

THE AFIB REPORT

Your Premier Information Resource for Lone Atrial Fibrillation!

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In this issue we complete the evaluation of the responses to our 3rd LAF survey (May 2002). The most interesting finding is that hypothyroidism (underactive thyroid gland) could possibly be an underlying cause of LAF just like hyperthyroidism (overactive thyroid gland) is known to be. It would also appear that eating a large meal is a frequent trigger for afib episodes.

We also discuss the various medical therapies available for the treatment of LAF. Pharmaceutical drugs and implantable pacemakers/defibrillators are used to control LAF, but they do nothing to cure it. The maze procedure and radiofrequency ablation do cure the disorder, but ablation has not yet been perfected to the point where success can be guaranteed. There are also still some questions regarding the risk of stenosis inherent in some of the variants of the ablation procedure. AV node ablation does help control the symptoms of LAF, but does not eliminate it and necessitates long-term warfarin therapy.

The quest for a safe, effective and affordable cure for LAF is, by no means, over so the mission of "The AFIB Report" and the Bulletin Board remains intact – to discover and disseminate suggestions for alternative preventive or curative approaches while closely following relevant developments in the medical world.

*Yours in health and sinus rhythm,
Hans Larsen*

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Findings from LAFS III – Part 3

In this issue we present the final results of the May 2002 questionnaire.

31. Have you ever been diagnosed as having a high CRP (C-reactive protein) level and if so, what was the level?

Only 5 (7%) of the 75 respondents who answered this question had been diagnosed with a high CRP value. The values considered high were 0.6, 1.0, 2.2 and 4.0 mg/L. The 5 respondents were more likely to have taken Valium and to have had a dysfunctional childhood, but these observations must, due to the small sample size, be taken with a very large grain of salt. It is also not clear whether the respondents who answered no to this question did so because they had never had their CRP level tested or because their level was low. So from this data it is not possible to deduce whether high CRP levels are associated with atrial fibrillation.

32. What is your basal body temperature?

Twenty-two respondents answered this question. Nine (41%) had a normal value (97.6 - 98.2 degrees F or 36.4 - 36.8 degrees C), 10 (45%) had a low value and the remaining 3 had a high value.

Of the 10 with low values 4 had actually been diagnosed with hypothyroidism; their basal temperatures were 96.5, 97.0, 97.1 and 97.3 degrees F respectively. The 6 undiagnosed respondents with low values had values of 96.4, 96.8, 97.0, 97.1, 97.2 and 97.3. This may indicate that the 6 respondents with low values also have hypothyroidism. I would urge them to have it checked out.

The 3 respondents with high values had basal temperatures of 98.6, 98.8 and 100.0 degrees F. This may indicate an overactive thyroid gland (hyperthyroidism) and again should be checked out. Hyperthyroidism is a recognized trigger of atrial fibrillation and it is possible, but to my knowledge yet unproven, that hypothyroidism could also be a trigger.

Four of the respondents had no data for episode severity so was left out of the following analysis. The remaining 18 respondents consisted of 2 adrenergic, 8 mixed, 6 vagal and 2 permanent afibbers. The 16 paroxysmal afibbers were separated into 3 groups – those with low basal temperatures, those with normal and those with high. There was a clear difference between the 3 groups in the number of episodes over the 6-month study period. The normal group had an average of 4 episodes lasting 7 hours and spent 29 hours in fibrillation. The low temperature group had an average of 8 episodes lasting 7 hours and spent 53 hours in afib. The high temperature group had an average of 9 episodes lasting 22 hours and spent 249 hours in fibrillation. The 2 permanent afibbers both had abnormally low basal temperatures.

This analysis implies that both low (hypothyroidism) and high (hyperthyroidism) basal temperatures worsen the severity of LAF. Unfortunately, the analysis was only based on 18 data points so it must be viewed with considerable caution. Nevertheless, the result is intriguing and certainly worthy of further investigation.

33. Have you noticed any correlation between your diet and the severity of your episodes?

Forty-eight (47%) of the 102 respondents had noticed a correlation (52% vagal, 48% mixed, 41% permanent and 33% adrenergic). The most common factors that seemed to initiate or prolong an episode were:

- A large meal reported by 44%
- High glycemic index food reported by 10%
- Eating as such reported by 10%
- Alcohol reported by 8%
- MSG reported by 8%
- Starchy meal reported by 5%
- Salmon reported by 5%

Gluten, salt and a fatty meal were each mentioned by one respondent. There was no indication that afibbers who had noticed a correlation between their diet and afib had more or fewer episodes or shorter or longer ones than fibbers who had not noticed a correlation. Afibbers who had noticed a dietary correlation were also more likely to have been diagnosed with a bowel disorder or GERD.

35. Do you have rheumatoid arthritis, fibromyalgia, chronic fatigue or thyroid problems?

Sixteen (15%) of 105 respondents (21% mixed, 17% adrenergic, 13% vagal and 11% permanent) reported that they had one of the above disorders. Six had thyroid problems (4 specified hypothyroidism), 4 fibromyalgia, 3 osteoporosis, and 1 each had arthritis or fatigue. Osteoporosis, depression, and irritable bowel syndrome have all been linked to elevated cortisol levels while low levels have been linked to rheumatoid arthritis, fibromyalgia and chronic fatigue syndrome[1-7]. It is thus possible that these disorders could affect LAF severity through the cortisol connection, but there was no statistically significant indication that afibbers with these disorders have more or less episodes than those without. There is also no indication that any of the above disorders, with the exception of thyroid problems, could be an important underlying cause of LAF.

37. Were you brought up in a dysfunctional home (alcoholism, frequent parental fights, etc.) or were you physically or sexually abused as a child?

At least 2 studies have found that adverse conditions that produced frequent elevated cortisol levels in childhood may contribute to low levels in adulthood and that these low levels may make the adult hypersensitive to stress and thus a good candidate for LAF[8,9].

Twenty-six (25%) of 104 respondents (28% mixed, 28% vagal, 25% adrenergic and 12% permanent) reported that they had been brought up in a dysfunctional home. Is this high when compared with the general population? I have been unable to find out, but my guess would be that it probably is not. There was no indication that being raised in a dysfunctional home affected LAF severity, however, it did affect other parameters. Afibbers brought up in a dysfunctional home were more likely to be women. They were also more likely to smoke, have a bowel disorder, have been on Valium, and have a high CRP level. Quite a legacy!

Conclusions from LAFS III

This completes our evaluation of the results of the LAFS III (May 2002) survey. It has certainly turned up some interesting clues, but most of them will need to be verified by larger studies with matched, afib-free controls. Our 3rd LAF survey confirmed the main findings of the previous two surveys.

- The vagal variety of LAF is the most common followed by mixed, adrenergic and permanent.
- Vagal afibbers tend to develop LAF at a slightly younger age.
- Fifty per cent of lone afibbers have their first episode between the ages of 40 and 55 years. Only 7% are diagnosed at age 65 or older.
- Women tend to be diagnosed with LAF at a later age than men and tend to have less severe episodes.
- Episode severity tends to increase with age and number of years since diagnosis.
- About a quarter of the respondents have an enlarged atrium (42-50 mm), but this was not associated with increased episode severity.
- Bowel disorders and GERD (gastroesophageal reflux disease) are common among afibbers. Flare-ups of GERD could be an important trigger of LAF.
- The prevalence of previous tonsil removal was considerably higher among the survey respondents than would have been expected. It is thus possible that tonsillectomy could predispose to the later development of LAF; this, however, needs to be confirmed in a much larger study comparing age and sex-matched afibbers with afib-free controls.
- Over 90% of the respondents considered themselves highly or somewhat physically active.
- Cardiac arrhythmias were more common among close relatives of afibbers than would be expected. However, a much larger study involving afib-free controls is needed to confirm or disprove a possible genetic connection.
- There is some indication that hypothyroidism (low basal temperature) could be an afib trigger just as hyperthyroidism is. Again, this needs to be confirmed in a larger survey.
- Almost half of all respondents had noticed an association between their diet and LAF episodes. A large meal was the most common trigger.
- About a quarter of all respondents had a dysfunctional childhood. This did not significantly affect episode severity, but did have an impact on several other parameters.

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Medical Therapy for LAF

Medical intervention in lone atrial fibrillation is aimed at preventing episodes, ameliorating the symptoms of episodes, converting the fibrillation to normal sinus rhythm (NSR), and reducing the risk of stroke. With the exception of surgery (the maze procedure) and catheterization (radiofrequency and ultrasound ablation) medical intervention is not designed to eliminate (cure) the disorder, but rather to control (manage) it over the long-term.

Lone atrial fibrillation is a chronic disorder like diabetes or arthritis rather than an acute disorder like the flu or a bout of pneumonia. It comes in three "flavours" – paroxysmal, persistent, and permanent. Paroxysmal AF converts to normal sinus rhythm on its own and episodes last less than 7 days (most less than 24 hours); persistent AF episodes last more than 7 days, but cardioversion is effective in conversion to NSR; permanent AF is permanent and does not respond to cardioversion.

There is substantial evidence that "AF begets AF"; in other words, the more frequent and longer lasting episodes are the more likely they are to become even more frequent and longer lasting. This process appears to be reversible at least for paroxysmal and persistent AF[1]. Most afibbers can agree that "AF begets AF", but I want to emphasize that many lone afibbers have been able to reverse the trend and actually have fewer episodes now than they experienced a year or two ago. So the remodelling of the heart leading to more and longer episodes is definitely reversible in paroxysmal LAF. Whether permanent LAF is reversible through lifestyle and dietary modifications is not known.

Researchers at the University of Bologna have categorized the various stages of remodelling and shown that they depend on the duration of the episode. Short-term episodes (seconds/minutes) lead to minor changes involving the myocyte ion channels. Medium-term episodes lasting hours or days cause altered gene expression and protein synthesis while very long-term episodes (months/years) cause irreversible structural damage, fibrosis and enlargement of the atrium[2].

Prevention of episodes can be accomplished, albeit not very successfully, by pharmaceutical drugs or by the use of implantable pacemakers. Symptom relief during an episode can be obtained through the use of drugs and conversion to normal sinus rhythm can be achieved either chemically (with drugs) or by exposing the heart to an electrical shock at the appropriate moment. Stroke prevention is achieved through the use of antiplatelet (aspirin) or anticoagulation (warfarin) agents or by physically blocking off the left atrial appendage of the heart where blood clots may form during an extended (greater than 48-hour) fibrillation episode[3].

Undergoing the maze procedure or having ablation therapy eliminates the need for all of the above as the LAF is no longer present once these procedures have been successfully completed.

Pharmaceutical Drugs

Pharmaceutical drugs are used for four purposes - to prevent episodes, to ameliorate symptoms during an episode, to achieve conversion to NSR, and to prevent stroke. Antiarrhythmics are used to prevent episodes, beta and calcium channel blockers to ameliorate symptoms, ibutilide (Corvert), dofetilide (Tikosyn), flecainide (Tambocor), and propafenone (Rythmol) to convert to NSR, and aspirin or warfarin (Coumadin) to prevent stroke.

Chemical conversion to NSR usually takes place in the hospital and involves infusion of one of the above-mentioned drugs. It is most effective if started within a couple of hours of the onset of the episode. Anecdotal evidence reported on the afibbers.org bulletin board indicates that "at-home" conversion with flecainide or propafenone can also be quite effective[4,5]. The procedure involves crushing either flecainide tablets (200 mg) or propafenone tablets (225 mg) and then swallowing them with a glass of lukewarm water. The procedure works best, or perhaps only, if it is performed within 5 minutes of the onset of an episode. Both flecainide and propafenone are very powerful and potentially fatal drugs so the "on-demand" approach should only be attempted with a doctor's approval.

The efficacy of antiarrhythmics has been discussed in several issues of The AFIB Report[6-9]. They are generally not effective for preventing or shortening episodes except in the case of vagal afibbers where

flecainide and disopyramide (Norpace) are often effective. Amiodarone (Cordarone) can also be effective, but its side effects are so serious that the medical profession considers it the “drug of last resort”[10]. Beta-blockers and calcium channel blockers like verapamil (Verelan, Calan) and diltiazem (Cardizem, Tiazac) are quite effective in ameliorating symptoms during an episode by lowering the heart rate. Aspirin and warfarin (Coumadin) are somewhat effective for stroke prevention, but as discussed in The AFIB Report are not necessary for many lone afibbers[11,12].

Implantable Pacemakers/Defibrillators

Pacemakers have long been used to assist ailing hearts in maintaining a steady beat. The most common reason for implanting a pacemaker is because the heart’s own pacemaker (the sino-atrial node) has become too slow because of heart disease, age or the use of heart medications. Pacemakers are also used to provide a regular electrical impulse directly to the ventricles in case the AV (atrioventricular node) is blocked. A pacemaker can only increase the heart rate; it cannot slow it down[13-16].

The pacemaker consists of a small metal housing containing a battery and a small computer and is connected to one or two leads (wires) that are threaded through a vein beneath the collar bone and positioned in the heart using an x-ray monitor. The pacemaker itself is placed in a pocket beneath the skin just below the collar bone. The entire installation procedure takes about an hour or two and is performed under local anaesthesia. About 600,000 new pacemakers are installed worldwide every year[13]. The pacemaker is powered by a lithium battery the lifetime of which can be as much as 10 years for a very simple pacemaker, but a little as 3 years for a highly sophisticated one. Battery replacement requires renewed surgery. The implantation can result in complications such as blood clot formation and complications caused by dislodgement of the leads (1.6-3.8%)[13,14,17].

From its original application as a means of supporting ailing hearts, the pacemaker has now emerged as a viable means of preventing cardiac arrhythmias. Their first application was in the prevention and termination of potentially fatal ventricular arrhythmias, but they are now also used in the prevention of atrial fibrillation. Some later models also have the capability of terminating fibrillation episodes by rapid bursts of pacing or by delivering an internal shock to the heart at much lower energy levels than what is required for the conventional external cardioversion procedure. These new devices are called implantable cardioverter-defibrillators or ICDs and are highly sophisticated and expensive electronic devices[15,16].

A typical, modern ICD such as the Medtronic AT500 DDDR Pacing System acts as a miniature Holter monitor, has electronic circuitry that will provide precisely-timed impulses to the atria, thus preventing afib from beginning and if an episode does start has the capability to terminate it[18]. The ICD works in two ways to prevent arrhythmia. It continuously senses the heart beat and if it drops below a certain rate transmits electrical impulses to one or more electrodes implanted in the heart and connected to the ICD. This speeds up the heart beat and prevents bradycardia (dangerously slow heart beat) and the subsequent compensatory atrial fibrillation. This approach would work particularly well for vagal afibbers whose episodes are almost always preceded by bradycardia.

The ICD also continuously senses the heart rhythm and quickly notices the occurrence of PACs (premature atrial complexes) that often are a prelude to an afib episode, particularly among mixed and adrenergic afibbers. When the ICD senses PACs it speeds up the heart rhythm in a step-wise fashion until the PACs no longer appear. It then slowly reduces the heart rate to its original level and leaves the heart to do its own pacing again[17,19-21].

Modern ICDs are programmable just by holding a special wand in front of them and the built-in Holter monitor, which can store lengthy heart beat records, can be read in the same way. They are indeed highly sophisticated devices and, as such, take extremely competent cardiologists to install and program.

The only question that matters to afibbers is do they work? The jury is still out on this. Some studies have found them reasonably effective, especially for vagal afibbers while others report rather disappointing results[1,17,20,21]. A trial carried out at the Mayo Clinic found that pacing at two sites in the atrium was more effective than pacing at just one site and that both methods were somewhat more effective than no pacing in increasing the time space between afib episodes in a group of 9 patients with paroxysmal AF. The pacing also

resulted in a very substantial decrease in the number of PACs observed during 24-hour Holter monitoring before and after pacemaker implantation[22].

Atrial pacing can be, and often is, combined with treatment with antiarrhythmic drugs. One trial of this approach reported a reduction of the incidence of afib episodes of about 34% when dual-site pacing was combined with drug treatment – not terribly impressive[15].

The use of the defibrillation feature is also problematical. Depending on electrode size and position the electrical energy required to shock the heart back into sinus rhythm might produce intolerable pain. Many patients find a shock exceeding 0.5-1 joule exceedingly uncomfortable. Patients with frequent episodes or with short, self-terminating episodes are not good candidates for an implantable defibrillator. There is also the possibility that a wrongly timed shock may send the heart into ventricular fibrillation. A built-in ventricular defibrillator may take care of this problem, but – I am sure you get the drift – this is starting to get horribly complicated[1,20,23].

To further underscore this important point, quite recent research raises the possibility that ICD recipients with atrial fibrillation may actually increase their risk of experiencing ventricular fibrillation or tachycardia[24].

Some ICD experts caution that full contact sports, exposure to arc welding equipment or high voltage transformers and exposure to MRI scanners should be avoided by ICD wearers and that the use of cell phones can be problematical. They advise keeping the phone away from pockets near the pacemaker and to hold the phone to the ear furthest away from the pacemaker. ICD wearers are also advised not to stand near the doorways of stores with electronic theft-detection devices or airport security devices (although they may pass through them)[13,19].

Perhaps the best indication of the uncertainty still surrounding the efficacy and safety of ICDs is the fact that the British government prohibits ICD wearers from driving for 6 months after implantation of the device. If the defibrillator actually activates after implantation then the waiting begins all over again. If the doctor needs to reprogram the ICD driving is prohibited for a month after[25].

The reliability of the ICDs is also much in question. During the period 1990-2000 over half a million pacemakers and ICDs were recalled for mechanical or electrical problems. Over one million device checks and 36,187 device replacements resulted from the recalls and cost approximately \$870 million[26]. There is also some indication that ICD implantation may result in an infection requiring removal of the device[27].

In conclusion, would I personally consider an ICD at this stage of its development? The answer I'm afraid is no. There are still too many unresolved problems.

AV Node Ablation

Another approach to eliminating the effects of the fibrillation of the atrium on ventricular beats is to isolate the AV node (the ventricular beat controller) from any extraneous impulses and feed it its marching orders from an implanted pacemaker. This procedure has two very major drawbacks[28]:

- It does nothing to stop the fibrillation of the atria, which in itself can be quite uncomfortable, and necessitates continuing anticoagulation (warfarin) therapy.
- It makes the patient entirely dependent on the pacemaker. If it malfunctions or the batteries run out the patient dies.

AV node ablation is performed in much the same way as the RF ablation except that it is the area around the node that is ablated. A recent study found the procedure to be relatively safe for patients with lone atrial fibrillation, but another more recent study concluded that the mortality rate is about 2.1%[29,30]. Nevertheless, AV node ablation and pacemaker implantation is still considered a last resort approach especially for lone afibbers[1].

Maze Procedure

This is open-heart surgery with a price tag of about \$60,000 US. After making a foot long incision and cracking open the ribs, scar tissues are created on the surface of the heart to make a new pathway through which signals

travel directly from the sinus node to the AV node. The procedure is performed under general anaesthesia and takes about 3 hours. This is followed by a week in the hospital and 6 to 8 weeks recovering at home. It can take 3 months or more to return to full energy levels[31,32].

Nevertheless, if performed by a competent surgeon, the procedure is very effective in eliminating atrial fibrillation. Dr. James Cox at the Georgetown Cardiovascular Institute developed maze surgery. During the past 10 years Dr. Cox has operated on 346 patients with 94% success rate[33]. Swedish surgeons recently reported that the quality of life of 48 patients (80% with lone AF) who had undergone maze surgery improved very significantly after the procedure to equal the level of a healthy Swedish population. However, 12 patients had fairly serious complications. Two required a permanent pacemaker installed and 3 needed a temporary pacemaker. None of the patients died during one year of follow-up[34].

In conclusion, the maze procedure, although very effective for lone atrial fibrillation, is very major surgery and probably best left alone unless you are really desperate and can find a surgeon who has performed many successful ones.

Ablation Therapy

Ablation therapy, if successful, results in a complete cure of lone atrial fibrillation. The procedure begins with an electrophysiology study (EPS), an invasive test designed to map the electrical activity of the heart during fibrillation. Small tubes (catheters) are inserted into the veins in the groin, arms or neck, or under the collar bone and then directed into the heart. Once the measuring electrodes are in place fibrillation is usually induced and the electrophysiologist is then able to pinpoint the areas where the rogue (ectopic) beats originate. These areas are often found at the junction between the left atrium and the pulmonary veins[23,35]. The study can be somewhat uncomfortable and can last from 1 to 3 hours. At the end of it the electrophysiologist may report nothing to ablate if they have not located any foci of rogue cells or they may go directly to the next step and ablate the active area(s).

Radiofrequency (RF) ablation utilizes radio frequency energy to heat the tip of a special catheter inserted through one of the tubes used in the EPS. The cardiologist or electrophysiologist places the catheter next to the area initiating the fibrillation and then “zaps” this area. This produces a scar, which destroys the offending area or prevents impulses originating in it from going anywhere.

The ablation procedure is generally fairly painless (except for the cost) and lasts 4 hours or less. Its success rate for atrial fibrillation is currently around 80%, but with improved mapping and ablation techniques this is bound to improve[36,37]. There are potential, but generally rare adverse events though[28]:

- Bleeding or infection can occur at the catheter insertion site.
- Heart and blood vessels can suffer damage.
- Blood clots can form.
- The heart's normal electrical pathway can be damaged requiring the insertion of a permanent pacemaker.

As with any invasive procedure, the key to success is an experienced surgeon with lots of successful procedures to his credit. Centers of excellence such as the Cleveland Clinic, the University of Michigan in the USA, and Dr. Haissaguerre's group in Bordeaux, France probably have success rates of 80-90%. However, our survey of 14 afibbers who had undergone RF ablation revealed a success rate of only 50%[38]. Experience and skill is imperative for this procedure.

Finding ways to improve ablation techniques is one of the hottest research fields in electrophysiology today. Cardiologists at the University of Michigan have 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 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 while 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[35].

In a later communication the University of Michigan researchers report that about 35% of patients undergoing pulmonary vein isolation experience recurrence of atrial fibrillation in the week following the procedure. They point out that this early recurrence of AF is associated with a poorer prognosis of an ultimate cure, but that 30% of these patients do eventually gain complete freedom from AF[39].

Stenosis (narrowing of the pulmonary vein by 50% or more as a result of trauma experienced during the ablation) can be a problem with pulmonary vein isolation and may require balloon dilation (similar to angioplasty) to correct[40].

Dr. Andrea Natale and his team at the Cleveland Clinic have tackled this problem and developed a new pulmonary vein ablation procedure that employs ultrasound rather than RF energy to isolate the veins. The procedure uses an ultrasound transducer in a saline-filled balloon[41]. This approach reduces the amount of heating of the surface of the vein and the heat is not conducted to the heart tissue as it is with RF ablation. The result would seem to be a complete lack of stenosis. The new procedure still has some hurdles to overcome before it can become an "industry" standard. There are some problems fitting the balloon into the veins and in larger vein orifices it is difficult to achieve adequate heating to accomplish the isolation[41].

Research is continuing to improve ablation techniques with cryoablation and various laser techniques being high on the list of possible new approaches[42].

In conclusion, the maze procedure and radiofrequency or ultrasound ablation performed by extremely skilled hands are, at the moment, the only procedures that can permanently eliminate atrial fibrillation. However, ablation procedures have not yet been fully optimised and stenosis may turn out, at least for some procedures, to be a more serious and more frequent complication than originally thought. Still, many afibbers are very uneasy about having someone poking around in their heart while others do not have access to centers of excellence nor do they, in many cases, have the funds to pay for one or two costly ablation procedures. So the mission of The AFIB Report and the Bulletin Board remains intact – to discover and disseminate suggestions for alternative preventive or curative approaches while closely following relevant developments in the medical world.

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