

# THE AFIB REPORT

*Your Premier Information Resource for Lone Atrial Fibrillation!*

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## **Editorial**

*We continue our tabulation of the results of LAFS II. Based on data from 130 paroxysmal afibbers it is clear that the most important triggers for episodes are emotional or work-related stress, alcohol consumption, digestive problems, exercise, specific foods and food additives, fatigue and lack of sleep, caffeine and cold drinks, and exposure to cold temperatures. Fully one third of all vagal afibbers reported that resting or sleeping could precipitate an episode whilst no adrenergic afibbers mentioned sleeping or resting as a trigger.*

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*Our feature article this month questions the use of the term “idiopathic”, that is, of no known cause, to describe lone atrial fibrillation. Dr. Philippe Coumel’s ground-breaking work provided clear proof of an autonomic nervous system connection and more recent research has provided evidence of an association between LAF and inflammation and between LAF and oxidative stress. It is also clear*

*that there is a strong connection, most likely through the autonomic nervous system, between stress and LAF. So it would appear that there are now several plausible causes of LAF. This, of course, makes finding a cure for LAF a whole lot easier. It is very difficult to develop a rational treatment protocol for a disease with no known cause. I believe the digestive connection will turn out to be exceptionally critical. A diagnosed or undiagnosed colon disorder could well be a powerful trigger for AF. In future issues of The AFIB Report we will cover effective approaches to the treatment of colon disorders. Enjoy!*

*Yours in health and sinus rhythm,  
Hans Larsen*

## **Findings from LAFS II – Part 2**

### **Triggers**

The most important trigger of an atrial fibrillation episode was found, as in previous surveys, to be emotional or work-related stress. 46% of the 133 respondents who listed their triggers had stress on their list. It was by far the most important trigger for adrenergic afibbers (94%) and mixed afibbers (56%), but of less importance for vagal afibbers (29%). Overall, the following triggers were most important. Please note that the percentages do not add up to 100% as most respondents listed more than one trigger.

	Vagal	Adrenergic	Mixed	Total
Stress and anxiety	29%	<b>94%</b>	<b>56%</b>	<b>46%</b>
Alcohol	<b>33%</b>	19%	<b>36%</b>	<b>32%</b>
Digestive problems/overeating	28%	6%	33%	<b>27%</b>
Exercise	17%	<b>44%</b>	<b>36%</b>	26%
Specific foods & food additives*	25%	<b>38%</b>	20%	25%
Fatigue/lack of sleep	<b>36%</b>	25%	4%	24%
Caffeine	22%	31%	20%	23%
Resting/sleeping	<b>33%</b>	0%	7%	20%
Cold and cold drinks	7%	6%	11%	8%
Other causes**	22%	25%	33%	26%

\* cheese, spicy foods, rice, chocolate, sugar, wheat, MSG  
\*\* overwork, inflammation, viral infection, dehydration, medications, hypoglycemia, bending over, sleeping on left side, air travel, migraine, sex

It is clear that adrenergic and mixed afibbers in particular could benefit substantially from getting their stress level under control and going easy on the exercise. Vagal afibbers, on the other hand, could probably avoid a fair number of episodes by taking it a bit easier, getting adequate sleep, cutting back on alcohol, and avoiding overeating.

### **Effect of Interventions**

#### **A. Radiofrequency Ablation**

Eight vagal afibbers have undergone radiofrequency ablation. Three of the procedures were successful with the patients completely eliminating their atrial fibrillation and no longer requiring antiarrhythmics to prevent further episodes. The successful procedures were done by Dr. Robert Bock at the Presbyterian Hospital (?), at the Good Samaritan Hospital in San Jose (Dr. Coggins), and at the Cleveland Clinic (Dr. Natale); this last procedure had to be repeated.

Three vagal afibbers underwent ablation for atrial flutter; they also had atrial fibrillation so an attempt was made to eliminate the fibrillation foci at the same time. The flutter ablations were successful in all cases. One of the fibrillation ablations, done at the Virginia Mason Hospital in Seattle (Dr. Chris Fellow), was successful but the patient is still taking beta-blockers to prevent further afib episodes. The remaining two afib ablations were not successful and the patients are still taking antiarrhythmics to prevent episodes. These last two procedures were done at the Seton Medical Center in Austin and the Ottawa Heart Institute in Canada.

Two of the procedures involving vagal afibbers were done within the last six months. One, done at Johns Hopkins (Dr. Calkins), appears to have been at least partly successful although the patient is still on Tambacor. The other has, so far, not been successful and the patient is still on antiarrhythmics.

No adrenergic afibbers reported ablation therapy. It is not clear whether this is because they did not attempt it or because the electrophysiologist could not find an active area to ablate.

Five afibbers with the mixed variety of LAF underwent ablation. Two of these operations were clearly not successful; one was performed at Saint Joseph's Hospital in Tampa, and one at St. Thomas Hospital in London, England. It is too early to tell whether the remaining three were successful, but a least one, done at the Cleveland Clinic (Dr. Natale) appears to have been.

Two chronic afibbers had ablation therapy. One operation, done at the Duke Medical Center (Dr. Marcus Wharton), was successful. The other was done very recently and it is not clear whether it was successful or not.

In conclusion, four of the 15 ablation surgeries reported were completely successful and the patients are off all antiarrhythmics. One procedure was successful, but the patient is still on beta-blockers. Four operations were clearly not successful, and six were done so recently that it is difficult to say whether they were successful. It is not clear whether the lack of success with ablation for atrial fibrillation was due to poor procedure or because there was nothing to ablate (no focal points).

### **B. Maze Procedure**

Two afibbers with mixed LAF had undergone maze surgery. Both operations were completely successful and the patients are no longer taking antiarrhythmics. The procedures were performed at the Cleveland Clinic (Dr. Patrick McCarthy) and at St. Joseph's Hospital (Dr. Enrique Cuenza Lopez).

### **C. Amalgam Replacement**

The question as to whether afibbers without amalgam dental fillings have fewer or less severe episodes than do those with amalgam fillings is not clear. One major reason for this is the difficulty in sorting out the individual effects of age, drug use, and other preventive treatments with that of having amalgam fillings.

Nevertheless, there does not appear to be any significant amalgam-related difference in number and severity of episodes as far as adrenergic and mixed afibbers are concerned. There is, however, an indication that vagal afibbers with amalgam fillings have more episodes (median of 4 versus 2 per 6 months) and spend more time in fibrillation per month (median of 5 hours versus 1.7 hours for those without amalgams). This possible effect needs to be confirmed in a detailed correlation analysis.

A total of 10 people (6 vagal, 3 mixed, 1 adrenergic) with paroxysmal LAF have had their amalgam fillings replaced. Five of them underwent proper detoxification while four did not. The detoxification status of the remaining person is unknown. Detoxification did not seem to affect the number of episodes, but it did affect their duration. The average time spent in fibrillation for the detox group was 3 hours per month versus 21 hours for those who had not detoxified. Considerable caution is needed though in interpreting these numbers since the sample size is very small.

Subjectively, some people felt that amalgam removal had been highly beneficial whilst others had observed no benefits. Here are their comments:

- "I have had no afib episodes since removal of my amalgam fillings and gold alloy bridges in December 2000. I would absolutely recommend this procedure to other afibbers provided it is carried out by an outstanding holistic dentist and is followed up by complete detoxification. Watch out for residual amalgam under old bridges and crowns." – vagal afibber
- "I have had no episodes since June 2001 when I had all my amalgam (silver) fillings replaced. I am still undergoing detoxification, but hope to stop this once my mercury levels are back to normal. I feel the replacement has been beneficial." – vagal afibber
- "I had all my amalgam fillings replaced in 1992, but did not undergo detoxification. I still have dissimilar metals in my mouth. I have had no episodes in the past 6 months, but do not believe that this is due to the replacement. Nevertheless, I would, for other reasons, recommend replacement." – vagal afibber
- "I had all my amalgam fillings replaced in 1995 and followed up with chelation therapy. I did not find the amalgam replacement beneficial." – mixed afibber
- "I had all my amalgam fillings replaced in 1997 and am now doing detoxification. I feel this was beneficial and would recommend the procedure to other afibbers." – mixed afibber
- "I had all my amalgam fillings replaced in 2000 followed by detoxification. I have not noticed much difference in the number and severity of episodes." – mixed afibber

- “I had all my amalgam fillings replaced in the latter part of 2001. I did not follow up with detoxification and am not sure the replacement has made any difference.” – vagal afibber
- “I had my fillings replaced in September 2000. No detoxification though. The frequency of episodes did not decrease, but the duration did. I think the procedure was beneficial and would recommend it to other afibbers.” – vagal afibber
- “I had my fillings replaced in November 2001. I am not sure yet whether it has decreased my episode severity. So I cannot yet recommend the procedure for atrial fibrillation relief, but I am sure my general all round health will be improved.” – vagal afibber
- “I had all my fillings replaced in July 1999 and followed up with partial detoxification – not long enough though to bring my mercury levels to normal. I don’t think the removal helped a lot.” – adrenergic afibber

So, it’s a fairly mixed bag of results. Amalgam replacement and detoxification would likely have overall health benefits including a reduction in the risk of developing neurodegenerative diseases such as Alzheimer’s and Parkinson’s. However, it is not clear that it should be the first measure taken against lone atrial fibrillation unless you have proven toxic levels of mercury in your body and/or are especially sensitive to mercury. In any case, amalgam replacement should always be performed under strictly controlled conditions and followed up by thorough detoxification. See the July 2001 issue of The AFIB Report for more details.

#### **D. Anti-inflammation Protocol**

Four people had tried the anti-inflammatory protocol (The AFIB Report, September 2001). Here are their comments:

- “When I started the anti-inflammation protocol both my allergies and my adrenergic afib disappeared and I was able to work out again. But it seems like when you think it’s gone it comes back. Now I have vagal instead; it comes on right after a meal. I do believe the protocol helped though.” – vagal/mixed afibber
- “It certainly helped for the first couple of months and according to my C-reactive protein test I no longer have an inflammation. It did not completely eliminate my afib episodes.” – adrenergic afibber
- “I started the anti-inflammation protocol in September 2001. It has definitely helped and I would recommend it to other afibbers.” – mixed afibber
- “I started the protocol in December 2001 using just the Moducare. I have not had an episode since so I think it would be worth a try.” – vagal afibber

I would conclude that the anti-inflammation protocol (published in the September 2001 issue of The AFIB Report) is worth a try if a C-reactive protein (CRP) test shows a higher than normal value indicating the presence of an inflammation.

*That’s all for now. In the next issue we will begin the evaluation of the effectiveness of antiarrhythmic drugs in the prevention of LAF episodes.*

## **Is LAF Really Idiopathic?**

It is becoming increasingly clear that calling atrial fibrillation with no underlying heart disease “idiopathic”, i.e. of no known cause, is no longer appropriate. Recent research has uncovered at least three conditions that can initiate and promote atrial fibrillation in people free of heart disease. Inflammation, oxidative stress, and a dysfunctional autonomic nervous system (ANS) have all been found to be potential causes of atrial fibrillation (AF). Emotional stress has also been linked to the initiation of AF, but may act through its effect on the

autonomic nervous system (heightened adrenergic tone), the immune system (inflammation) or by creating additional oxidative stress.

### **AF and Oxidative Stress**

The role of oxidative stress was discussed in the March 2002 issue of The AFIB Report. I have since come across two articles that further support the idea that oxidative stress is involved in atrial fibrillation. Researchers at the Cleveland Clinic and the Ohio State University have found that AF patients show signs of extensive oxidative injury to their myofibrillar creatine kinase (MM-CK). MM-CK controls the contraction of individual heart cells (myocytes). The researchers also determined that the oxidative damage was caused by peroxynitrite, a highly potent free radical. They conclude that peroxynitrite-induced oxidative stress can damage individual heart cells to such an extent that their normal function is disrupted and atrial fibrillation results[1].

The Cleveland Clinic researchers later followed up these initial findings by an experiment designed to show whether reducing the level of peroxynitrite through the use of an antioxidant (vitamin C) would prevent surgery-induced atrial fibrillation. Their clinical trial involved 50 bypass surgery patients who were given 2 grams of ascorbic acid (extended release) the night before surgery, followed by 500 mg doses twice daily for 5 days after surgery. The incidence of postoperative AF in the vitamin C group was 16.3% as compared to 34.9% in a comparable group not given vitamin C. The researchers conclude that AF episodes are sustained by oxidative stress and increased peroxynitrite generation caused by the rapid heart rate[2].

The conclusion is clear, oxidative stress is involved in both the initiation and maintenance of AF and supplementation with vitamin C, a potent antioxidant, helps prevent AF. I personally believe that gamma-tocopherol and lycopene are as effective, if not more effective than vitamin C, in neutralizing peroxynitrite radicals. Gamma-tocopherol and lycopene are both fat-soluble antioxidants found in the membranes of cells whereas vitamin C is water-soluble and found in the watery part of the cell. For optimum prevention of AF a combination of the 3 antioxidants is required.

### **Inflammation**

The role of inflammation was discussed in the September 2001 issue of The AFIB Report. The idea that an inflammation of the heart lining can cause AF has since been supported by work done at the Cleveland Clinic and the University of Athens (The AFIB Report, February 2002). Both groups of researchers found a clear association between the level of C-reactive protein (CRP), a marker for inflammation, and the presence and severity of lone atrial fibrillation[3,4].

The junctions of the pulmonary veins and the left atrium are prime sites for the initiation of LAF. Creating a physical barrier between these sites (foci) and the rest of the heart via radiofrequency or ultrasound ablation is now accepted as a highly effective means of preventing LAF[5]. The origin of the offending foci is not known, but it seems plausible that they could be sites of local inflammation.

Systemic inflammation can be successfully controlled with prednisone. This drug, however, has many serious adverse effects and is not recommended for extended use. There are also several natural supplements (The AFIB Report, September 2001) that are quite effective in combating inflammation. Giving them a try is worthwhile if an inflammation (high CRP level) is present.

### **Autonomic Nervous System Dysfunction**

The heart begins beating on its own in the early embryonic stage and continues to do so until death. The nervous system develops at a later stage and extends its control to the heart primarily through its connection with the sinoatrial node. Both sympathetic (adrenergic) and parasympathetic (vagal, cholinergic) nerves are involved in heart rate control. It must be emphasized that the heart is perfectly capable of beating on its own without any help from the ANS. Transplanted hearts have no ANS connection and yet keep beating just fine. The autonomic nervous system basically serves as an override-control mechanism that helps the heart and circulatory system adjust to changes in the external or internal environment. A classic example of this is the fight or flight reaction where a whole cascade of body changes, including an accelerated heart rate, is "ordered" by the ANS to meet an actual or perceived threat.

The main body functions controlled by the ANS are vision (pupil dilation), breathing, heart rate, skin temperature, and digestion. The ANS is basically a two-way communication system with the control centers for the different organs (pupils, lungs, heart, blood vessels, digestive tract and liver) both receiving input from and sending instructions to the individual organs. The nerve fibres transmitting information about the status of an organ to its control center are called afferent fibres whilst those that transmit instructions for actions are called efferent. The two types often run alongside each other and both terminate in the control centers.

Maintaining a blood pressure sufficient to ensure an adequate blood supply throughout the body but low enough to avoid bursting small capillaries in the brain is perhaps one of the most important tasks of the ANS. The cardiac control center of the ANS constantly receives input from baroreceptors. These specialized muscle fibres are located in the walls of the heart and the major arteries and they “measure” the blood pressure by stretching and relaxing as the blood flows past them. A lower than desired pressure will cause the ANS to activate the sympathetic nervous system and thus make the heart beat faster and the blood vessels constrict whilst too high a pressure will activate the parasympathetic system slowing down the heart and dilating the blood vessels.

The override mechanism of the ANS usually functions flawlessly and most people are not aware that it even takes place. However, if the adrenergic and vagal inputs to the heart become unbalanced atrial fibrillation can result. There is ample evidence that AF episodes are preceded by an abnormally high level of either adrenergic (sympathetic) or vagal (parasympathetic) activity[6,7]. It is not clear though whether these disturbances cause AF on their own or whether an inflammation or excessive oxidative stress is also required in order to initiate and sustain AF.

Sorting out the relative “contributions” of inflammation, oxidative stress and ANS dysfunction to atrial fibrillation is actually quite difficult. Oxidative stress and inflammation are closely linked as is inflammation and ANS dysfunction. Nevertheless, it may prove fruitful to take a closer look at the ANS/heart connection.

The heart muscle itself and the sinoatrial node, in particular, are supplied by both sympathetic and parasympathetic postganglionic nerve fibres. The sympathetic (adrenergic) speeds up the heart beat whilst the parasympathetic (vagal, cholinergic) fibres slow it down. The sympathetic nerve fibres originate in the spinal cord whilst the parasympathetic fibres originate in the brain stem (medulla oblongata) as the 10<sup>th</sup> cranial or vagus nerve. Some very recent research has shown that the actual control center for the heart is located in the medulla oblongata right in the area where the vagus nerve terminal is located[8]. This finding may have far reaching consequences and may provide an important clue as to the origin of the ANS dysfunction that helps precipitate AF.

### **The Digestion Connection**

The digestive system, from initial saliva generation, through the generation of digestive enzymes and gastric juice, nutrient absorption in the small intestine, and final defecation, is controlled by the vagus nerve with minor input from the sympathetic branch of the ANS. Could disturbances in the digestive system somehow translate into heart rhythm problems through the common factor of the vagus nerve? Recent research would indicate that there could indeed be a connection. Argentine researchers have discovered a strong association between the presence of diverticular disease (diverticulosis) of the colon and vagal atrial fibrillation. They performed barium enema examinations on 16 patients under the age of 50 years with documented vagal AF and found that 14 (87.5%) of them had diverticulosis. This compares to an incidence of 5% among the general population. The researchers conclude that there is a strong association between vagal AF and diverticular disease of the colon[9].

I asked Dr. Vladimir Shusterman, a member of the team which discovered the exact location of the “cardiac control center”, the question, “Could your finding, that the “cardiac control center” is located very close to the vagus nerve, explain the recently discovered association between diverticular disease of the colon and vagal atrial fibrillation? In other words, is it possible that digestive problems could cause feedback that would activate the stimulation point you discovered and thus cause heart rhythm irregularities?” Dr. Shusterman replied, “The idea that intestinal problems can lead to the activation of vagus, which, in turn, would activate a cascade of central responses perturbing cardiac rhythm makes a lot of sense”[10].

The possibility that digestive tract problems can cause ANS disturbances ultimately leading to atrial fibrillation is indeed an intriguing one and opens up a whole new perspective on the prevention of LAF.

Many afibbers, particularly those with the vagal variety, have episodes shortly after eating their evening meal. It is as if the mere mobilization of the digestive system is enough to "irritate" the vagus nerve to such an extent that the feedback causes the heart rhythm to become irregular. Some vagal afibbers have found that taking pancreatic enzymes before dinner helps in avoiding this type of episode. The body's own production of pancreatic enzymes is entirely controlled by the vagus nerve. This means that if more enzymes are called for a more active vagal response is required. The effect of this higher vagal tone probably extends to the heart and could conceivably result in the initiation of a vagal episode. Supplementing with pancreatic enzymes would reduce the need of the pancreas to produce them and might therefore reduce vagal tone. Add to this the proven anti-inflammatory effects of pancreatic enzymes and it is clear that they could be a very important supplement for lone afibbers. Cotazym and Zypan are two effective pancreatic enzyme preparations.

Another good remedy for an "over-excitabile" digestive system is peppermint oil. This essential oil seems to calm the intestines and prevent spasms in the colon. It needs to be taken in the form of enteric-coated capsules in order to reach the intestines intact. I have found that taking 2 capsules about an hour before dinner is very effective.

Quite apart from diverticular disease there is a whole raft of digestive system disorders, which are associated with an ANS disturbance either directly or through the intermediary of an inflammation or the formation of excessive amounts of free radical (oxidative stress).

### **Food sensitivities and allergies**

Several researchers have found a link between food sensitivities/allergies and an abnormal ANS reaction to the offending foods. Japanese researchers found that the ingestion of allergic foods increases the parasympathetic (vagal) response[11-13]. Many people are allergic to wheat and gluten-containing products or dairy products. Some afibbers have experienced significant improvement by totally eliminating these foods from their diet for a 2 to 3 week period.

### **Indigestion (dyspepsia)**

It is often caused by an inflammation linked to a Helicobacter pylori infection. However, there is also evidence that an ANS dysfunction can be involved. Brazilian researchers recently reported that a third of patients with functional dyspepsia had impaired vagal function and that the majority of these patients also showed abnormalities in cardiovascular reflexes[14].

### **Gastritis**

Gastritis or inflammation of the stomach lining is also frequently linked to an infection caused by Helicobacter pylori and involves a great deal of oxidative stress[15]. Russian researchers have reported a strong association between chronic gastritis and abnormalities in the functioning of the ANS[16].

### **GERD (gastroesophageal reflux disease)**

GERD, also known as heartburn, is extremely common in the Western world. Recent research has shown that it is associated with both an ANS dysfunction and an excessive generation of oxygen-based free radicals (oxidative stress)[17-19]. Several afibbers have reported that they suffer from GERD and at least one has found that controlling the disorder with the drug Nexium (esomeprazole) helps reduce the number of AF episodes.

### **Irritable bowel syndrome**

IBS, another very common ailment in the Western world, has been linked to an ANS dysfunction specifically an enhanced vagal response[20-24]. Several afibbers have reported that their AF episodes tend to be more frequent when they experience a flare-up of their IBS. Recent research has established that many people with IBS often suffer from celiac disease as well. This makes it absolutely imperative to stay away from wheat and other gluten-containing products[25]. Enteric-coated peppermint oil capsules have been found highly effective in reducing the pain often associated with IBS[26].

### **Inflammatory bowel diseases (Crohn's disease and ulcerative colitis)**

These conditions involve an inflammation of the colon as well as a dysfunction of the ANS[27]. In 1993 Swedish researchers reported that patients with Crohn's disease had a sympathetic system (adrenergic) dysfunction whilst those with ulcerative colitis had a primarily vagal dysfunction[28]. It is also clear that an oxidative stress mechanism is involved. Crohn's disease and ulcerative colitis patients have been found to have impaired antioxidant defenses and free radical- induced DNA damage[29,30]. There is also evidence that ulcerative colitis by itself generates reactive oxygen species[31].

### **Conclusion**

There is substantial evidence that digestive disorders are associated not only with inflammation and oxidative stress, but also with autonomic system dysfunction. It is plausible that the ANS dysfunction could be a factor in initiating AF episodes. In future issues we will take a look at how digestive disorders can be effectively managed and will also devote considerable space to exploring the connection between LAF and emotional stress.

I believe that it will ultimately be possible to reduce the frequency of LAF episodes by avoiding the triggers, eliminating inflammation, excessive emotional stress and oxidative stress, and by paying close attention to the digestive connection. It is clear that the heart "remembers" episodes and that the more frequent they are the more likely it is that another one will occur. Professor Michael Rosen, MD at Columbia University recently discovered that the heart remembers arrhythmias for at least a month and is more prone to go into arrhythmia for as long as the memory lasts[32]. So the goal should be to avoid episodes for one or, even better, two months so as to completely erase the memory of the last episode. Nevertheless, I do believe that "eternal vigilance" is going to be required to remain afib-free.

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## **AFIB News**

**AF linked to colon disorder.** Argentine researchers have discovered a close association between vagal atrial fibrillation and the presence of diverticular disease (diverticulosis). Their study involved 16 vagal afibbers under the age of 50 years who underwent barium enema examinations to check for colon problems. The researchers found that 14 out of the 16 (87.5%) had diverticular disease. This incidence is far higher than the 5% rate found in the general population under 50 years of age. They warn that their findings have significant implications in regards to anticoagulation (warfarin) therapy for vagal afibbers. Diverticular disease is commonly associated with enteric bleeding in the colon (10-30% of all patients). The bleeding risk would clearly be increased by warfarin. They conclude that vagal afibbers should be examined for diverticular disease before they are put on warfarin (Coumadin).

*American Journal of Cardiology*, Vol. 89, February 15, 2002, pp. 475-77

**Magnesium depletion linked to chronic stress.** Yugoslavian researchers report that young men exposed to chronic stress (military duties, political intolerance, and permanent stand-by duty) or everyday mortal danger have significantly decreased magnesium levels and a high degree of oxidative stress. They conclude, "These findings support the need for magnesium supplementation and antioxidant vitamins for people living in conditions of chronic stress."

*Magnesium Research*, Vol. 13, No. 1, March 2000, pp. 29-36 [abstract]

**Rate control best for chronic afibbers.** Cardiologists have long debated whether it is best for afibbers to stay in fibrillation and just take beta or calcium channel blockers to keep their heart rate down (rate control) or whether it is best to attempt to convert them to sinus rhythm and maintain them in sinus rhythm through the use of antiarrhythmic drugs (rhythm control). The recently released results of the Atrial Fibrillation Follow-up

Investigation of Rhythm Management (AFFIRM) trial clearly indicates that rate control is at least as effective, if not more effective, as rhythm control. The AFFIRM trial involved 4060 AF patients with a mean age of 69.7 years. 39% of them were women, 71% had a history of hypertension, and 38% had coronary artery disease. Half the patients were randomised to rate control plus anticoagulation whilst the other half was randomised to rhythm control plus anticoagulation. After 5 years of follow-up the mortality in the rate control group was slightly lower than in the rhythm control group. The researchers conclude that it is time to accept rate control as the primary strategy in the treatment of AF. NOTE: Although the AFFIRM trial did not include lone afibbers it is likely that its conclusions are equally valid for afibbers without structural heart disease.

*Circulation, Vol. 105, No. 12, March 26, 2002, pp. e9085-90*

**Pulmonary vein isolation is safe and effective.**

Cardiologists at the University of Michigan have just released a comprehensive report on the effectiveness of pulmonary vein isolation. This procedure is a variant of radiofrequency ablation and involves the creation of a ring of scar tissue in the left superior, left inferior and right superior

pulmonary veins. Electrophysiological studies have shown that these 3 veins are the most likely sources of the ectopic beats that generate AF. By isolating them from the left atrium via the scar tissue AF is prevented. The cardiologists treated 58 patients with paroxysmal AF and 12 with chronic AF. Most (93%) had lone AF (no structural heart disease). Five months after the procedure 70% of the patients with paroxysmal and 22% with chronic AF were completely free of episodes whilst another 13% of paroxysmal afibbers had improved significantly. The researchers point out that other studies have found that most (85-95%) AF episodes are triggered from foci located in the pulmonary veins. They also conclude that pulmonary vein isolation is usually not effective for chronic AF indicating that the offending foci are located in the atrium rather than in the pulmonary veins. Of particular interest to afibbers contemplating ablation is the fact that the researchers found no correlation between left atrial size and the success of the procedure. Left atrial sizes of the patients varied between 30 and 57 mm. This would indicate that it is not so much left atrial size, but rather the overall structural soundness of the heart that determines the success of ablation.

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