

THE AFIB REPORT

Your Premier Information Resource for Lone Atrial Fibrillation!

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9th YEAR



It is becoming increasingly evident that relying on mass media headlines, or even “official” abstracts of published medical journal articles, to present accurate and truthful information is fraught with peril. Two abstracts in this month’s issue illustrate the point:

- *Researchers at the Mayo Clinic and the Utah School of Medicine concluded (in the abstract of their published article) that the presence of atrial fibrillation is an independent risk factor for stroke. What the data actually shows is that AF increases stroke risk only in patients who have already experienced a stroke or TIA, and in patients with two risk factors such as hypertension and congestive heart failure.*

There was no statistically significant contribution by AF to stroke risk in patients with 0 or 1 risk factor.

- *Irish hospital physicians reported the case of a 55-year-old woman who felt dizzy after supplementing with potassium citrate and say that, “this highlights the potentially serious consequences of unmonitored use of potassium citrate”. What the published abstract of the article failed to mention was that the patient had impaired kidney function, and had been ingesting about 10 grams (10,000 milligrams) of elemental potassium every day for 6 months.*

Also in this issue we report that fish oils may reduce PVCs, that pre-procedure inflammation lowers success rates for PVI, that catheter ablation is remarkably safe, that experiencing afib episodes immediately following a PVI is not a bad sign, and that habitual consumption of green or black tea significantly reduces the risk of stroke.

Last, but by no means least, we have a fascinating and uplifting afib “journey” by Richard Webster – “My Travels Along the AFIB Road”.

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

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Fish oils and PVCs

DURHAM, NORTH CAROLINA. Ventricular ectopy (premature ventricular complexes or PVCs) affects many heart attack victims, but is also a problem for atrial fibrillation patients. Ventricular ectopy, if sustained, can lead to ventricular fibrillation in patients with heart disease and ventricular fibrillation, in turn, can result in sudden cardiac death (SCD). There is substantial evidence that EPA (eicosapentaenoic acid) and DHA (docosahexaenoic acid), the main components of fish oil, protect against the development of coronary

heart disease (CHD) and help prevent SCD in patients who have suffered a heart attack (myocardial infarction or MI). There is also evidence that the plant-based omega-3 fatty acid alpha-linolenic acid (ALA) may be cardioprotective. Researchers at Duke University now report that omega-3 fatty acids may be helpful in reducing ventricular ectopy in recovering MI patients.

Their study involved 260 MI patients who were enrolled in the study within 72 hours of their heart attack. Most of the study participants were male (62.9%) with an age ranging between 27 and 86 years, and an average left ventricular ejection fraction of 52%. Most of the patients (88.5%) were taking beta-blockers at the time of their assessment and, not surprisingly, most had one or more comorbid conditions. Thus, 59% had hypertension, 41% diabetes, 12% chronic obstructive pulmonary disorder (COPD), and 7% congestive heart failure. Seventy-three percent had a history of smoking and 57% were current smokers.

All participants completed the Harvard food-frequency questionnaire to determine their average dietary intake of omega-3 fatty acids during the year preceding their MI. They also underwent 24-hour Holter monitoring either during or immediately following their hospital stay. Evaluation of the data

collected showed a clear inverse relationship between the intake of omega-3 fatty acids and the number of daily PVCs (including couplets, triplets, bigeminy, etc). Based on a daily energy intake of 1000 kcal, the researchers observed that an average daily intake of 0.6 gram of n-3 fatty acids was associated with 450 PVCs/day, while an intake of 0.9 gram/day was associated with only 235 PVCs/day. They estimate that a 1-gram/day increase in the intake of EPA + DHA could reduce the number of PVCs by as much as 800/day. ALA reduced PVCs in a similar fashion, but although it has been shown to help prevent CHD, there is no evidence that it reduces SCD. The researchers conclude that future randomized, controlled trials are needed to investigate whether fish oil supplementation during hospitalization for MI will reduce the number of sudden cardiac deaths.

Smith, PJ, et al. Association between n-3 fatty acid consumption and ventricular ectopy after myocardial infarction. American Journal of Clinical Nutrition, Vol. 89, May 2009, pp. 1315-20

Editor's comment: Although lone afibbers by definition do not have heart disease, it is conceivable that supplementation with fish oil could help in reducing PVCs, especially following an ablation or maze procedure.

Predicting ablation success

BAD KROZINGEN, GERMANY. There is abundant evidence that inflammation and atrial fibrillation are associated. However, what is much less clear is whether inflammation causes afib or afib results in inflammation. Researchers at the German Heart Centre now report that pre-ablation inflammatory status may be an important predictor of the long-term success of PVI ablation.

Their study involved 72 afib patients with an average age of 55 years, 81% of whom were male, and 14% of whom had structural heart disease. Thus, 86% of the group were lone afibbers (64% paroxysmal and 36% persistent), but 52% had high cholesterol (17% were on statin drugs), 19% had hypertension, and 22% had diabetes. The study participants underwent a circumferential PVI (using the *EnSite NavX* system with a *Thermo-Cool* catheter) with the endpoint being the absence or dissociation of potentials in the isolated area. Antiarrhythmic drugs were continued for one month

following the procedure and then discontinued if no symptoms of arrhythmia had been experienced. Warfarin was stopped after 3 months. The patients were evaluated 1, 3, 6, and 12 months after the ablation. At the 1-year follow-up point, 61% were in normal sinus rhythm without the use of antiarrhythmics.

In looking at the pre-ablation data observed for each patient, the German researchers observed that afibbers with pre-ablation hypertension were 3 times more likely to still experience afib than were normotensive participants. Other factors predicting an unsuccessful procedure were a high white blood cell (WBC) count (42% increased risk of failure) and, to a limited extent, an enlarged left atrium - 7% increased risk of failure with a left atrium diameter greater than 43 mm (4.3 cm). There was also a trend for elevated body mass index (BMI) and a low left ventricular ejection fraction to be associated with failure. A high-sensitivity C-reactive protein (CRP)

value was also associated with a greater incidence of failure, but as the sensitivity of the test was only 0.3 mg/dL (3 mg/L) and the average CRP level among the participants was 0.3 mg/dL, this finding should obviously be treated with caution. Age, afib type (paroxysmal or persistent), AF duration (years), presence of structural heart disease, medications, and fibrinogen levels were not associated with ablation outcome. The researchers conclude that hypertension and a WBC above 6280 mm³ are significant predictors of PVI failure.

Letsas, KP, et al. Pre-ablative predictors of atrial fibrillation recurrence following pulmonary vein isolation: the potential role of inflammation. Europace, Vol. 11, 2009, pp. 158-163
Kourliouros, A and Camm, AJ. Does inflammation influence atrial fibrillation recurrence following catheter ablation? Europace, Vol. 11, 2009, pp. 135-37

Editor’s comment: An elevated WBC and, of course, an elevated CRP are indicators of systemic inflammation. It would seem prudent to take steps to eliminate such inflammation prior to undergoing a PVI.

CHADS2 score and C-reactive protein

SALT LAKE CITY, UTAH. Several studies have observed a direct correlation between atrial fibrillation and elevated value for the inflammation marker hs-CRP (high-sensitivity C-reactive protein). This correlation applies to both lone afibbers and afibbers with underlying heart disease. There is also evidence that permanent afibbers tend to have higher CRP values than persistent afibbers who, in turn, have higher values than paroxysmal afibbers.

correlated CRP results from 2340 patients with suspected coronary artery disease (CAD) with the patients’ CHADS2 score. The CHADS2 score is an estimate of the risk of ischemic stroke in which congestive heart failure, hypertension, age over 75 years, and diabetes are each assigned a point score of 1, while having experienced a stroke or a transient ischemic attack (TIA) is assigned a point score of 2.

A study of 5000 healthy individuals without afib found that CRP values varied between 0.01 mg/dL (0.1 mg/L) and 0.38 mg/dL (3.8 mg/L) with a median of 0.16 mg/dL (1.6 mg/L). A CRP value above 0.38 mg/dL (3.8 mg/L) is generally considered a sign of a systemic inflammation. There is also evidence that a high CRP value increases the risk of ischemic stroke.

The study group included 3288 patients who underwent coronary angiography for suspected CAD. Of these, 61% did indeed have CAD, 56% had hypertension, 19% diabetes, 15% congestive heart failure, and 2% had suffered a prior stroke or TIA. Ten percent of the group had atrial fibrillation (AF) who, on average, had a higher CRP level (14.0 mg/L or 1.4 mg/dL) than did study participants without AF (9.1 mg/L or 0.9 mg/dL). A higher CHADS2 score was found to be associated with a higher CRP value.

Researchers at the Mayo Clinic and the University of Utah School of Medicine have now quantified the relationship between CRP and stroke risk. They

CHADS2 Score	hs-CRP, mg/L
0	1.99 (0.2 mg/dL)
1	2.91 (0.29 mg/dL)
2	3.49 (0.35 mg/dL)
3	3.89 (0.39 mg/dL)
4 – 5	4.82 (0.48 mg/dL)

Patients with AF tended to have higher CRP values for equivalent CHADS2 scores, but this increase was only statistically significant for a CHADS2 score of 2 indicating that afib, on its own, does not increase CRP in patients with a low risk of ischemic stroke (0 or 1 risk factor).

Editor’s comment: The authors of the study conclude that the presence of AF increases hs-CRP across the CHADS2 score strata and that this means AF is an inflammatory process and may convey an independent risk of ischemic stroke. It is indeed unfortunate that their data does not support this conclusion, i.e. patients with AF and a CHADS2 score of 0 or 1 (none or one risk factor) do not have a statistically significant higher level of CRP than do patients without AF. Furthermore, the CRP levels

Crandall, MA, et al. Atrial fibrillation and CHADS2 risk factors are associated with highly sensitive C-reactive protein incrementally and independently. PACE, Vol. 32, May 2009, pp. 648-52

associated with CHADS2 scores of 0 and 1 (2 mg/L and 2.9 mg/L) are not generally considered a sign of systemic inflammation, but are within the range observed in healthy people. It would seem that the conclusions drawn from this study are flawed, but

as the headlines reporting the results in the mainstream media will no doubt read “AF is an independent risk factor for stroke”, I thought it useful to debunk this study before its results attain the status of dogma.

Disappointing results for valsartan (Diovan)

FLORENCE, ITALY. It is now generally accepted that there is a connection between atrial fibrillation (AF) and the renin-angiotensin-aldosterone system (RAAS). Aldosterone causes inflammation and fibrosis of the heart tissue by direct action on the mineralocorticoid (MC) receptors in the myocardium; this results in both electrical and structural remodeling of the atria. Aldosterone also causes an increased level of reactive oxygen species in the heart, promotes the loss through the urine of potassium and magnesium, and is associated with an unbalanced (overly sympathetic) autonomic nervous system.

Not surprisingly, several researchers have speculated if blocking the action of the RAAS would prevent, or at least reduce, the frequency of afib episodes. Angiotensin II type 1 receptor antagonists (AT1R) are potent blockers of MC receptors and thus should, in theory, reduce the adverse effects of aldosterone on the heart. There is also evidence that AT1R antagonists, specifically valsartan (Diovan) reduces plasma aldosterone levels. A group of Italian researchers (GISSI-AF investigators) now report on a large clinical trial aimed at determining if valsartan is effective in preventing the recurrence of AF.

The trial involved 1442 AF patients who were randomized to receive 320 mg/day of valsartan (gradual dose increase over 4 weeks) or a placebo for one year. All participants were in sinus rhythm at the start of the trial, but 88% had undergone an electrical or pharmacological cardioversion within 2 weeks prior to randomization. The majority (85%)

of participants had reasonably well-controlled hypertension, 16% had diabetes, 57% were taking ACE inhibitors, 35% were on amiodarone, and 32% had been prescribed a class I antiarrhythmic (flecainide, propafenone, disopyramide). After 8 weeks, when all participants (except placebo group) were on the full dose of valsartan, 42.7% in the valsartan group and 44% in the placebo group had experienced recurrence of afib. Corresponding percentages after one year was 51.4% and 52.1%. More than one episode during the year was recorded by 26.9% in the valsartan group and in 27.9% in the placebo group. None of the above differences were statistically significant. The conclusion was that treatment with valsartan does not reduce the incidence of AF in this patient population.

Valsartan for prevention of recurrent atrial fibrillation. New England Journal of Medicine, Vol. 360, April 16, 2009, pp. 1606-17

Gillis, AM. Angiotensin-receptor blockers for prevention of atrial fibrillation – a matter of timing or target? New England Journal of Medicine, Vol. 360, April 16, 2009, pp. 1669-70

Editor’s comment: It is fairly conclusive that valsartan did not prevent the recurrence of afib episodes in this fairly unhealthy group of afibbers. In other words, it did not reverse existing electrical and structural remodeling. However, this does not mean that valsartan or other AT1R antagonists could not be effective in **preventing** the remodeling from starting or progressing in the first place. Hopefully, primary prevention trials will be conducted to prove or disprove this possibility.

Safety of catheter ablation

MILAN, ITALY. Catheter ablation with the intent of curing atrial fibrillation (AF) is one of the most complex procedures in medical practice. It involves the introduction and manipulation of several catheters in a space smaller than the inside of a golf ball, the puncture of the wall between the right and left atrium without puncturing the heart wall itself in

the process (cardiac tamponade), the close control of anticoagulation, and the accurate placing of multiple lesions in the left atrium. One would expect a fairly high casualty rate with such a complex procedure. A team of electrophysiologists from around the world now reports that the mortality rate

associated with catheter ablation for AF is less than 0.1% (0.98 per 1000 patients).

Their study included data from 45,115 procedures performed on 32,569 patients in 162 ablation centers between the years 1995 and 2006. During or within 30 days of the procedure 25 patients died (0.077%) and another 7 procedure-related deaths (0.021%) occurred more than 30 days following the procedure. Thirteen of the deaths occurred during the procedure itself of which 5 were due to tamponade, 2 to stroke, and the remaining due to heart attack, torsades des pointes or other causes. During the following 30 days, 5 patients died from atrioesophageal fistula, 2 from cardiac arrest, and another 2 from massive antibiotic-resistant pneumonia.

Somewhat surprisingly, the study group found no difference in fatality rates associated with the volume of procedures performed in a center, nor was there any difference attributable to the type of procedure (segmental or circumferential) or size of the catheter. Overall, the 0.1% procedure-related deaths were most commonly associated with cardiac tamponade, atrioesophageal fistula and stroke or heart attack.

Cappato, R, et al. Prevalence and causes of fatal outcome in catheter ablation of atrial fibrillation. **Journal of the American College of Cardiology**, Vol. 53, No. 19, May 12, 2009, pp. 1798-803

Belhassen, B. A 1 per 1,000 mortality rate after catheter ablation of atrial fibrillation. An acceptable risk? **Journal of the American College of Cardiology**, Vol. 53, No. 19, May 12, 2009, pp. 1804-06

Editor's comment: Although clearly a cause for concern when deciding to undergo an ablation for afib, a 0.1% fatality rate is actually quite low and on par with the rate observed for relatively simple cataract surgery. Appendectomy (removal of the appendix) is associated with a 0.2% mortality rate, hip replacement with a 1% mortality rate, and bypass surgery with a 3% risk of death. It should also be kept in mind that the observed fatality rate for afib ablation is per patient not per procedure. If looking at the rate per procedure, the risk of a fatal tamponade is 0.016% (1.6 deaths per 10,000 procedures), that of atrioesophageal fistula 0.011% (1.1 deaths per 10,000 procedures), and that of stroke and heart attack 0.009% (0.9 deaths per 10,000 procedures). The all-inclusive fatality rate per procedure is 0.07% - really an amazingly low number when considering the complexity of the procedure.

Inflammation and AF recurrence after ablation

TSUKUBA, IBARAKI, JAPAN. Experiencing PACs (premature atrial contractions) and episodes of tachycardia or atrial fibrillation immediately following a pulmonary vein isolation (PVI) procedure is not uncommon and, not surprisingly, is a cause of great concern for newly-ablated afibbers. A team of Japanese researchers recently set out to answer the question, "Does the occurrence of one or more sustained afib episodes during the 3 days after a PVI indicate that the procedure was unsuccessful?"

Their study included 186 paroxysmal afibbers (average age of 60 years, 79% men) who underwent a segmental PVI (double-lasso technique). The end-point of the procedure was complete bidirectional block between each pulmonary vein and the wall of the left atrium. During the 3 days immediately following the procedure 45 patients (24%) experienced afib episodes lasting longer than 30 seconds (immediate recurrence group), another 27 patients (15%) experienced sustained afib episodes in the period between 4 days and 1 month following their PVI (early recurrence group), and the remaining 114

patients (61%) experienced no afib episodes (lasting longer than 30 seconds) at all during the first month.

The Japanese research team recorded body temperature (baseline and then every 6 hours after PVI), C-reactive protein levels, occurrence of frequent PACs (more than 10 per minute), and sustained (longer than 30 seconds in duration) and non-sustained (shorter than 30 seconds) afib episodes during the initial 3 days post-PVI procedure. Highlights of their findings are:

- An elevated body temperature after the procedure and a significant increase in post-procedural body temperature compared to baseline were significantly associated with immediate afib recurrence as was an increase in CRP level indicating that inflammation plays an important role in immediate recurrence.
- Frequent PACs and non-sustained afib episodes during the first 3 days were

significant predictors of early recurrence (4-30 days post-procedure) and were substantially more frequent than in the immediate and no-recurrence groups.

- At the end of the 6-month follow-up period, 76% of the members of the immediate recurrence group were free of afib without the use of antiarrhythmic drugs. In contrast, only 30% of the early recurrence group was in normal sinus rhythm at the end of follow-up. In the group that experienced no afib episodes during the first month post-ablation, 96% were afib-free without antiarrhythmics at the end of follow-up. All told, 79% of ablatees were in NSR without the use of antiarrhythmics at the 6-month follow-up.

The researchers suggest that immediate afib recurrence may be associated with the process of

healing the myocardial damage caused by the PVI procedure, while early recurrence is likely due to recovery of conduction between the pulmonary veins and the left atrium.

Koyama, T, et al. Comparison of characteristics and significance of immediate versus early versus no recurrence of atrial fibrillation after catheter ablation. American Journal of Cardiology, Vol. 103, 2009, pp. 1249-54

Editor's comment: The finding that having one or more sustained afib episodes during the first 3 days following a PVI procedure is, by no means, a sign of procedural failure is indeed a comforting one. I would venture a guess that the high inflammation level associated with immediate recurrence is the result of a more aggressive ablation approach, which would be expected to be associated with a better long-term outcome.

A case of too much potassium

LONDONDERRY, NORTHERN IRELAND. Cardiologists at a Londonderry hospital report the case of a 55-year-old woman who was admitted due to a spell of dizziness whilst out walking. Prior to admission her GP had done an ECG (electrocardiogram) and noticed an abnormal heart rhythm (markedly widened QRS complexes). The patient had hypertension and had suffered one previous episode of atrial fibrillation. Her medications included a beta-blocker, amiodarone, amlodipine (a dihydropyridine prescribed for angina and hypertension), warfarin, and recently prescribed cephalexin (a cephalosporin antibiotic) for a urinary tract infection.

At admission the patient's creatinine level was 114 micromol/L or 1.3 mg/dL, which is well outside the normal range of 0.5 to 1.0 mg/dL and thus would indicate impaired kidney function. Her serum potassium level was 9.6 mmol/L, which is well outside the reference range of 3.5 – 5.0 mmol/L. Questioning by the attending physician revealed that the patient had been prescribed potassium citrate for painful urination 6 months earlier. She

had continued to take it every day ever since in a dose of 200 mmol/day which corresponds to 7800 mg/day. The patient was treated with calcium gluconate, insulin and dextrose after which her heart rhythm (ECG) returned to normal.

Lyons, KS and McGlinchey, P. Hyperkalaemic cardiac arrhythmia due to prolonged ingestion of potassium citrate. International Journal of Cardiology, Vol. 131, 2009, pp. e134-e136

Editor's comment: This case illustrates the danger of supplementing with excessive amounts of potassium when kidney function is impaired. The patient's total potassium intake, including the amount obtained from the diet, would likely approach 10 grams/day, which is more than twice the recommended intake of 4.7 grams/day. The case also underscores the importance of having kidney function checked before embarking on any potassium supplementation protocol at all. Most afibbers, who have found potassium supplementation beneficial, use about 1.5 grams/day and maintain their serum level in the 4.1- to 4.5-mmol/L range.

Tea may help prevent stroke

LOS ANGELES, CALIFORNIA. Several studies have shown that habitual consumption of green tea is protective against cardiovascular disease, breast cancer, and prostate cancer. Now researchers at the UCLA School of Medicine report that habitual tea (green or black) drinking is also associated with a reduced risk of stroke.

The researchers performed a meta-analysis of 10 studies from 6 different countries (China, Japan, Finland, the Netherlands, Australia and the USA). Mortality from stroke (ischemic and hemorrhagic) among 35- to 74-year-old men in these countries ranged from 3 per 10,000 in Australia to 24 per 10,000 in rural China. The 10 studies covered 4378 strokes (fatal and non-fatal) that occurred in a total population of 194,965 men and women. The annual incidence of stroke in a subgroup of 162,700 individuals was 0.7% a year. The researchers pooled the results of the 10 studies and arrived at the conclusion that individuals drinking 3 or more cups of green or black tea every day had a 21%

lower risk of stroke than did those consuming less than one cup a day. They speculate that the protective effect of tea may be due to its ability to improve endothelial function and thereby increase blood flow to the brain, or to the ability of one of its components (theanine) to reduce the size of the area impacted by a stroke. They also point out that the protective effect of tea is primarily evident in the case of ischemic stroke and is likely to be considerably less important, if important at all, in the prevention of hemorrhagic stroke.

Arab, L, et al. Green and black tea consumption and risk of stroke: a meta-analysis. Stroke, Vol. 40, May 2009, pp. 1786-92

Editor's comment: Although there is, as yet, no conclusive evidence that green tea extract in capsule form provides the same benefits as green tea itself, it may be advisable for those who do not drink tea to supplement with a green tea extract containing its two most important components epigallocatechin and gallate (EGCG) and theanine.

Incidence of thrombi prior to ablation

BALTIMORE, MARYLAND. Catheter ablation for atrial fibrillation (AF) carries a low (0.3 – 0.7%), but still significant, risk of ischemic stroke and transient ischemic attack (TIA). The two major causes of stroke or TIA occurring during the procedure are the formation and subsequent embolization (blocking of a blood vessel) of char or thrombi (blood clots) formed on the ablation catheter and the disturbance of existing thrombi in the left atrium or left atrial appendage (LAA). This disturbance can be caused by catheter manipulation or be related to the more forceful heart beat accompanying restoration of normal sinus rhythm. The risk of a stroke or TIA during the procedure can clearly be reduced by ensuring that there are no clots in the left atrium or LAA prior to starting the procedure. This is the reason why most major ablation centers insist that patients scheduled for an ablation undergo a TEE (transesophageal echocardiogram) shortly before the procedure. A TEE is the "gold standard" for determining if clots are present in the left atrium or LAA, but the procedure is invasive, uncomfortable for the patient and relatively costly. Thus, researchers at Johns Hopkins University School of Medicine recently undertook a study to determine if all afib patients scheduled for ablation actually needed a pre-procedural TEE.

Their study involved 585 patients who underwent a total of 732 ablation procedures (repeat rate of 25%). All patients were anticoagulated with warfarin for at least 4 weeks prior to their procedure except for the last 5 days when warfarin was replaced by enoxaparin (a low molecular weight heparin). All patients also underwent a TEE within 24 hours prior to the procedure. The researchers found thrombi in the LAA in 12 cases (1.6%) requiring cancellation of the procedure. All 12 patients had been on warfarin for at least 6 months prior to the procedure and all had a left atrial diameter equal to or greater than 4.5 cm (45 mm). Nine of the patients had persistent afib and 3 had paroxysmal AF.

Analysis of all the TEE data collected revealed that the risk of finding a clot in the left atrium or LAA was significantly associated with left atrial diameter and stroke risk as measured with the CHADS2 score. NOTE: The CHADS2 score assigns a risk of 1 point each for congestive heart failure, hypertension, age 75 years or older and diabetes, and 2 point score if having suffered a previous stroke or TIA. Thrombi were present in 0.3%, 1.4% and 5.3% in patients with CHADS2 scores of 0, 1 and 2 or greater. None of the cases where thrombi were observed had a left atrial diameter less than 4.5 cm. In contrast, no

thrombi were observed in patients with a CHADS2 score of 0 and a left atrial diameter of less than 4.5 cm.

The researchers suggest that a pre-procedural TEE may be unnecessary in this group of patients provided they have been properly anticoagulated prior to the procedure.

Scherr, D, et al. *Incidence and predictors of left atrial thrombus prior to catheter ablation of atrial fibrillation.* **Journal of Cardiovascular Electrophysiology**, Vol. 20, April 2009, pp. 379-84

Cappato, R. *Searching for left atrial thrombi prior to catheter ablation of atrial fibrillation.* **Journal of Cardiovascular Electrophysiology**, Vol. 20, April 2009, pp. 385-87

Editor's comment: This study confirms that warfarin is by no means 100% effective in preventing clot formation in the left atrium and, on a more positive note, that healthy afibbers with no stroke risk factors and a left atrial diameter less than 4.5 cm have a very low risk of forming clots in the left atrium.

My Travels Along the AFIB Road

by Richard Webster

I am presently 69 years of age. My history of athletics was high school, college and military basketball and baseball with normal training routines. Later in my 30s I began jogging about 10 miles a week for many years. I did not run marathons but did run some 10k events – not a hard runner, but more than the casual jogger.

The Road Down

In the late 1990s and throughout 2001 I kept having periods of a racing heart beat and associated tiredness. I learned that in some cases the condition would go away with 4 to 8 hours of rest. At the advice of my GP, on one occasion, I drove to the local ER and had an EKG where the condition was diagnosed as afib. I was placed on Coumadin, 25 mg/day atenolol and 180 mg/day Cartia, a calcium channel blocker. After some deliberation, I stopped the Cartia but continued the other two.

I set out to learn all I could about this condition and believe that I am a lone afibber, as all of my other tests and blood chemistry are normal. I was 61 years at the time. I am 6'5" and weighed 205 lbs. My cardiologist did not agree I had LAF, as my blood pressure was in the 130 to 140 over 80 to 90 range. He said that, although not real high, he thought that qualified as an additional condition. My first attempt to eliminate afib was to avoid all the known triggers as best I could, but that did not seem to help, as an episode would start, apparently for no common reason that I could determine. I also thought that being in a stressful job and about to retire contributed to the condition. As an aside, now knowing this was afib, I can remember having short episodes in my early 20s. On occasion, while playing basketball we would call a timeout. During the timeout I would notice my heart race, but it would always stop when play resumed. And in my late 30s, during a stressful divorce, it reappeared. After an electrocardioversion to NSR, it did not reappear.

Now between 1999 and the spring of 2005, episodes were happening about every 3 to 4 months and would last 16 to 20 hours. By May 2005, episodes were down to every 2 months and lasting from 24 to 30 hours, and I started in earnest to track my condition and try some natural approaches. Shortly thereafter I found www.afibbers.org and started taking vitamins. I started on a program of 200 mg Magnesium Aspartate, 100 mg Potassium Aspartate, 150 mg COQ-10, 1000 mg Vitamin C, a B-50 complex and Omega-3 with 360 mg EPA and 240 mg DHA. I also eat plenty of bananas. My potassium serum levels were normal, but on the lower end of the scale. For the year, May 2005 to May 2006, my afib burden (% of total time spent in afib) was 2.31%. For the next 12 months it became 3.14%. From May 2007 to May 2008, it increased to 5.00%. Episodes were now in the every 3-week range and, in some cases, were lasting 24 to 36 hours.

The Road Up

Coincident with a cholesterol level that had crept up to the 220 range and a decrease in my HDL, I started to change diet and reestablished a mild but significant exercise program. I was intrigued by the case study in the May 2008 issue of *The AFIB Report* as to the benefits of the Paleo diet. I began to follow the dietary recommendations in the book *The 8 Week Cholesterol Cure* by Robert E. Kowalski. I started to exercise regularly on an elliptical machine or tread mill for 20 minutes each day. Within three months I lost 20 lbs., mostly

belly fat. Coincidentally my blood pressure dropped to 110 to 120 over 65 to 70 ranges. At this point, I reduced the atenolol dosage to 12.5 mg daily. During the next few months I had one stretch of 70 days without an episode.

In September 2008 I was given a drug load test of 600 mg propafenone and found it to be acceptable to use as a pill-in-the-pocket method to try and reduce the duration of my afib episodes. Since that time I have had 3 episodes where I took a 300 mg dose to see if it would reduce the durations. Prior to these last 3 episodes, my standard duration was around 24 hours but the last few of the summer and fall of 2008 had increased to 36- to 40-hour duration. For the first episode I waited 17 hours and took 300 mg and converted to NSR one hour later. The second episode, I waited 12 hours and took 300 mg and converted 4 hours later. The third episode I took 300 mg one hour after onset and there was no conversion. I waited until 18 hours and took another 300 mg and converted 4 hours later. Again a laboratory of one is not a significant conclusion, but it would suggest that Rythmol is effective in reducing my afib episode durations, but that the body does need some time in afib to "reset" to a condition where NSR is possible.

Next, the case study in the October 2008 issue of *The AFIB Report* again mentioned the Paleo diet and the addition of 4000 mg taurine daily with a cessation of episodes for 55 months. In late October, I added 3000 mg of taurine daily to my dietary supplements. The results have been phenomenal. Since beginning the taurine I have only had three episodes. All three were definitely shortened in duration by taking 300 mg propafenone. One interval between episodes was 73 days and, as I write this, it has been 131 days since my last one. My afib burden from May 2008 to May 2009 dropped to 2.11%. But the best news is that my burden for 2009, so far, is 0.00%!

THE AFIB REPORT is published 10 times a year by:

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

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