanent AF (6%) all study participants were prescribed warfarin with an INR target of 2.0 – 3.0. Management of warfarin dosage was carried out by the hospital's own anti-coagulation clinic. More than 10,000 INR measurements were made during the 1-year follow-up period. The time spent within the prescribed INR range (2.0 – 3.0) was only 58% with 29% being spent below 2.0 and 13% above 3.0.

The overall incidence of major hemorrhage was 7.2% and that of intracranial hemorrhage (hemorrhagic stroke) was 2.5%. A third of the hemorrhagic strokes were fatal and 89% of them occurred in patients 75 years or older. The incidence of major hemorrhage was particularly high (13.1%) among patients 80 years or older. Age and an INR greater than 4 were strong risk factors and 58% of the major hemorrhages occurred within the first 90 days after initiation of warfarin therapy. Concomitant use of aspirin was also a significant risk factor for major bleeding and there was no indication that taking 81 mg/day was any safer than taking the standard 325 mg/day.

During the study 26% of participants aged 80 years or older were taken off warfarin – 81% because of safety concerns and 19% because they regained normal sinus rhythm. The Boston researchers conclude that the risk of major bleeding among older AF patients on warfarin has been significantly underestimated in previous trials. They also point out that the rate of bleeding observed in their closely controlled clinical trial would likely be significantly lower than that experienced in the "real world".

In an accompanying editorial Dr. George Wyse of the Health Sciences Center in Calgary, Canada states, "*there is reason to be sceptical about net benefit when warfarin is used in some elderly patients with AF*." Dr. Wyse also points out that warfarin therapy would appear to be over-utilized in patients with low to moderate risk of ischemic stroke. A recent European study found that 50% of AF patients with no risk factors for stroke were being treated with warfarin or similar anticoagulants.

Hylek, EM, et al. Major hemorrhage and tolerability of warfarin in the first year of therapy among elderly patients with atrial fibrillation. **Circulation**, *Vol. 115, May 29, 2007, pp. 2689-96*
Wyse, DG. Bleeding while starting anticoagulation for thromboembolism prophylaxis in elderly patients with atrial fibrillation. **Circulation**, *Vol. 115, May 29, 2007, pp. 2684-86*

Editor's comment: This study adds to the growing evidence that warfarin therapy is far from ideal for AF patients. It would appear to be over-prescribed for patients who don't need it and of no overall benefit for older patients with one or more risk factors for ischemic stroke.

Metabolic syndrome implicated in atrial fibrillation

NIIGATA, JAPAN. Metabolic syndrome (MS) is defined as a combination of risk factors for atherosclerosis including obesity, high blood pressure (hypertension), insulin resistance (glucose intolerance), and dyslipidemia (elevated cholesterol and triglycerides). The diagnosis of MS is made when at least 3 of the following criteria are met:

- Elevated body mass index (BMI) – greater than 30 kg/sq m*
- Elevated triglycerides – greater than 150 mg/dL
- Low high-density cholesterol (HDL) – less than 40 mg/dL in men and 50 mg/dL in women
- Elevated blood pressure – greater than 130/85 and/or history of treated hypertension
- Impaired glucose tolerance

*NOTE: Because the incidence of obesity in Japan is only 2-3% as compared to 20-30% in western countries, a cut-off value for obesity of only 25 kg/square meter was used in this study.

A group of American and Japanese researchers now report that metabolic syndrome is not only a risk factor for atherosclerosis and cardiovascular disease, but also significantly increases the risk of developing atrial fibrillation (AF). Their study involved 28,449 Japanese men and women enrolled in the Niigata Association for Comprehensive Health Promotion and Research Study. The mean age of enrolment in the study was 59 years and 66% of participants were women. At baseline MS was diagnosed in 13-16% (depending on definition of impaired glucose tolerance) of the participants, but none had AF. After a 4.5-year follow-up period, AF had been diagnosed in 265 subjects corresponding to an annual incidence rate of 0.41% in men and 0.13% in women. The annual incidence rate of AF was 0.19% in participants without MS and 0.33% in those with MS, thus indicating that MS along with age and gender (males were 3 times more likely to develop AF than were women) are potent risk factors for AF.

The risk of developing AF increased with the number of MS components diagnosed in individual participants. The relative risk increase associated with the components were as follows:

- Obesity – 64%
- Hypertension – 69%
- Low HDL cholesterol – 52%
- Impaired glucose tolerance – 44%

An elevated level of triglycerides was not associated with an increased risk of AF. The researchers speculate that inflammation and oxidative stress may be common factors in both MS and AF and that hypertension and obesity can cause atrial stretch and dilation resulting in a structural substrate predisposing to AF.

Watanabe, H, et al. Metabolic syndrome and risk of development of atrial fibrillation. **Circulation,** *Vol. 117, March 11, 2008, pp. 1255-60*

Left atrium function after RF ablation

BARCELONA, SPAIN. Many afibbers, myself included, have wondered just how efficient the pumping action of the left atrium is after undergoing an extensive RF ablation. Researchers at the University of Barcelona obviously wondered about this too and now report on a clinical trial to find the answer. They measured the left atrial ejection fraction (LA EF%) using cardiac magnetic resonance imaging (MRI) in 55 patients (in normal sinus rhythm) who had undergone one or more circumferential pulmonary vein ablations for paroxysmal or persistent AF. The researchers defined LA EP% as (LAmax – LAmin/LAmax) x 100 where LAmax is the volume of the left atrium immediately before the mitral valve opens to allow blood flow into the left ventricle, and LAmin is the volume immediately after closure of the mitral valve.

MRI was performed prior to the ablation and 4-6 months following the ablation. After an average of 1.2 ablations, 60% of the patients were in normal sinus rhythm (NSR) without the use of antiarrhythmics, 9% were in NSR with the use of antiarrhythmics, and the remaining 31% were still experiencing episodes. Patients were prescribed an antiarrhythmic for the first 4 weeks following the procedure and remained on warfarin for a minimum of 2 months post-procedure.

The researchers found that the LA EF% increased or remained stable in 68% of the patients whose ablation had been successful (with or without antiarrhythmics). In contrast, patients whose ablation had not been successful experienced an average drop of 11% in their LA EF%, indicating that the contractibility of the left atrium had been impaired by the unsuccessful ablation. The decline in LA EF% was associated with a smaller decrease in LAmin after the ablation among patients with an unsuccessful procedure than among those with a successful one. The contractibility of the left atrial appendage was not affected by the ablation irrespective of outcome.

Perea, RJ, et al. Left atrial contractibility is preserved after successful circumferential pulmonary vein ablation in patients with atrial fibrillation. **Journal of Cardiovascular Electrophysiology**, *Vol. 19, April 2008, pp. 374-79*

Editor's comment: It is indeed comforting to know that the pumping action of the left atrium and the left atrial appendage is not likely to be impaired following a successful RF ablation.

Variables affecting PVI success

BARCELONA, SPAIN. It is known that the success or failure of a circumferential pulmonary vein ablation (Pappone method) depends on the amount of existing left atrial scarring observed during mapping, the area ablated in the left atrium, whether or not vagal denervation is performed, and the absence of AF inducibility after the procedure. There is also some evidence that the success rate in the case of non-paroxysmal (persistent and permanent) AF is less than that observed for paroxysmal AF.

Spanish researchers now add to our knowledge regarding pre-procedure factors that affect the final outcome of an anatomically-guided (CARTO) circumferential PVI (CPVA). Their study involved 148 patients, of which the majority (60.8%) had paroxysmal afib, while the remaining had either persistent (23.6%) or permanent (15.6%). The average age was 52 years and 82% were male. Eighty percent experienced lone atrial fibrillation (no underlying structural heart disease) and 33.8% had hypertension.

The patients all underwent a standard CPVA procedure using the CARTO mapping system and an 8-mm irrigated *Navistar* catheter. The procedure involved lesions encircling both left- and right-sided pulmonary veins as well as linear lesions along the posterior wall of the left atrium and along the mitral isthmus. The patients were followed for an average of 13 months at which time 73.6% were free of afib recurrences. A second procedure was needed in 22 patients (14.8%) because of recurrent afib or the development of left atrial flutter (9.5%), and a third procedure was needed in 4 patients (overall 18% repeat rate). Two patients suffered a TIA during the procedure, 6 developed post-procedural pericarditis, and 3 experienced cardiac tamponade, thus resulting in an overall major complication rate of 7.4%.

The researchers noted that the success rate of the procedure was negatively affected by advanced age, hypertension, permanent afib, elevated left atrial antero-posterior diameter (LAD), and elevated left ventricular end-systolic diameter. However, in multivariable analysis only hypertension (2.8 times risk) and elevated LAD (1.1 times risk) proved to be independent predictors of failure. They conclude that patients with a LAD at or below 45 mm and no hypertension could expect a favourable outcome in at least 85% of cases, while among those with hypertension and an enlarged left atrium (LAD >45 mm), a success rate of only about 50% could be expected.

Berruezo, A, et al. Pre-procedural predictors of atrial fibrillation recurrence after circumferential pulmonary vein ablation. **European Heart Journal**, *Vol. 28, 2007, pp. 836-41*

Editor's comment: The finding that hypertension is associated with an almost three times greater likelihood of failure is indeed a sobering one. It is known that hypertension causes left atrial enlargement and fibrosis, but it is not clear whether controlling hypertension (through medications or supplements) will result in a better outcome.

Left atrial scarring predicts ablation failure

CLEVELAND, OHIO. Both electrophysiological and anatomical mapping has shown that a significant proportion of afibbers have significant scarring in the left atrium. Researchers at the Cleveland Clinic now report that such scarring is a strong predictor of failure of the pulmonary vein antrum isolation (PVAI) procedure for the elimination of AF. Their study involved 700 patients who underwent a PVAI during the period January 2002 to August 2003. The average age of the patients was 53 years and a little over half had lone AF (no structural heart disease). All underwent a standard PVAI guided by intracardiac echocardiography (ICE). Prior to creating the ablation lesions, the electrical potentials of the left atrium wall were carefully mapped using a multipolar Lasso catheter (later checked by the use of a NaviStar (CARTO) catheter).

Areas with no electrical activity were classified as scarred tissue and patients exhibiting 3 or more such areas were classified as having left atrial scarring (LAS). On average, scar tissue covered about 21% of the total left atrium surface area. The 42 patients (6%) with LAS had, on average, a larger left atrium diameter, a lower left ventricular ejection fraction, and a much higher level of C-reactive protein (CRP) – 5.93 mg/L vs 0.31 mg/L in the non-LAS group. NOTE: A CRP level of 0.31 mg/L is at the lower end of the normal range thus indicating that the majority of afibbers in the non-LAS group (94% of total) did not have an elevated CRP level. There was also a trend for LAS patients to be less likely to have paroxysmal afib (26% vs 40% in the non-LAS group), but this trend was not statistically significant. Mean age, AF duration, and the incidence of structural heart disease were no different in the two groups.

All patients were prescribed an antiarrhythmic (dofetilide, flecainide, propafenone or sotalol) for a 2-month period (blanking period) after the completion of the PVAI and were then followed for an average of 16 months. The complete success rate (no afib, no antiarrhythmics) was 81% in the non-LAS group, but only 43% in the LAS group. Of the patients with afib recurrence, 17 of 24 in the LAS group and 117 of 128 in the non-LAS group underwent a second procedure. Complete success rate after the repeat procedure was 52% in LAS patients and 90% in non-LAS patients.

The researchers conclude that the presence of LAS is a strong predictor of PVAI failure with patients experiencing LAS having a 3.4 times greater risk of failure than non-LAS patients. They suggest that for those with LAS combining ablation with long-term drug therapy may be the most effective approach. It is also possible that more extensive ablation of the left atrial wall itself may prove helpful.

Verma, A, et al. Pre-existent left atrial scarring in patients undergoing pulmonary vein antrum isolation: an independent predictor of procedural failure. **Journal of the American College of Cardiology**, *Vol. 45, No. 2, January 18, 2005, pp. 285-92*

Editor's comment: Left atrial scarring, unfortunately, can only be determined by an electrophysiologic study, so it is not possible to say whether a patient is a good candidate for pulmonary vein ablation before the procedure is actually underway. None of the study participants had undergone a previous catheter ablation, so from this study it is not possible to conclude whether scar tissue originating from previous ablation(s) may also reduce the chance of success.

Early recurrence and final ablation outcome

VIENNA, AUSTRIA. Early recurrence of afib (within 48 hours of procedure completion) is not uncommon following a pulmonary vein isolation (PVI) procedure. However, it is not known whether early recurrence is associated with a poorer long-term prognosis. EPs at the Medical University of Vienna recently completed a study to investigate this.

The study included 234 patients undergoing catheter ablation for symptomatic paroxysmal or persistent AF. The average age of the patients was 57 years, 72% were men and 71% had paroxysmal AF with 82% experiencing daily or weekly episodes. Twenty-two percent had structural heart disease, so the majority (78%) of the group would be classified as lone afibbers. Thirty-five percent of the group underwent a segmental PVI (Haissaguerre method), while the remaining 65% underwent an anatomically-guided (CARTO) circumferential PVI (Pappone method) including roof line and mitral isthmus line. Total procedure time was 173 minutes for the segmental procedure versus 142 minutes for the circumferential procedure with total fluoroscopy times of 64 and 46 minutes respectively.

Early afib recurrence (within 48 hours) was observed in 37% of the segmental group patients versus 46% in the circumferential group. After an average (median) follow-up of 12.7 months, 35% of study participants were free of afib without the use of antiarrhythmics, 23% were free of afib while on antiarrhythmics that had previously failed, and the remaining 42% still experienced episodes. Among paroxysmal afibbers, 64% were free of afib (with or without antiarrhythmics) at the end of the follow-up as compared to only 45% of persistent afibbers.

Early recurrence was a significant predictor of failure. Among paroxysmal afibbers, the long-term success rate (with and without antiarrhythmics) was 70% for those without early recurrence and 53% for those with early recurrence. Corresponding numbers for persistent afibbers were 59% and 32%. Early recurrence was a predictor of failure in both the segmental PVI group and in the circumferential group. Overall, ablated afibbers who experienced early recurrence experienced twice the risk of failure, as did those not experiencing early recurrence. However, the researchers emphasize that, despite this, 46% of those who experienced early recurrence still were free of afib at the end of the study period.

Richter, B, et al. Frequency of recurrence of atrial fibrillation within 48 hours after ablation and its impact on long-term outcome. **American Journal of Cardiology**, *Vol. 101, 2008, pp. 843-47*

Editor's comment: The success rates in this study are certainly not impressive (complete success rate of 35% after one procedure); however, there was no significant difference between the outcome of segmental (Haissaguerre) and circumferential (Pappone) procedures. It is of interest that the characteristics of the Vienna group – age at ablation: 57 years, male: 72%, and paroxysmal: 71% – are quite similar to those of the 516 afibbers responding to the 2007 ablation/maze survey. Here the average age at ablation was 56 years with 78% being male, and 78% being paroxysmal. The complete success rate achieved by the Austrian group (35%) is also very close to the average success rate (34%) found in the 2007 survey.

Fosamax (alendronate) implicated in AF

SEATTLE, WASHINGTON. A recent (2007) clinical trial of once-a-year osteoporosis treatment with zoledronic acid (a bisphosphonate) revealed that this treatment significantly increased the risk of atrial fibrillation from 0.5% to 1.3%, or a 2.6-fold risk increase. The obvious question at the time was, "Do more common orally-administered bisphosphonates such as alendronate (Fosamax) also increase the risk of atrial fibrillation in postmenopausal women with osteoporosis?" Clinicians at the University of Washington in Seattle, the University of California, and the Center for Health Studies, Group Health also in Seattle now provide an answer to this question.

Their study involved 719 women with diagnosed afib and 966 matched controls who had been receiving health care at Group Health for a median of 20 years. The median age was 75 years for afib patients and 71 years for controls. The women were observed for a 6-month period during which 45.6% were diagnosed with persistent or recurrent paroxysmal AF – 41.6% experienced just one self-terminating episode and 11.5% were found to have permanent AF. Women who had ever used alendronate (irrespective of how long they had used it, or how much their cumulative intake had been) were found to have an average 86% greater relative risk of being diagnosed with afib. The risk of being diagnosed with permanent afib was almost 6 times greater among ever users of alendronate than among those never using it. The relative risk increase for women with persistent/recurring afib was 25% and for those with just one self-terminating episode the increase was 93%. Adjustment for possible confounding variables such as cardiovascular disease, BMI, cholesterol levels, estrogen therapy, osteoporosis, etc. did not change these risk estimates. However, the researchers did note that the combined use of alendronate and statin drugs was associated with a 13-times increased risk of developing AF.

The researchers conclude that 3% of the AF diagnoses made during the 6-month study can be attributed to alendronate use. They further state that the benefits of fracture prevention in high-risk patients will generally outweigh the possible risk of AF. However, in women with only modestly increased fracture risk the benefits may be less clear.

Heckbert, SR, et al. Use of alendronate and risk of incident atrial fibrillation in women. **Archives of Internal Medicine,** *Vol. 168, No. 8, April 28, 2008, pp. 826-31*

Editor's comment: Alendronate (Fosamax) can also cause stomach ulcers, particularly in combination with naproxen and has been strongly implicated in rotting of the jawbone after dental work. Add a substantial risk of atrial fibrillation and it is clear that alendronate is by no means an innocuous drug. Fortunately, there are highly effective alternative means of preventing and treating osteoporosis. Lifestyle changes and supplementation with calcium, magnesium, strontium, boron, zinc and vitamins C, D and K can go a long way towards dealing effectively with osteopenia (the forerunner of osteoporosis) and osteoporosis itself. You can find out more about the natural approach at http://www.yourhealthbase.com/osteoporosis.htm

Elimination/Reduction Protocol

Case No. 779

Female afibber – **65 years** of age with **vagal AF** of **7 years standing;** no underlying heart disease
No. of episodes in 6 months prior to starting protocol: **4-5**
Afib burden in 6 months prior to starting protocol: **4 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: **58 months**
Episodes since protocol implementation: **3 episodes (from ½ to 12 hrs)**
Still need to avoid triggers?: **No**

Main components of effective protocol

Trigger avoidance: **MSG, caffeine, high glycemic index foods, dehydration, emotional stress**
Diet changes: **Eliminated gluten and wheat. Sharply reduced intake of dairy products. Switched to paleo diet.**
Supplementation: **Magnesium, potassium, taurine, low-sodium V8 juice**
Drug therapy: **Lisinopril (Zestril) – 10 mg/day**
Stress management: **Avoided stressful relationships**
Approaches to shorten episodes: **None**
Approaches to reduce ectopics: **Low-sodium V8 juice and potassium supplementation**

Background and details of protocol

My first brush with afib was in late '99 while working a stressful job. I was 57 years old, seriously overweight, had that year gone through a lot of stressful life changes, was eating poorly [whatever I could pick up in the convenience store I worked in], it was hot weather and I had no air conditioning, and I was surviving on coffee. I had a couple of short episodes that went away before I could get to a doctor, and of course when I did get to the doctor he found nothing wrong. Then I had one that did not go away, and ended up in the hospital for 3 days.
I changed jobs after that, and worked more normal hours, dropped the coffee and ate better [more vegetables, less junk], and had no more afib until August 2000, when I was hospitalized again with another "just-won't-go-away" episode. This again was associated with caffeine [green tea this time, dozens of cups of it, trying to stay awake at work] and hot weather, compounded by lack of sleep. After that I dropped caffeine altogether, and got an air conditioner.

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For the next several years I had short episodes occasionally, but they always went away by themselves, and in any case, I was getting turned off by hospital emergency rooms. I had learned a little about using computers by that time, and was researching better nutrition. I retired and moved back to Maine, and eventually got my own computer, and found Hans' site. Here I found there were a lot of people taking various drugs, and none of these drugs seemed to be curing their afib. They were still getting afib attacks, trading drug advice, going on different drugs, and still getting afib. Some of them were talking about, and some even resorting to, heart surgery. I couldn't blame them for doing this, because their afib had started small and gradually increased until it ruled their lives. I was afraid mine would do that too.

Worse yet, by no means all of those ablation patients had gotten rid of their afib either. Two of them had had near-death experiences, and I was pretty sure that the reason there were not more stories like that was because most ablations that went bad had resulted in death, and of course, we are not very likely to hear from those people. And then there were 2 people posting who claimed to have gotten rid of their afib by diet and supplements. These were Fran and Erling.

Well, I thought, if these 2 people so different from one another can do that, maybe I can too. Food choices are something I can control. So I changed to a mostly paleo diet, and sent away for some Carlson's magnesium glycinate. At first I still did get some short, mild afib episodes, but then I began seeing posts about low sodium V8 juice, 850 mg potassium per 8 oz. glass. I was having trouble consuming enough vegetables and fruits to get in 3-5 grams K a day, and this seemed like just what I needed, and sure enough it was. The taurine I added later when I began to experience loose bowels from my usual dosage of magnesium glycinate.

The Paleo Diet

The paleo diet is based on the premise that the human body thrives best on the diet of our hunter/gatherer forebears of 10,000 years ago, ie. before the introduction of agriculture. The proponents of the diet point out that the human genomic make-up is very slow to change and has not had a chance to adjust to the very major changes in diet that have occurred since the Stone Age. The hunter/gatherers of the Stone Age consumed a diet based on fish and meat from wild animals, vegetables, berries, fruits and nuts. Grains and dairy products were not available. The paleo diet thus emphasizes the above food sources and excludes dairy products, grains, starchy vegetables, sugar and legumes, and of course, chemical food additives. The paleo diet is described in detail in the book "The Paleo Diet" by Loren Cordain, PhD or at www.paleodiet.com

Concerning those few short, mild episodes, I think a lot of what paleo did for me was eliminate postprandial hypoglycemia. A paleo diet pretty much prohibits high glycemic load foods. Jackie and others had called my attention to the fact that a lot of my afib symptoms were the same symptoms as those of reactive hypoglycemia - shaky, lightheaded, cold sweat, panic - and sure enough, the minor episodes I got soon after converting to paleo lacked just those features. I wasn't sorry to see them go.

Also, I need to mention that those last episodes, mild though they were, appeared right after use of a seasoning containing MSG. I had never had an afib episode that I could tie to MSG before, but then I had never been without it for any period of time before either. For all I really know, they could have all had to do with MSG, in combination with stress, hypoglycemia, dehydration, electrolyte deficiency, caffeine, and any of the other myriad stressors of modern life.

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Any paleo diet purist will point out that I ingest a lot of stuff that isn't paleo. The V8 certainly isn't, and neither are the supplements I take. I do eat a little cheese, too, though not the plasticized processed cheese. I cannot afford organic food, so I make do with what I can find in the local supermarket, cheapest first. I go out to eat sometimes, and on those occasions I commit excesses like baked potato and gravy, or bread on sandwiches. I cheat outrageously sometimes, too, particularly with chocolate baked goods.

Speaking of bread, gravy, and bakery goodies, if I hadn't gone to paleo I would also never have realized that I have a bad reaction to wheat. Since taking up the paleo diet my antacid consumption has gone way down, except when I eat anything with wheat in it. That will have me eating antacids for a good 12 hours and sometimes more.

Another good thing about the paleo diet is that I fit the classic profile for insulin resistance - fat, high blood pressure, relatively inactive, cholesterol a bit on the high side - and the paleo diet is good for insulin resistance. I hope to avoid type 2 diabetes this way, or at least to slow it down.

For those concerned about whether my afib is "really cured", I do not think I can expect to be cured of needing proper nutrition, any more than cars are cured of needing gasoline. I don't think I am going to ever again be just like I was in my 20's either. To use the same metaphor, old cars are never again just like they were when new.

I think afib is one of the long latency deficiency diseases, and that is why, in most people, it does not appear until a relatively 'older' age, and why it appears in the context of stress so often. I am still old, fat, and lame in the knees, but I don't have afib any more. If I can do this, you can too.

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