THE AFIB REPORT

Your Premier Information Resource for Lone Atrial Fibrillation!

NUMBER 79 **MAY 2008** **8th YEAR**

The pros and cons of participating in vigorous endurance exercise are very much on the minds of many afibbers, especially vagal ones. Is sustained, vigorous exercise good or bad? Does it predispose to the development of afib and may cutting back be of benefit in managing LAF? I have researched these questions for this issue and present the results in my report "Endurance Exercise – Is It Worth It?"

Also in this issue we present evidence that most vagal afibbers receive the wrong medication, and some interesting findings regarding the perils of discontinuing aspirin therapy. We also try to help answer the questions – is lone afib recurrence inevitable, and does cryoablation work?

Last, but by no means least, we present another encouraging report in our series of effective elimination/reduction protocols using trigger avoidance, diet changes, supplementation, or medications.

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

Wishing you good health with lots of NSR,

Hans

Highlights

Most vagal afibbers receive wrong medication

MAASTRICHT, THE NETHERLANDS. There is still widespread denial among North American cardiologists as to the existence of vagally-mediated AF (atrial fibrillation) and a pronounced tendency to treat all AF patients the same. Hopefully, this will now change with the publication of the results of the Euro Heart Study. This study involved over 5000 AF patients treated in 182 hospitals in 25 different countries.

A total of 1517 of the patients experienced paroxysmal (intermittent) afib and was studied in detail. Among this group, 42% (640 patients) had a distinct, physician-verified, autonomic pattern as far as triggering an episode was concerned. Another 35% reported no clear trigger patterns, while in the remaining 23%; the physician did not verify the presence of triggers. The authors of the study classified the trigger pattern as vagal if episodes occurred after a meal or during the night, and as adrenergic if initiated by exercise or emotional stress. Afibbers with no clear trigger pattern were classified as mixed.

Sixteen percent of the group had lone AF defined as afib without the presence of hypertension, coronary artery disease, or heart failure. Somewhat surprisingly, the researchers found no difference in the incidence of heart disease among vagal and adrenergic afibbers. Among the group with clearly defined trigger patterns, 18% were classified as vagal, 46% as adrenergic, and the remaining 36% as mixed. (NOTE: The distribution in our most

recent LAF survey was 30% vagal, 6% adrenergic, and 64% mixed).

The major conclusions reached from the study are as follows:

- Exercise and emotional stress were the most common triggers followed by electrolyte imbalances, and alcohol and caffeine consumption.

- The majority (72%) of vagal afibbers received non-recommended drugs (beta-blockers, sotalol, digoxin or propafenone) – 57% were prescribed beta-blockers or sotalol.

- Vagal afibbers who were prescribed non-recommended drugs were more likely to progress to persistent or permanent AF than were vagal afibbers prescribed recommended drugs (primarily flecainide). After 1 year of follow-up, 19% of vagal afibbers prescribe non-recommended drugs had developed persistent or permanent afib as compared to 0% in the group prescribed correct drugs.

- Among adrenergic afibbers, 20% did not receive the medication recommended in the 2006 ACC/AHS/ESC Guidelines for the Management of Atrial Fibrillation. However, there was no indication that the type of medication affected progression to persistent or permanent in this group.

- Quality of care would appear to vary considerably between the regions in Europe. In the Mediterranean region 41% of patients received the recommended treatment as compared to 20% in Central Europe, and only 19% in Western Europe. Similarly, in the Mediterranean region physicians verified the presence of triggers in 75% of cases as compared to 79% in Central Europe and only 46% in Western Europe. *Editor's comment*: It would seem that afib care in Western Europe is substandard, but probably no worse than in North America.

- The authors point out that beta-blockers are often given in conjunction with class 1C antiarrhythmics (flecainide and propafenone) in order to prevent 1:1 conduction in the case of atrial flutter induced by the class 1C drug. They suggest that verapamil and diltiazem could be used as safer alternatives.

The authors conclude, "*Physicians do not seem to choose rhythm or rate control medication based upon autonomic trigger pattern of AF. However, the role of autonomic influences should be taken into consideration in order to achieve an optimal management of the disease as non-recommended treatment may result in aggravation of the arrhythmia.*"

de Vos, CB, et al. Autonomic trigger patterns and anti-arrhythmic treatment of paroxysmal atrial fibrillation: data from the Euro Heart Survey. **European Heart Journal**, *Vol. 29, 2008, pp. 632-39*

Editor's comment: Although not specifically directed at lone AF, this new European study is clearly a landmark and emphasizes the importance of determining trigger pattern (vagal, adrenergic or mixed) before prescribing medication for paroxysmal afibbers. It is interesting that our first LAF Survey (February 2001) revealed that 50% of vagal afibbers had been prescribed non-recommended drugs. This resulted in an average afib burden (# of episodes times their duration) more than twice as high than the burden among vagal afibbers taking flecainide or disopyramide. As far as propafenone (Rythmol) is concerned, the situation may not be as clear-cut as suggested in the Euro Heart Study. Some vagal afibbers have found this drug quite useful. Some fairly recent research have found that the degree of beta-blocking effect exhibited by propafenone depends markedly on how fast it is metabolized, so this may explain why it works for some vagal afibbers, while it is contraindicated in most others.

N-acetylcysteine helps prevent post-operative AF

ISPARTA, TURKEY. N-acetylcysteine (NAC) has anti-inflammatory properties and is the precursor of glutathione, the body's main indigenous antioxidant. It is effective in counteracting the effects of acetaminophen (Tylenol, Paracetamol) poisoning and is protective against kidney damage caused by exposure to x-ray dyes (contrast media). Turkish researchers now report that NAC is also highly effective in preventing post-operative atrial fibrillation (AF). The development of AF following cardiac valve surgery and coronary artery bypass surgery is quite common affecting between 10% and 65% of patients. Considering that well over 500,000 bypass operations (half of which are likely unnecessary) are performed each year in the US alone, post-operative AF is clearly a major problem.

The Turkish researchers reasoned that since cardiac surgery is accompanied by inflammation, substantially increased oxidative stress, and a loss of glutathione, it would perhaps be beneficial to pre- and post-treat patients with a potent antioxidant and glutathione precursor such as NAC. Their randomized, double-blind, placebo-controlled clinical trial involved 115 patients 107 of which underwent bypass surgery alone, while the remaining 8 underwent valve surgery with or without accompanying bypass surgery.

Half of the study participants received an intravenous infusion of NAC one hour prior to their procedure followed by an infusion for 48 hours after the procedure. The amount of NAC infused prior to surgery was 50 mg/kg (3500 mg for a person weighing 70 kg – 154 lbs), while the amount infused during the 2 days following the procedure was 50 mg/kg per day. The other half of the group just received a standard saline infusion. All patients were monitored with 12-lead electrocardiography. No side effects of NAC administration were observed.

During follow-up 21.1% of patients in the placebo (saline infusion) group developed AF as compared to only 5.2% in the NAC-treated group – a 4-fold reduction in risk. All afib episodes converted to sinus rhythm either spontaneously or with the use of amiodarone. The researchers conclude that pre- and post-treatment with NAC substantially reduces the risk of post-operative AF and speculate that NAC therapy may be of value in AF patients.
Ozaydin, M, et al. N-acetylcysteine for the prevention of postoperative atrial fibrillation. **European Heart Journal**, *Vol. 29, 2008, pp. 625-31*

Editor's comment: There is little question that bypass surgery is a potent "recruiting ground" for afib patients. Whether or not these patients go on to have subsequent episodes is not clear, but certainly, anything that will help prevent that first episode is most welcome. Could regular supplementation with NAC help prevent future episodes? I am not aware of any evidence to this effect, but it certainly is an intriguing possibility. It is, unfortunately, not clear from the article whether the NAC was infused in a saline solution or in an aqueous solution. If indeed it was infused in the aqueous form (no salt), then it is possible that the observed benefits of NAC were at least partly due to the lack of salt (sodium chloride) in the infusion. Somehow, it does not seem like a great idea to infuse a salt solution after the patient has suffered the significant potassium loss always accompanying cardiac surgery.

Perils of aspirin discontinuation

LAUSANNE, SWITZERLAND. It is estimated that more than 50 million Americans now take a daily aspirin for the prevention of cardiovascular disease. While there is evidence that this practice may help prevent heart attacks in high-risk populations, there is no evidence that it may help prevent a first stroke or TIA (transient ischemic attack) in low-risk patients such as lone afibbers. Nevertheless, the ritual of the daily aspirin is clearly very popular and it is therefore of concern that interrupting this ritual may result in an increased risk of stroke.

Researchers at the University Hospital in Lausanne report a 3-fold increased risk of ischemic stroke in a group of high-risk patients who discontinued their aspirin therapy prior to scheduled surgery, because they experienced bleeding complications or interactions with other drugs, or because they or their physician decided that they no longer needed the aspirin. The study included 309 patients with an average age of 72 years who had suffered a recent stroke or TIA, and a control group of 309 patients who had a history of stroke or TIA, but had not suffered an event in the last 6 months. Neither

group was particularly healthy with about 70% having hypertension, and 36% and 18% (control group) respectively having coronary heart disease. Thirteen participants in the patient group and 4 in the control group had discontinued aspirin at least 4 weeks prior to their TIA or stroke (patient group) or 4 weeks prior to being interviewed (control group).

The researchers found (after correcting for possible confounding variables such as coronary heart disease) that those who discontinued aspirin were 3.4 times more likely to experience a TIA or ischemic stroke than were patients who remained on the aspirin. Seventy percent of the strokes occurred within 10 days after discontinuation (mean: 9 days). The researchers conclude that the discontinuation of aspirin therapy could increase the risk of ischemic stroke in patients with multiple cardiovascular risk factors, mainly in those with coronary heart disease.

Maulaz, AB, et al. Effect of discontinuing aspirin therapy on the risk of brain ischemic stroke. **Archives of Neurology**, *Vol. 62, August 2005, pp. 1217-20*
Llinas, RH. Could discontinuation of aspirin therapy be a trigger for stroke? **Nature Clinical Practice Neurology**, *Vol. 2, June 2006, pp. 300-01*

Editor's comment: The patient groups evaluated in this study had multiple cardiovascular risk factors including hypertension, coronary heart disease, and diabetes. Thus, it is not at all clear whether the increased stroke risk accompanying aspirin withdrawal applies to afibbers with no underlying heart disease or other stroke risk factors. My guess would be that it probably does not. Nevertheless, if an afibber wishes to wean off the daily aspirin it may be prudent to replace it, at least for a couple of months, with one or more natural antiplatelet aggregation agents such as vitamin C, vitamin E, vitamin B6, niacin, fish oil, ginkgo biloba, or garlic.

Is lone AF recurrence inevitable?

BARCELONA, SPAIN. Lone atrial fibrillation (LAF) is defined as AF occurring in the absence of structural heart disease. Idiopathic AF is defined as lone AF of no known cause; ie. thyroid disorders, hemochromatosis, alcoholism, and electrolyte disturbances have been ruled out. Although LAF patients, in most cases idiopathic, constitute between 10 and 30% of all afib patients, comparatively few studies have been done dealing specifically with this condition. A recently released study by researchers at the University of Barcelona is, hopefully, a harbinger of a trend to focus greater efforts on determining the causes and likely progression of LAF.

The study involved 98 patients (71% men with an average age of 48 years) who were admitted to the University hospital's emergency room with AF of no known cause (idiopathic). Most (64.3%) had experienced previous episodes, while the remaining 35.7% showed up with their first episode. Half the patients reverted spontaneously to normal sinus rhythm (NSR) or did so after oral flecainide administration (classified as paroxysmal afibbers), while the other half required electrical cardioversion to convert (classified as persistent afibbers). First-occurrence patients were discharged with no medication, while recurrent patients were discharged on whatever medications they had used prior to the index episode (the episode at which they first were admitted to the ER), or on a class 1C antiarrhythmic (mostly flecainide). Patients for whom class 1C drugs had clearly not worked were recommended to try amiodarone. None of the patients were discharged with a prescription for anticoagulants (warfarin).

During the following 6 months, 57% of the entire patient group experienced at least one subsequent afib episode. Recurrent afib was more common among those with prior episodes before the index episode (65.1%) than among "first-onset" patients (34.9%); this despite the fact that 70% of the "veteran" afibbers were taking antiarrhythmics. As a matter of fact, taking amiodarone or a class 1C antiarrhythmic did not significantly influence the risk of recurrence in this group. The researchers also observed that an enlarged left atrium (dilated anteroposterior LA diameter) was associated with a 30% increased risk of AF recurrence. However, they found no association between recurrence risk and afib type (paroxysmal or persistent).

They conclude that for lone (idiopathic) afibbers who have a recurrent episode and an enlarged left atrium (indexed for body surface area), the probability of another episode is about 90% despite the use of antiarrhythmics. On the other hand, the probability of another episode is only 30% in a patient with normal LA diameter who has just experienced one episode.

Arriagada, G, et al. Predictors of arrhythmia recurrence in patients with lone atrial fibrillation. **Europace**, *Vol. 10, 2008, pp. 9-14*

Editor's comment: Several findings stand out in this excellent report:

- First-onset patients were not put on medication after their first episode. This is in accordance with the 2001 ACC/AHA/ESC recommendations.
- No patients were prescribed warfarin.
- The use of antiarrhythmics was not effective in preventing further episodes in most cases.

Of particular interest is the finding that first-time afibbers with a non-dilated left atrium have only a 30% chance of experiencing another episode in the 6 months following the first one. My guess is that these patients may well be able to hold off subsequent episodes for a long time through trigger avoidance, supplementation, and dietary and lifestyle changes.

Cryoablation outcome

MAASTRICHT, THE NETHERLANDS. Pulmonary vein isolation (PVI) using cryoablation is a procedure very similar to the standard PVI using radiofrequency energy for lesion creation except that it uses a nitrogen-cooled (–90^{o} C) catheter rather than an electrically-heated catheter. Cryoablation is potentially safer than RF ablation in that the risk of pulmonary vein stenosis and esophageal injury is pretty well non-existent. The procedure also has the advantage that, since no pain is felt during lesion creation, it does not require conscious sedation or anaesthesia.

EPs at the Academic Hospital in Maastricht recently reported on the long-term success of the procedure. Their study included 70 patients (54 men and 16 women), 77% of whom had lone AF with the remaining having arterial hypertension (14%) or minimal heart disease (9%). The age of the patients ranged between 21 and 65 years (average of 40 years), their average left atrial diameter was 38 mm, and the left ventricular ejection fraction averaged 59% – in other words, a pretty healthy group. The patients had all failed one or two antiarrhythmic drugs (none had been on amiodarone), and had suffered from symptomatic afib episodes for an average of 4 years.

All patients underwent a segmental PVI using cryoablation. The Maastricht EPs were able to locate the specific offending vein(s) in 14% of cases and isolated only that vein or veins. In other cases, all veins were targeted. The patients were followed for an average of 33 months; the first 180 days via a transtelephonic event recorder and the following months via periodic Holter monitoring. At the end of the follow-up period, 49% were still in sinus rhythm without the use of antiarrhythmics, 22% were afib-free with the use of antiarrhythmics, 11% were improved more than 50% with the use of antiarrhythmics, and the remaining 18% had not benefited from the procedure. The researchers point out that the 10 patients in which the offending vein could be identified were all free of afib at the end of the follow-up period. They make the following interesting statement:

> *Atrial fibrillation is a disease with different stages. In early stages, paroxysmal and nonsustained episodes are the rule. In this stage, the triggers, mostly located in the pulmonary veins, are the main culprit of AF. Over time, atrial remodeling starts to occur, and more substrate becomes available to sustain longer episodes. Therefore, self-perpetuation of AF (AF begets AF) leads to the idea that a treatment strategy employed early in the disease would be more likely to succeed.*

The average procedure time was almost 6 hours with a fluoroscopy time of 88 minutes. One patient suffered a stroke during or after the procedure, another experienced a pulmonary embolism, and a third experienced transient phrenic nerve paralysis. No cases of stenosis or esophageal injury were detected.

Moreira, W, et al. Long-term follow-up after cryothermic ostial pulmonary vein isolation in paroxysmal atrial fibrillation. **Journal of the American College of Cardiology**, *Vol. 51, No. 8, February 26, 2008, pp. 850-55*

Editor's comment: A total success rate of 49% in a group of prime PVI candidates is not impressive; thus, there would seem to be no advantage of choosing cryoablation over a RF ablation carried out by a top-rated EP.

RESEARCH REPORT

Endurance Exercise – Is It Worth It?

by Hans R. Larsen

There is ample evidence that being physically fit reduces the risk of heart disease, stroke, metabolic syndrome, osteoporosis, hypertension, diabetes, prostate cancer, breast cancer, colon cancer, depression, anxiety, and many other conditions. There is also evidence that physically fit people live longer than do sedentary people. It is also clear that the only way to become and remain physically fit is by being physically active. The question is, "How much physical activity is required to be considered fit, and is there such a thing as overdoing the physical fitness"?

How Much is Enough?
Ten years ago researchers at the Royal Free Hospital School of Medicine in London, England reported that middle-aged men who regularly engaged in light to moderate physical activity experienced a 40-50% lower mortality than did those who were largely inactive.[1] Researchers at Harvard Medical School found that women who walked for at least one hour a week at a moderate pace had a 50% lower risk of developing coronary artery disease than did those who did not walk regularly. The pace of walking (exercise intensity) was found to be less important than the time spent in walking, and increasing pace or walking time (beyond 1.5 hours/week) did not provide added protection.[2] Clearly, regular exercise is important, but how much is required and what are the optimum ways of getting it?

An expert panel endorsed by the American Heart Association and the American College of Sports Medicine recommends that all healthy adults aged 18 to 65 years engage in at least 30 minutes of moderate-intensity aerobic physical activity on 5 days each week, or vigorous-intensity aerobic activity for a minimum of 20 minutes on 3 days of the week. Combinations of moderate and vigorous exercise are also acceptable and the 30 minutes of moderate physical activity can be met, for example, by 3 individual bouts of 10 minutes each. The panel emphasizes that physical exercise over and above the recommend minimum can be expected to lead to reduced premature mortality and further health improvements, particularly in regard to cardiovascular health. The panel also recommends activities that maintain and increase muscular strength for a minimum of 2 days each week. Such activities would include stair climbing, weight training, and weight-bearing calisthenics.

The intensity of physical exercise is usually expressed in terms of energy expenditure which, in turn, is expressed in **metabolic equivalents** (MET). One MET represents an individual's energy expenditure while sitting quietly for 1 minute (equivalent to about 1.2 kilocalories/minute for a person weighing 160 lbs). Moderate activity is associated with a MET equivalent of 3-6 METs per minute, while vigorous exercise is associated with METs greater than 6. METs for some common activities are given below:

• Walking at 3 mph (5.0 km/h)	3.3 MET
• Walking at very brisk pace of 4 mph (6.4 km/h)	5.0 MET
• Bicycling on flat surface at 10-12 mph (16-19 km/h)	6.0 MET
• Bicycling fast at 14-16 mph (22-26 km/h)	10.0 MET
• Golfing (walking and pulling clubs)	4.3 MET
• Swimming (leisurely)	6.0 MET
• Swimming (moderate to hard)	8.0-11.0 MET
• Hiking at moderate pace with light or no pack	7.0 MET
• Hiking at steep grades and heavy pack	7.5-9.0 MET
• Jogging at 5 mph (8 km/h)	8.0 MET
• Cross-country skiing (slow)	7.0 MET
• Cross-country skiing (fast)	9.0 MET
• Competitive soccer	10.0 MET

Thus, 30 minutes of walking at 3.0 mph would accumulate 99 METs (3.3x30) and jogging for 20 minutes at 5 mph would accumulate 160 METs (8x20). The panel suggests a minimum weekly MET accumulation of 450 to 750 METs be achieved through specific physical exercise.

The panel makes the interesting observation that exercise is relatively ineffective in achieving weight loss, but that a very much increased level of activity is required to maintain a weight loss achieved by other means. They also acknowledge that the risk of musculoskeletal injury increases substantially with increased physical activity and can affect as many as 55% of people involved in jogging programs and US Army basic training. The risk of cardiac arrest and heart attack also increases during vigorous physical exercise, especially among infrequent exercisers. Nevertheless, the panel concludes that, in the case of healthy individuals, the benefits of regular moderate to vigorous physical activity far outweighs the risks. They also suggest that healthy men and women do not need to consult with a physician or other healthcare provider prior to embarking on a regular exercise program. However, those with cardiovascular disease, diabetes, or other chronic diseases should clearly do so.[3]

I an accompanying article Miriam Nelson of Tufts University and other members of a separate panel outline physical activity recommendations for those above the age of 65 years and adults aged 50-64 years with clinically significant chronic disease conditions or functional limitations. The recommendations are identical to those discussed above, except that the definition of *moderate* and *vigorous* exercise is tailored to the individual's basic fitness level rather than given as specific MET targets.[4]

How Much is Too Much?

So, regular exercise is clearly a good thing, but like all good things it can be overdone. British researchers followed 20 veteran athletes for 12 years and concluded that high intensity lifelong endurance exercise is associated with altered cardiac structure and function, especially the development of left ventricular hypertrophy (thickening of the muscles of the left ventricle) and profound bradycardia. Two of the athletes ended up having to have a pacemaker implanted.[5] NOTE: Endurance exercise is usually defined as vigorous exercise for more than 45 minutes per session.

Swedish sports medicine experts found that elderly men with a lifelong history of regular, very strenuous exercise were more likely to suffer from complex ventricular arrhythmias than were men who had been only moderately physically active.[6]

A recent study involving 134 former Swiss professional cyclists concluded that these former athletes were more likely to suffer from sinus node disease and atrial fibrillation and flutter than were an age-matched group of golfers. The two groups were examined at age 66 years, which for the cyclists was an average of 38 years from their last professional race (Tour de Suisse). The Swiss researchers also observed that ventricular tachycardias were more common in the cyclists than in the golfers (15% vs 3%). They conclude that, "The elderly athlete may not be as healthy as believed."[7]

In 1998 Jouko Karjalainen and colleagues at the University of Helsinki reported that the prevalence of lone atrial fibrillation in a group of elite orienteers was 6 times higher than in a control group of less active men (5.3% vs 0.9%). The first afib episode among the orienteers occurred at a mean age of 52 years after an average training history of 36 years. Although the orienteers were more likely to develop lone atrial fibrillation, they were significantly less likely to develop heart disease (2.7% vs 7.5% in control group) and experienced lower mortality during the observation period (1.7% vs 8.5% in control group). The Finnish researchers conclude that vigorous, long-term endurance exercise is associated with atrial fibrillation in healthy, middle-aged men despite protecting against coronary heart disease and premature death. They speculate that the increased risk for afib is related to enhanced vagal tone, atrial enlargement, and left ventricular hypertrophy.[8]

Medical researchers at the University of Barcelona have found that men who engage in vigorous physical exercise of many years have an increased risk of developing lone (vagal) atrial fibrillation. A review of the records of 1160 patients seen at an outpatient arrhythmia clinic revealed that the incidence of lone AF among long-term exercisers was 60% as compared to only 15% in the general population of Catalonia.[9] The same group of researchers also concluded that lone afib was about 3 times more prevalent among men who reported

former and current sport practice than among men who did not. They observed a particularly strong correlation for men who reported more than 1500 hours of lifetime sports activities.[10]

More recent research by the Spanish group confirmed the strong association between LAF risk and accumulated moderate and heavy physical activity. Those with a lifetime accumulated moderate plus heavy physical activity of more than 9300 hours had 15 times the prevalence of LAF than did those with less than 2100 hours accumulated. More than 564 hours of accumulated heavy, vigorous physical activity was associated with a 7 times increased prevalence of LAF.

The researchers speculate that the negative effects of moderate and particularly vigorous physical activity may be related to the chronic volume and pressure overload caused by the increased activity. They conclude,

> "*The fact that physical activity is a risk factor for AF does not argue against exercise as a way of preventing coronary artery disease. It only offers a word of caution suggesting that the benefits obtained by physical activity, if excessively intense and over a great many hours, may be counteracted by the risk of AF.*"[11]

The evidence that heavy, sustained physical exercise is associated with an increased risk of lone atrial fibrillation is indeed substantial. The only study disputing this connection is the one carried out by Antonio Pelliccia and colleagues at the National Institute of Sports Medicine in Rome. These researchers found no difference in the prevalence of atrial fibrillation in a group of competitive athletes as compared to the general population.[12] However, the average age of this group of athletes (24 years) was substantially lower than the average age in the studies discussed earlier, so the results are not comparable, especially since it is well known that the incidence of afib increases with age, and that the average age at diagnosis is about 48 years for lone afibbers.

Why would long-term, vigorous endurance exercise increase the risk of developing atrial fibrillation? Long-term endurance training profoundly affects the body's physiology. Among other things it significantly reduces the heart rate and testosterone levels.[13,14] It is also known that, while exercise in the short-term increases adrenergic tone, its long-term effect is an increase in vagal tone.[15,16] Vigorous, long-term endurance exercise has also been associated with an increased risk of inflammation. Greek researchers observed that participants in a 36-hour long distance run experienced a 152-fold increase in C-reactive protein (CRP) levels and an 8000-fold increase in the level of interleukin-6 (IL-6), another important marker of systemic inflammation. They conclude that the increases in the inflammation markers noted, "amount to a potent systemic inflammatory response".[17] Finally, there is ample evidence that long-term endurance training tends to increase the size of the left atrium and is also likely to lead to left ventricular hypertrophy.[5,7,8]

Taken together, all these effects of vigorous, long-term endurance training is likely to combine to form a potent breeding ground for the development of atrial fibrillation. It would seem logical that continuing vigorous endurance training after experiencing a first afib episode would be a poor choice.

Several studies have found a convincing association between inflammation and afib.[18] There is also evidence that vigorous endurance sports such as participating in marathons can result in a very pronounced systemic inflammation.[17] Andrea Frustaci and colleagues at the Catholic University of Rome have found that inflammation of the heart lining (myocarditis) is an almost universal feature among lone afibbers.[19] Further exercise will fan an inflammation and Swedish sports medicine experts are adamant that exercise should be avoided when myocarditis is suspected.[20]

Does Detraining Help Prevent AF?

Does refraining from heavy exercise actually work for lone afibbers? Says the late Professor Philippe Coumel,

> "*It is known that in well-trained people suffering from vagal AF, the first step of therapy should be deconditioning by discontinuing high-level training. It may be sufficient to bring about an improvement in the patient and it is often a necessary adjuvant to facilitate pharmacological therapy*."[21]

In the same paper Dr. Coumel also makes the following statement of interest to vagal afibbers,

> "*Not only are beta-blockers ineffective,* [for vagal afibbers] *but they usually make patients worse and inhibit the efficacy of antiarrhythmics.*"

British researchers support Dr. Coumel's observation about the beneficial effects of detraining. They report the case of a 53-year-old athlete whose symptoms of palpitations, ectopics, and atrial tachycardia completely resolved after detraining.[22] Spanish researchers report that detraining for 2-4 weeks results in an increase in heart rate and adrenergic tone – both changes beneficial in regards to vagally-induced afib.[23] At least one member of our afib group has found that forgoing exercise one week out of every four significantly reduced his frequency of episodes. Of course, abruptly ceasing all exercise may carry with it a whole new set of problems, so a gradual approach is definitely in order. This might be worth experimenting with if you are a vagal afibber.

There is some evidence that patients who have been ablated for right atrial flutter are more likely (81% increased risk) to develop atrial fibrillation post-ablation if they have a history of active engagement in endurance sports. Those continuing endurance sports after their ablation are also more likely (68% increased risk) to develop post-ablation AF. The Belgian researchers reporting these findings conclude that there is a 10% and 11% increased risk of developing AF per weekly hour of sport performed pre- and post-ablation for flutter.[24]

Several ablated afibbers who resumed their pre-ablation training schedule too early have reported a relapse and required a second ablation to achieve a final cure. There is now evidence that repeat ablations may be the norm rather than the exception for competitive athletes with afib. Italian researchers found it took an average of 2.3 PVIs to prevent afib recurrences in athletes who had been disqualified from competition due to their afib.[25,26]

Somewhat paradoxically, actions that may promote afib in vagal afibbers may also help to terminate an episode already in progress. About 27% of male vagal afibbers reported (in LAF Survey 14) that they were able to terminate an afib episode by exercise. This finding is supported by a case history involving a 45-year-old physician with vagally-mediated, paroxysmal AF. The patient was able to convert to normal sinus rhythm by exercising for 20 minutes on a cross-country ski machine (pulse rate of 170 bpm).[27]

Conclusion

So, is exercise good or bad? There is no question that the overall benefits of a regular, moderate exercise program far outweigh any possible adverse effects. However, when it comes to long-term, vigorous endurance exercise, the benefit/risk ratio is less clear. Such exercise can lead to undesirable cardiac modifications and an increased risk of developing atrial fibrillation. In those who already experience vagally-mediated afib, refraining from such exercises, or substantially cutting back may prove highly beneficial. To again quote Professor Coumel,

> "*Excessive training is harmful when it exaggeratedly modifies the ANS balance beyond the sympathetic and parasympathetic physiological values. It is a major mistake to think that the man in the street must be as trained and fit as the professional sportsman. Any common sense driver knows that if he wants to make his car last, he must avoid handling it as a rally or Formula One driver.*"[28]

References

1. Wannamethee, SG, et al. Changes in physical activity, mortality, and incidence of coronary heart disease in older men. The Lancet, Vol. 351, May 30, 1998, pp. 1603-08
2. Lee, IM, et al. Physical activity and coronary heart disease in women: is "no pain, no gain" passé? JAMA, Vol. 285, March 21, pp. 1447-54
3. Haskell, WL, et al. Physical activity and public health. Updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. Circulation, Vol. 116, August 28, 2007
4. Nelson, ME, et al. Physical activity and public health in older adults. Updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. Circulation, Vol. 116, August 28, 2007
5. Hood, S and Northcote, RJ. Cardiac assessment of veteran endurance athletes: a 12-year follow up study. British Journal of Sports Medicine, Vol. 33, 1999, pp. 239-43
6. Jensen-Urstad, K, et al. High prevalence of arrhythmias in elderly male athletes with a lifelong history of regular strenuous exercise. Heart, Vol. 79, 1998, pp. 161-64

7. Baldesberger, S, et al. Sinus node disease and arrhythmias in the long-term follow-up of former professional cyclists. European Heart Journal, Vol. 29, 2008, pp. 71-78
8. Karjalainen, J, et al. Lone atrial fibrillation in vigorously exercising middle aged men. British Medical Journal, Vol. 316, June 13, 1998, pp. 1784-85
9. Mont, L, et al. Long-lasting sport practice and lone atrial fibrillation. European Heart Journal, Vol. 23, March 2002, pp. 477-82
10. Elosua, R, et al. Sport practice and the risk of lone atrial fibrillation. International Journal of Cardiology, Vol. 108, No. 3, April 14, 2006, pp. 332-27
11. Mont, L, et al. Physical activity, height, and left atrial size are independent risk factors for lone atrial fibrillation in middle-aged healthy individuals. Europace, Vol. 10, 2008, pp. 15-20
12. Pelliccia, A, et al. Prevalence and clinical significance of left atrial remodeling in competitive athletes. Journal of the American College of Cardiology, Vol. 46, No. 4, August 16, 2005, pp. 690-96
13. Steinacker, J.M., et al. Training of junior rowers before world championships: effects on performance, mood state and selected hormonal and metabolic responses. Journal of Sports Medicine and Physical Fitness, Vol. 40, December 2000, pp. 327-35
14. Hackney, A.C. Endurance exercise training and reproductive endocrine dysfunction in men: alterations in the hypothalamic-pituitary-testicular axis. Curr Pharm Des, Vol. 7, March 2001, pp. 261-73
15. Matsuo, S., et al. Cardiac sympathetic dysfunction in an athlete's heart detected by 123I-metaiodobenzylguanidine scintigraphy. Japanese Circ J, Vol. 65, May 2001, pp. 371-4
16. Hautala, A., et al. Changes in cardiac autonomic regulation after prolonged maximal exercise. Clin Physiol, Vol. 21, March 2001, pp. 238-45
17. Margeli, A, et al. Dramatic elevations of interleukin-6 and acute-phase reactants in athletes participating in the ultradistance foot race Spartathlon. Journal of Clinical Endocrinology & Metabolism, Vol. 90, No. 7, July 2005, pp. 3914-18
18. Swanson, DR. Atrial fibrillation in athletes: implicit literature-based connections suggest that overtraining and subsequent inflammation may be a contributory mechanism. Medical Hypotheses, Vol. 66, 2006, pp. 1085-92
19. Frustaci, A, et al. Histological substrate of atrial biopsies in patients with lone atrial fibrillation. Circulation, Vol. 96, August 19, 1997, pp. 1180-84
20. Friman, G and Wesslen, L. Special feature for the Olympics: effects of exercise on the immune system. Immunol Cel Biol, Vol. 78, No. 5, October 2000, pp. 510-22
21. Coumel, P. Atrial fibrillation: one more sporting inconvenience? European Heart Journal, Vol. 23, No. 6, March 2002, pp. 431-33
22. Obel, OA and Davidson, C. Arrhythmias in an athlete: the effect of de-training. Postgraduate Medical Journal, Vol. 81, January 2005, pp. 62-64
23. Mujika, I and Padilla, S. Cardiorespiratory and metabolic characteristics of detraining in humans. Medicine and Science in Sports and Exercise, Vol. 33, March 2001, pp. 413-21
24. Heidbuchel, H, et al. An endurance sport is a risk factor for atrial fibrillation after ablation for atrial flutter. International Journal of Cardiology, Vol. 107, No. 1, February 8, 2006, pp. 67-72
25. Furlanello, F, et al. Radiofrequency catheter ablation of atrial fibrillation in athletes referred for disabling symptoms preventing usual training schedule and sport competition. Journal of Cardiovascular Electrophysiology, published online, February 8, 2008
26. Lampert, R. Atrial fibrillation in athletes: toward more effective therapy and better understanding. Journal of Cardiovascular Electrophysiology, published online, March 21, 2008
27. Ragozzino, MW, et al. Self-cardioversion of paroxysmal lone atrial fibrillation with exercise. New England Journal of Medicine, Vol. 347, No. 25, December 19, 2002, pp. 2085-86
28. Larsen, HR. Lone Atrial Fibrillation: Towards A Cure. IHN, Victoria, BC, Canada, 2002, p. ii

Elimination/Reduction Protocol

Case No. 853

Female afibber – **47 years** of age with **vagal AF** of **25 years standing**; no underlying heart disease
No. of episodes in 6 months prior to starting protocol: **Permanent**
Afib burden in 6 months prior to starting protocol: **Permanent**
No. of episodes in most recent 6 months after starting protocol: **0**
Afib burden in most recent 6 months after starting protocol: **0**
Time on protocol: **7 ½ years**
Episodes since protocol implementation: **3 episodes lasting approx. 2 hours each early on**
Still need to avoid triggers?: **No**

Main components of effective protocol
Trigger avoidance: **MSG, aspartame, alcohol, caffeine, dehydration, high glycemic index foods**
Diet changes: **Changed to paleo diet**
Supplementation: **None**
Drug therapy: **None**
Stress management: **None**
Approaches to shorten episodes: **Not applicable**
Approaches to reduce ectopics: **Paleo diet**

Background and details of protocol

I experienced AF at 22 years of age. It came out of the blue after the birth of my first son. Unfortunately, sometimes when it happened I would pass out. Witnesses said I convulsed, so the diagnosis came as epilepsy. I was put on anticonvulsants which never worked. In the beginning my AF was maybe twice or three times a week. Always at rest. It was short-lived, well the really fast racing part was short lived – maybe 2 to 4 hours (in the end it could go on for days, then became permanent). Every so often I would visit my GP and complain, but was told it was just palpitations and I was being over anxious. So decided they must be panic attacks and gave up on doctors.

Nine years later I was finally diagnosed with atrial fibrillation and was put on digoxin which I stayed on for 10 years. In 2000 I finally saw a new cardiologist who said that I should never have been put on digoxin. I was then put on Rythmol (propafenone) and when this did not work sotalol, flecainide, and atenolol followed. All these drugs had terrible side effects, so in December 2001 I stopped taking all medications.

In October of 2000 I had started a program of trigger elimination (notably MSG and food additives) and had also adopted the paleo diet. In hindsight my diet had been very refined which led to leaky gut (with no digestive symptoms) so that my body was very low on all nutrients. The paleo diet cut out the problem foods, helped heal the gut, and didn't feed bad bacteria and allowed good bacteria to flourish, thus allowing absorptions of all the major nutrients. Excitatory neurotransmitters such as MSG, aspartame, etc. played havoc since there were not enough minerals, vitamins, etc to make inhibitory neurotransmitters, hormones, etc. and the liver was under undue stress and unable to break down and eliminate toxins. Starchy foods such as grains and potatoes played havoc with blood sugar levels as there were no glycogen stores in the liver or muscle to fall back on. This was verified by hair tissue analysis which showed that I was still low on all minerals except vitamin K which was very high (and had an inverse ratio with Na – very low), meaning that K is not readily available for use in the body. The lack of available K meant it was difficult for insulin to be delivered into cell walls for storage, and also couldn't polarize nerve impulses. As mineral levels increased the problems subsided. Obviously, the whole scenario is a lot more complex than discussed above; however, it is my opinion that my AF was a culmination of long-term malnutrition that could not be sorted out by taking supplements since they could not be metabolized, and the underlying reasons had to be dealt with first.

Since then I have played around with my diet, as after curing my AF, I became aware of reactive hypoglycemia. Happily, I have overcome this, but of course, I have to stick to my paleo diet. I prefer it this way since I have regained my health. I have cured more than just AF – don't suffer from fibromyalgia, headaches, tremors, seizures, and fainting. The only thing I have not solved is low blood pressure, but I can live with that!

THE AFIB REPORT is published 10 times a year by:

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