THE AFIB REPORT

Your Premier Information Resource for Lone Atrial Fibrillation!

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It is now abundantly clear that permanent or long-lasting, persistent atrial fibrillation is significantly more difficult to cure with catheter ablation than is paroxysmal (intermittent) AF. Prof. Haissaguerre and his group at Hopital Cardiologique du Haut Leveque in Bordeaux now report that the likelihood of a permanent afibber experiencing a successful procedure can be determined with good accuracy from a standard electrocardiogram. They also suggest that patients with long-lasting, persistent afib should wait no longer than 2 years before being referred for an electrophysiology study and possible ablation.

Also in this issue, we report that dronedarone (Multaq) is likely safer, but less effective than amiodarone (Cordarone), that athletes are at greater risk of developing afib, that severe mitral regurgitation and mitral valve prolapse actually decrease, rather than increase, the risk of ischemic stroke, and that Framingham researchers have developed a risk score for atrial fibrillation.

We conclude with an intriguing story from Tom Voll, P.Eng. who observed that statin drugs may provoke vagal afib due to their tendency to increase vagal tone. Thanks Tom for sharing!

To find the answers to most of your questions about lone AF don't forget to check out "Frequently Asked Questions" at http://www.afibbers.org/faq.htm.

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

Wishing you lots of NSR,

Hans

Highlights

Permanent afibbers should not wait for an ablation

BORDEAUX, FRANCE. It is well-established that permanent or long-lasting, persistent atrial fibrillation (AF) is significantly more difficult to cure with catheter ablation than is paroxysmal (intermittent) atrial fibrillation. Paroxysmal AF is usually triggered by rogue P-cells located in the pulmonary veins; thus, electrically isolating the veins from the left atrium is often enough to terminate the arrhythmia permanently (pulmonary vein isolation [PVI] procedure). However, once episodes become long-lasting and no longer subject to spontaneous self-conversion (persistent or permanent AF), then it becomes much more difficult to cure the arrhythmia with catheter ablation. This is due to the fact that triggers are now also active outside the pulmonary veins and substantial substrate modification has likely taken place as well. Thus, although a standard PVI (segmental or circumferential) is still the starting point, it is necessary to also perform electrogram-based

ablation and linear ablation in order to achieve a reasonable degree of success.

It is becoming increasingly clear that the demand for catheter ablations is increasing substantially faster than the capacity to provide them. This is especially true when it comes to persistent and permanent AF where exceptional skill and longer procedure times are required to ensure success. A team of researchers from Hopital Cardiologique du Haut-Leveque and Lund University in Sweden now reports that a standard surface electrocardiogram (ECG) taken prior to an ablation can provide a surprisingly accurate indication as to whether the procedure is likely to be successful or not.

Their study involved 90 patients (average age of 57 years, 84% men) with long-lasting, persistent AF defined as continuous afib lasting longer than one month and resistant to either electrical or pharmaceutical conversion. The majority (75%) of the patients had no structural heart disease, so would be classified as lone afibbers. All patients underwent a standard segmental PVI plus whatever other ablations were necessary to terminate the arrhythmia. Overall success, measured as being afib-free without antiarrhythmics, was 84%; however, an average of 1.8 procedures per patient was required to achieve this. Prior to starting the actual ablation, all patients had a standard ECG in which particular attention was paid to the AF cycle length (AFCL) observed at lead V_1. This cycle length was found to correlate well with cycle lengths measured inside the heart at the right and left atrial appendages.

The researchers noted that the pre-ablation AFCL was a good indicator of whether or not afib would be terminated by the ablation procedures. Thus, the average pre-procedure AFCL among patients whose arrhythmia terminated was 154.3 milliseconds (corresponding to 389 atrial contractions per minute) as compared to only 131.7 ms (corresponding to 456 atrial contractions per minute) in those whose arrhythmia did not terminate. This difference was highly significant.

It was also clear that patients with a smaller left atrial diameter (47 mm vs 54 mm) and a shorter duration of persistent afib (22 months vs 60 months) had a significantly better chance of termination. However, the only independent predictor of termination success was a long AFCL.

Eighteen months after the last ablation, 84% of patients were in normal sinus rhythm (NSR) without the use of antiarrhythmics. At one year, 93% of patients with an AFCL greater than 142 ms were in NSR as compared to only 68% among those with a cycle length less than 142 ms. Also at one year, 95% of patients who had been in persistent afib for less than 21 months were in NSR as compared to only 72% of those who had longer-standing afib.

The authors conclude that measuring the AFCL with a standard ECG prior to deciding on treatment is useful for predicting which patients are most likely to benefit from an ablation. They also suggest that patients with long-standing, persistent (permanent) afib should wait no longer than 2 years before being referred for an electrophysiology study and possible ablation.

Matsuo. S, et al. Clinical predictors of termination and clinical outcome of catheter ablation for persistent atrial fibrillation. **Journal of the American College of Cardiology**, *Vol. 54, No. 9, August 25, 2009, pp. 788-95*

Wilber, DJ. Pursuing sinus rhythm in patients with persistent atrial fibrillation: when is it too late? **Journal of the American College of Cardiology**, *Vol. 54, No. 9, August 25, 2009, pp. 796-98*

Editor's comment: This study resulted in two important findings. Firstly, afibbers whose episodes change from paroxysmal to persistent or permanent should get in line for a possible ablation as soon as possible and those already in permanent afib should delay as little as possible in order to improve their chances of a successful outcome. Secondly, a measurement of AFCL on a standard surface ECG will give a good indication of whether an ablation procedure is likely to be successful in the case of permanent afib. Put another way, if the AFCL is below 142 ms then as many as 4 procedures may be required.

Porphyria associated with atrial fibrillation

LANSING, MICHIGAN. Porphyrias are a group of inherited disorders that interfere with the synthesis of heme, a crucial component of hemoglobin. Acute hepatic porphyria can be triggered by drugs, alcohol, fasting or treatment with sex hormones, and its main symptoms are abdominal pain and neuropsychiatric symptoms. Erythropoietic prophyrias are characterized by a distinct skin rash. There is substantial evidence that porphyria is

associated with a dysfunction of the autonomic nervous system.

Now a group of researchers from Michigan State University, the Mayo Clinic, Harvard Medical School and Assiut University in Egypt reports that porphyria is also associated with an increased incidence of paroxysmal atrial fibrillation. Their study involved 56 patients with porphyria diagnosed in the period 2000 – 2008 and 56 age- and gender-matched controls. About 70% of the study participants had the results of one or more electrocardiograms. Five patients in the porphyria group were diagnosed with atrial fibrillation and 2 were diagnosed with atrial flutter. Overall, 7 (18%) of porphyria patients with an available ECG were diagnosed with atrial flutter or afib as compared to only one patient (2.5%) in the control group. This difference was statistically significant. All but one of the 7 patients with porphyria and afib or flutter had acute intermittent porphyria and acute episodes of porphyria correlated with paroxysmal episodes of afib or flutter. None of the 7 patients had hyperthyroidism, a family history of afib/flutter, or left atrial enlargement.

The researchers suggest that the occurrence of afib/flutter in porphyria patients is due to a dysfunction of the autonomic nervous system resulting from impaired heme biosynthesis within the nerve cells, or accumulation of porphyrin precursors or their derivatives in the body. They also point out that the incidence of afib and flutter may have been even higher than reported due to the fact that the diagnosis in many cases were based on only one electrocardiogram.

Dhoble, A, et al. Relation of porphyria to atrial fibrillation. **American Journal of Cardiology**, *Vol. 104, 2009, pp. 373-76*

Editor's comment: It is interesting that the authors of this study believe that the link between porphyria and afib/flutter is a dysfunction of the ANS and that this dysfunction may be caused by oxidative stress and free radical injury of autonomic nerves. It is tempting to further speculate that the free radicals are generated by iron or iron-containing porphyrin precursors, which are left free to "roam around" because their incorporation into heme is inhibited.

Comparison of amiodarone and dronedarone

DURHAM, NORTH CAROLINA. Amiodarone (Cordarone) is the most effective antiarrhythmic drug on the market today, although a recent trial found that its efficacy in keeping atrial fibrillation patients afib-free for a year is only 34%. Apart from questionable efficacy, amiodarone also has a long list of potentially very serious side effects including thyrotoxicosis, hypothyroidism, pulmonary toxicity (fatal in 10% of cases), liver toxicity, optic neuropathy (including loss of vision), and blurred vision.

The amiodarone molecule contains 37.5% by weight of iodine and it is widely believed that it is the iodine that causes most of the adverse effects of the drug. Thus, it is not surprising that much research has been devoted to finding a drug similar to amiodarone (a benzofuran derivative), but without the iodine component. This search has now resulted in the development of dronedarone (Multaq). Dronedarone has undergone several large-scale clinical trials, which, with the exception of one (ANDROMEDA) involving patients with severe congestive heart failure, have found it to be safe and with no significant adverse effects after one year of use. However, an increase in serum creatinine level (an indicator of possible kidney toxicity) has been observed in some trials, as have gastrointestinal problems like diarrhea, nausea and vomiting.

Two large-scale clinical trials (EURIDIS and ADONIS) evaluated the effect of 400 mg of dronedarone in 1237 AF patients. At the end of the trial (12 months from the start), 24.8% of the placebo group were still in normal sinus rhythm as compared to 35.9% in the dronedarone group.

A group of researchers from Duke University Medical Center has now performed a comparison of amiodarone and dronedarone to establish their relative efficacy and safety. Unfortunately, their study does not involve a head-to-head comparison between the two drugs in which half of the study group is randomized to one drug, while the other half receives the other drug. Rather, the researchers selected 4 trials comparing amiodarone to placebo and 4 comparing dronedarone to placebo. Also included was one small trial of amiodarone vs. dronedarone. Unfortunately, the study population was quite heterogeneous in that the 4 dronedarone/placebo studies excluded patients with permanent AF, while 2 of the 4 amiodarone studies excluded patients with

paroxysmal afib. None of the studies included patients with highly symptomatic afib and none paid any attention to quality-of-life factors or to the incidence of stroke and bleeding.

Nevertheless, the researchers, not unexpectedly, found amiodarone to be about twice as effective as dronedarone in maintaining sinus rhythm. On the other hand, more patients discontinued amiodarone than gave up on dronedarone and there was a trend towards overall greater mortality in the amiodarone group. In interpreting this finding, it should be kept in mind that members of the amiodarone group tended to be in worse shape health-wise than those in the dronedarone group.

The researchers conclude that for every 1,000 patients treated with dronedarone (800 mg/day @ a cost of $9.00/day) rather than with amiodarone (200 mg/day @ a cost of $3.50/day), there would be 228 more recurrences of afib at one year, offset by 9.6 fewer deaths and 63 fewer adverse events requiring discontinuation of drug therapy.

Piccini, JP, et al. Comparative efficacy of dronedarone and amiodarone for the maintenance of sinus rhythm in patients with atrial fibrillation. **Journal of the American College of Cardiology**, *Vol. 54, No. 12, September 15, 2009, pp. 1089-95*

Chan, PS, et al. Amiodarone or dronedarone for atrial fibrillation: too early to know the winner? **Journal of the American College of Cardiology**, *Vol. 54, No. 12, September 15, 2009, pp. 1096-98*

Editor's comment: Due to the obvious lack of attention paid to ensuring that the groups of patients compared were indeed comparable, i.e. paroxysmal afibbers compared with paroxysmal afibbers, and permanent afibbers compared with other permanent afibbers, I am not really certain that one can draw any hard and fast conclusions from this study other than perhaps that dronedarone is likely less effective, but safer than amiodarone.

Atrial fibrillation and endurance exercise

COPENHAGEN, DENMARK. Several studies have concluded that men who regularly engage in vigorous exercise for extended periods of time are at increased risk of developing atrial fibrillation (AF), notably lone AF. A Danish research team has just completed a meta-analysis of 6 case-control studies comparing the prevalence of AF among athletes and gender- and age-matched controls.

The studies involved 655 athletes (regularly engaged in intense physical endurance sports) and 895 controls with an average (mean) age of 51 years (93% men). During a follow-up period ranging from 6 to 28 years, 147 cases of afib were observed among the athletes as compared to 116 cases among the controls. This corresponds to a prevalence of 23% among athletes as compared to 12.5% among controls, and a 5.29 times higher risk of developing afib among athletes. The researchers suggest that left atrial enlargement and increased vagal dominance are the most likely factors involved in predisposing athletes to the development of atrial fibrillation.

Abdulla, J and Nielsen, JR. Is the risk of atrial fibrillation higher in athletes than in the general population? **Europace**, *Vol. 11, No. 9, 2009, pp. 1156-59*

Editor's comment: According to this recent meta-analysis, the prevalence of afib among controls was 12.5% versus 23% for athletes. A recent study involving almost 17,000 male American physicians concluded that about 10% of the participants had been diagnosed with afib with athletes, especially joggers, having increased risk. The officially accepted number for the prevalence (total number of cases of a disease in a given population at a specific time) of AF in American adults over the age of 20 years is about 1%. The total adult population (over the age of 20 years) in the USA (February 2009) is 221 million, which means that about 2.2 million people in the USA have atrial fibrillation. In addition, it is estimated that 4.3 million individuals in the European Union, and 8 million in China also suffer from AF. Yet here we have a study (the Copenhagen study) which found 263 cases of afib among 1550 participants, or a prevalence of 17%. The Physicians Health Study found that during 12 years of follow-up, 1661 men out of 16,921 developed AF, which means that the prevalence of AF among this group at 12 years follow-up was 9.8%. Something does not add up! Here we have two carefully controlled studies, one involving almost 17,000 physicians, concluding that the prevalence of AF is anywhere between 10 and 17%, and yet, the official figure is 1%. Am I missing something here, or is it more likely that in the USA in excess of 20 million people rather than 1 million actually have AF – obviously mostly undiagnosed?

Mitral regurgitation and stroke risk

TOYAMA, JAPAN. Extensive research has shown that the main factor determining left atrium clot formation and associated stroke risk is the rate at which blood flows in and out of the left atrial appendage (LAA). If this rate is high then the formation of a clot or the precursors of a clot (smoke-like echoes [spontaneous echo contrast or SEC] on a transesophageal echocardiogram) is very unlikely.

Factors that can reduce the flow rate through the LAA include aging, congestive heart failure, poor left ventricular ejection fraction, elevated levels of hematocrit or von Willebrand factor, and a prior ischemic stroke or transient ischemic attack (TIA). Fortunately, a recent Japanese study concluded that lone afibbers and patients with atrial flutter are at very low risk for thrombus formation in the LAA. Now a group of researchers from Toyama University report that the presence of severe mitral regurgitation (MR), including mitral valve prolapse, increases the flow of blood in and out of the LAA and thus is associated with a substantially lower risk of clot formation.

Their study included 271 patients (average age of 67 years) with permanent atrial fibrillation who underwent both transthoracic and transesophageal echocardiography (TEE) and also had blood samples taken to measure markers of blood clotting, notably D-dimer. NOTE: Fibrin monomers and dimers polymerize into blood cots through the action of activated coagulation factor XIII. The patients investigated were by no means lone afibbers – about 40% had hypertension, 25% had heart failure, 25% had a history of stroke or TIA, and 56% were on warfarin. Of the 271 patients, 20 (7%) had severe MR including 9 patients with mitral valve prolapse, 45 (17%) had moderate MR, and 92 (34%) had mild MR as determined with TEE. The TEE study found that patients with severe MR had a higher average LAA peak flow velocity (35.2 cm/s) than did those with no or only mild MR (25.5 cm/s). In addition, the severity of SEC was significantly lower in patients with severe MR (0.7) than in those with moderate (1.7), mild (2.2) or no MR (1.9). The level of D-dimer was surprisingly low (0.76 mcg/mL) in patients with severe MR and highest (1.72) in those with moderate MR. Patients with no MR had an average D-dimer level of 0.82. Warfarin therapy had no effect on d-dimer levels or presence of SEC.

The Japanese researchers conclude that patients with severe MR, including mitral valve prolapse, have a lower thromboembolic risk than do those with mild or moderate MR. They speculate that the more "chaotic" blood flow resulting from severe MR helps prevent blood stasis in the LAA. They suggest that the increased D-dimer levels found among patients with moderate MR may be associated with their heart failure, but caution that the higher D-dimer levels and SEC values found among these patients could increase their risk of thromboembolism.

Fukuda, N, et al. Relation of the severity of mitral regurgitation to thromboembolic risk in patients with atrial fibrillation. **International Journal of Cardiology**, *August 5, 2009 [Epub ahead of print]*

Editor's comment: In an earlier LAF survey 7% of respondents reported that they had been diagnosed with mitral valve prolapse, while 14% had mild regurgitation. Although there is no evidence that lone afibbers with intact left ventricular ejection fraction are at increased risk for LAA blood stasis, SEC or thrombus formation, it is comforting to learn that the presence of severe mitral valve regurgitation, including mitral valve prolapse, does not increase stroke risk, but is actually protective against thromboembolism.

Antiarrhythmics post-ablation: Useful or not?

PHILADELPHIA, PENNSYLVANIA. Early recurrence of atrial arrhythmias (atrial fibrillation, flutter or tachycardia) is common following a pulmonary vein isolation (PVI) procedure, but does not necessarily indicate failure of the procedure. Nevertheless, such recurrences are disturbing to the patient and may result in the need for cardioversion and hospitalization. Some electrophysiologists (EPs) prescribe antiarrhythmics to be taken for a certain period after the ablation, while others just prescribe beta- or calcium channel blockers, or no drugs at all. Now EPs at the University of Pennsylvania report that taking antiarrhythmics for 6 weeks following a PVI markedly reduces the recurrence of long-lasting atrial arrhythmias as well as the need for cardioversion and hospitalization.

Their study involved 110 fibbers (average age of 55 years, 71% male, 50% with hypertension and 13% with coronary artery disease) who underwent a PVI during the period 2006 to 2008. Half the patients were randomized to receive an antiarrhythmic (60% received propafenone or flecainide) plus an AV node blocking agent (beta-blocker or calcium channel blocker), while the other half received only a blocker. All were monitored continuously for 4 weeks with a trans-telephonic event monitor and re-evaluated at the end of 6 weeks.

During the 6 weeks substantially more patients (28%) in the blocker only group experienced an arrhythmia lasting longer than 24 hours or needed cardioversion or hospitalization as compared to those in the antiarrhythmic group (13%). The authors conclude that treatment with an antiarrhythmic during the 6 weeks following a PVI reduces the risk of early recurrence of atrial arrhythmia and the need for cardioversion or hospitalization. They emphasize that antiarrhythmic therapy after ablation should always be coupled with an AV node blocking agent to prevent the rapid ventricular response that may occur in connection with atrial flutter occurring after the ablation. As a matter of fact, the incidence of post-procedural flutter or tachycardia recorded on the trans-telephone monitoring strips was actually higher in the antiarrhythmics group (28% vs. 11%), but presumably did not cause symptoms serious enough to require medical attention.

Roux, J-F, et al. Antiarrhythmics after ablation of atrial fibrillation (5A Study). **Circulation**, *Vol. 120, September 22, 2009, pp. 1036-40*

Risk score for atrial fibrillation

FRAMINGHAM, MASSACHUSETTS. It is estimated that atrial fibrillation (AF) now affects over 2 million individuals in the USA, some 4 million in Europe, and 8 million in China. By 2050 the prevalence of AF in the USA alone is expected to approach 16 million – truly a growth of epidemic proportions! As long as the cause(s) of the epidemic is largely unknown it is difficult to see how a program can be established to effectively prevent its continued growth. Nevertheless, it is important to determine the major risk factors for the development of AF in the general population. This is what the developers of the Framingham risk score for cardiovascular disease have now done.

Their study involved 4764 participants in the Framingham Heart Study enrolled between June 1968 and September 1987. The participants were monitored regularly for the first event of AF over a 10-year period following enrolment. About 55% of the study cohort were women, age range was 45 to 95 years, and at enrolment fewer than 5% had heart disease as defined by significant cardiac murmur, heart failure or previous myocardial infarction (heart attack). During the 10-year follow-up 457 (10%) participants developed AF with the incidence being almost twice as high among men than among women. After analyzing the data obtained during over 8,000 medical examinations, the Framingham researchers concluded that there are seven measurable factors which materially affect the risk of developing AF. These are:

- Age (risk increased with age)
- Body mass index (risk increased with a BMI greater than 30)
- Systolic blood pressure (risk increased if over 160 mm Hg)
- Being treated for hypertension (increased risk)
- PR interval[1] (risk increased if over 160 ms)
- Age at first sign of heart disease[2] (the earlier the greater the risk)
- Age at heart failure (the earlier the greater the risk)

[1] PR interval is the interval between the onset of the P-wave and the beginning of the QRS complex. It ranges between 120 ms and 200 ms with a higher value indicating vagal dominance.
[2] Age at which a significant cardiac murmur is first detected.

The age score ranges from –3 for women between the ages of 45 and 49 years to +8 for both men and women above the age of 85 years. A BMI greater than 30 (obesity) carries a risk score of 1, as does systolic blood pressure above 160 mm Hg, being treated for hypertension, and having a PR interval between 160 and 199 ms. A PR interval of 200 ms or higher is given a risk score of 2. The age at which heart disease is first diagnosed is given a score between 0 and 5, with the highest score given for age 45 to 54 years. Having heart failure

diagnosed between the ages of 45 to 54 years carries a risk score of 10, while being diagnosed at age 75 years or later carries a risk score of 0.

Thus, a woman between the ages of 55 and 59 years with no other risk factors would have a less than 1% risk of developing AF over the next 10 years. On the other hand, an obese man between the ages of 55 and 59 years with high systolic blood pressure and being treated for hypertension would have a 30% risk of developing AF if he was also diagnosed with heart disease or had experienced a heart attack. The scoring system was validated with a white, middle-aged to elderly cohort so may not be applicable to a different population such as younger adults.

The researchers point out that echocardiographic measurements such as left atrial diameter, left ventricular wall thickness, and left ventricular fractional shortening also affect the risk of developing AF, but not nearly to the same extent as do the seven major risk factors identified.

Schnabel, RB, et al. Development of a risk score for atrial fibrillation (Framingham Heart Study): a community-based cohort study. **The Lancet**, *Vol. 373, February 28, 2009, pp. 739-45*

Editor's comment: It is, as usual, unfortunate that no attempt was made to separate the risk of developing lone AF from that of developing the heart disease-associated from of AF. If this had been done it is likely that such factors as sleep apnea, GERD, and excessive physical exercise would also have emerged as important risk factors. It is interesting that the researchers associate a PR interval of 200 ms or greater as a sign of vagal dominance. Perhaps this readily obtainable ECG component could be used to distinguish between adrenergic and vagal AF among lone afibbers?

Ablation of ganglionated plexi

OKLAHOMA CITY, OK. Dr. Warren Jackman and his group at the University of Oklahoma Health Sciences Center present an excellent overview of the history of catheter ablation for atrial fibrillation (available at www.jafib.com). Dr. Jackman and colleagues cover developments from the first use of catheter ablation in 1994 to cure relatively simple arrhythmias, through Prof. Haissaguerre's 1998 discovery of the crucial role of pulmonary vein triggers in AF initiation, to the latest developments involving the ablation of areas exhibiting low-level complex fractionated atrial electrograms (CFAE) during AF episodes (Nademanee protocol).

Although standard pulmonary vein isolation (PVI) procedures carried out by highly skilled EPs are successful in about 90% of cases among paroxysmal afibbers, there is still considerable room for improvement when it comes to persistent and permanent afib. Dr. Jackman and his group now proposes that ablation of ganglionated plexi in the left atrium either by itself or in combination with a PVI procedure, a maze procedure or a mini-maze procedure may further improve success rates.

Ganglionated plexi (GPs) are the hubs or "command modules" of the intrinsic cardiac autonomic nervous system (ICANS). They are particularly well enervated with both adrenergic and vagal nerve endings and are housed in so-called "fat pads" which are mainly located in the area where the pulmonary veins enter the left atrium. Dr. Jackman and colleagues believe that afib episodes can be initiated by excessive vagal or adrenergic stimulation of the GPs and that ablation of the fat pads can substantially increase the chance of a successful ablation. The GPs can be located either on the inside (endocardial) or outside (epicardial) of the heart wall by high-frequency electrical activation.

A recent trial by Dr. Carlo Pappone's group in Milan found that a combination of a circumferential PVI and GP ablation resulted in 99% of 102 patients being free from afib after one year. A very small trial involving 18 patients claims a 94% success rate after one year with GP ablation alone. Similar promising results of GP ablation have been reported from cardiothoracic surgeons performing mini-maze procedures. Dr. Onorati and colleagues at the University of Catanzaro in Italy found that ablating GPs during mitral valve surgery resulted in 93% success as compared to 73% success in the group not undergoing GP ablation.

The Oklahoma researchers conclude that concomitant GP ablation may increase the success rate of catheter ablation and surgical procedures aimed at curing AF.

Scherlag, BJ, et al. The autonomic nervous system and atrial fibrillation: the roles of pulmonary vein isolation and ganglionated plexi ablation. **Journal of Atrial Fibrillation**, *Vol. 1, August 2009, pp. 471-86*

Editor's comment: The addition of GP ablation to the standard PVI procedure certainly looks promising. My only concern would be if this additional ablation, which obviously destroys parts of the heart's intrinsic regulatory mechanism, would result in the emergence of tachycardia or – not supported by any evidence I know of – increase the risk of sudden cardiac death.

Do Statin Drugs Precipitate Vagal AF?

by Tom Voll, P.Eng.

I have been much empowered through your research and *The AFIB Report* to learn and assume responsibility for my AF care, alongside my doctors. As a fellow engineer, I thought you might be interested in my latest observations.

I do a lot of international travel. During March this year I was on a trip which had to be extended a week. During that time I ran out of my Lipitor prescription. After a few days without Lipitor (normally 20 mg/day), I noticed that I seemed to be less likely to have ectopics and seemed further away from that "feeling" prior to having AF.

Then I recalled that I have been having trouble with AF almost as long (8 years) as I have been taking Lipitor (about 10 years now). So I decided to go without Lipitor to see how much my AF might be a side effect of taking Lipitor. Sure enough, the longer I was without Lipitor, the better I felt. I even cut my flecainide prescription in half (150 mg/day to 75 mg/day) with no problems.

I have classic "vagal" AF (starts during sleep, worse when sleeping on left side, or letting down after exercise, after eating, esp. caffeine, salt, alcohol). Also, alpha and beta-blockers almost killed me. So I did some research in medical journals and found three different articles connecting statin use with "improved" (increased) vagal tone (from increased vascular Nitric Oxide), as well as reduced LDL. My theory is that the increased (hyperactive) vagal tone is what was initiating my AF. Since "vagal" afibbers constitute a minority of AF patients, most researchers and MDs probably assume that increased vagal tone is better for everyone, which is why the connection between statins and vagal AF might have been missed. My holistic MD has tried a few statin substitutes (red yeast rice, pantethine, krill oil), but they all seemed to stimulate AF events. I am now trying niacin as a statin substitute.

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