THE AFIB REPORT

Your Premier Information Resource for Lone Atrial Fibrillation!

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I recently came across the abstract of an article entitled "The Influence of Weather Conditions on the Occurrence of Paroxysmal Atrial Fibrillation". Unfortunately, the article is in Polish, but the English abstract provides some intriguing clues. The Polish researchers studied 1153 patients with paroxysmal afib over a 10-year period. The majority (84%) had underlying heart disease with the remaining 16% being lone afibbers. The researchers found that abnormally high or low air temperatures, high humidity, and a rapid increase in atmospheric pressure all had a definite impact on the occurrence of episodes, especially in patients with hypertension or heart disease. Other researchers have found that afib frequency increases substantially in areas affected by the desert wind known as the Sirocco. The finding that certain weather conditions can affect the risk of experiencing an afib episode is indeed intriguing and hopefully will help lead to a better understanding of the mechanism underlying atrial fibrillation.

In this month's issue we report that vitamin B6 and a diet rich in vegetables and fruits helps protect against ischemic stroke, bisoprolol plus magnesium proves effective in preventing afib resulting from heart surgery, a high pulse pressure may be a risk factor for afib, but regular alcohol consumption does not appear to be so. Also, hyperaldosteronism has been linked to periodic afib episodes, and ultrasound pulmonary vein isolation makes its debut.

Last, but not least, 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 very much appreciated.

Wishing you good health and lots of NSR,

Hans

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Diet and venous thromboembolism

MINNEAPOLIS, MINNESOTA. Venous thromboembolism (VTE) in which blood clots form in the veins of the lower part of the legs is becoming increasingly common among air travellers. VTE is also a common feature after certain surgical procedures and can progress to pulmonary embolism, which can be fatal. VTE is believed to involve an increase in blood coagulability, blood stasis (lack of circulation), and damage to the wall of the vein. This makes the condition somewhat similar to embolic stroke in afibbers resulting from blood stasis in the left atrial appendage. The main risk factors for VTE are high levels of homocysteine, factor VIII, and von Willebrand factor.

A team of American and Norwegian researchers now reports that a diet high in fruit and vegetables, and fish, and low in red meat is an excellent defense against VTE. Their study involved almost 15,000 middle-aged adults who participated in the Atherosclerosis Risk in Communities study. The participants (average age of 54 years, 68% overweight or obese) were followed-up for an average of 12 years during which 196 were

diagnosed with VTE. The analyses of food frequency questionnaires completed at baseline and year six showed that even a moderate daily intake (more than 2.5 servings) of fruit and vegetables reduced the risk of VTE by 27% to 53% (compared to less than 2.5 servings a day). One or more weekly servings of fish were associated with a 30% to 45% lower risk as compared to consuming fish rarely.

Individuals eating red or processed meat more than 1.5 times a day had a 2 times higher risk of developing VTE than did those consuming less than ½ serving a day. The researchers also observed that participants with a moderate folate intake (more than 160 micrograms a day) had a 34% to 51% lower risk of VTE than did those with intakes below 160 micrograms a day. Those with the highest intake (> 2.26 mg/day) of vitamin B6 had a 63% lower risk of VTE as compared to those with an intake below 1.25 mg a day. Finally, those with an omega-3 fatty acid intake (mainly from fish) above 390 mg a day had a 30% to 46% reduced risk when compared to participants whose intake was less than 100 mg a day.

The researchers conclude that a diet including more plant food and fish, and less red and processed meat is associated with a lower incidence of VTE.
Steffen, LM, et al. Greater fish, fruit and vegetable intakes are related to lower incidence of venous thromboembolism. **Circulation**, *Vol. 115, January 16, 2007, pp. 188-95*

Editor's comment: Folic acid, vitamin B12, and vitamin B6 are all essential for maintaining low homocysteine levels. Vitamins C and E are highly effective in lowering the level of von Willebrand factor – a key factor in the coagulation sequence leading to VTE. There is also growing evidence that vitamin B6 is highly effective in preventing ischemic stroke. Researchers at Harvard Medical School and Massachusetts General Hospital have discovered a strong association between stroke risk and low blood levels of pyridoxal-5'-phosphate (PLP), the main metabolite of vitamin B6. This increased risk of stroke with low PLP levels was entirely independent of homocysteine levels confirming that vitamin B6, on its own, has significant stroke prevention properties. The researchers found that study participants with a plasma level of PLP of more than 80 nanomol/L had a 90% lower risk of stroke and transient ischemic attacks (TIAs) than did participants with a level below 20 nanomol/L. The risk decrease was independent of the presence of other risk factors such as hypertension, diabetes, and atrial fibrillation[1]. The researchers also noted a strong inverse correlation between C-reactive protein level and PLP level indicating that vitamin B6 may also have strong anti-inflammatory properties – an added plus for afibbers.

The 90% relative reduction in stroke risk among people with high PLP levels is very significant and compares extremely favourably with the oft-quoted relative risk reduction afforded by warfarin (64%) and aspirin (25%). Clearly, ensuring adequate blood levels of PLP is a must for all afibbers. Vitamin B6 is converted to its active metabolite PLP in the liver and there is some evidence that the liver can only handle about 50 mg of vitamin B6 at a time. Experiments have shown that the plasma concentration of PLP does not increase further if 100 mg rather than 50 mg of pyridoxine (vitamin B6) is ingested at any one time. So it is assumed that the conversion to PLP is limited by the liver's conversion capacity[2]. Other experiments have shown that supplementing (orally) with 40 mg of vitamin B6 will increase average plasma concentration from about 23 nmol/L (range: 18-37 nmol/L) to about 230 nmol/L within 3 days of beginning supplementation. No further increases were observed with 40 mg/day supplementation for a 12-week period[3].

The 230 nmol/L concentration achieved is well above the 80 nmol/L concentration associated with the 90% reduction in stroke risk observed by the Harvard researchers[1]. So 40-50 mg/day would seem to be sufficient for stroke protection and is considered entirely safe[3]. Vitamin B6 itself is, however, water-soluble and any excess is totally eliminated in the urine within about 9 hours. To keep the vitamin B concentration up, it would be necessary to take two or three 50 mg doses per day. However, in the case of stroke protection, one 50 mg dose per day is likely to be quite adequate, as PLP concentration does not vary much during the day once steady state conditions are achieved. Adequate amounts of vitamin B2 and magnesium are required in order to convert vitamin B6 to PLP.

[1] Kelly, PJ, et al. Low vitamin B6 but not homocysteine is associated with increased risk of stroke and transient ischemic attack in the era of folic acid grain fortification. Stroke, Vol. 34, June 2003, pp. e51-e54
[2] Khaw, KT and Woodhouse, P. Interrelation of vitamin C, infection, haemostatic factors, and cardiovascular disease. British Medical Journal, Vol. 310, June 17, 1995, pp. 1559-63
[3] Zempleni, J. Pharmacokinetics of vitamin B6 supplements in humans. Journal of the American College of Nutrition, Vol. 14, 1995, pp. 579-86

Vitamin B6 helps protect against stroke

UDINE, ITALY. Italian researchers now confirm that low blood levels of vitamin B6 (pyridoxine) are a strong risk factor for heart attack, angina, and ischemic stroke. Their study involved 1021 healthy middle-aged subjects (490 men and 531 women) who were followed for 12 years after having undergone a baseline medical examination complete with extensive blood sampling. During the follow-up members of the group experienced 30 heart attacks (of which 15 were fatal), 36 cases of unstable angina, 29 ischemic strokes, and 14 transient ischemic attacks (TIAs) for a total of 109 coronary and cerebrovascular events (0.9% a year).

The researchers found that study participants with a vitamin B6 level of 40 nmol/L had a 31% lower risk of a cardiovascular or cerebrovascular event than did patients with a level of only 15 nmol/L. They also observed that an elevated homocysteine level (14 micromol/L) was associated with a 34% greater risk than a level of 9.8 micromol/L. There was no indication that the blood level of folate and vitamin B12 had any correlation with the risk of stroke or heart attack, nor was there any indication that the plasma concentration of C-reactive protein did. A combination of low vitamin B6 and high homocysteine levels was found to be particularly dangerous with this combination conferring an 18 times greater risk of experiencing an event than the risk associated with a low homocysteine level and a high vitamin B6 level.

The researchers conclude that high homocysteine and low vitamin B6 plasma levels are long-term independent risk factors for coronary and cerebrovascular events.

Vanuzzo, D, et al. Both vitamin B6 and total homocysteine plasma levels predict long-term atherothrombotic events in healthy subjects. **European Heart Journal,** *Vol. 28, 2007, pp. 484-91*

Editor's comment: A daily intake of 40 mg of vitamin B6 (pyridoxine) will result in a plasma level of about 230 nmol/L. Most well-formulated multivitamins contains about 25-30 mg of B6 so should provide adequate protection.

Bisoprolol + magnesium helps prevent post-operative AF

COLOGNE, GERMANY. Atrial fibrillation is a common post-operative complication following coronary bypass graft surgery (CABG). It is estimated that as many as 40% of patients suffer this complication most often during the first two days after surgery. It is common practice to give patients beta-blockers to prevent post-operative AF, but this approach is generally not very effective.

Now researchers at the University of Cologne report that a combination of bisoprolol, a highly cardio-selective beta-blocker, and intravenous and oral magnesium is highly effective in preventing post-operative AF. Their clinical trial involved 100 patients scheduled for elective CABG surgery. The trial participants were randomized to either the prophylaxis group (n=50) or the control group (n=50). Members of the control group remained on whatever beta-blocker they had been on prior to the operation, while those in the prophylaxis group received 2.5 mg of bisoprolol twice a day starting on the day of the surgery. In addition, they received a single intravenous infusion of 100 mL saline solution containing 2000 mg of magnesium sulfate within 30 minutes of arriving in the ICU following surgery. The magnesium component of the bisoprolol + magnesium combination was changed after the infusion to 600 mg of magnesium oxide three times daily for a week following the operation.

The incidence of new afib during the first week was 42% in the control group as compared to only 20% in the prophylaxis group. The difference was even more startling in those over 65 years of age. In this group 65% in the control group developed post-operative afib as compared to only 17% in the prophylaxis group. The bisoprolol/magnesium combination also had an immediate effect on cost effectiveness in that the average hospital stay in the control group was 9 days versus only 7 days in the prophylaxis group. It is interesting to note that 40% of control group participants were treated with bisoprolol before and after their surgery thus perhaps indicating that magnesium is the most important player in the combination.

Behmanesh, S, et al. Effect of prophylactic bisoprolol plus magnesium on the incidence of atrial fibrillation after coronary bypass surgery. **Current Medical Research and Opinions**, *Vol. 22, No. 8, 2006, pp. 1443-50*

Editor's comment: This study brings two thoughts to mind. An intravenous infusion of 2000 mg

magnesium sulfate in 100 mL saline solution is obviously safe and effective. Personally, if I experienced intolerable, unmanageable afib this is the first approach I would try. The bisoprolol + magnesium combination is obviously effective in controlling new onset afib. Could it be effective in controlling paroxysmal afib? It certainly would be worth a try for adrenergic and perhaps mixed afibbers, but may not be a good idea for vagal afibbers. The daily oral dose of magnesium (1800 mg magnesium oxide) used in the trial would only have provided about 45 mg/day of absorbable elemental magnesium, so it would certainly be easy to up this amount by using a much more absorbable magnesium compound such as magnesium glycinate or citrate.

Inflammation and atrial fibrillation

TRABZON, TURKEY. There is considerable evidence that inflammation is an underlying cause of atrial fibrillation. Turkish researchers now report that patients with AF have considerably higher levels of the inflammation markers C-reactive protein (hs-CRP) and interleukin-6 (IL-6) than do normal controls. Their study involved 85 patients with afib (30 paroxysmal or new-onset, 24 persistent, and 31 permanent). Eighteen were classified as lone afibbers since they did not have hypertension or structural heart disease.

The average CRP and IL-6 levels for controls were 0.23 mg/dL and 11.6 pg/mL respectively as compared to 0.63 mg/dL and 29 pg/mL for the entire afib group. Persistent afibbers had the highest CRP levels at 0.83 mg/dL followed by permanent afibbers at 0.60 and paroxysmal at 0.51 mg/dL; the CRP level among lone afibbers was 0.44 mg/dL. IL-6 levels were highest among permanent afibbers (37.3 pg/mL) followed by paroxysmal at 28.4 and persistent at 21.8 pg/mL. IL-6 level for lone afibbers was 26 pg/mL. The researchers conclude that inflammation may play a significant role in AF although its role in lone afib is still controversial.

Gedikli, O, et al. Inflammatory markers according to types of atrial fibrillation. **International Journal of Cardiology**, *2007 [Epub ahead of print]*

All fish are not equally healthy

MONTREAL, CANADA. The American Heart Association and similar organizations have long extolled the virtue of consuming fish once or preferably twice a week as a powerful preventive measure against cardiovascular disease. Although there is increasing evidence that most fish now contain mercury (especially methylmercury), it is still felt that regular fish consumption is beneficial overall.

University of Quebec researchers now question the assumption that fish consumption is universally beneficial. They do not question whether an increased intake of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) is beneficial – this has pretty well been proven beyond a doubt. However, they do question whether all fish actually ends up contributing to the body's stores of EPA and DHA when consumed.

Their study involved 243 moderate consumers of fish living in the areas surrounding four lakes in the province of Quebec. The participants were interviewed to determine their consumption of 12 freshwater and 30 marine (saltwater) fish over the preceding three months and then had blood samples drawn for the determination of fatty acid (especially EPA and DHA), mercury, and selenium content. The age of the participants ranged between 18 and 74 years, 53% were men and 26% were considered obese (BMI >30). No relation was observed between fish intake and BMI, or between fish intake and alcohol intake. The 243 participants were divided into 4 groups according to daily consumption (occasional – < 24 grams/day, low – 24-41 grams/day, moderate – 41-66 grams/day, and high – > 66 grams/day). The average (mean) estimated intake of DHA + EPA from fish was 223 mg/day and that of omega-6 fatty acid (from fish) was 95 mg/day giving a very healthy ratio of omega-6 to omega-3 of 0.32. Much to their surprise, the researchers found no correlation between the total intake of fish or the intake of locally caught fish and serum levels of EPA and DHA. They did, however, find a strong correlation between the intake of salmon and trout and serum levels of EPA and DHA.

They conclude that, even though food tables may show that some lean fish do contain EPA and DHA, for some reason consuming these fish does not increase serum levels of EPA and DHA. Thus, the

advice to eat fish on a regular basis needs to be revised to apply to only saltwater fish (especially salmon, mackerel, trout, sardines, and herring). The researchers also noted an increased blood methylmercury concentration in frequent fish eaters. They conclude that no matter how many locally caught freshwater fish are eaten, serum EPA + DHA levels are not affected.

Philibert, A, et al. Fish intake and serum fatty acid profiles from freshwater fish. **American Journal of Clinical Nutrition**, *Vol. 84, December 2006, pp. 1299-1307*

Editor's comment: This study clearly shows that one needs to be very selective in what kind of fish or shellfish one eats in an attempt to obtain the benefits of the fish oils EPA and DHA. For example, lobster, flounder, sole, plaice, haddock, scallops, cod, halibut, and oysters are unlikely to supply any meaningful amounts of EPA and DHA. Thus, while fish consumption should probably still be encouraged, although mercury contamination is an ever-growing concern, it would seem prudent to ensure an adequate daily intake of EPA + DHA by supplementing with a high quality (molecular distilled) fish oil.

High pulse pressure – A new risk factor for atrial fibrillation?

WALTHAM, MASSACHUSETTS. Atrial fibrillation (AF) is associated with advancing age, increased systolic (the high value) blood pressure, diabetes, hypertension, heart failure, valvular disease, heart attack, obesity, left atrial enlargement, ventricular hypertrophy, and impaired ejection fraction. Now researchers at Boston University School of Medicine report that increased pulse pressure (difference between systolic and diastolic pressure) is also a strong risk factor for the development of AF.

Their study involved 5331 participants in the Framingham Heart Study who were free of AF at time of enrolment. The median age of the participants was 57 years and 55% were women. They were followed for 16 years during which time 363 men and 335 women developed AF. Prior to developing afib 182 participants (54% of afibbers) had either experienced a heart attack or developed heart failure.

The researchers found a highly significant correlation between pulse pressure and the risk of developing AF. Thus, while participants with a pulse pressure of 40 mm Hg or less had a risk of 5.6%, those with a pressure above 61 mm Hg had a risk of 23.3%. After adjusting for mean arterial pressure and known clinical risk factors for AF, the researchers conclude that each 20 mm Hg increase in pulse pressure is associated with a 24% increase in the risk of developing afib. They speculate that increased arterial stiffness (hardening of the arteries) and abnormalities in left ventricular function lies behind the increase in pulse pressure and the development of abnormal left atrial structure and function. They also point out that blockage of the renin-angiotensin system has been shown not only to reduce pulse pressure, but also to reduce the incidence of new and recurrent AF.

Mitchell, GF, et al. Pulse pressure and risk of new-onset atrial fibrillation. **Journal of the American Medical Association**, *Vol. 297, February 21, 2007, pp. 709-15*

Editor's comment: Blood pressure readings prior to ablation were available for 41 afibbers who participated in LAFS-8. All were lone afibbers (no heart disease) with no hypertension and no use of anti-hypertensive medications. The mean systolic blood pressure of the group was 116 mm Hg (median of 120 mm Hg, range of 90-150 mm Hg). The mean diastolic pressure was 74 mm Hg (median of 70 mm Hg, range of 60-90 mm Hg). Overall, 54% of the group had a pulse pressure of 40 mm Hg or less. This compares to only 25% of participants in the Framingham group having a pulse pressure of 40 mm Hg or less. Also, while 24% of the Framingham group had a pulse pressure higher than 61 mm Hg, no one in the LAFS-8 survey had a pulse pressure above 60 mm Hg. These differences may add another piece of evidence to the idea that lone afib really is different from the more common form of afib, which involves structural heart abnormalities (my speculation). There were no differences in pulse pressure between vagal, mixed, adrenergic, and permanent afibbers, nor was pulse pressure associated with age or age at diagnosis.

Alcohol consumption and atrial fibrillation

BOSTON, MASSACHUSETTS. There is evidence that binge drinking, particularly among younger people, can trigger atrial fibrillation. However, it is not clear if there is a relationship between moderate alcohol consumption and the risk of developing AF when it comes to older people. Researchers at the Beth Israel Deaconess Medical Center now provide answers to this question.

Their study included 5609 adults 65 years and older who reported their use of wine, beer, and spirits on an annual basis. The study participants were grouped according to their drinking habits – abstainers, less than 1 drink a week, 1-6 drinks a week, 7-13 drinks a week, and more than 14 drinks a week. During the 9-year follow-up, 1232 participants (22%) developed afib. Advanced age, male gender, hypertension, coronary heart disease, heart failure, large left atrial size, high total cholesterol level, and a high creatinine level were the main risk factors for AF. There was no indication that alcohol consumption over the range evaluated had any statistically significant effect on the risk of developing AF in this group of older afibbers. Nor was there any significant association between alcohol consumption and mortality. However, the researchers did note that former drinkers had a higher mortality and risk of AF than did the remainder of the group. They speculate that this is because former drinkers may have given up drinking when they became aware of some serious deterioration in their health.

The researchers conclude that moderate alcohol consumption is not associated with an increased risk of developing AF, or with an increased mortality among patients with diagnosed AF.

Mukamal. KJ, et al. Alcohol consumption and risk and prognosis of atrial fibrillation among older adults: The Cardiovascular Health Study. **American Heart Journal,** *Vol. 153, February 2007, pp. 260-66*

Ultrasound PVI makes its debut

OKLAHOMA CITY, OK. Although pulmonary vein isolation (PVI) using radiofrequency ablation is now highly successful and safe in experienced hands, it is not necessarily so when less skilled operators perform the procedure. It can be difficult to obtain complete isolation of the pulmonary veins using standard 4 mm or 8 mm ablation catheters and pulmonary vein stenosis, thrombus formation, and penetration of the heart wall (tamponade) are still potential complications. A team of American, British, and German researchers now reports on the first trial of a high-intensity-focused ultrasound (HIFU) balloon catheter.

Their study population involved 19 patients with paroxysmal and 8 patients with persistent afib (21 males and 6 females between the ages of 39 and 68 years). Ten of the patients had structural heart disease and left atrial diameter ranged from 30 – 54 mm. After undergoing a magnetic resonance angiogram or a CT scan to determine pulmonary vein location and size the patients underwent a PVI procedure using a balloon HIFU catheter with a sonicating ring diameter of 20, 25 or 30 mm depending on the size of the veins. The procedure was performed under heavy sedation or general anesthesia. Complete antrum isolation was achieved in all left superior and inferior veins, in 92% of right superior veins, but in only 11% of right inferior veins. A range of 1 – 26 (median of 3) sonic applications was used to isolate the successfully isolated veins.

The patients were followed for at least 15 months after their procedure. At the 12-month follow-up 56% of the 27 patients were free of afib; however, 20% of the group were only achieving this by the continued use of antiarrhythmics. Thus, the complete success rate (no afib, no antiarrhythmics) at the 12-month checkup was 45%, and the partial success rate was 11%. Another 22% had a better than 50% reduction in the number of episodes while using antiarrhythmics that had previously proven ineffective. No cases of stenosis, stroke or transient ischemic attack (TAI) were observed during or after the procedures; however, one case of (non-permanent) phrenic nerve injury and one case of pulmonary hemorrhage did occur.

Nakagawa, H, et al. Initial experience using a forward directed, high-intensity focused ultrasound balloon catheter for pulmonary vein antrum isolation in patients with atrial fibrillation. **Journal of Cardiovascular Electrophysiology**, *Vol. 18, February 2007, pp. 136-44*

Editor's comment: Although a complete success rate of 45% is not overly impressive it will no doubt improve as further experience is acquired. The lack

of success in isolating the right inferior veins is, nevertheless, a concern. Only 11% of these veins were isolated. Recent studies have shown that failure to isolate the right inferior veins is a major cause of late afib recurrence (beyond 12 months).[1]

[1] Mainigi, SK, et al. Incidence and predictors of very late recurrence of trail fibrillation after ablation. Journal of Cardiovascular Electrophysiology, Vol. 18, January 2007, pp. 69-74

Antiarrhythmic drugs after PVI

MADDALONI, ITALY. It is fairly common practice to prescribe antiarrhythmic drugs to patients who have undergone a pulmonary vein isolation (PVI) procedure. These drugs are usually discontinued after 2-3 months if the patient remains afib-free, but may be continued indefinitely if the patient still experiences episodes. Italian researchers recently completed a study to determine if continuing antiarrhythmics beyond the first month is of benefit.

Their study included 107 afibbers (60% paroxysmal and 40% persistent) who had suffered AF episodes for a mean of 4.5 years (64% men). Fifty-seven per cent of the study participants had hypertension. The patients underwent an anatomically-guided (CARTO) PVI procedure and were then divided into two groups. Group A received no post-procedure antiarrhythmics, while group B received either amiodarone (71%), flecainide (19%), propafenone (6%), or sotalol (4%).

During the first month following the PVI, 35% of the members in group A had a recurrence of AF as compared to only 17% in group B. However, during the following 12 months, 34% of group A had experienced one or more AF episodes as compared to 30% in group B. This difference, however, was not statistically significant. The percentage of afibbers with one or more asymptomatic episodes (detected by transtelephonic ECG) was 63% in group B, but only 18% in group A.

The researchers conclude that, while antiarrhythmic therapy may be useful during the first month following the PVI, it would seem unnecessary to continue it beyond the first month in order to attempt the prevention of AF recurrences unless the therapy is effective in alleviating symptoms.

Turco, P, et al. Antiarrhythmic drug therapy after radiofrequency catheter ablation in patients with atrial fibrillation. **PACE**, *Vol. 30, January 2007, Suppl. 1, pp. S112-S115*

Aldosterone and atrial fibrillation

MINEAPOLIS, MINNESOTA. Medical doctors at the Veterans Affairs Medical Center report the case of a 58-year-old African American man whose afib episodes were found to be clearly related to low serum levels of potassium resulting from Conn's syndrome (primary hyperaldosteronism). The man presented with palpitations, dizziness, chest pain, and muscle cramps. He reported having AF episodes occurring every 1 to 2 weeks. Upon admission his serum potassium level was found to be 2.7 mEq/L (reference range: 3.5 – 4.5 mEq/L). He was treated with intravenous potassium chloride (40 mEq [1560 mg]) every 4 hours for an 8-hour period as well as an esmolol (beta-blocker) infusion (100 mcg/kg/min).

The atrial fibrillation ceased once the potassium serum level normalized. After a diagnosis of Conn's syndrome (serum aldosterone of 82.7 ng/dL [normal range: 4 – 31 ng/dL] and renin of 0.07 ng/mL/hr [normal range: 0.7 – 5 ng/mL/hr]) he was discharged with a prescription for 25 mg/day of spironolactone and 10 mEq/day (390 mg/day) of potassium chloride. On this regimen his palpitations disappeared and he has now been afib-free for a year. The doctors conclude that hypokalemia (low potassium levels) may increase the risk of developing paroxysmal AF in patients with hyperaldosteronism (Conn's syndrome).

Aloul, BA, et al. Atrial fibrillation associated with hypokalemia due to primary hyperaldosteronism (Conn's syndrome). **PACE**, *Vol. 29, November 2006, pp. 1303 05*

Editor's comment: this paper is of particular interest to me as I had a similar experience myself prior to my successful ablation. My aldosterone was elevated and my renin was substantially suppressed, particularly immediately prior to an episode; my potassium level was also at the lower end of the normal range (3.5 mEq/L). Unfortunately, supplementation with spironolactone

and potassium did not solve my problem. You can read more about the connection between hypokalemia, hyperaldosteronism and AF in Session 26 of the Conference Room Proceedings http://www.afibbers.org/conference/session26.pdf

My Experience with Traditional Chinese Medicine and LAF

Wanda Craig

It was a beautiful spring day in Charlotte, North Carolina about 4 years ago. My husband and I were eating chicken Caesar salads at a sidewalk café and enjoying the warm sunshine. All was well in my world. Suddenly, my heart began racing. I was concerned but not alarmed, so I did not go to the doctor until the next day. My doctor ordered a series of tests and heart monitoring. I was diagnosed with arrhythmia—lone atrial fibrillation (LAF). My heart was strong, my thyroid was functioning well, my blood chemistry was normal—they found nothing to blame. "It's just one of those things," she said. I was immediately put on Coumadin, a blood thinner, and also Diltiazem. Later, because I was only 52 at the time, my cardiologist took me off Coumadin but prescribed low-dosage aspirin therapy. I told him I had been on the Atkins Diet for about a month and asked if that could have triggered it. He did not think it did, but to this day I am convinced that the change in diet upset my body's balance.

For about six months, I felt much better and had episodes about once a month that lasted a couple of hours. Then my ankles began swelling to twice their size, so my cardiologist put me on flecainide, which I took for over three years. Even on medication, I continued to have intermittent episodes several times a week that lasted for several hours. Common triggers were cold drinks, alcohol, caffeine, and stress. My cardiologist said "break-throughs" while on medication were normal. But I felt tired all the time and could sense that my body was getting weaker. One morning I woke up and decided I was going to heal myself.

After reading some research by Dr. Lam I increased my dosage of CoQ10 to 200 mg/day and added magnesium, fish oil, L-Carnitine, and Lipoic Acid. I also decided to try acupuncture to return my body to balance. Li Jie Chu, a wonderful acupuncturist in Charlotte, felt confident she could help me. She prescribed two acupuncture treatments a week and Chinese herbs to take twice daily. After two months, I could tell I was getting stronger, so I decreased my flecainide by taking one pill each night instead of the usual twice a day. My heart went crazy, so I had to go back to taking the prescribed dosage twice a day. After a few weeks, I then tried taking half of a pill twice a day to keep the dosage more even, but I began having frequent AF episodes, so I returned again to taking two 50 mg pills a day. I was getting discouraged. After three months of acupuncture, I was no closer to being drug free, so I decided to stop the treatments. Then my inner "wise woman" voice said, "Keep going." So I kept going and tried again to wean myself off the flecainide. This time when I took just one pill each night, I had no problems. I continued this dosage for about a month, and then began taking one pill every other night. At first, I had short, mild AF episodes as my body was learning its rhythm again. As Li Jie reminded me, my heart had "forgotten" how to function without drugs. After another month, I stopped the flecainide altogether. I took my last pill on October 31, 2006—Halloween night. My heartbeat has continued to grow steady, and after eight months of acupuncture, I feel great. I am finally drug free.

A couple of words of caution are in order. It is never wise to stop your medication without medical supervision. I asked my doctor and cardiologist to help me find a natural treatment. Neither signed on to help me, so I did it on my own out of desperation. I understood the risk, but I felt the risk of staying on medication the rest of my life was worse. While I am happy to share my happy ending, I also do not want to be responsible for anyone taking risks without understanding the potential consequences. I chose not to live in fear and dependence, but you have to make those decisions for yourself.

Another thing to keep in mind is that I also made significant lifestyle changes. I began yoga and learned to breathe properly, which helps regulates the nervous system. I continue to take all the supplements listed above plus the Chinese herbs and eat a balanced diet of fish, chicken, fruit and vegetables. I gave up alcohol

completely, limit caffeine, and drink lots of water. The benefits are definitely worth the small sacrifices I have made.

I encourage anyone suffering from LAF to do the research, try acupuncture with a certified practitioner, take heart-healthy supplements, pay attention to your body's reaction to what you eat and drink, and above all—do not give up. I believe LAF is a symptom of the body's imbalance and can be managed without drugs. My story has a happy ending, and I believe yours can too.

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