THE AFIB REPORT

Your Premier Information Resource for Lone Atrial Fibrillation!

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The main feature of this issue is the excellent and thought-provoking article by Kerry Acker "My Amazing Maze". I always find it fascinating and a great privilege to be allowed to share a fellow afibber's journey. We can indeed learn so much from each other!! Kerry did an extensive amount of research before deciding on the full maze procedure using cryoenergy to create the lesions. He has now been completely afib-free for over 6 months and highly recommends the procedure and the surgeon who performed it. Thank you Kerry for sharing your story.

Success rate for ablation and maze procedures continues to be a major point of discussion. How should it be defined? How should it be measured (ECG, Holter, event recorder)? At what point in time should the judgment be made? Should only symptomatic episodes enter into the picture, or should asymptomatic episodes be considered as well? There are many question and so far, no consistent answers.

About the only thing the experts in the field agree on is that the first 3 months post-procedure should be considered a "blanking period". In other words, the occurrence of afib episodes during this time does not necessarily mean that the procedure was a failure. Personally, I believe that it is possible to get a fairly good idea of the final outcome of a procedure after the first, often unstable month. The recent survey of 314 afibbers who had undergone a PVI showed that complete success (no afib, no antiarrhythmics) was associated with only a 9% incidence of continuing afib after the first month. Failure, on the other hand, was associated with a 66% incidence of continuing episodes after the first month. This difference is extremely significant ($p < 0.0001$).

It is also becoming clear that the EP/surgeon performing the procedure often looks at success differently from the patient undergoing it. Some physicians consider a procedure successful if the patient is free of afib with or without the use of antiarrhythmics, and some even consider success as having reduced the patient's afib burden (time spent in afib) by 90% or better. The patient, in most cases, just wants to be rid of the whole thing and get on with a normal life. In other words, success is no afib with no medications and preferably, no need to avoid previous triggers.

In our surveys we have consistently categorized the outcome of an ablation (at 6 months post-procedure) as either:

- *Complete Success: No afib – no antiarrhythmics*
- *Partial Success: No afib, but still on antiarrhythmics*
- *Failure: Continuing afib episodes with or without antiarrhythmics.*

In an article covered in this issue the claimed 86% success rate is defined as free of afib with or without antiarrhythmics, or at least a 90% reduction in afib burden. According to our definition 56% of patients had complete success, 20% had partial success, and the remaining 24% were failures. The difference between a success rate of 86%, as defined in this article, and the actual 56% complete success rate is substantial and, once again, points out the need to always read "the small print".

Also in this issue we present a summary of the highlights of the 2007 EP conference in Venice, a novel means of predicting the success of cardioverting persistent afibbers, and a fascinating discussion of the association between resting heart rate and lifespan.

As most of you will know by now, this month also marks the release of my latest book "Lone Atrial Fibrillation: Toward A Cure – Volume IV". This 250-page book contains the information published in the 2006 issues of "The AFIB Report" arranged in logical sections. The comprehensive subject index makes it easy to locate that elusive, but important piece of information you know is there - somewhere.

Volume IV covers subjects ranging from the latest ablation procedures, their outcome and potential complications, to the safety and efficacy of antiarrhythmic drugs. The latest insights into the mechanism of atrial fibrillation as well as important information about stroke risk and prevention are also covered. The results of the 2006 Ablation/Maze Survey, a comprehensive evaluation of the outcomes of almost 500 ablation and maze procedures, and the personal stories of a number of afibbers who have cured or at least managed to control their condition round out this book -- a worthy companion to the original "Lone Atrial Fibrillation: Towards A Cure" and Volumes II and III.

You can order your copy of Volume IV at http://www.afibbers.org/volume4.htm

Wishing you good health and lots of NSR,

Hans

Highlights

Prediction of effectiveness of cardioversion

LUND, SWEDEN. Electrical cardioversion is generally not very effective in converting persistent afib (episodes lasting longer than 7 days and not converting on their own) to normal sinus rhythm (NSR). Although the immediate success rate may be 90% or better, as few as 25% of patients remain in NSR one year after the cardioversion. Even when using antiarrhythmic drugs only about 50% of cardioverted patients remain in NSR after a year. It is clearly of interest to be able to predict whether cardioversion is likely to be worthwhile in specific individuals.

Researchers at Lund University Hospital now report that a simple measurement of atrial fibrillatory rate (AFR) derived from a standard electrocardiogram (ECG) can predict the long-term success of cardioversion. AFR is essentially a measurement of atrial refractory period and can be determined by identification, templating and removal of the ventricular components of the ECG. The Swedish study involved 175 patients with persistent afib (37% had lone AF) who had an ECG prior to undergoing standard electrical cardioversion. The mean AFR of the entire group was 383 fpm (fibrillations per minute) and the average arrhythmia duration prior to cardioversion was 94 days.

The majority (71%) of the study participants were on a beta-blocker, while 29% were on digoxin (digitalis, Lanoxin). Congestive heart failure and a low left ventricular ejection fraction were both associated with a lower chance of remaining in NSR after cardioversion. After adjusting for possible confounding variables the researchers concluded that an AFR greater than 384 fpm was associated with a 3.2-fold increase in the risk of relapse, while being on digoxin was associated with a 2.3-fold increase. The researchers also observed that patients who had been in persistent afib for 30 days or less were more likely to be symptomatic when arriving at the hospital (97% vs. 62% for patients with longer-standing afib). The short duration (less than 30 days) afibbers were also significantly less likely to be on digoxin (7% vs. 33%) or beta-blockers (45% vs. 77%) and were also more likely to have lone AF. The difference in AFR between short duration afibbers who remained in NSR (348 fpm) and those who relapsed (424 fpm) was statistically highly significant (p=0.003). The difference in cardioversion outcome was also highly significant in the 16 patients who were on antiarrhythmic drugs (mainly sotalol) with those maintaining NSR having an AFR of 292 fpm vs. 382 fpm for the relapsers.

The Swedish researchers conclude that AFR prior to cardioversion is higher in patients relapsing into afib than in those remaining in NSR. They speculate that a higher AFR is associated with more extensive electrical remodeling.

Holmqvist, F, et al. Atrial fibrillatory rate and sinus rhythm maintenance in patients undergoing cardioversion of persistent atrial fibrillation. **European Heart Journal**, *Vol. 27, 2006, pp. 2201-07*

Editor's comment: This study, once again, demonstrates that digoxin (digitalis, Lanoxin) is a very bad drug for afibbers. Not only is it associated with a poorer cardioversion outcome, but it is also clear that there is a very strong (p=0.003) association between its use and the increased duration of persistent afib episodes. The same applies to beta-blockers where use is associated with a longer duration of episodes (p=0.001). The Swedish researchers point out that digitalis is known to increase intracellular calcium overload which, in turn, is believed to be the key process of atrial remodeling. The morale of this story is my oft-repeated assertion that digitalis has no place in the treatment of lone afib and that the use of beta-blockers on a continuous basis should be postponed as long as possible.

Resting heart rate and lifespan

ZURICH, SWITZERLAND. Resting heart rate (RHR) usually averages 60-80 beats a minute (bpm), but can occasionally exceed 100 bpm in unconditioned, sedentary individuals and be as low as 30 bpm in highly trained endurance athletes. It has long been known that patients with coronary heart disease and an elevated RHR have a shorter life expectancy than do those with a normal or low RHR.

Swiss researchers now suggest that an elevated RHR may also be associated with a reduced lifespan in the general population. They point out that among mammals RHR is generally inversely proportional to life expectancy – small animals have a higher RHR and shorter lifespan than do larges ones. As a matter of fact, the average number of heartbeats per lifetime in most mammalians is pretty well constant at about 7×10^8. The researchers provide the fascinating comparison of a shrew (weighing 2 g) and having a heart rate of 1000 bpm with a blue whale (weighing 100,000 kg) and having a heart rate of 6 bpm. The lifespan of the shrew is 1 year, while that of the blue whale is 118 years. Interestingly, the total lifetime oxygen consumption of the two species is very similar at about 37,000 litres/kg/lifetime.

For some unknown reason, humans deviate from the normal semi-logarithmic relationship between RHR and life expectancy in that they (barring any fatal diseases or accidents) are good for about 30×10^8 heartbeats per lifetime. The researchers provide pretty compelling arguments for their hypothesis and pose the fascinating question, "Does RHR causally determine lifespan and if so, what can be done to maximize it?"

It is estimated that a high RHR is genetically ordained in about 21-26% of cases. A high body mass index, hypertension, smoking, alcohol consumption, and the metabolic syndrome probably account for at least another 20%. Some very recent research postulates a common link between an elevated RHR and the metabolic syndrome, namely, a lack of nitric oxide, which modulates the autonomic control of RHR. It is also an established fact that regular endurance training increases parasympathetic activity and decreases sympathetic activity in the human heart resulting in a significant reduction in RHR.

Unfortunately, it is not clear whether the use of beta-blockers or calcium channel blockers to lower an elevated RHR will actually increase the lifespan of people without heart disease. However, it is known that preventing anxiety and stress, and avoiding toxics (caffeine, alcohol, nicotine, amphetamines, cocaine) can all reduce RHR – as can pet ownership. The search for new drugs that will reduce RHR without the side effects of beta- and calcium channel blockers is ongoing with one of the more promising candidates being ivabradine (Procoralan) which acts specifically on the sinus node to reduce RHR.

The researchers conclude their intriguing article by calling for large clinical trials to determine if any drug-induced reduction in RHR will indeed extend the life expectancy of healthy people.

Cook, S, et al. High heart rate: a cardiovascular risk factor? **European Heart Journal**, *Vol. 27, 2006, pp. 2387-93*

Editor's comment: I found this article absolutely fascinating but, you may well ask, what has it got to

do with lone atrial fibrillation? Several related questions immediately come to mind:

1. Can afibbers with asymptomatic permanent afib (without underlying heart disease) and uncontrolled RHR expect a shorter lifespan?
2. Can permanent afibbers (with no underlying heart disease) with RHR controlled by beta- or calcium channel blockers expect a shorter lifespan?
3. Many afibbers experience an elevated RHR for months, if not years, post-ablation or maze procedure. Will this, albeit temporary, rapid depletion of their lifetime heartbeat "capital" reduce their lifespan? Would medication with beta- or calcium channel blockers prevent the effects of this depletion? Should endurance exercise be "prescribed" for this group of afibbers?
4. There is some evidence that non-inherited elevated RHR is associated with a defect in the bioavailability of nitric oxide (NO). Would taking NO-generating medications such as NO-ASA (nitroxy-butyl-acetylsalicylate) or the amino acid, l-arginine, help reduce an elevated RHR experienced after an ablation or maze procedure?

These are all vital and intriguing questions. Hopefully, future medical research will address them.

Pill-in-the-pocket approach endorsed in the UK

LONDON, UNITED KINGDOM. It is estimated that 1.5% of the population of the UK now suffer from atrial fibrillation. About 200,000 of these patients have recurrent episodes and frequently visit emergency departments in order to have their episodes terminated by chemical or electrical cardioversion. Cardiologists at the University of London now suggest that many patients with paroxysmal afib can safely cardiovert themselves by taking a 300-mg dose of flecainide or a 600-mg dose of propafenone preferably within 5 minutes of experiencing the first signs of an afib episode. They base their recommendation on a study involving 210 patients with paroxysmal atrial fibrillation, less than 12 episodes a year, and no serious underlying heart disease. The researchers noticed a very significant drop in emergency department visits by the 210 patients once they began using the pill-in-the-pocket approach (form an average of 45 visits a month for all 210 patients to 5 visits a month). They point out that the on-demand approach should also be applicable to patients with mild hypertension or well controlled ischemic heart disease.

However, it is important that the first dose of the antiarrhythmic (propafenone or flecainide) be administered in a hospital setting. After taking the pill, the patient should rest seated or supine until the palpitations cease or for up to 4 hours. If the heart rate noticeably quickens or if dizziness or blackouts occur, the patient should be hospitalized immediately.

Camm, AJ and Savelieva, I. Some patients with paroxysmal atrial fibrillation should carry flecainide or propafenone to self treat. **British Medical Journal**, *Vol. 334, March 24, 2007, p. 637*

Editor's comment: Many afibbers have been using the pill-in-the-pocket (on demand) approach to good effect ever since our first two courageous experimenters (Patrick Chambers and Gert Mueller) tried it for the first time 5 years ago.

Atrial flutter after modified maze procedure

MAYWOOD, ILLINOIS. A modified maze procedure is often performed in patients with atrial fibrillation as part of corrective valve surgery. The procedure involves the creation of wide area lesions circumferentially encircling the pulmonary veins with the addition of any combination of a posterior wall, roof, mitral isthmus, septal and/or cavo-tricuspid isthmus lines. The lesions may be created with radiofrequency energy, cryothermy or microwave energy. It is now becoming apparent that although the procedure is quick (about 20 minutes) and quite effective in eliminating atrial fibrillation it is often followed by the appearance of other tachyarrhythmias especially right or left atrial flutter.

Electrophysiologists at the Loyola Medical Center now report on a series of nine patients who developed atrial flutter following a modified radiofrequency maze procedure. The researchers found that conventional ECGs were unable to distinguish between right and left atrial flutter. However, by using electrophysiological entrainment mapping they determined that three of the nine patients had typical counterclockwise right atrial flutter while the remaining six had left atrial flutter. This despite the fact that the maze procedure had included ablation of both the cavo-tricuspid isthmus (right atrial flutter) and the mitral isthmus (left atrial flutter). The researchers observed extensive scaring in both the right (38%) and left (65%) atria and speculate that this could explain the inability of surface electrocardiograms to distinguish between right and left flutter. All 9 patients underwent conventional percutaneous catheter ablation and after a mean follow-up of 8 months eight of the nine patients were back in normal sinus rhythm including three patients who remained on antiarrhythmic drugs.

In an accompanying editorial electrophysiologists at the University of Virginia point out that in the case of three of the patients who developed post-maze flutter, cryoablation was used in ablating the tricuspid and mitral isthmuses during the original maze procedure.

Akar, JG, et al. Surface electrocardiographic patterns and electrophysiologic characteristics of atrial flutter following modified radiofrequency maze procedures. **Journal of Cardiovascular Electrophysiology**, *Vol. 18, April 2007, pp. 349-55*

Mason, PK and DiMarco, JP. Atrial tachycardias after surgical ablations for atrial fibrillation: an incoming tide. **Journal of Cardiovascular Electrophysiology**, *Vol. 18, April 2007, pp. 356-57 (editorial)*

Editor's comment: It is becoming increasingly clear that the development of flutter after a modified maze procedure or a circumferentially guided catheter ablation is fairly common and is usually a result of incomplete lesion creation. Incomplete lesions around the pulmonary veins are of course also often responsible for recurrence of atrial fibrillation after a seemingly successful catheter ablation. In a recent study of eight patients who developed atrial tachyarrhythmias after a modified maze procedure German researchers observed that complete linear lesions may be difficult to obtain with cryoablation[1]. Thus, while cryoablation is generally considered very safe, there is now some question as to whether it, at its present stage of development, is uniformly able to create lesions that achieve complete isolation.

[1] Chun, KRJ, et al. Pulmonary vein conduction is the major finding in patients with atrial tachyarrhythmias after intraoperative maze ablation. Journal of Cardiovascular Electrophysiology, Vol. 18, April 2007, pp. 358-63

Sleep apnea and obesity linked to AF

ROCHESTER, MINNESOTA. Both obesity and obstructive sleep apnea (OSA) have been linked to an increased risk of atrial fibrillation. It is not known, however, whether these two conditions act in concert or whether they are independent risk factors. Researchers at the Mayo Clinic recently released a study aimed at determining risk factors for AF with particular emphasis on obesity (BMI>30) and OSA. Their study included 3542 Olmsted County adults without past or current AF who were referred for polysomnography to determine the quality of sleep. The study participants were followed for an average of 4.7 years in order to determine the incident of new onset AF. During the follow-up period 133 participants experienced a first incidence of atrial fibrillation. The researchers observed the following risk factors for participants under the age of 65 years: age, male gender, hypertension, smoking, coronary artery disease, elevated BMI (body mass index) and a decrease in nocturnal oxygen saturation (a cardinal feature of OSA). In contrast, in men over the age of 65 years only heart failure predicted the development of AF. After adjusting for other risk factors the researchers concluded that among subjects under the age of 65 years the cumulative probability of developing afib was about twice as high in subjects with OSA as compared to those without OSA. Furthermore, the use of continuous positive airway pressure (CPAP) did not affect the incidence of a first afib episode. For participants over 65 years of age only heart failure predicted the development of AF. In this group participants with heart failure had an 8 times higher risk of developing AF than did those without heart failure. Obesity (BMI>30) was independently associated with the development of AF with about a 7% added risk for each BMI unit above 25.

Gami, AS, et al. Obstructive sleep apnea, obesity, and the risk of incident atrial fibrillation. **Journal of the American College of Cardiology**, *Vol. 49, February 6, 2007, pp. 565-71*

Ablation improves left ventricular ejection fraction

PHILADELPHIA, PENNSYLVANIA. The left ventricular ejection fraction (LVEF) is a measure of the heart's pumping capacity with a low value being indicative of heart failure. Researchers at the University of Pennsylvania (Marchlinski/Callans Group) now report that low LVEFs improve markedly in afibbers after pulmonary vein isolation (PVI). Their clinical trial included 366 patients scheduled for a PVI; 67% of these patients had impaired left ventricular function with an average LVEF of 42%. The average LVEF for the remainder of the group was 61%. After one or more ablations 56% of the patients were free of afib and off all antiarrhythmics, another 20% were free of afib, but still on antiarrhythmics, while 10% had experienced a greater than 90% decrease in their afib burden (time spent in afib). The researchers equate this with an 86% success rate. The success rate was similar for patients with and without low LVEF.

A comparison of LVEF on the day after the PVI with the value obtained six months post-procedure showed that the average LVEF in the group with low LVEF had increased from 43% to 56%. The researchers conclude that a PVI may reverse AF-induced ventricular cardiomyopathy in patients with atrial fibrillation and depressed left ventricular function.

Gentlesk, PJ, et al. Reversal of left ventricular dysfunction following ablation of atrial fibrillation. **Journal of Cardiovascular Electrophysiology,** *Vol. 18, January 2007, pp. 9-14*

Editor's comment: This article provides a clear example of the confusion surrounding the definition of ablation success. In our surveys we have consistently categorized the outcome of an ablation (at 6 months post-procedure) as either:

- Complete Success: No afib – no antiarrhythmics
- Partial Success: No afib, but still on antiarrhythmics
- Failure: Continuing afib episodes with or without antiarrhythmics.

In this article the 86% success rate is defined as free of afib with or without antiarrhythmics or at least a 90% reduction in afib burden (time spent in afib). According to our definition 56% of patients had complete success, 20% had partial success and the remaining 24% were failures. The difference between a success rate of 86%, as defined in this article, and the actual 56% complete success rate is substantial and, once again, points out the need to always read "the small print".

Highlights from the Venice EP conference

VENICE, ITALY. During the *VeniceArrhythmias2007* conference a group of world-recognized experts in the field of atrial fibrillation ablation gathered to develop a document expressing the consensus reached by these experts regarding the current state of afib ablation. Among the EPs participating in the discussions leading to the so-called Venice Chart were Dr. Andrea Natale, Pr. Michel Haissaguerre, Dr. Pierre Jais, Dr. Carlo Pappone, Dr. Francis Marchlinski, Dr. Douglas Packer, Dr. Richard Schilling (UK), and Dr. Atul Verma (Canada). The discussions ranged from the detailed anatomy of the pulmonary veins to recommendations for post-ablation anticoagulation. Major highlights are presented below:

- The main anatomical structures, which may initiate ectopic beats resulting in afib, are the pulmonary veins, the vein (ligament) of Marshall, the musculature of the coronary sinus, and the posterior wall of the left atrium.

- Most patients (20-60%) presenting for ablation have 4 distinct pulmonary vein opening (ostia) into the left atrium. However, some have 3, some have 5 and their location relative to other features of the left atrium are, by no means, uniform. This provides the rationale for a CT scan or MRI prior to the ablation so as to be better prepared to deal with an unusual configuration.

- The onset and maintenance of atrial fibrillation, irrespective of the underlying mechanism, requires an event (trigger) that initiates the afib and a predisposing substrate that perpetuates it. Inflammation and autonomic nervous system dysfunction may also act to facilitate initiation and maintenance of AF.

- Most patients (about 90%) with AF have underlying structural heart disease or hypertension. Only about 10% have no evident cardiac disorder (so-called "lone" AF). **Editor's note** – The term "lone" AF as defined by this group of experts does not include a reference to age, but only to the absence of evident cardiac disorder.

- Atrial fibrosis and loss of myocardial tissue are common findings in patients with AF. There is some indication that ACE inhibitors and angiotensin receptor blockers may help prevent AF by inhibiting the angiotensin II promoted formation of collagen.

- There is now evidence that lone AF may be associated with certain gene mutations that reduces the atrial refractory period (AERP).

- Vagal denervation, produced by delivering RF energy to the sites of autonomic ganglia, may help control AF. **Editor's note** – It is also likely that extensive vagal denervation may result in a substantially elevated resting heart rate post-ablation.

- Atrial fibrillation perpetuates itself by electrical and structural remodeling. The electrical remodeling involves a shortening of AERP. The structural remodeling involves enlargement of the atria and changes to individual myocytes (heart cells) – more specifically, an increase in cell size, accumulation of glycogen, myolysis, changes in mitochondrial shape, and alterations in connexin expression.

- There are currently 5 main approaches to catheter ablation for AF – PV isolation, electrogram-based ablation, linear lesions, ablation of autonomic ganglionated plexi, and the sequential ablation strategy. **Editor's note** – The Venice Chart contains detailed descriptions of these procedures.

- The vast majority of procedures currently use radiofrequency energy to create the lesions. New approaches under development include balloon-shaped catheters powered by cryoenergy, high-intensity-focused ultrasound (HIFU), and laser energy.

- The working group emphasized that adequate support personnel and facilities must be available in hospitals performing ablations. These would include facilities for rapidly testing anticoagulation efficacy (ACT) during the procedure, competence and experience to perform needle pericardiocentesis immediately if needed, anesthesiology expertise to manage procedural sedation/general anesthesia and provide resuscitory support if required, cardiac surgery personnel to perform emergency surgical procedures (including open heart surgery) as needed, and the capacity for urgent bedside echocardiographic examination primarily for diagnosing pericardial tamponade (perforation of the heart wall).

- The EP performing the procedure must have adequate training and experience. Two-thirds of the workshop participants felt that a prospective candidate entering a training program for performing AF ablations should have performed at least 100 other ablation procedures and have attended at least 20 AF ablations before being allowed to assist in doing an actual procedure. More than 50% of the experts felt that a "rookie" EP should have performed at least 40 procedures under the guidance of an experienced EP before he/she could begin doing the procedure on their own.

- The general consensus in regard to anticoagulation prior to the procedure was that patients with stroke risk factors ($CHAD_2$ score equal to or greater than 1) and patients with persistent afib should receive warfarin for at least 3 weeks with documented INR between 2 and 3. Patients who arrive for the procedure in AF should have a TEE (transesophageal echocardiogram) on the day of the procedure or the day before to rule out the presence of left atrial thrombi. Patients with paroxysmal AF and a $CHAD_2$ score of 0 may be treated with warfarin or aspirin (75-325 mg/day). Warfarin should be continued in all patients for 3-6 months after the procedure.

- There is no consensus as to how the ultimate success of the procedure should be measured. There is general agreement that about 30-50% of patients with documented or symptomatic episodes during the first 3 months post-ablation will ultimately be free of afib. Thus, most experts now consider the first 3 months as a "blanking period" during which the success or failure of a procedure cannot be predicted.

- The most feared complications (approximate incidence in brackets) during AF ablations are stroke (1%), cardiac tamponade (0.1 – 1.0%), severe pulmonary vein stenosis (0.5 – 2.0%), phrenic nerve injury – usually resolving on its own (0.1 – 0.5%), atrio-esophageal fistula (very rare, but usually fatal), periesophageal vagal injury (1%), and catheter entrapment in mitral valve (0.01%).

- Post-ablation left atrial tachyarrhythmias (flutter and inappropriate sinus tachycardia) are quite common (3 – 50%), particularly among patients having undergone the circumferential, anatomically-guided (Pappone) procedure. About 50% of these tachycardias resolve on their own, but others may require repeat ablations if they are highly symptomatic.

- Reports of long-term success rates for AF ablation procedures range from 45% to 95%. In centers with the greatest experience the success rate for a first ablation was recently found to average 80.5% (no afib, no drugs). Repeat ablations added another 5-15% success to this number. Longer term recurrence is usually due to the recovery of electrical conduction between the pulmonary veins and the left atrium. Success rates tend to be lower in patients with structural heart disease and in those with permanent AF.

The Venice Chart, with its 20 fact-filled pages and 169 references, is clearly a must-read for anyone interested in the current status of atrial fibrillation ablation.
Natale, A, et al. Venice Chart international consensus document on atrial fibrillation ablation. **Journal of Cardiovascular Electrophysiology***, Vol. 18, May 2007, pp. 560-80*

My Amazing Maze

Kerry Acker (kacker4@hotmail.com)

I was diagnosed with afib in early January of 2003. A full battery of medical tests showed no heart disease, a normal sized left atrium and no other medical conditions that would cause my afib. It took me six months of consultations, research and trial and error to determine the most effective medical and lifestyle regimen. With the help of this board, I learned that I was a lone vagal persistent afibber and settled upon the following regimen:

1. Cardizem, which kept my heart at a safe and relatively comfortable rate during my episodes, which usually lasted 15-20 hours. Although my rate was comfortably low, the arrhythmia was usually very uncomfortable and resulted in considerable anxiety, discomfort and feelings of dread.
2. Flecainide, taken only at the onset of an episode and then twelve hours later. I usually converted within a few hours of my second dose. (Thanks to Hans for informing me about this option.) My EP did not believe this occasional use of flec would be effective, although she did not otherwise object to my plan to use it in this manner. She was wrong and a year or so later, the "pill in the pocket" became an accepted medical practice.
3. High doses of fish oil, together with a good multivitamin.
4. One full aspirin (325mg) per day.

In addition to the above, I found that the use of benzodiazepines on an intermittent basis helped to control my episodes. After I read a note in Hans' book, I tried using Ativan immediately upon inception of PACs, and found that it would terminate the PACs, and would thereby prevent the onset of an episode. I later switched to Valium, a longer acting benzo, which had the same effect. While I could not prevent all episodes, since many of my episodes were not preceded by PACs, I did obtain some measure of relief with this use of benzos. However,

once an afib episode had started, I found that the Valium did not help in converting me to NSR, although in some cases it made me more comfortable. In any event, I recognized that long-term use of benzodiazepines was not a result I wanted or that would be medically desirable.

During 2003, my episodes occurred about once every two weeks. Then, miraculously, without any changes in my meds or lifestyle, I had two years of relative calm. I experienced about nine episodes each year in 2004 and 2005, a major improvement over 2003. Then, literally on the first day of January 2006, my episodes returned with abandon, and I suffered more afib in the first three months of that year than I did in all of 2004 and 2005 combined. I decided that it was time to do heavy research into all available interventional options.

Over the next six months, I met with the EP group at the University of Pennsylvania, a top tier catheter ablation center. I also had a consultation with the Wolf group in Cincinnati about their "mini-maze" procedure. Finally, I met with a surgeon who performs the full Maze procedure. In between these visits around the country, I did daily reading and research into the available procedures, and I read posts from others who had had them done, queried doctors (and their assistants) through online websites, etc.

Even though I am a relatively young (50) lone afibber, I eventually decided not to undergo a catheter ablation or the less comprehensive "mini-maze". Rather I opted for a full Maze. Even though both of the former procedures are less invasive than the full Maze, I decided, based on the extensive research I had done and discussions I had had, that a comparison of the risk factors and success rates for all three procedures, led to the inescapable conclusion that a full Maze was my best option.

I concluded that both the catheter ablation and the full Maze presented specific and sometimes different risk factors, but in the case of a healthy, fifty year old lone afibber the relative overall risks were not necessarily greater with the Maze. According to my surgeon, the risks of stroke, bleeding or infection are no higher for the full Maze procedure than for an ablation or mini-maze, and some of the other risks of an ablation –esophageal perforation, pulmonary vein stenosis and extensive radiation exposure -- are non-existent in the full Maze procedure.

I was further influenced by the length of time that the Maze procedure has been around. It was developed in 1987 and twenty years of research and practice has resulted in a uniform set of lesions used by all of the top tier Maze surgeons, with the major difference in technique being the energy source used to create the lesions.

I was struck by the fact that while the procedures performed at the top ablation centers have a number of similarities, there are substantial differences in the nature of the ablation procedure, depending on who is performing it. This made me uncomfortable, because it seemed clear that there was not yet uniformity among the EPs as to the best way of doing the procedure. I concluded that more time was needed for the best protocols to be established and adopted by most top EPs, and frankly, I did not want to be one of the case studies that helped them to develop that protocol. (With respect to the modified, or "mini" Maze procedures, although they hold great promise, I believe they are too new to consider right now and, in any event, the success rates are even lower or at best the same as catheter ablations.)

Moreover, I was not particularly impressed with the overall success rate of first catheter ablations (around 70%), with a slightly higher success rate for touch-ups after a first one. On the other hand, when I first met in March of 2006 with the surgeon who later did my Maze, he stated that he personally had had an overall 90% success rate, defined as afib- free, without meds, for at least one year following the surgery. Although he did not have formal data, he expressed the opinion that my age, health and nature of my afib would probably give me an even greater likelihood of success. While this seemed to be an extraordinarily high success rate, I felt confident that he had given me honest statistics after I queried him at length and followed up with my own research. Since my worst nightmare would be to have a procedure done (whether ablation or surgery) and then find that I still had afib, the success rate was a key factor for me.

After careful consideration, I concluded that the full Maze offered me the most time- tested procedure along with the best overall chance of success with a relatively low risk profile.

I chose one of the best Maze surgeons in the world, Dr. Niv Ad who had trained with the founder of the original Maze, Dr. James Cox. Dr. Ad (pronounced "Ahd") has performed more than 500 full Cryosurgical Maze procedures to date. Approximately 200 of those have been performed with his surgical team at Inova Fairfax Hospital in Falls Church, Virginia. Contrary to popular belief, the procedure does not have to be performed using a sternal incision and is far less complex than the original "cut and sew" procedure since the scalpel has been replaced by traditional energy sources, i.e., cryoenergy and bi-polar radiofrequency. Ultrasound and microwave energy are also used. Dr. Ad uses only cryoenergy as he feels it is the safest technology in which transmurality of the lesions can be seen and verified.

Initially I assumed that I would have the minimally invasive Maze procedure, as I had been told by Dr. Ad that I was an ideal candidate for it (no underlying health or cardiac issues, optimal weight, etc.) However, in my initial visit with him, we had an extensive discussion about the pros and cons of doing the Maze using the minimally invasive incision versus a median sternotomy. During that discussion, Dr. Ad told me that a major advantage of a sternal incision is that the time on heart-lung bypass is far less than with the minimally invasive procedure. In addition, he acknowledged the common sense conclusion that the surgeon would have better visualization of the heart with a sternal incision. Finally, he also advised that the sternal incision is generally less painful during recovery than the incision used in the minimally invasive procedure (which is very similar to the one used in the Wolf procedure). However, he also assured me that the full Maze can be performed safely and effectively using the minimally invasive right side incision. Over the next few months, as my afib continued to plague me and I did more research and reading, I started to seriously consider the median sternotomy. When I scheduled my procedure with Dr. Ad, it was with the assumption that I would be having the minimally invasive procedure done, but he let me know that I could change my mind right up until the moment before I went to the OR. Finally, the day before the surgery, I made up my mind that I would opt for the median sternotomy, because I liked the idea that the surgeon would have better visualization, and I particularly was more comfortable with the idea of significantly less time on bypass. When I saw Dr. Ad on the morning of surgery, I told him that I wanted a median sternotomy, and therefore the procedure went ahead in that manner. However, I stress that this was a personal decision, and that the procedure, in all likelihood, would have been just as effective had I elected to have the right side incision instead.

The actual surgery takes about an hour, and I was hospitalized for about four days. I had a few minor, expected and totally controllable complications of the surgery which were dealt with while I was still in the hospital. Because I had traveled from out of town to see Dr. Ad in Virginia, I remained in the area for about a week after I was discharged, just in case of any complications.

Post-operatively, I was fortunate to have the luxury of taking plenty of time to limit my activities, primarily due to the sternal incision. I was restricted from lifting more than 5 pounds, and many other normal daily activities (such as driving, or even sitting in the front seat of a car) had to be put on hold for about 6-8 weeks, although I could have returned to work in two. (With the lesser invasive incision, full activities can be resumed much more quickly.) I experienced some discomfort in the area of my incision for a few weeks, but it was controllable with medications, and, as indicated above, the pain with a minimally invasive incision is often worse because of the cutting of certain nerves. The only pain I experienced was when I coughed or laughed but this went away in about two weeks.

I recently marked the six-month anniversary of my surgery, and I have been afib-free since day one. Up to 50% of patients experience some post-op afib for up to three months after surgery. This is expected and if the procedure has been successful, the afib generally disappears after about three months. I was very fortunate to be free from afib immediately after the surgery, as well as from PACs and other uncomfortable symptoms that had previously been associated with my afib.

Other than fatigue and weakness for about four weeks after the surgery, the recovery has gone very well and I now feel back to normal. I have resumed my cardiovascular and weight training and I am no longer concerned about eating or drinking anything that might trigger an episode.

One of the other advantages of the Maze surgery is the ability of the surgeon to deal with potential causes of afib if discovered during the procedure. In my case, Dr. Ad tested my Ligament of Marshall and, not surprisingly, got a very strong vagal response. This small ligament is vestigial so snipping it does not affect heart function but

eliminates any possible role that it may have in atrial fibrillation. However, deactivation of the Ligament of Marshall can cause a temporary increase in resting heart rate, which I did experience. That has slowly subsided and will probably improve over the next few months, although the current rate is perfectly acceptable and given the vagal nature of my afib, may even be preferable.

The final step in the procedure is closure of the left atrial appendage, which is known to be a primary source of clotting, and hence stroke. Many surgeons opt to remove it entirely, but because there may be some hormonal functions performed by this appendage, Dr. Ad chooses instead to oversew it. I am well aware of the studies showing that lone afibbers are not at any greater risk of stroke than the general population. Nevertheless, if my afib does ever return, I will sleep just a little better at night knowing that a primary source of clotting has been shut down.

I have no doubt that I made an unusual decision to opt for a full Maze, in that most relatively young lone afibbers would probably go for an ablation before considering surgery. However, given the relative risks, options, and likelihood of success, I certainly would encourage any chronic afibber who is considering some form of interventional therapy to at least consider and research the full Maze.

Only a handful of surgeons in this country qualify as top-notch Maze surgeons, and they are primarily located in major metropolitan areas. However, most of these surgeons do not have the extended waiting time that is the usual for top ablation EPs. Shorter waiting time might also be a factor for those whose afib may have taken a sudden turn for the worse and might be looking at a year-long waiting list, with the possibility of a second procedure, in the case of an ablation.

I lived with afib for several years, some worse than others. In the end, I could no longer tolerate the uncertainty and discomfort of this condition as I entered my sixth decade of life. Some of the people closest to me could not understand how I would elect to have what they thought of as “open heart surgery,” when my afib was only intermittent, and probably not life-threatening. However, I was always aware that another episode was just around the corner, and even when it wasn’t, I always felt that it was. I found myself restricting enjoyable activities, modifying my diet in ways that were not acceptable to me (and often didn’t make any difference anyway), and in general, I always felt as though my afib was controlling me, instead of the other way around. Once I met a surgeon whom I felt completely comfortable with and confident in, who was willing to answer my many questions, I felt that a full Maze was the way to go. I went into the procedure knowing that I was taking a risk – a low, but not insignificant one– which I had weighed against the toll that afib was taking on my life. I went ahead, and today, six months later, I am glad I did. It was well worth it.

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