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Publisher: Hans R. Larsen MSc ChE**

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VIRTUAL LAF CONFERENCE

Proceedings of 64th Session
May 21st, 2008 – June 6th, 2008

SUBJECT: Exercise and Afib

Exercise and afib is clearly a very important subject and deserves a full Conference Room session. It has been discussed at length in my recent research report (The AFIB Report - May 2008) as well as in several Bulletin Board threads. So the purpose of this Conference Room Session (Number 64) is to consolidate what has already been posted and also hopefully, add new significant material to the deliberations. So, I'll start out with my May 2008 Research Report.

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]

In 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]

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Date: 07-28-07 06:42

I've been reading this post for 2 months now and it has really helped understanding this issue. I am 44, standing HR of 38-40bpm, and a competitive cyclist since 1985. I have experienced proximal A-Fib since 2001. Early in the spring, after the usual week long training camp in the mountains of NC, my palpitations became unbearable when lying down and A-fib activity increased. Only a low heart rate coupled with lying down causes extended periods of palpitations. I get them while sitting down, however, not nearly as noticeable. Furthermore, my palpitations can be terminated by standing up. All this devilish activity in my chest makes sleeping very difficult. This increased heart activity and anxiety caused me to seek out a cardiologist. He told me the usual heart medications are out of the question because of my low standing heart rate. I have scheduled a consultation appointment with an Electrophysiologist in August. My fear is coming out of an ablation with something worse than I have now.

A-Fib History

I have experienced intermittent, harmless palpitations since the late 1980's. During my first episode of A-fib in 2001 my initial reaction was flight, which terminated the A-fib episode (take it from me: running on a freeway isn't advisable). In my case, A-fib never manifests itself during exercise- only during a relaxed state. In all cases the A-fib episodes can be self terminated by raising my heart rate with vigorous exercise. It isn't easy at 2am nor comfortable, however, beats the alternative. I jump rope for 5min (in a-fib), let my ventricular heart rate go up to 130, stop exercising, and let my rate come down. If I don't convert the first time I repeat the process until the A-fib abates. A couple of times I have let my A-fib continue without practicing this method the rational being: A) To find out if it was more stubborn than me B) Investigate if it would mysteriously self terminate C) To get it recorded on the EKG.

Actions I am currently taking

Since April, I have attenuated my cycling and keep my HR below 140 when exercising; furthermore, started in June taking supplements outlined in this forum. Since I am de-tuning the engine, my standing heart rate has increased to 50bpm. Haven't noticed much improvement with the flutter, however, the A-fib episodes have decreased slightly and easier to get out of them. Sleeping is the real challenge with the palpitations (flutter) so I take Ambien at night. If I could sleep while standing up I would. Perhaps I will design a vertical straight-jacket-bed that will allow me to vertical sleep.

Thanks for taking the time reading this. Perhaps it can help someone else or you may have an opinion.

Author: [Wil Schuemann](mailto:---.dial.gorge.net) (---.dial.gorge.net)

Date: 07-28-07 08:04

As an athlete, if you discovered that because of a genetic deficiency, your ankle was predisposed to degenerate, and your athletic activity was aggravating the degeneration: then an intelligent person would probably cease all athletic activity except for the normal minimal activity associated with living, because they would recognize they have only three choices: (1) eliminating the athletic activity completely and immediately would decrease the probability of further degeneration and/or medical intervention; (2) stubbornly continue their athletic activity (full speed ahead and damn the torpedoes), denying (or ignoring) that doing so will aggravate the ankle's degeneration; or (3) pursue heroic medical intervention in the hope that doing so can somehow make them "normal" again, and which will allow them to continue being athletic, hopefully without making the degeneration situation even worse in the future.

There are a lot of gamblers here who have chosen (3). The jury is still out on whether (3) will neutralize the genetic deficiency. Those of us who have both given up on the athletic activity and have chosen heroic medical intervention, still worry whether the medical intervention will be sufficient to keep us in nsr for the rest of our life.

I personally am amazed by those who are convinced, in spite of continuing to engage in the athleticism which caused

their a-fib, that they will be forever remain a-fib free, because the medical intervention has somehow inoculated them against a-fib.

Were you a violinist whose right hand was smashed, it would be easy to "decide" to change to another activity. But, a-fib is insidious, because it develops slowly, and it therefore lets you continue to deny what seems so obvious to others, at least for a time.

A-fib is more like lung cancer. For decades, many smokers, who found they had a genetic deficiency which led to lung cancer, and who were "successfully" operated on for their lung cancer, continued to smoke because there was no "proof": that smoking had caused their lung cancer; and/or would do so again.

Author: [Gary](mailto:---.triad.res.rr.com) (---.triad.res.rr.com)

Date: 07-28-07 09:05

Point well taken, however, how am I too assume that A-fib is the result of being athletic? Furthermore, I am not convinced that not being athletic has caused my A-fib to decrease substantially. In the past the palpitations (flutter) decreased as a results of vigorous exercise. Only recently has the flutter remained (and stronger) even though I was vigorously exercising. In your example it is very easy to come to the conclusion: cut out exercise. However, things are not always as they seem. If you found out that exercise wasn't the cause at all wouldn't you fix the root cause by medical intervention? God gave us an intelligent mind which allows us to fix ourselves. So why not pursue this ?

Bottom line is: I have changed my life style by attenuating my level of exercise and duration. I feel that making radical change (a step change) from a active state to a sedentary state would be worst thing imaginable for any "type A" sane creature. I am sure I would end up in an insane asylum sans at least a little endorphins.

Author: [Jack](mailto:---.dsl.pltn13.pacbell.net) (---.dsl.pltn13.pacbell.net)

Date: 07-28-07 10:10

Gary--I, for one, agree w/your "bottom line" 100%. I have battled AFIB sans drugs & surgery for several years now, while continuing my exercise program, but have been using all of the supplements discussed on this BB. There have been some slightly scary times, but now I think I am coming out on top of the affliction--no AFIB for a while now. I could never even think of a sedentary lifestyle---
May the force be with you!

Author: [uncletommy](mailto:209.115.255.---) (209.115.255.---)

Date: 07-28-07 10:28

Gary: You are coming to the conclusion that many of us have drawn. All this exercise and focus on cardio vascular health may have detrimental effects on how our heart functions. I was a competitive swimmer, played hockey, enjoyed long bike rides and finished off refereeing Senior men's hockey (not for the faint of heart here in Canada). I have always prided myself with being able to keep up with these 30 and 40 year old weekend warriors for a full 60 minutes going red line to red line. That was until I ended up in hospital with an out of control Afib episode on top of hypertension. Afib is an anomaly to normal heart functioning. The fact that you have recognized your condition and are keeping track of what brings it on is just the beginning of your journey to controlling it. I am convinced that there is a very strong correlation between vigorous physical activity and the development of A-fib. Experience will show that the simplest things, from eating to climbing stairs to mowing the lawn may bring it on. Don't go the drug route if you can possibly avoid it. This forum has much of what you require to wrestle this monster to the ground. Ask questions and be willing to experiment. Also realize that you will reach an age where you don't have to prove yourself, physically, to anyone including yourself. I have found lawn chairs and a good book a very good alternative to abusing myself in a hockey arena. I'm not advocating that you roll over and opt for couch potato status, but rather put your physicalness in perspective. My wife walks 6 miles a day and doesn't have any of the problems I have and still looks pretty good at 59.

It's all relative to what you want out of life and how you feel about it. Maybe it's the difference between a road bike and mountain bike. Keep digging and you'll find the solution. Tom

Author: Jackie (---.neo.res.rr.com)

Date: 07-28-07 10:33

Gary - I have been collecting some data on this topic and your post gives me the opportunity give some input.

Having been active on this BB for over 5 years, I have seen a lot of postings similar to yours and I can really appreciate your dilemma. One thing seems to be fairly consistent - heavy exercisers like runners, bikers, etc all seem to be in denial about the 'chink' in their otherwise impervious armor and is a mental attitude that they are invulnerable. Coming to grips with a vulnerability seems to be extremely difficult for many and the younger the person, the more difficult...and understandably so. We never want to give in to a frailty. Even some diehard older athletes seem to have trouble finding a sensible exercise level.

We all were warriors of one sort or another in our pursuit of life and we all were set back on our heels when AF entered our lives. Without exception, everyone admits it has a huge impact on one's life.

However, there is enough science out there to warrant an investigation on your part to learn just how exercise to the extreme - or even just a significant amount of aerobic exercise - can be damaging to the body and especially the heart and related cardiovascular system. I'm not even going to mention the wear and tear on the skeletal system. You need to check out in a Google search terms like oxidative stress damage, reactive oxygen species and free-radical damage, and attempt to understand how that impacts the heart. (I've provided some links below as a start to get you motivated).

It's not at all uncommon for athletes like runners or marathoners to eventually have heart issues and some are fatal – not to mention the brain issues mentioned below in Dr. Blaylock's clip.

I do have some questions for you:

What are you doing to replenish the nutrients your body needs to support your level of exercise? Do you know what your lab values are for magnesium and potassium? Do you take measures to augment with essential electrolytes that your heart and your body need to function well? Do you avoid sports drinks that may contain chemicals that the body considers toxins? Do you take abundant anti-oxidants to help reverse the free radical damage brought on by intense aerobic activity?

Here's a clip on Over-Exercise - Maybe we should title it "Exercise – a double-edged sword? How about a Goldilocks program?"

Over-exercise & Endorphin levels

An interesting section in Julia Ross' book, *The Mood Cure*, is on endorphins, those feel-good hormones that take away pain, give satisfaction to meals, improve mood and act like an opiate in our system.

In a subsection titled, *Why Not use Exercise, Sweets or other Comforts to Raise your Endorphins?* she says while it's true that you should be able to stay high on your own endorphins since things like sunlight, music, romance, exercise and nature can all raise endorphin levels, it can't help if your basic levels are so low you have nothing to work with.

Without adequate levels, you are stuck with an addictive need for exercise, sex, sugar or alcohol and drugs that *force* a brief release of endorphin at a very high price. As an example, she says sex raises endorphin levels 200% and while that sounds great – 200% of nothing is nothing.

She says, "when your natural levels are low, a temporary boost from sex, chocolate or pot won't take you very far for very long."

"Regarding exercise, the short answer is this: If you've exercised hard enough to get an endorphin 'high,' you've exercised too long. Runners' "highs" typically kick in after they have hit the wall of exertion that should have been a signal to stop running. She says do not push with exercise past pain and to forget about that kind of gain as you can be

fit and feel great using moderate exercise and smart nutrition.”

If you are addicted to the exercise high, use DLPA (which she discusses in another section) and taper back to more moderate levels. If you've stopped feeling the high after intense exercise, your endorphin levels have definitely bottomed out. This was documented in studies of endorphin levels in endurance athletes. She says the solution is DLPA, the basic nutrients recommended and plenty of protein....and to reconsider the extent of your exercise.

She gives a Sample of an Endorphin-Building Menu

Breakfast: Three-egg omelette with vegetables and potatoes in the morning; or a protein powder and fresh fruit shake with coconut milk.

Lunch: Plenty of turkey or other meat and cheese on a whole-grain sandwich or in a big salad with plenty of Olive oil and balsamic vinegar salad dressing

Dinner: A substantial piece of fish chicken or lamb with asparagus in lemon butter and a buttered baked potato; or if you are vegetarian – rice and beans with cheese, cashews and vegetables stir-fried in ghee or coconut oil.

Reference:

pp 113-114

The Mood Cure

Julia Ross, MA

Penguin Books

C 2002

Julia Ross, MA, is a nutritional psychologist. Her practice is in Mill Valley, California, and as a result of her books, The Diet Cure and now The Mood Cure, she says over a hundred thousand people have reported success. She wrote the book with the intention to make it reader friendly to self-test and make adjustments in diet and lifestyles to correct mood disorders.

Julia Ross, MA Nutritional Psychologist

Recovery Systems – 415-383-3611 www.moodcure.com

Mill Valley, CA

In another reference:

What is exercise abuse?

Not everyone who exercises becomes obsessive about working out. What separates healthy exercisers from those who abuse exercise is their attitude. For addicts, exercise encompasses their thoughts and moods and dictates their lives.

Exercise is the No. 1 priority in life for exercise abusers, and all activities are scheduled around it. "They make certain life choices based on their exercise routines," says Randi Rotwein, M.A., M.F.C.C., a personal trainer and licensed therapist in Los Angeles, California, who specializes in treating eating disorder and exercise abuse patients.

Exercise abusers may even lie about their exercise patterns. Maybe they weren't able to exercise in the morning, so they make up some excuse and cancel a date that night so they can work out. The more involved they get with exercise, the deeper their addiction becomes. They often feel a loss of control and become so dependent on their workouts that they can't and won't stop, regardless of the cost. In many cases, their dependency on exercise isn't evident until their job, family, injury, etc., interferes with their routine.

Exercise abusers also tend to disregard sickness and injury. No matter how sick they feel or how much they hurt, they continue to push themselves. Sometimes, the consequences of exercising while injured can lead to permanent physical disability which prohibits future exercise, Raglin says.

Like addicts who abuse drugs and alcohol, when exercise levels diminish, exercise abusers experience withdrawal symptoms. Even casual exercisers who don't abuse exercise experience withdrawal symptoms when they can't work out. They may feel antsy, irritated and sometimes become severely depressed. They may also experience sleeping problems, muscle soreness, changes in appetite and mood swings.

Why do people abuse exercise?

Currently, there are no known causes for exercise abuse, or what some call "exercise addiction" and/or "compulsive exercising." Some researchers believe people become addicted to the body's natural release of endorphins when exercising. Those endorphins produce a natural high. However, Raglin says that's unlikely.

"Your body does release endorphins when you exercise," he says. "However, there's no strong evidence that endorphins change the way you feel." Instead, Raglin suggests that psychology plays a larger role than physiology.

In other words, personalities may predispose people to developing an addiction to exercise. People with obsessive-compulsive disorder, for example, may be more prone to developing a heavy reliance on exercise. For others, exercise is a coping mechanism. "They deal with their stress through exercise," Raglin says.

Addicted to Sweat - American Fitness, Nov, 1999

http://findarticles.com/p/articles/mi_m0675/is_6_17/ai_61182000

Also - important for you to read the responses in these threads and consider the use of d-ribose since hearts are muscles and need energy repletion just as skeletal muscles do.

http://www.afibbers.net/forum/read.php?f=6&i=10722&t=10588#reply_10722

http://www.afibbers.net/forum/read.php?f=4&i=17456&t=17456#reply_17456

http://www.afibbers.net/forum/read.php?f=6&i=11450&t=11441#reply_11450

http://www.afibbers.net/forum/read.php?f=6&i=11441&t=11441#reply_11441

Consider that ROS damages cellular DNA –

<http://environ.spawar.navy.mil/randd/BiomarkerLab/DNAdamage.htm>

Here are some comments on exercise by neurosurgeon, Russell Blaylock, MD.

Subject: Exercise

Some notes by neurosurgeon, Russell Blaylock MD on exercise and the heart.

The Blaylock Wellness Report

(Published by Newsmax.com (\$48/year)

Advantages of exercise:

Increases antioxidant enzymes that protect from disease conditions including atherosclerosis.

Improves blood flow

Protect against brain degeneration

Increases, alertness and mental acuity and improves memory

Lowers blood pressure

Reduces risk of diabetes,

Improves lung function

Inhibits depression.

The least fit men have six times more chance of dying of heart attack compared to most fit men...

However... exercise can also be bad for you – can increase disease – even brain disorders.

Studies show exercise increases free radical generation and lipid peroxidation – both contribute to most disease conditions including cardiovascular disease.

Extreme exercise especially, increases metabolism and therefore more generation of free radicals; the higher the metabolism, the more free radicals.

Exercise physiologists warn intense athletes must take high amounts of antioxidants compared to less active people. But vitamin-based antioxidants (vitamins C, E and carotenoids from vitamin A) do not neutralize many of the more destructive free radicals.

Magnesium's role in enzymatic functions are extremely effective in preventing heart attacks and strokes and extreme athletes are told to supplement with magnesium.

Case studies involving marathon runners indicate that at least six months are required to replace the amount of magnesium lost in a single marathon race.

As we know, magnesium prevents inflammation, improves blood flow through vessels, thus the blood acts as an antioxidant and prevents coronary artery spasm.

Case studies point out that marathon runners require at least six months to replace the amount of magnesium lost in a single marathon race. Magnesium is vital as it prevents inflammation, improves blood flow through blood vessels, thus the blood, acts as an antioxidant, and prevents coronary artery spasm.

Most of us have been told at one time or another that we should exercise to prevent heart disease and strokes. But exercise also has many other health advantages.

Exercise increases the antioxidant enzymes that protect us from a number of diseases, including atherosclerosis. In addition, it improves blood flow, protects the brain against degeneration, increases alertness and mental acuity, improves memory, lowers blood pressure, reduces the risk of diabetes, improves lung function, strengthens immunity and inhibits depression.

One study found that the least fit men were six times more likely to die from a heart attack than the most fit men. Yet an increasing number of studies show that excessive exercise is actually bad for you and can greatly increase disease, even brain disorders.

Careful studies have shown that this is because exercise increases free radical generation and lipid peroxidation, both of which are associated with most diseases, including cardiovascular disease. During exercise, especially extreme exercise, the body's metabolism is dramatically increased. Since the major source of free radicals in the body is metabolism, the higher metabolism, the greater the generation of free radicals.

Based on these studies, exercise physiologists are warning intense athletes that they should take a higher amount of antioxidants than less active people. Unfortunately, vitamin-based antioxidants, such as vitamins C, E and the carotenoids, do not neutralize some of the more destructive free radicals.

Athletes undergoing intense, prolonged training or participating in endurance races such as marathons have a high incidence of infection, especially upper respiratory tract infections. Prolonged, exhaustive exercise can lower plasma levels of glutamine, which is an important fuel for certain cells of the immune system.

Glutamine and the Effects of Exhaustive Exercise Upon the Immune Response," Castell LM and Newsholme EA, Can J Physiol Pharmacol, 1998;76:524-532.

From the Ribose Post:

What will ribose do for someone who exercises on a regular basis?

Scientific research shows that three or four workouts per week may not allow enough rest time between sessions for heart and muscle energy pools to return to normal levels. Taking Ribose shortens the time needed by heart and muscle tissue to replace energy that is lost through vigorous exercise. Keeping energy pools full helps to keep heart and muscles in good physiological condition, increase power and endurance, and reduce fatigue. Recent research has also shown that ribose supplementation during exercise reduces free radical formation and lowers cardiac stress associated with hypoxia.

Dr. Johnson says: "In normal healthy hearts that have been stressed with ischemia or hypoxic insult, research shows that it takes more than 10 days to fully recover the energy charge. That's in a normal healthy heart. In muscle, we know it takes more than 3 days because we've done all the studies to show the ATP recovery rate in skeletal muscle following exercise and we know it to be greater than 3 days.

So, with a chronic case of oxygen deprivation, like in heart disease or what I call chronic exercise – when people exercise every day or when high intensity exercise every day or every other day, and the oxygen deprivation is repeated, the tissue simply can't make ribose fast enough to ever recover. [note – emphasizing "ever"]. That's why for example in congestive heart failure, the energy level of the heart continues to go down and down until the patient finally dies.

Type in a PubMed search marathons to read more.

This should give you some food for thought in considering that you need to figure out a way to satisfy your exercise needs but do it in moderation.

I'll be especially interested in learning the answers to the questions posed on what you are doing to replenish nutrients in your body with such extensive exercise.

Author: J (---.dynamic.mts.net)

Date: 07-28-07 12:09

Afib always ends up in the same place for everyone, quality of life. Eventually your quality of life erodes to the point where you have had enough, and ablation and/or maze procedures are entertained. The major difference for all of us is the point at which this decision comes. Some are quite happy to be sedentary as they have been essentially sedentary all their lives. Others, used to an active life style, will not accept being shut down by Afib. Mario Lemeiux, of the Pittsburgh Penguins, was a good example of the latter case. My father, an inactive person, has been on Rythmol for the past 30 years successfully warding off his Afib. I, however, would view his quality of life as a failure in my eyes. To each his own. As far as exercise is concerned, what is the right amount of exercise to maintain your heart muscle in good working order? Are you better off training with occasional LAF or sitting on your duff without it? I believe the latter to be far more life threatening.

Author: [Mark S.](#) (---.bhsn.org)

Date: 07-28-07 13:24

Gary --- I believe the advice of uncltommy mirrors mine as well. I'm reminded of two quotes, the first a Genoese proverb: "It is better to wear out one's shoes than one's sheet." The other, whose author I have forgotten, goes "I cannot have survival as my only goal." I've been an athlete most of my fifty-seven years and a lone atrial flutterer for the last fifteen or so. Give up climbing a mountain to catch a halycon sunset? Avoid a 5K run with autumn leaves swept by winds? Or leave alone a brisk bike along a rushing river? Rubbish. Sure, we get older, our physiology changes, and pathologies creep up and bite us in the butt. But despite my bouts of atrial flutter, I'm still loving what comes natural, still reaping all the benefits of regular exercise, and still wearing out my running shoes and preserving my sheets, and still making something more than survival my goal. My succinct advice: 1) Form a bond with a competent sports-minded physician 2) Have regular checkups to monitor your health. 3) Learn everything you can about atrial

flutter/fibrillation, and absorb the information from forums like this. 4) Adapt, adapt, but don't throw in the towel (I'm convinced that exercise is not the bogey deed that some make it out to be, though it can have byproducts that may require some adjustments -- think electrolyte imbalances, fatigue, dehydration, etc. 5) Keep a positive outlook and avoid people with negative attitudes.

Author: PeterL (---.ri.ri.cox.net)

Date: 07-28-07 13:54

Gary:

I am a 41yo ex-heavy exerciser with afib/flutter.

You said:

"Point well taken, however, how am I too assume that A-fib is the result of being athletic? Furthermore, I am not convinced that not being athletic has caused my A-fib to decrease substantially. "

You need to find out if your exercise is causing your afib/flutter - and if it is, you will be better off in the long run if you stop it now. Taking a week or so off will not do it. Afib/flutter tends to get worse over time. You may come to a point where you can no longer self-convert and you start having to go to the ER for a cardioversion. Then after a few times of that nonsense, ablation starts looking better and better and finally you get one and hope you are cured.

But after the ablation you wonder" hey can I exercise now?". I went through the above chain of events - all the way to the ablation 3mo.s ago for flutter. I wished I had chosen to take 3-4 mos. off from exercise to see if that was the problem - I tried everything else but I was hooked on the way heavy exercise made me feel. They cured my flutter. I can do non-strenuous exercise now without a problem - walking and basic callisthenics (situps pushups etc) without problem, but whenever I get my heart rate up in the 140's 150's I get palps and short afib runs (usually overnight or the next day). The ablation does not cure the underlying issue that makes exercise a no-no.

Take a chance if you can and quit the exercise for a few months.

Author: [Carol A.](#) (---.proxy.aol.com)

Date: 07-28-07 14:14

Gary.

"Sleeping is the real challenge with the palpitations (flutter) so I take Ambien at night. If I could sleep while standing up I would. Perhaps I will design a vertical straight-jacket-bed that will allow me to vertical sleep. "

Have you thought about elevating the head of your bed with blocks (3,4,5" high, whatever works for you) to see if that helps with sleeping. GERD or esophageal reflux can be a trigger for afib?

Also, are you drinking 8 or more glasses of water a day - more when you exercise? Dehydration also triggers afib with some of us.

Carol

Author: [Wil Schuemann](#) (---.dial.gorge.net)

Date: 07-28-07 16:32

Mark S makes an "argument" that the only choices are: (1) "It is better to wear out one's shoes" or; (2) ... "survival as my only goal".

Gary makes a similar "argument". He defines the two choices as: (1) continue to exercise vigorously; or (2) end up in an insane asylum.

This is like "arguing" that the only two choices are: (1) regularly eat with reckless abandon; or (2) always sleep. Such an argument is a transparent attempt to make it look like there is really only one choice.

There are better choices available, but those better choices do not include vigorous exercise. Those better choices will optimize the rest of life, which should be the goal now that a-fib has announced that your heart has deteriorated and will not be fully reliable and trouble free in the future.

You have walked through a door, and it locked behind you.

Welcome to a-fib...

Author: kageyd (---.taconic.net)

Date: 07-28-07 18:05

Wil, you can be relentless - fortunately. And because you are so often dead right on target, certainly this time, this reader is totally supportive. It is what I think the logicians call a "false dilemma." I didn't take the time to look it up, but I believe that is the formal name. Implying that there is no other choice available, and thus being in a "dilemma," when there are many other options available. Thanks, Wil.

Author: J (---.dynamic.mts.net)

Date: 07-28-07 18:36

I think this thread shows that the optimization of one's life can vary widely from person to person. Everyone has their own journey through LAF. We all respond differently to diet and exercise. No generalities can be drawn from either in my opinion. In my personal 18 years with LAF, exercise (Not sure what vigorous means) has reduced my instances of afib. I currently lift weights 3 times/week as well as mountain biking 10 hours per week. I guess, depending on my age, this exercise could be categorized all the way from moderate to vigorous. These terms are vague at best. My last Afib was three years ago and I take no meds.

Author: toni (---.bagu.cable.ntl.com)

Date: 07-29-07 01:49

Gary

You are going to have to strike a balance between exercising and healing just taking supplements and carrying on with your level of exercise is not going to help repair any of the structures in your heart cell membranes they may not be replaced at the level you need or be allowed to heal at your level of activity, you may also have lowered enzyme function within the heart you may need to look further into sports nutrition.

see Jackie's ribose posts

look at amino acids and supplements rich in RNA (brewers yeast and Chinese Chorella)

don't expect a typical healthy diet to fuel an athletes needs

having AF doesn't mean you have to give up exercise but you are going to have to consider changing a few things if you want to get better.

Author: [PeggyM](#) (---.usadatanet.net)
Date: 07-29-07 02:29

I remember how tentative and fragile i felt the first few months when i had, thru diet and supplementation, gotten the afib boogies to go flap their leathery wings someplace else. To my amazement, though, i felt very, very well on the paleo diet, and found my self getting more exercise and just feeling generally vigorous and healthy. Had not felt that way for quite a while. Once i became convinced it was not coming back, though, the feeling of empowerment was tremendous. Keep in touch, Jack.

Author: [GeorgeN](#) (---.hlrn.qwest.net)
Date: 07-29-07 09:10

From my perspective, there is perhaps a middle ground on exercise, between doing nothing and maximum exertion. It is most likely the last 10% or so of effort that causes the most damage.

For example, one can exert oneself for hours at a modest heart rate, and not "hit the wall" or get a runner's high from endorphins. From my perspective, it is the "pushing to the limit" that is most damaging.

As to exercise being a risk, in the Finnish Orienteer study, the orienteers had a 5.5 times greater risk of afib than the controls. <http://www.bmj.com/cgi/reprint/316/7147/1784.pdf> In addition, the controls with afib were also chronic exercisers!

Though I spend a lot of time being active, most times I'm not tired at the end of an activity, because I don't push myself to the limit. That being said, the only people who consider me deconditioned are my mates who think nothing of a 500 mile (805 km) week on their bicycles in the Colorado mountains. However I can still play and keep up with them for most reasonable activities.

Most of my daily activity is at a heart rate less than 130. A significant amount is at 90-100 BPM. Do I let my heart rate exceed 130? Yes, but only in "fun" activities like skiing.

Author: [Matt](#) (---.gv.shawcable.net)
Date: 07-29-07 09:20

Gary, afib, only starts at rest, goes away with exercise, your symptoms seem very similar to mine. After cardioversion and several scary attacks within one year of 1st attack I found this forum and searched for my "triggers" by eating more of things, turns out it was not chocolate :-)

What I did find was 2-3 days after having maybe only one or two beers I would go into afib mildly. I quit drinking and started eating lots of pumpkin seeds (source of magnesium which alcohol depletes) and nuts and fish, salmon mostly. I also cut out all junk food and hydrogenated foods. Not drinking Alcohol means I drink tea and juice more when out at the bar etc.

3 yrs later I have had no afib events and only a few very mild erratic beats for a while but nothing that would stop me sleeping or relaxing.

I do not run but do surf a fair bit and have not noticed any link between surfing for 2-3hrs and afib.

Its no cure but clearly for me it has helped to change my diet and if it can buy me more years without the need for treatment then when I do need treatment technology will have improved hopefully.

Author: J (---.dynamic.mts.net)
Date: 07-29-07 11:02

Hi Peggy,

I have been following the Zone Diet since its origins in 1995 and have been taking PG Fish Oil for four years. Lately, through the influence of many of the kind folks here, I have been following the Paleo Diet and just switched over to NKO Krill Oil as it seems to be slightly more bioavailable than fish oil. I might be a slight oddity here as my occurrences of AF have decreased over time as my fitness has increased. I am 46 with my first occurrences of AF just prior to 30. I have had a total of 5 instances of AF with one cardioversion when I was suffering from Pneumonia. My AF only occurs during the day despite my heart rate of sub 30 BPM during sleep. Daytime resting 48-52 BPM. Hope this helps.

Author: [PeggyM](#) (---.usadatanet.net)

Date: 07-29-07 14:23

Matt, 3 years without afib deserves serious recognition. You quit junk foods and foods containing hydrogenated oils, if i understand you right. Quitting processed foods would rid you of all the additives in these fake foods, as well as the hydrogenated oils and the high glycemic load of these "foods".

And i am sure quitting alcohol helped too, and trigger avoidance. Good for you on all counts. My most sincere congratulations.

Author: [PeggyM](#) (---.usadatanet.net)

Date: 07-29-07 14:28

Hello, J. Your afib decreasing as your fitness rose may have to do with your paleo diet during those years. You are far from the first one to find relief from afib via paleo, as you surely know. Most sincere congratulations.

Author: [James Driscoll](#) (---.bethere.co.uk)

Date: 07-30-07 02:31

Hi George I've always been intrigued by the study you mention...

<http://www.bmj.com/cgi/reprint/316/7147/1784.pdf>

but am concerned that we are lacking some data. This was a study specifically looking at Lone AF but wasn't clear on reporting other types of AF.

The diagram on page 1784 is not complete. We know that from the 228 orienteers with no risk factors 12 developed Lone AF but we are not told how many of the 27 that reported risk factors developed AF.

Similarly although only 2 of the 212 in the control group had LAF we don't know how many of the 133(!) with risk factors developed AF.

The relative risk of developing LAF may indeed be 5.5 but we are not told what the absolute risk of developing all types of AF are (which is actually the number most will be interested in).

and just to really put the cat amongst the pigeons....

The cause and effect is even harder to pin down. I suspect my AF is due to very large pulmonary veins. Large pulmonary veins might mean you can get more oxygenated blood around the body and may predispose someone to be good at orienteering.

Did too much exercise in my early years make me develop large PVs? Would stopping exercise now reduce the size of my PVs?

the study concludes

We do not know whether stopping exercise would have prevented recurrence of atrial fibrillation. Most of the orienteers with commonly relapsing cases had responded adequately to antiarrhythmic drugs and continued competing

Having all 12 respond so well to meds that they could keep competing seems astonishing to me. (and makes me wonder how many different types of Lone AF are out there!)

I'm just trying to highlight we are a long way away from a definitive answer on this topic and, for now(!), am agreeing with you that the 'middle ground' is as good a place as any until we know more. I don't subscribe to the idea that anyone should completely stop exercise (or aim for the minimum amount of walking to tic over) nor do I subscribe to the idea that professional athletes are healthy - they clearly push the bar beyond that limit and induce all sorts of damage.

What appears to quite hard to define is how large this middle ground is. I suspect it covers quite a large range and will accommodate most personalities with only the extremely sedentary and the extremely competitive having to modify their behaviour.

Author: jeff gould (---.bulldogdsl.com)

Date: 07-30-07 04:04

Gary, I was a competitive cyclist for 40 years, the last 8 of which were accompanied by afib. Like you, my afib never occurred during hard exercise, but only when I relaxed. A lot of your correspondents have urged you to stop the exercise. I don't. Some of my best rides were done immediately after regaining normal rhythm. I wouldn't have missed those for anything. I would still be cycling now but for the nerve damage that accompanied a ruptured aneurysm. Afib got worse after that surgery, but I've been pretty well clear of it now for 3 years.

Author: toni (---.bagu.cable.ntl.com)

Date: 07-30-07 05:52

Toni

when you think about the AF coming on after exercise could it be to do with a protein synthesis deficit
i'm not really that well up on the exercise issues and AF but when reading about branch chain aminos they promote protein synthesis after exercise which support cell recovery

i've not read that much yet but if you think about this share of nutrients that we have when you are exercising the heart will be selfish for it needs until you stop and cool down AF starts, but then if you start exercising again you can get yourselves out of AF, is it getting you back into beat because the heart is saying i need the nutrients again for enzyme function and cell recovery

protein synthesis makes enzymes, enzymes are the bodies chemical catalyst which lower activation energy for chemical reactions to happen, the body makes them from the eight essential amino acids and proline, without enzymes in the body no chemical reactions would happen in the body because it runs at 37 degrees it also needs a good source of nucleotides to get the code off DNA to make the enzyme from amino acids (don't forget your tinned sardines)

they are one of the main products of protein synthesis every single chemical reaction the in the heart will require one and they are all coded for in DNA branch chain aminos might be something for the exercisers to look into anyway and

Ribose (important in respiration and makes up part of the backbone of the Messenger RNA molecule see Jackie's Posts).

Author: toni (---.bagu.cable.ntl.com)

Date: 07-30-07 08:02

James

i think you are right about finding a middle ground healthy exercise encourages cell renewal at a normal rate and will have many health benefits but exercising to the point of where you simply haven't got enough nutrients around to do this because you are constantly burning them off is another story who's more likely to have a healthy level of enzymes in the heart cells and healthy turnover and renewal of structures in the heart cell membranes like the sodium ion channels and the Na⁺ K⁺ pump

me who swims for 30 mins three times a week and does a couple of sessions of yoga and has an active job or a competitive athlete that is overdoing things and not listening to their body ??

i'm not saying you can't be a competitive athlete but you will probably need a degree in sports nutrition to make sure you are fuelling your needs.

Author: [Scott](#) (---.hfc.comcastbusiness.net)

Date: 07-30-07 10:38

Gary

I have been watching this board and paying close attention to topics regarding endurance athletes and afib. Had previously been doing marathons, a few ultras, etc till about age 45 when I became more sedentary. At age 50 had first afib in the evening after working hard outside weed whacking with a chain saw. Too much sweating, poor liquid consumption, etc. Electrolytes out of whack. Out of shape as well from my prior level of conditioning.

After this session, and finding this BB, I of course did the research that we all did, and had to make some decisions. From reviewing this BB I soon realized that triggers for some were solutions for others.

My response was to get my self back in shape, eliminate the caffeine, limit alcohol severely, and resume my athletics. Two years later I have had only a single pseudo reoccurrence that I attribute more to falling off the wagon with caffeine, BP control etc. I am racing in triathlons at competitive levels for my age.

I don't know if my endurance training has contributed to the development of my Afib. I do know that with my training now I am in the best shape of my life, and I have a better basis for controlling the other risk factors in my life. Endurance athletes can get heart disease (ask Alberto Salazar), are likely more prone to skin cancer and to being the victim of a hit and run driver while training. I suspect however that our level of conditioning, weight management, cardiovascular health, etc, makes us significantly more healthy than our peers. I also know that as I have gotten back into training I have necessarily paid more attention to my nutrition, and I would not have done this otherwise.

In reviewing some of the responses to this board I hear many people that think you are crazy or taking extreme risks with what you are doing. You, and I, are on a search for our balance. It's easy to say we shouldn't do what we do, but being human as we are, people look for things to fill their life and time, and to stay motivated. At the races I do I see many men in their late 50s thru 70s still competing and great shape. On balance I feel that what I am doing is significantly more constructive than destructive.

As I run nursing homes, I also encounter many people in the same age range come through my facilities, and I can tell you that I have never seen anyone come in from overtraining. All of my "premature" clients suffer from similar combinations of disuse syndrome (little if any exercise as they aged) and poor lifestyle (nutrition, drugs, weight) decisions. Many of these people also suffer from Afib. I know this isn't scientific, but it is my experience.

We may be making a bad decision, who knows. For me, however the risk is worth it given some of the potential alternatives.

Author: [GeorgeN](#) (---.biz.mindspring.com)
Date: 07-30-07 10:51

James,

"What appears to quite hard to define is how large this middle ground is. I suspect it covers quite a large range and will accommodate most personalities with only the extremely sedentary and the extremely competitive having to modify their behaviour."

I would agree. There are obvious benefits to exercise. The orienteers were otherwise more healthy than the controls.

http://www.afibbers.net/forum/read.php?f=6&i=11492&t=11489#reply_11492

In addition, assuming that quite a few of the Olmstead County LAF'ers were exercisers, like the rest of us, they are also healthy compared to controls.

So the question is, "how much is too much?" This will be hard to study effectively. There have been many studies that show that the greatest benefit from exercise is in just getting off the couch (more fit than the bottom 20% of your age group). However most of us that are prone to exercise would like to partake at a higher level. So is being in the top 2% of fitness for your age group too much? How about 5% or 10%?

Your point about whether exercise is actually a cause, or just correlated is also a good one. I do recall that larger hearts were more likely to sustain the reentrant of wavelets. Larger people and exercisers both have larger hearts. Then there is also the issue of atrial stretch and exercise.

Good points.

Author: [Matt](#) (---.gv.shawcable.net)
Date: 07-30-07 23:43

Peggy, its hard to know if its luck or diet. I deliberately avoided magnesium or other supplements as I continue to read reports of the body simply not being able to use many supplements properly.

Seems to me that the long gaps between many peoples attacks could be a indication that its not one thing that triggers there attack but a slow build up of many factors that ends up only needing to be tipped by an event like cold ice drinks or other triggers.

In the 2 mths prior to my 1st attack I had gone from eating meat to vegetarian and as well intentioned as this may have been i feel it is very significant and most likely caused such a huge change in my body that it could not cope and the result was a heart out of balance.

If it is a slow build up then the best response is a slow but permanent change of diet, low fat meat is back on my plate :-) as is more Salmon... yum

http://www.eurekalert.org/pub_releases/2005-01/nhla-cte011005.php (supps and warfarin and aspirin)
http://www.eurekalert.org/pub_releases/2001-01/MC-CaLs-0401101.php (Lead in Calcium supps)
http://www.eurekalert.org/pub_releases/2004-03/acs-os031604.php (fish oil supps)

I continue to check back on this forum and sure think myself lucky, or is it luck?

Author: [PeggyM](#) (---.usadatanet.net)
Date: 07-31-07 02:39

Matt, my bet would be on diet.

" In the 2 mths prior to my 1st attack I had gone from eating meat to vegetarian..."

GeorgeN, another List member, is a vegetarian and is supplementing with Taurine, an amino acid not found in vegetable foods. He uses potassium and magnesium supplements as well, of course. Many of the List members use taurine, myself included, and swear by it to keep us in NSR. The ability to synthesize taurine from diet is variable in different individuals, with some doing better at it than others. My guess is that it is one of the many factors that can tip the balance toward afib in susceptible individuals.

About magnesium supplements being poorly absorbed, that is why we recommend and use magnesium glycinate rather than the common, very cheap, magnesium oxide found in most supplements. It [the oxide form] is as cheap as dirt, while the Mg glycinate is much more expensive, but only about 4% of the oxide form can be absorbed by one's body, making it next to useless for our purposes.

Matt, whatever you are doing, if it keeps you out of afib then it must be right for you. My most sincere congratulations, and thank you for taking the time to answer my post to you.

Author: [Wil Schuemann](#) (---.dial.gorge.net)
Date: 07-31-07 13:21

Human beings don't handle long term slow health problems well because we mostly think short term. If we equate how humans handled smoking/lung cancer and are now handling exercise/a-fib we see similarities.

Sometime in the middle of the 20th century the association between smoking and lung cancer was found. Initially the smokers didn't believe the association. Next they had to accept the association, but thought it was related to something other than the smoking. Next they had to accept that it was something related to smoking, but thought that it was caused by something other than the tobacco, for instance an impurity or compound which was added during manufacturing. Next they had to accept that it was the tobacco itself, but contended that there was no causal proof, so we couldn't be sure you had to stop smoking to reduce the risk of lung cancer. The argument was also raised that smoking conferred benefits as well as possibly causing a problem, and that the benefits outweighed the liability. Along the way many who survived lung cancer via a medical operation, went on smoking believing that getting lung cancer twice in a row was unlikely. Eventually our culture accepted that if you smoked your risk of lung cancer would be some number of times greater than for non-smokers. This decades long sequence was possible because cancer develops slowly. That is important because it lets humans imagine that today's behavior hasn't changed their situation noticeably, therefore their behavior today, which is important to them, probably isn't hurting them all that much.

A-fib is similarly a long term (heart) deterioration problem, also involving decades before noticeable symptoms are noted. In the early 21st century the association between a-fib and exercise was found. Many a-fibbers still refuse to believe the association. Some have progressed to accepting the association, but believe that with "moderation", and supplements, the deleterious effect of exercise can be eliminated. Others have decided that a successful ablation inoculates them from the effect of exercise. Others quibble about the amount of exercise needed to damage the heart, or argue that occasional vigorous exercise is ok. Others argue that exercise confers benefits as well as possibly causing increasing heart electrical system deterioration, and the benefits outweigh any possible increased deterioration. Some are postulating future cures that will solve any problems their vigorous exercise now might be causing.

The same decades long sequence of attitude adjustment that occurred with smoking will likely have to be duplicated with a-fib.

The cultural conditioning and habits associated with a culturally "desirable" behavior (the socially important benefits of smoking then, and social/health benefits of vigorous exercise now) can take a long time to reverse.

Author: [James Driscoll](mailto:---.bethere.co.uk) (---.bethere.co.uk)

Date: 07-31-07 15:12

Wil, that's all fine and dandy but exercise has also been strongly associated with good health and absence of exercise with poor health, so comparing it with smoking is rather misleading. I'm positive that there is overwhelming evidence saying we are better off exercising than not exercising.

We are into this range debate again where too little or too much of thing can be bad for you. These ranges appear to be really poorly researched as far as LAF is concerned so coming to any conclusion now is, in my opinion, premature (particularly given the tenuous link in the sparse research on this topic).

I'm 38 years old and am currently swimming about 30km a month (at around 3kmh). Is this too vigorous ? I suspect it's more than the recommended minimum but a long way away from me worrying Michael Phelps. Until I see some research suggesting I'm doing a bad thing or I get some negative feedback from my heart I'll continue on what I think is healthy ground.

Perhaps a competitive athlete has a much harder decision to make (good luck Gary!) but I suspect the vast majority of us are not in that boat and have little to fear about moderate exercise, whatever 'moderate' means.

Author: [GeorgeN](mailto:---.biz.mindspring.com) (---.biz.mindspring.com)

Date: 07-31-07 15:33

James,

Looking at some of Wil's posts from the 5th archive, he says he exercises at 125-130 bpm.

Here is one thread:

http://www.afibbers.net/forum/read.php?f=4&i=22753&t=22538#reply_22753
http://www.afibbers.net/forum/read.php?f=4&i=22753&t=22538#reply_22753

My guess is that your activity is in the same range, so you really aren't that far apart in how you actually exercise on a daily basis.

Author: Jackie (---.neo.res.rr.com)

Date: 07-31-07 16:23

Wil - Having sat through many presentations that dental professionals needed to know about the pitfalls of smoking so we could educate dental patients who were smokers, I seem to recall that it wasn't until the tobacco companies began tweaking the pure leaves to create cravings etc that suddenly 'they' discovered that tobacco causes lung cancer and, actually, it was the additions of chemicals to tobacco that has been determined to be much of the culprit in tobacco use. While the burning of the tobacco, itself produces harmful chemicals, apparently those added by the tobacco companies add significantly to the potential of the carcinogenicity caused by the burning of the both the tobacco and the chemicals additives. I recall something like several thousand chemicals or toxins are in cigarettes and about 500 or so were actually approved by the Government as additives. Insane.

It's all bad - don't misunderstand. I just think that the tobacco industry helped along the potential of a disease condition resulting from tobacco use.

I'm not a smoker and I don't approve of smoking. I saw many oral pathologies in smokers. We constantly educated on

the pitfalls of smoking as well as smokeless tobacco, but various people 'claim' that smoking pure tobacco isn't anywhere near as harmful as the chemical concoctions offered today by the tobacco industry.

Both exercise to get the endorphin effect or 'runner's high' and smoking are addictions and can be considered in the compulsive behavior realm because they stimulate the same or similar opiate-like receptors in the brain. Often there is a deficiency of neurotransmitters in the brain so the person needs to get the endorphin hit in order to feel decent or human and it's usually serotonin related.

All that said, I don't see at all how smoking and exercise are comparative in terms of health related activities. We know moderate exercise is healthy on many levels. I can't think of one healthy attribute smoking has going for it so I'm confused as to why you picked that as a comparison - other than the denial issue. Did I miss something?

Author: [Wil Schuemann](mailto:---.dial.gorge.net) (---.dial.gorge.net)

Date: 07-31-07 21:12

I picked cultural attitude toward smoking because it illustrated how a culture slowly and reluctantly changes important beliefs when evidence appears that questions the net value of the belief. The cultural change about smoking was one that many of us here lived through and can remember in some detail.

In a similar way, the a-fibber's cultural beliefs about the net value of exercise may go through a slow reluctant cultural change as evidence accumulates.

I will check with my daughter-in-law/physical-therapist, who is deeply involved with the US Olympic teams, and have her give me chapter and verse about how much exercise is necessary to obtain essentially 100% of the health benefits of exercise. It is a trivial amount compared to what most of the a-fibbers do, but I don't know the references and details.

Last week she was in Argentina supporting the Women's Olympic Volley Ball team. She was taking care of some Argentine team member's physical problem. He saw the name Schuemann on her name tag, and asked her if she was related to the Schuemann in the name of my business. She's a very private person who obsessively keeps her private and professional life completely separate. Even in Argentina she couldn't keep her private world separated. I was amused, but she was not.

Author: [James Driscoll](mailto:---.bethere.co.uk) (---.bethere.co.uk)

Date: 08-01-07 00:41

Wil wrote :

In a similar way, the a-fibber's cultural beliefs about the net value of exercise may go through a slow reluctant cultural change as evidence accumulates

Hi Wil, I'm not sure if you meant to say 'Lone AFers' but I believe that word is highly significant....

There's a population of AFers.

There's a subset with Lone AF ('otherwise healthy').

You could split both groups into sedentary, moderate and vigorous exercisers. (hopefully with a good definition of what moderate and vigorous actually means)

There's a population of people without AF.

There's a subset who are otherwise healthy.

Again you can split these into sedentary, moderate and vigorous exercisers.

We could then have all sorts of fun with comparisons. I don't believe any of us would be that surprised to see a lot of people who exercise in the 'otherwise healthy' population WITHOUT AF (I'd love to know if vigorous exercisers have about a 5.5 times greater risk of finding themselves in that group :)

It may well be that some of us would be better off modifying our exercise regimes but I really don't have a good feel for how many us fit into that group.

Author: [James Driscoll](mailto:---.bethere.co.uk) (---.bethere.co.uk)

Date: 08-01-07 03:30

Just in case this very interesting thread gets bumped into the conference room at some point for archiving I thought it may be a good place to also record what's currently in my bookmarks on this subject, I hope it's not too biased :)...

Long-lasting sport practice and lone atrial fibrillation

<http://eurheartj.oxfordjournals.org/cgi/reprint/23/6/477.pdf>

Left Atrial Enlargement in Competitive Athletes Common but Not Pathologic

http://www.medscape.com/viewarticle/510877_print

Prevalence and Clinical Significance of Left Atrial Remodeling in Competitive Athletes

<http://content.onlinejacc.org/cgi/content/full/j.jacc.2005.04.052v1>

Endurance sports is a risk factor for atrial fibrillation after ablation for atrial flutter.

http://www.ncbi.nlm.nih.gov/sites/entrez?db=pubmed&cmd=Retrieve&dopt=AbstractPlus&list_uids=16337500&query_hl=13&itool=pubmed_docsum

Recommendations for competitive sports participation in athletes with cardiovascular disease

<http://eurheartj.oxfordjournals.org/cgi/content/full/26/14/1422#EHI325TB9>

Paroxysmal atrial fibrillation in male endurance athletes. A 9-year follow up

<http://europace.oxfordjournals.org/cgi/content/full/6/3/222>

Atrial fibrillation in elite athletes

http://www.ncbi.nlm.nih.gov/sites/entrez?cmd=Retrieve&db=PubMed&list_uids=9727678&dopt=Citation

Lone atrial fibrillation in vigorously exercising middle aged men: case-control study (this is the same article as the pdf George points to above)

<http://www.bmj.com/cgi/content/full/316/7147/1784>

Author: J (---.dynamic.mts.net)

Date: 08-01-07 06:57

Unfortunately, I see LAF as a symptom having some parallels to headaches. Many people experience headaches but, as we know, there are numerous and varying causes requiring very different treatments. Are the ultimate causes of LAF as broad and varied as with headaches? Not sure, but we will have to be happy treating the symptom for some time. It is no wonder EPs are reluctant giving us advice on diet, supplements and exercise. They don't have a causal clue and neither do we. Hopefully, we will all continue to conduct our individual experiments with this underlying complexity in mind.

Author: [GeorgeN v/52/na](mailto:---.hlrn.qwest.net) (---.hlrn.qwest.net)

Date: 08-01-07 07:02

Here are data from a prospective study of fitness & mortality. Subjects were divided into quintiles of fitness. Here is the data as deaths per 10,000 person-years of follow up:

Fitness - low to high,
Quintiles, 1, 2, 3, 4, 5
Male, 64.0, 25.5, 27.1, 21.7, 18.6
Females, 39.5, 20.5, 12.2, 6.5, 8.5

There is certainly a diminishing return to added fitness, the biggest gain is going from quintile 1 to 2, or "getting off the couch." However there is generally added benefit at each step up the fitness ladder. The optimal quintile for females appears to be the 4th quintile.

<http://jama.ama-assn.org/cgi/content/abstract/262/17/2395>

I got the detailed data from a source other than the abstract.

Author: PeterL (---.ri.ri.cox.net)
Date: 08-01-07 08:40

This is a very important thread to many people who exercise and I think there are two questions that we are trying to answer:

1. For people who do not currently have lone afib, what is the effect of moderate/heavy exercise on causing afib problems to start?
2. For people who already have lone afib, what effect does continuing moderate/heavy exercise have on the progression of the afib?

I think we should look at these questions outside of considering the overall health benefits of exercise. It would seem that if the answer to question 1 is that exercise causes lone afib (as was somewhat shown in the Finnish Orienteer study where the orienteers had a 5.5 times greater risk of afib than the controls) then the answer to question 2 would be that continuing moderate/heavy exercise will continue the progression of afib.

Possibly, if a person is genetically destined to get afib sometime in their 60's that heavy exercise cause the problem to start much sooner, say in their 40's or 50's.

Author: [James Driscoll](#) (---.bethere.co.uk)
Date: 08-01-07 09:38

Hi Peter - I think there's a few of us interested in question 3.

3. for those lone AFers who are now back in NSR after successful management what effect will moderate/heavy exercise have on AF recurrence.

and with all due respect I don't believe the Finish study does answer question 1 because of poor control group selection and some questionable logic.

Author: PeterL (---.ri.ri.cox.net)
Date: 08-01-07 10:41

Hi James D.

I agree with you on questioning the validity of that Finish study, but I don't know of any other studies that have tried to answer the question.

I notice you said "successful management" rather than "cure" for afib. I don't think the "successful management" of afib we have so far (drugs, supplements and ablation) does anything to hit the root cause of lone afib. In contrast, my gut feeling is that heavy exercise does aggravate the root cause of the lone afib.

A possible question 4 is: If exercise aggravates the root cause of lone afib, stopping exercise should reverse or slow down the process. How long will it take for a measurable change to occur?

As an aside, I think when we talk about "lone afib" there seems to be a notion sometimes that the heart is physically / electrically normal except for the fact that it occasionally goes into afib. I think we need to remember that the real definition of lone afib is that its cause is unknown and there may in fact be a physical problem.

Author: [Gunnar 62/v/na](mailto:Gunnar.62@v.na) (---.12-2-64736c11.cust.bredbandsbolaget.se)

Date: 08-01-07 10:42

I do not see exercise as a factor for hurting the heart, as tar in the cigarette smoke will cause lung cancer. The problem is already there, as ectopic foci around the pulmonary veins already exist, due to imperfections, when the heart was built in the foetus or due to genetic problems. The exercise is simply damasking this imperfection by setting up the conditions for AF by bigger geometrical dimensions of the heart, which results in lower heart rate. The lower heart rate removes protection of the overdrive suppression and the bigger mass of the heart favours the creation of wavelets and AF is there and starts its own remodelling process.

Atrial Fibrillation: From Cell to Bedside

<http://www.medscape.com/viewarticle/422901>

Overdrive suppression

<http://cvphysiology.com/Arrhythmias/A018.htm>

At an echocardiogram the EP will not find the heart too big if he knows that nation the patient is heavy exerciser, as he also am aware of that the enlargement will go back, if the exercise stops or is reduced. However the hypertrophied heart could mean the difference between having AF and not having AF as the geometrical dimensions permit a wavelength, that promotes AF. It will take several month of no exercise for the heart to get back to a more normal size. My own heart has reduced its size since 2000 every year, but I had hypertension and also was doing a lot of bike riding.

Author: [Scott](mailto:Scott@hfc.comcastbusiness.net) (---.hfc.comcastbusiness.net)

Date: 08-01-07 10:49

I want to thank James for the links to the studies. I also want to thank Wil, for his ideas, while I will admittedly hope he is not entirely correct.

As I had my AF episode after a prolonged period of seriously undertraining, and was never a consistent athlete, I am not sure if my experience fit the extreme model when I had my session, and I have been more vigorous since without further episode.

As I have a pretty high stress job I do at times suffer from pre-hypertension. I have adapted my diet to deal with this - try to follow a DASH program, etc. I am usually able to keep my BP without normal range however at times it goes up a level of about 135/85. I can usually attribute this to something I am dealing with at the time.

I find that my Systolic will drop significantly more than my Diastolic. I tend to have about a 40 point variance when the Systolic goes down to 120/80. Generally, when I feel my BP has gone up, it is more the Diastolic that tends to be

toward the high end for the normal range, rather than the Systolic.

With this thread, I was wondering if there was any correlation between a higher Diastolic and being more athletic. One of the studies that James quoted seemed to imply that.

Anyone know?

Author: [Wil Schuemann](mailto:---.broadband.gorge.net) (---.broadband.gorge.net)

Date: 08-01-07 13:02

The following thought is embedded in the above discussion, but I thought it would be useful to make it explicit.

(State 1) If you are a member of the general population there is information pertaining to the effect of exercise for the general population.

(State 2) If you are a member of the population of lone a-fibbers, the information pertinent to (State 1) may not be valid for lone a-fibbers. Lone a-fibbers may eventually be able to obtain information specifically pertaining to the effect of exercise for lone a-fibbers.

(State 3) If you a member of the population of permanent a-fibbers, information pertinent to (State 1) and/or (State 2) may not be valid for permanent a-fibbers. Permanent a-fibbers may eventually be able to obtain information specifically pertaining to the effect of exercise for permanent a-fibbers.

(State 4) If you are a member of the population of successfully ablated a-fibbers, information pertinent to (State 1) and/or (State 2) and/or (State 3) may not be valid for successfully ablated a-fibbers. Successfully ablated a-fibbers may eventually be able to obtain information specifically pertaining to the effect of exercise for successfully ablated a-fibbers.

My instinct is that the information(s) about the effect of exercise pertinent to (State 2), (State 3), and (State 4) will be similar, but all these information(s) will be quite different from the information applying to (State 1). However, the (State 2) information will be least different from the (State 1) information, and (State 3) and (State 4) progressively more different.

Statistically, for the entire general population, progressively more exercise progressively improves longevity. My guess is that statistically, for a-fibbers (State 2, State 3, and State 4), a little exercise will improve lifespan, but progressively increasing exercise above a little exercise will progressively decrease lifespan. The optimum amount of exercise for (State 2) will be greater than for (State 3) and (State 4), with the latter States having similar low optimum levels of exercise.

Author: [GeorgeN](mailto:---.biz.mindspring.com) (---.biz.mindspring.com)

Date: 08-01-07 13:34

From one of James's references:

"Cox proportional hazard calculations revealed a 10% and 11% increased risk for AF development per weekly hour sport performed before and after ablation respectively ($p < 0.01$ for both)."

http://www.ncbi.nlm.nih.gov/sites/entrez?db=pubmed&cmd=Retrieve&dopt=AbstractPlus&list_uids=16337500&query_hl=13&itool=pubmed_docsum

My own bias is that it would be interesting to see if there was any accounting for exercise intensity, as well as duration.

Author: [Wil Schuemann](mailto:---.broadband.gorge.net) (---.broadband.gorge.net)
Date: 08-01-07 16:01

Another interpretation is that the same historical/genetic/behavioral factors dispose an athlete for both a-fib and a-flutter. This assumes that the a-flutter and a-fib develop in parallel (are not dependent on each other), rather than a-fib being the result of the a-flutter or the flutter ablation.

Then, the successful ablation treatment of the a-flutter would not necessarily affect the probability of developing a-fib. If so, then the probability of developing a-fib before and after the ablation would be expected to be the same in the study population, as they found to be true.

The conclusion from the study: "A history of endurance sports activity is associated with the development of AF after ablation of atrial flutter", should maybe have been worded; "A history of endurance sports activity is associated with the development of AF [both before and] after ablation of atrial flutter".

Author: [Eric Oddleifson](mailto:---.hsd1.ma.comcast.net) (---.hsd1.ma.comcast.net)
Date: 04-27-08 06:52

Hi all. I am brand new to this board but the topic really interests me. I have had LAF for about 10 years now (I'm 40) It developed during a relatively sedentary time of my life. I would get episodes only a few times a year and they would last for 4-8 hours. A few years ago, the episodes increased dramatically to every 1-2 weeks for 12 hours on average.

As my sense of control began to deteriorate, I decided I could not let LAF 'decide' my activities for me. So, I started racing mountain bikes. Yes, sometimes after a race when my HR is decelerating, I go into AF. But, overall I feel hard exercise on an intermittent basis is preventative. I can go 6-8 weeks without AF when I am actively training. When I stop, AF episodes increase in frequency. I tried propafenone a couple of years ago with no positive effect. It made it worse. I would get AF every 2-3 days while on the med. I don't have a special diet or take supplements at this time and realize I will probably have to undergo surgery in the future if I want to try and cease it for good. At least that is my thinking at this point.

My point is here that I do think too much intense exercise will predispose you to an episode of AF but it is so individual specific that it is difficult to make sweeping generalizations. Also, I can understand how fatiguing the heart during intense exercise could lead to an aberrant signal from a PV or some other place gaining the upper hand in pacing the atria but why would moderate exercise in general increase that chance? I can get AF sitting on the couch at night doing nothing. That has always been the case. I would not conclude that exercise is negatively affecting my condition.

I think all people who have LAF need to try and do something to gain that sense of control. At least that is true for me. Two weeks ago, my race was about to start, then boom..AF. I had to drop out after .5 mile. That sense of unknowing and frustration is difficult to deal with so I have to do something for my own mental health. It seems 'that something' is different for different people.

Author: [GeorgeN](mailto:---.hlrn.qwest.net) (---.hlrn.qwest.net)
Date: 05-21-08 21:47

Hans,

I also suggest you add this thread (or most of it) on Exercise, inflammation, oxidative stress, training and supplements to the PDF: http://www.afibbers.net/forum/read.php?f=7&i=7874&t=7874#reply_7874

In addition, Jackie has posted on oxidative stress and exercise. Jackie, is there data regarding the change in oxidative stress and exercise. In other words, how much is too much? I know from how my body reacts that there is a huge difference between going for a stroll for an hour and running at maximum pace for 4 or 6 hours. I have almost instantaneous recovery from the first. In other words, Jackie, is there a way to quantify the oxidative stress. I'm guessing that this is a major component of the negative effects of exercise and afib.

Author: [Dick](#) (71.94.185.---)
Date: 05-09-08 15:08

EXERCISE AND ATRIAL FIBRILLATION

There has been considerable discussion here on the relationship between various levels of “exercise burden” -- defined as session duration x intensity x frequency x number of years -- and AF.

For many, a high level of exercise confers benefits in addition to oft-reported effects on cardiac and physical health, including self-esteem, status, the camaraderie of competition and a uniquely good feeling that has been labeled “the runner’s high”. Because of the special significance of exercise in so many people’s lives, it is understandable that people do not want to give up their “positive addiction”.

Alarms were raised as early as the late 1990’s, most popularly in Ken Cooper’s book “The Antioxidant Revolution” published in 1997, which pointed out the dangers of the free radicals generated by prolonged exercise. He counseled both cutting back on aerobic exercise -- to less than 40 minutes a day, if I remember correctly -- and taking antioxidants to minimize free radical damage. (You may remember that Dr Cooper was an early advocate of long-distance running and other aerobic exercise.)

A recent article by Hans Larsen in the May 2008 AFIB Report has surveyed the literature on the effects of exercise on heart structure and function and the potential of these changes to contribute to AF.

Here are some of the things you will find in this article:

>> Specific guidelines for the amount of exercise needed for optimum health and cardiac benefit for most people.

>> Convincing evidence that a heavy exercise burden is a risk factor for the development of AF. Especially thought-provoking are reports that detraining (cutting back on exercise) may be a helpful or even crucial part of treatment.

The studies cited leave a big grey area between the moderate exerciser who might spend several hours per week walking vigorously and doing a couple of hours of weight training, and the serious or professional endurance athlete whose training might consist of, say, five days a week running 5-10+ miles a day, or bicycling 20-50 miles a day, with a race on the weekend, for most of the year, over a period of many years. The training would include “hard” days which could be close to a race pace that most amateurs could not approach.

So, what is the person who falls in this grey area to do?

I do not find that research can answer this question definitively, but there may be some ways to cut the risk of exceeding the bare minimums noted in Han’s article.

A brief summary and conclusions section is next, followed by excerpts from relevant studies.

SUMMARY AND CONCLUSIONS

1) Exercise creates oxidative stress and inflammation, which can create significant damage in people who increase their level of exercise too rapidly or to a level too high for their body to adapt. Gradually increasing your exercise level may reduce or prevent such damage.

The CRP test for inflammation is readily available. If CRP levels are elevated, either sustaining to high a level of exercise or increasing your level too fast might be the reason? I have not heard of its being used for this purpose.

Markers for ROS (“reactive oxygen species”, which include free radicals) are more difficult to measure. There are symptoms of overtraining (see the very end of this article) which can be heeded by the person who wishes to avoid the emergence – or re-emergence-- of AF.

2) Use of antioxidants (vitamins C, E and others) may help prevent the effects of exercise to which the individual has not adapted. Diet (i.e. the DASH diet) may be equal or superior to supplementation.

Substances as diverse as statins, aspirin, and possibly fish oil and ACE inhibitors may be helpful anti-inflammatories.

Such measures may be especially important for older people.

FURTHER WORK IS NEEDED...

Evaluate or refine the usefulness of physiological or behavioral markers for inflammation or oxidative stress that can help to define the limits to which a person can push his workouts.

Figure out how to determine the changes to the heart that have already occurred from years of exercise.

Determine the usefulness of antioxidant supplements, anti-inflammatories or diets in preventing damage from exercise-induced inflammation or oxidative stress.

Finally, there is this from the Medical Hypotheses site:

"Further searching and literature analysis revealed that excessive endurance exercise or overtraining can lead to chronic systemic inflammation and, separately, that there is a solid association between CRP and AF and that anti-inflammatory agents have been reported to lower CRP and ameliorate AF. No articles were found that brought together all three concepts – AF, inflammation, and exercise.

The following hypothesis is plausible, readily testable, and apparently novel: Older athletes diagnosed with AF but otherwise healthy who have engaged in rigorous aerobic endurance exercise for more than a decade will have CRP levels that are higher than those of a similar population of athletes without AF. Corroboration of this hypothesis would then justify a prospective clinical trial of anti-inflammation therapy. It is of particular interest to extend recent studies of inflammation in AF to athletes; athletic behavior that can induce inflammation may contribute to understanding the origins of AF."

<http://www.medical-hypotheses.com/article/PIIS0306987706000478/fulltext>

The topics that are covered in the excerpts below are:

- 1) Exercise and inflammation or oxidative stress in trained and untrained individuals
- 2) The relationship between inflammation or oxidative stress and AF
- 3) Exercise and the immune system
- 4) What you can do

Definitions:

Inflammation (note that fibrosis is a possible outcome):

<http://en.wikipedia.org/wiki/Inflammation>

Oxidative stress:

http://en.wikipedia.org/wiki/Reactive_oxygen_species

THE CONNECTION BETWEEN EXERCISE AND INFLAMMATION OR OXIDATIVE STRESS IN TRAINED AND UNTRAINED INDIVIDUALS

Indeed, there is evidence for the inflammation-suppressing effects of exercise. A recent review concluded that a single bout of strenuous exercise produces a short-term, transient increase in plasma levels of C-reactive protein. The C-reactive protein increase is due to an exercise-induced acute phase response, mediated by the cytokine system and mainly IL-6.² In contrast, exercise training may blunt the acute pro-inflammatory response, and even suppress the inflammatory process, thereby contributing to the beneficial effects of habitual physical activity.² There also appears to be an acute homeostatic anti-inflammatory response after a bout of strenuous exercise that counteracts the pro-

inflammatory response.2

<http://eurheartj.oxfordjournals.org/cgi/content/full/27/11/1385-a>

Physical activity is associated with a reduced incidence of coronary disease, but the mechanisms mediating this effect are not defined. There has been considerable recent interest in inflammation in the pathogenesis of cardiovascular disease. Some of the beneficial role of physical activity may result from its effects on the inflammatory process. We searched PubMed for articles published between 1975 through May 2004 using the terms exercise, physical activity, or physical fitness combined with C-reactive protein, inflammation, inflammatory markers, or cytokines. The review revealed 19 articles on the acute inflammatory response to exercise, 18 on cross-sectional comparisons of subjects by activity levels, and 5 examining prospectively the effects of exercise training on the inflammatory process.

Exercise produces a short-term, inflammatory response, whereas both cross-sectional comparisons and longitudinal exercise training studies demonstrate a long-term "anti-inflammatory" effect. This anti-inflammatory response may contribute to the beneficial effects of habitual physical activity.

http://content.onlinejacc.org/cgi/content/abstract/45/10/1563?ijkey=7dc1d010ac2ceae03d8dc7f29c35f9e36c24b36&keytype=tf_ipsecsha

The quicker the individuals were able to get back to their resting heart rate after a strenuous exercise test was inversely related to their CRP," Vieira said. "In other words, individuals who had better parasympathetic tone had lower levels of inflammation..."

"And the reason we're excited about this is that exercise is a great way to improve parasympathetic tone. When you exercise – that is the sympathetic/parasympathetic communication – your sympathetic goes up, and when you stop exercising, your parasympathetic kicks in to bring you back to normal. An untrained person will take a while to get their heart rate back down to resting. A trained person's heart rate will come back down very quickly."

<http://www.physorg.com/news102949890.html>

In summary, acute exercise increases oxidant levels and oxidative stress in untrained animals [4,5], but long-term exercise may counter this effect by increasing the activity of antioxidant enzymes and reducing oxidant production [64-67,21,22]. These defenses may be critical for preventing chronic oxidative damage to muscle during exercise and even at rest.

<http://plaza.ufl.edu/cleeuwen/CMC8.PDF>

Exercise-induced oxidative stress affects erythrocytes in sedentary rats but not exercise-trained rats

<http://jap.physiology.org/cgi/content/abstract/91/5/1999>

THE RELATIONSHIP BETWEEN INFLAMMATION OR OXIDATIVE STRESS AND AF

Inflammation may also contribute to the persistence of atrial fibrillation in non-surgical patients. We recently observed that the levels of C-reactive protein (CRP), a sensitive marker of the systemic inflammatory state, are elevated in patients with atrial fibrillation, and that the degree of elevation was related to the persistence of atrial fibrillation. That is, CRP levels were elevated in patients with paroxysmal AF relative to the controls. However, the levels were even more elevated in patients with persistent AF. (Ref 3)

We conclude from these studies that oxidative stress may have an important role in the atrial pathologies associated

both with rapid atrial rates, and with the inflammation-mediated postoperative arrhythmias.

http://www.clevelandclinic.org/heartcenter/pub/atrial_fibrillation/AFresearch.htm#oxidativestress

Patients who exhibit PAF after cardiac surgery have significantly increased acute oxidative stress, which translates into increased myocardial oxidation. Also, patients with PAF have a differential oxidative genomic response after cardiopulmonary bypass that may predispose them to oxidative stress.

http://www.sciencedirect.com/science?_ob=ArticleURL&_udi=B6T11-4PPMVNF-M&_user=10&_rdoc=1&_fmt=&_orig=search&_sort=d&_view=c&_acct=C000050221&_version=1&_urlVersion=0&_userId=10&md5=a7618147f88446493eefe28e75e0c719

There is growing evidence that oxidative stress is involved in the pathogenesis of atrial fibrillation. Many known triggers of oxidative stress, such as age, diabetes, smoking, inflammation, and renin angiotensin system activation are linked with an increased risk of the arrhythmia. Blockers of angiotensin II signaling and other drugs with anti-oxidant properties can reduce the incidence of atrial fibrillation. Now, studies in animal models and human tissue have shown directly that atrial fibrillation is associated with increased atrial oxidative stress. We review the evidence for a role of oxidative stress in causing atrial fibrillation and propose a unifying hypothesis that multiple triggers elicit oxidative stress which acts to enhance the risk of atrial fibrillation through ion channel dysregulation.

<http://www.ingentaconnect.com/content/ben/ccr/2006/00000002/00000004/art00002>

Recent interest has been directed to the stimulus or 'drive' that increases the risk of development and recurrence of AF and its complications, especially thromboembolism. Inflammatory cytokines have been postulated as possible candidate stimuli. Some evidence to support an association between AF and inflammation can be extracted from the frequent association of AF with inflammatory conditions of the heart, such as myocarditis and pericarditis.[7,8]

Indeed, Bruins et al.[9] were the first to propose a direct link between inflammation and AF by observing an increased frequency of AF after coronary artery bypass surgery, where the peak incidence of AF occurred on the second and third postoperative day, coinciding with the peak elevation of C-reactive protein (CRP).

Atrial biopsies taken from patients in AF have also demonstrated evidence of inflammatory infiltrates within the atrial tissue with evidence of oxidative damage or occult myocarditis, even among persons who were thought to have had lone AF.[10,11]

Whilst clinical data continue to be debated, cogitated and (maybe) regurgitated, perhaps attention should be drawn to the mechanism(s) leading to abnormal inflammation in AF. The precise mechanisms are uncertain, but the possibility remains that cytokines and CRP might reflect active participation of CRP in the local inflammatory response within the atrial myocardium. In human coronary disease models (where inflammation has been clearly linked to thrombosis), deposits of CRP have clearly been demonstrated, on immunohistochemical staining, in the vascular wall of active atherosclerotic plaques, where it is co-localized with the terminal complement complex.[22] Consistent with this, Roldan et al.[23] reported high levels of IL-6 in AF, but >>> this appears to be more related to clinical variables of the patients rather than to the presence of AF per se. Thus, the inflammatory state in AF may simply reflect associated vascular disease in AF. <<< [!!!???

An association between inflammation and AF is undoubtedly present, but unfortunately, association does not equate to causation. Further studies are clearly needed to better understand highly complex interaction. We have commenced a new and exciting chapter in the understanding of the pathophysiology of AF in which therapeutic options involving anti-inflammatory agents may potentially play a major role.

<http://www.medscape.com/viewarticle/509538>

Atrial fibrillation (AF) has been associated with myocardial oxidative stress, and antioxidant agents have demonstrated antiarrhythmic benefit in humans. We compared serum markers of oxidation and associated inflammation in individuals

with or without AF.

These data suggest that oxidative stress markers may have predictive value in AF management.

<http://www.clinchem.org/cgi/content/abstract/53/9/1652>

Atrial fibrillation (AF) is the most common cardiac arrhythmia, associated with a five- to six-fold increase in the incidence of stroke due almost exclusively to embolization of thrombus formed in the left atrial appendage. Whereas epidemiological data links AF to increased mortality, recent clinical trials show no improvement in outcomes with strategies designed to maintain sinus rhythm.

One possible explanation for this AF paradox is that AF is a manifestation of an underlying disease process that is not addressed by current treatment strategies. Several lines of evidence suggest that oxidant stress may contribute to the atrial remodeling and the hypercoagulable state associated with AF. These alterations involve an increase in reactive oxygen species and a concomitant decrease in the bioavailability of nitric oxide. Recently, we observed this situation in the left atrial appendage. Increased oxidant stress may be central to the perpetuation of AF, and failure to address this underlying pathology may explain the lack of efficacy seen in clinical trials.

<http://cat.inist.fr/?aModele=afficheN&cpsidt=15207286>

EXERCISE AND THE IMMUNE SYSTEM

http://sportsmedicine.about.com/od/injuryprevention/a/Ex_Immunity.htm

WHAT YOU CAN DO

The association between AF and inflammation has raised potential therapeutic implications. For example, Dernellis and Panaretou[24] were able to demonstrate that the use of >*low dose glucocorticoids [steroids such as prednisone]*< not only improved the efficacy of sinus rhythm maintenance postcardioversion but was reflected by a fall in CRP levels. Also, >*statins*< possess additional anti-inflammatory, antihypertrophic, antifibrotic and antioxidant properties, and these agents appear useful for the primary and secondary prevention of AF.[25-27]

Recent attention has also been focused on the potential anti-inflammatory, antioxidant and antiarrhythmic properties of >*oily fish*<, containing a high content of omega-3 fatty acids. In a prospective, population-based cohort of 4815 older (≥65 years) Mozaffarian et al.[28] showed that consumption of high levels of fish containing omega-3 fatty acids was associated with a lower incidence of subsequent AF development. Unfortunately, an even larger prospective study of 47,949 participants (mean age 56 years) found that consumption of omega-3 fatty acids from fish was not associated with a reduction in risk of AF.[29]

As inflammation is closely related to oxidative stress, perhaps some lateral thought is needed. Indeed, >*ascorbic acid (Vitamin C)*< is a water-soluble antioxidant that has been shown to attenuate atrial electrophysiological remodeling and reduce the incidence of postcoronary artery bypass surgery, possibly through scavenging peroxynitrite and other reactive oxygen species.[30,31]

<http://www.medscape.com/viewarticle/509538>

The observation that atrial tissue from patients with persistent AF is marked by signs of increased oxidative stress (ref 1) led us to hypothesize that treatments that either scavenge or prevent free radicals might alter the electrophysiological and/or structural remodeling processes associated with AF.

- In a recent study we have shown that the >*antioxidant ascorbate (vitamin C)*< can attenuate the electrical remodeling that accompanies rapid atrial pacing in an experimental model. In this study, the atrial tissue subjected to rapid atrial pacing showed direct evidence of increased oxidative stress (increased 3-nitrotyrosine formation), and ascorbate was able to minimize this effect. Further, >*supplemental ascorbate*< also helped to prevent tissue

depletion of endogenous ascorbate. (Ref 2)

• Following cardiac surgery, many patients experience transient episodes of atrial fibrillation. This arrhythmia follows a time course very similar to that of the inflammatory response following cardiac surgery. In the same study (ref 2), we reported the results of a pilot study in which we evaluated the impact of >supplemental ascorbate< on the occurrence of atrial fibrillation following coronary artery bypass graft surgery. In the patients receiving supplemental ascorbate, 7/43 (16%) had post-operative atrial arrhythmias. In contrast, 15/43 (35%) experienced postoperative arrhythmias in an age- and gender matched control population. Thus, in this pilot study, >supplemental ascorbate usage< was associated with a 50% reduction in the incidence of postoperative atrial arrhythmia. In view of this promising result, a new study, being performed in a randomized, blinded, and placebo-controlled fashion is underway to better evaluate the statistical significance of the pilot study.

http://www.clevelandclinic.org/heartcenter/pub/atrial_fibrillation/AFresearch.htm#oxidativestress

Atrial fibrillation (AF), the most common chronic arrhythmia, increases the risk of stroke and is an independent predictor of mortality. Available pharmacological treatments have limited efficacy. Once initiated, AF tends to self-perpetuate, owing in part to electrophysiological remodeling in the atria; however, the fundamental mechanisms underlying this process are still unclear.

We have recently demonstrated that chronic human AF is associated with increased atrial oxidative stress and peroxynitrite formation; we have now tested the hypothesis that these events participate in both pacing-induced atrial electrophysiological remodeling and in the occurrence of AF following cardiac surgery. In chronically instrumented dogs, we found that rapid (400 min⁻¹) atrial pacing was associated with attenuation of the atrial effective refractory period (ERP). Treatment with >ascorbate<, an antioxidant and peroxynitrite decomposition catalyst<, did not directly modify the ERP, but attenuated the pacing-induced atrial ERP shortening following 24 to 48 hours of pacing. Biochemical studies revealed that pacing was associated with decreased tissue ascorbate levels and increased protein nitration (a biomarker of peroxynitrite formation). >Oral ascorbate supplementation< attenuated both of these changes. To evaluate the clinical significance of these observations, supplemental ascorbate was given to 43 patients before, and for 5 days following, cardiac bypass graft surgery. Patients receiving ascorbate had a 16.3% incidence of postoperative AF, compared with 34.9% in control subjects.

In combination, these studies suggest that oxidative stress underlies early atrial electrophysiological remodeling and offer novel insight into the etiology and potential treatment of an enigmatic and difficult to control arrhythmia.

<http://www.circresaha.org>.<http://www.ncbi.nlm.nih.gov/pubmed/11557745?dopt=Abstract>

The most important result of our study was that the rate of vitamin E utilization increased during endurance exercise compared to the sedentary period. This is the first time that exercise has been reported to increase the rate of vitamin E utilization, a discovery that indicates increased oxidative stress during exercise. The increase in F2-isoprostane levels observed during the ultramarathon further supports our hypothesis that the runners experienced increased oxidative stress.

<http://lpi.oregonstate.edu/ss02/vitamine.html>

Several lines of evidence suggest that among different forms of vitamin E, >α-tocopherol (AT)< has potential beneficial effects with regard to cardiovascular disease. AT supplementation in human subjects and animal models has been shown to decrease lipid peroxidation, superoxide (O₂⁻) production by impairing the assembly of nicotinamide adenine dinucleotide phosphate (reduced form) oxidase as well as by decreasing the expression of scavenger receptors (SR-A and CD36), particularly important in the formation of foam cells. AT therapy, especially at high doses, has been shown to decrease the release of proinflammatory cytokines, the chemokine IL-8 and plasminogen activator inhibitor-1 (PAI-1) levels as well as decrease adhesion of monocytes to endothelium. In addition, AT has been shown to decrease CRP levels, in patients with CVD and in those with risk factors for CVD. The mechanisms that account for nonantioxidant effects of AT include the inhibition of protein kinase C, 5-lipoxygenase, tyrosine-kinase as well as cyclooxygenase-2.

Based on its antioxidant and anti-inflammatory activities, AT (at the appropriate dose and form) could have beneficial effects on cardiovascular disease in a high-risk population.

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The DASH trials featured controlled nutrient intake and repeated measurements, allowing for longitudinal analyses of biomarkers and traditional CVD risk factors. Those participants following the >*DASH diet*< had increases in serum antioxidant and ORAC levels, and reduced breath ethane levels. Similarly in the DASH Sodium Trial, those on the DASH diet had increases in serum antioxidant and ORAC levels, lowered F2-isoprostane levels and increased antibodies to oxidized LDL. Thus modulation of oxidative stress biomarkers was demonstrated to be achievable with dietary changes.

<http://www.nhlbi.nih.gov/meetings/workshops/oxidative-stress.htm>

Endurance exercise causes increased oxidative stress, which may be exacerbated if the exercise is irregular. Exercise training, however, has a positive effect upon antioxidant enzymes that act to reduce oxidative stress during exercise. While there is little definitive evidence to suggest that >*antioxidant supplementation*< is beneficial to athletic performance, it seems clear that >*dietary antioxidants*< can reduce the oxidative damage to muscles and other tissues caused by exercise. The long-term effects of antioxidant supplementation is not fully understood at present. But the evidence suggests that it may be beneficial for individuals performing regular heavy exercise.

<http://www.pponline.co.uk/encyc/0888.htm>

These results suggest that the dynamic handgrip exercise is a good model for studying the post-exercise oxidative stress and also that >*ASA (acetylsalicylic acid)*< seems to offer an efficient protection against oxidative stress and the changes in membrane permeability to potassium.

http://www.sciencedirect.com/science?_ob=ArticleURL&_udi=B6X16-45RDXDJ-8&_user=10&_rdoc=1&_fmt=&_orig=search&_sort=d&view=c&_acct=C000050221&_version=1&_urlVersion=0&_userid=10&md5=71117c2e230196795ff2b0ffd483940c

Athletes regularly participating in up to 40 min of acute high-intensity exercise may require higher intakes of exogenous antioxidants to defend against increased oxidative stress during exercise, which can be met through an >*adequate intake of high-antioxidant foods*<. Thus, there seems no valid reason to recommend antioxidant supplements to athletes participating in acute high-intensity exercise events up to 40 min in duration, except in those known to be consuming a low-antioxidant diet for prolonged periods.

<http://www.cababstractsplus.org/google/abstract.asp?AcNo=20053028744>

The results suggest that >*endurance exercise in combination with vitamin E*< reduces oxidative stress, improves aerobic fitness, and reduces BP and weight in older adults. Even sedentary participants who take vitamin E may reduce oxidative stress and lower BP.

<http://brn.sagepub.com/cgi/content/abstract/5/1/47>

Our findings indicate that >*consumption of tuna and other broiled or baked fish*<, but not fried fish or fish sandwiches, is associated with lower incidence of AF among older adults. These results suggest that fish intake may influence the risk of this common cardiac arrhythmia. Confirmation of these findings in other studies and evaluation of potential mechanisms of effect, such as effects on blood pressure, left ventricular diastolic function, systemic inflammation, or direct antiarrhythmic effects, are indicated.

<http://www.circ.ahajournals.org/cgi/content/full/110/4/368>

See the section by Stephen Byrnes in the LAF BB Conference Room Session 35 at

<http://www.afibbers.org/conference/session35.pdf>

Read this book for a method of breathing that may reduce the stress of physical training:

Body, Mind, and Sport : The Mind-Body Guide to Lifelong Health, Fitness, and Your Personal Best by John Douillard

You may want to pay attention to these signs of overtraining:

<http://sportsmedicine.about.com/cs/overtraining/a/aa062499a.htm>

Author: [Jackie](#) (---.neo.res.rr.com)

Date: 05-09-08 16:13

Nice work, Dick -

It's standard in Functional Medicine to Oxidative Stress by ordering a test through one of the labs such as Metametrix or Genova Diagnostics.

This test identifies markers of hydroxyl radical activity, urine lipid peroxides, reduced glutathione, superoxide dismutase, and glutathione peroxidase. The test indirectly measures via blood and urine your level of harmful free radicals.

High levels would call for an increased antioxidant protocol, changing diet and reduction of toxic exposure.

The nutritional circles talk about this all the time and emphasize preventive measures to counteract free radical damage.

While oxygen is considered the ultimate given of life, it is also a powerful killing agent....and is called "The Great Destroyer of Life.

Exercise, overactive thyroid, inflammation from fever, infection or other causes, radiation and certain chemicals all increase free-radical production.

A high rate of metabolism increases aging (free-radical damage)and is why a diet high in sugar is so harmful. One interview I just heard mentioned that marathoners have great free-radical damage and the speaker commented to note they always look older and that's because of the free-radical damage.

The body has a system to protect against free radicals called the antioxidant network. Groups of antioxidant enzymes function to neutralize free radicals... these are superoxide dismutase, glutathione peroxidase, catalase etc). All contain glutathione which is a very powerful neutralizer of free radicals.

Good support to reduce harm from free-radicals:

As reported in my Anti Aging Congress notes back in 04, Eric Braverman, MD, says the best way to fight free-radical damage is to take plenty of NAC (N-acetyl L-Cysteine)... along with ascorbate, melatonin, curcumin and numerous flavonoids from vegetables and fruits. Coenzyme Q10 provides major protection from free radicals and lipid peroxidation.

Quoting Russell Blaylock, MD, retired neurosurgeon, from Blaylock Wellness Report :

EXERCISE

Most of us have been told at one time or another that we should exercise to prevent heart disease and strokes. But exercise also has many other health advantages.

Exercise increases the antioxidant enzymes that protect us from a number of diseases, including atherosclerosis. In addition, it improves blood flow, protects the brain against degeneration, increases alertness and mental acuity, improves memory, lowers blood pressure, reduces the risk of diabetes, improves lung function, strengthens immunity

and inhibits depression.

One study found that the least fit men were six times more likely to die from a heart attack than the most fit men. Yet an increasing number of studies show that excessive exercise is actually bad for you and can greatly increase disease, even brain disorders.

Careful studies have shown that this is because exercise increases free radical generation and lipid peroxidation, both of which are associated with most diseases, including cardiovascular disease.

During exercise, especially extreme exercise, the body's metabolism is dramatically increased. Since the major source of free radicals in the body is metabolism, the higher metabolism, the greater the generation of free radicals.

Based on these studies, exercise physiologists are warning intense athletes that they should take a higher amount of antioxidants than less active people.

Unfortunately, vitamin-based antioxidants, such as vitamins C, E and the carotenoids, do not neutralize some of the more destructive free radicals. Magnesium plays a significant role in more than 300 enzymes in the body and has been shown to be as effective as statin drugs in preventing heart attacks and strokes. That is why extreme athletes are told to take magnesium.

Case studies point out that marathon runners require at least six months to replace the amount of magnesium lost in a single marathon race. Magnesium is vital as it prevents inflammation, improves blood flow through blood vessels, thins the blood, acts as an antioxidant, and prevents coronary artery spasm.

Metamatrix

DNA/Oxidative Stress Marker*

The interaction of reactive oxygen species with DNA and RNA produces oxidation products of these base rings. DNA repair enzymes must remove the damaged bases in order to maintain viability. Oxidation of guanine produces 8-hydroxy-2'-deoxyguanosine (8OHdG). The concentration of 8OHdG in urine has been shown to be an accurate measure of the rate of polynucleotide oxidative damage. Elevated 8OHdG is a sign that antioxidant nutrient intake may need to be increased. Toxicants and lifestyle stress factors may contribute to increased oxidative challenge.

Author: Mark Robinson (---.bb.sky.com)

Date: 05-11-08 13:12

Dick most people get confused about the "grey area of exercise". What must be understood is that quite simply it must always be relevant to the individual. If one tries generalize about the level of exercise which may be a risk factor one is doomed. It is the duration and intensity for the individual that is the issue.

E.g. my training group could jog along at five minute thirty pace and have an easy conversation. The oxidative stress load on these four minute milers (myself not included I was always maxed out) was probably very low. For other groups this pace would give them a high degree of oxidative stress. As would walking up a hill if you are 30 stone. Its all relative. In other words an amateur athlete will in many instances suffer more exercise induced oxidative stress than a professional athlete for a whole host of reasons. One top world class marathoner I knew off slept about 16 hours a day with excellent nutrition. So although the intensity of his exercise was very high indeed his overall daily oxidative stress load was probably lower than many.

I have not "exercised" now for over six years. As soon as I start up my heart becomes irritable (inflamed).

You gave a quote of "In other words, individuals who had better parasympathetic tone had lower levels of inflammation..."

Again my heart rate was about 38 on awakening suggesting a high parasympathetic tone but my inflammatory markers were probably through the roof. A double whammy for me with regards to my impending a.fib probs.

Author: [GeorgeN](#) (---.hln.qwest.net)
Date: 05-11-08 21:35

Hi Dick,

Good job!

Mark,

"It is the duration and intensity for the individual that is the issue." I heartily agree. My own philosophy is to keep my intensity to a moderate level.

I skied on Friday with a friend who just returned from a year as a MEDEVAC pilot in Iraq. Because of their work schedule, he could only exercise once every six days or so while he was there. In addition that time was at sea level. He is an expert skier, however even though our runs never had me out of breath, he was breathing heavily. He is not an afibber, but I'm sure his stress level was much higher than mine. When we do this again in November, I'm guessing that the same activity level will not stress him anymore than me.

Even if I were maxed out, there is no way I'd come close to keeping up with your training group, at this point.

Author: [Dick](#) (71.94.185.---)
Date: 05-12-08 18:30

In case anyone is still interested in this subject, here is an excerpt from another article which attempts to draw together findings on the effects of antioxidant supplements and diet on antioxidant levels and symptoms such as post-exercise soreness. It mentions something I hadn't heard of, the Oxygen Radical Absorbance Capacity (ORAC) of foods ...

Practical advice

How can the athlete make best use of the current antioxidant knowledge to maximise protection during training and competition? The first thing to say is that the evidence that taking single doses of antioxidant nutrients such as vitamin C or vitamin E is beneficial is rather patchy; some studies show that single nutrient supplementation can reduce levels of muscle damage and a couple of studies have indicated that vitamin C may help reduce post-exercise muscle soreness. However, plenty of other studies have produced inconclusive results. Supplementing combinations of antioxidant nutrients (eg vitamins A, C, E and selenium) may be more beneficial as antioxidant nutrients do not work in isolation in the body but synergistically; a multi-antioxidant nutrient supplement probably makes more sense.

However, athletes should take note of the rapidly growing body of evidence pointing to the protective benefits of phytochemical-rich foods, such as brightly coloured fruits and vegetables. These not only contain antioxidant nutrients but hundreds of other naturally occurring powerful antioxidant compounds.

While the strength and depth of colour gives a very rough rule-of-thumb guide to the antioxidant activity of plant foods, a more scientific approach has been developed that measures the Oxygen Radical Absorbance Capacity (ORAC) of foods. The higher the ORAC score, the higher the potential capacity of a food to 'quench' oxygen free radicals and render them harmless. Natural fruits typically score between 500 and 900 ORAC units per 100 grams and the US Food and Drug Administration (FDA) has recently suggested that a daily consumption of around 7,000 ORAC units may provide optimum antioxidant protection (that's around 5-10 servings of fruit and vegetables per day!).

However, some athletes with a high volume of training may struggle to include such large amounts of fruit and vegetables in their diet. This is because these foods are bulky and tend to contain relatively large amounts of water but low amounts of carbohydrate and very small quantities of protein. A large intake of fruits and vegetables increases satiety and could displace carbohydrate and protein-rich foods from the diet, making the task of muscle glycogen replenishment and recovery more difficult. The key then is to emphasise foods that are especially rich in antioxidant activity – ie with high ORAC scores.

But while ORAC scores give a better indication of antioxidant capacity of foods in vitro than mere colour, it's important to realise that the relationship between ORAC scores and antioxidant activity in the body is still poorly understood; for this reason, it's important not to sacrifice variety by consuming just one or two high ORAC foods in order to boost ORAC unit intake. Many lower scoring foods may offer particular benefits and work synergistically with other foods. Also beware of relying on some of the very high ORAC food extracts now coming onto the market claiming 20,000 ORAC units or more per 100g. It's not yet known whether such values are accurate or if such concentrated antioxidants can be absorbed by the human body as effectively as those found in natural foods.

Andrew Hamilton BSc, MRSC, trained as a chemist and is now a consultant to the fitness industry and an experienced science writer

[References from 1995-2006 follow the full article]

<http://www.pponline.co.uk/encyc/sports-nutrition-can-antioxidants-protect-athletes-35841>

Author: [GeorgeN](#) (---.hlrn.qwest.net)

Date: 05-12-08 19:40

Oxygen Radical Absorbance Capacity (ORAC) of Selected Foods – 2007

<http://www.ars.usda.gov/SP2UserFiles/Place/12354500/Data/ORAC/ORAC07.pdf>

Author: [Hans Larsen](#) (---.gv.shawcable.net)

Date: 05-22-08 09:49

Hi George,

Thank you for alerting me to this other very important thread. I must have missed it during a recent "off-line" period. Very impressive posting from Dick - thanks! I will add to the proceedings.

Dr. Kenneth Cooper, "the father of aerobics", believes that lower intensity exercise provides the optimum cardiovascular benefits. He recommends that one should exercise at least 30 minutes 3 times a week at one's personal target heart rate. He calculates a range for the target heart rate as 220 minus age multiplied by 0.65 and 0.80. Thus, the exercise target rate for a 40-year-old would be between 117 and 144 bpm. Dr. Cooper also suggests that to avoid free radical damage from exercise one should aim to earn no more than 50 "exercise points" per week.

For example, a 55-year-old jogger who jogs 3 miles in 32 minutes 5 times a week would earn 55 points, while a jogger under 30 years of age who jogs 3 miles in 24 minutes 5 times a week would earn 85 points - in Dr. Cooper's opinion - way too much.

Dr. Cooper also reports the fascinating finding that highly trained athletes, even at rest, exhibit a much higher level of free radicals (oxidative stress) than do untrained men. His recipe for healthy exercise is, "Avoid exercises or activities that place an undue or prolonged strain on your body or its oxygen production systems, such as the heart and lungs. In other words, do not push yourself to do frequent, high-intensity exercise, particularly to the point of total exhaustion and chronic fatigue."

Finally, Dr. Cooper points out that antioxidant supplementation (vitamins C and E) can help prevent some of the ravages caused by overtraining.

For those interested in the intricate relationship between exercise and oxidative stress, I can highly recommend Dr. Cooper's book "Antioxidant Revolution".

Author: [Jackie](#) (---.neo.res.rr.com)
Date: 05-22-08 10:07

George, I do have some current references...finding them and posting may take some time as I'm on another consult project at the moment. The best antioxidant is NAC - the more the better. I have some exercise physiology papers, books and notes that I'll check for study references that may be links to those stats. I also recently heard a couple of interviews with exercise physiologists on the ravages of exercise so I'll try to pull it together before this CR session ends.

One comment I recall was that endurance runners suffer the ravages of muscle wasting and the appearance issue - they always look older than they are because of the free radical damage. Interesting. I know several runner who do look remarkably older than their years. Overexercise also takes a toll on hormones - especially testosterone in males...(often found to be low). That was in another lecture.

Author: [Jackie](#) (---.neo.res.rr.com)
Date: 05-22-08 10:09

Also adding this from the recent BB entry:

Author: Jackie (---.neo.res.rr.com)
Date: 05-20-08 14:00

http://www.afibbers.net/forum/read.php?f=7&i=8351&t=8351#reply_8351

This was published in EP Digest (April 2007) - in conjunction with the feature article on

Advanced Mapping and Ablation: The Importance of the Coronary Sinus Catheter by Laurence M. Epstein, MD, Chief, Arrhythmia Service, Brigham and Women's Hospital, Boston, Massachusetts

Introduction:

In recent years, it has been recognized that rapidly firing ectopic foci, often arising from a muscular sleeve of atrial tissue extending into the pulmonary veins, can be responsible for triggering atrial fibrillation. Elimination or blocking conduction of these foci can prevent atrial fibrillation in selected patients. In this case study, a simplified approach to mapping and electrical isolation of the pulmonary veins is reviewed.

Case History

The patient is a 43-year-old man with a ten-year history of palpitations. The patient is very active, running at least 4 times per week. He describes initially feeling occasional skipped beats, which have increased over the years.

Approximately 5 years prior, the patient presented with a sustained episode of rapid, irregular palpitations after jogging. He was found to be in atrial fibrillation with rapid ventricular response. The patient received IV diltiazem for rate control and spontaneously converted to sinus rhythm. The patient was discharged on atenolol and did well, with continued occasional short bursts of AF.

Two years ago, the frequency and duration of the episodes increased. The patient was begun on propafenone and a stress test was benign. Over the past year, despite increasing doses of propafenone, the patient has been increasingly disabled by episodes of atrial fibrillation and was referred for consideration of catheter ablation. Given that the patient's highly symptomatic atrial fibrillation had failed an attempt of medical management, the patient was considered a candidate for catheter ablation. A limited approach was planned with isolation of the pulmonary veins and a left and right atrial flutter line.

(A brief description of the procedure was given and concluded with this summary)

The patient was monitored overnight and discharged the following day on warfarin, enoxaparin, atenolol, and propafenone. The patient is now 5 months post ablation, off all medications and back to an active life without atrial fibrillation.

Author: [GeorgeN](mailto:GeorgeN@---.biz.mindspring.com) (---.biz.mindspring.com)

Date: 05-22-08 12:01

Here is Dr. Cooper's point system. It is general, especially on activities like handball, basketball & etc. I've been in games where I was hardly working at all and others where my point value was probably closer to 1 point/minute (before my days of moderation).

From: <http://www.cooperaerobics.com/Printable.aspx>

Dr. Cooper's Aerobics Point System

Walking / Running

1.0 miles

Time - min/Points

Over 20:01 0

20:00-15:01 1.0

15:00-12:01 2.0

12:00-10:01 3.0

10:00-8:01 4.0

8:00-6:41 5.0

6:40-5:44 6.0

Under 5:43 7.0

1.5 miles

Over 45:01 0

45:00-30:01 0.5

30:00-22:31 2.0

22:30-18:01 3.5

18:00-15:01 5.0

15:00-12:01 6.5

12:00-10:01 8.0

10:00-8:35 9.5

Under 8:34 11.0

2.0 miles

Over 40:01 1.0

40:00-30:01 3.0

30:00-24:01 5.0

24:00-20:01 7.0

20:00-16:01 9.0

16:00-13:21 11.0

13:20-11:27 13.0

Under 11:26 15.0

2.5 miles

Over 50:01 1.5

50:00-37:31 4.0

37:30-30:01 6.5

30:00-25:01 9.0

25:00-20:01 11.5

20:00-16:41 14.0

16:40-14:19 16.5

Under 14:18 19.0

3.0 miles

Over 1:00:00 2.0
1:00:00-45:01 5.0
45:00-36:01 8.0
36:00-30:01 11.0
30:00-24:01 14.0
24:00-20:01 17.0
20:00-17:01 20.0
Under 17:09 23.0

3.5 miles

Over 1:10:01 2.5
1:10:00-52:31 6.0
52:30-42:01 9.5
42:00-35:01 13.0
35:00-28:01 16.5
28:00-23:21 20.0
23:20-20:01 23.5
Under 20:00 27.0

4.0 miles

Over 1:20:00 3.0
1:20:00-1:00:01 7.0
1:00:00-48:01 11.0
48:00-40:01 15.0
40:00-32:01 19.0
32:00-26:41 23.0
26:40-22:53 27.0
Under 22:52 31.0

4.5 miles

Over 1:30:01 3.5
1:30:00-1:07:31 8.0
1:07:30-54:01 12.5
54:00-45:01 17.0
45:00-36:01 21.5
36:00-30:01 26.0
30:00-25:44 30.5
Under 25:43 35.0

5.0 miles

Over 1:40:01 4.0
1:40:00-1:15:01 9.0
1:15:00-1:00:01 14.0
1:00:00-50:01 19.0
50:00-40:01 24.0
40:00-33:21 29.0
33:20-28:35 34.0
Under 28:34 39.0

6.0 miles

Over 2:00:01 5.0
2:00:00-1:30:01 11.0
1:30:00-1:12:01 17.0
1:12:00-1:00:01 23.0
1:00:00-48:01 29.0

48:00-40:01 35.0
40:00-34:19 41.0
Under 34:18 47.0

7.0 miles

Over 2:20:01 6.0
2:20-1:45:01 13.0
1:45:00-1:24:01 20.0
1:24:00-1:10:01 27.0
1:10:00-56:01 34.0
56:00-46:41 41.0
46:40-40:01 48.0
Under 40:00 55.0

8.0 miles

Over 2:40:01 7.0
2:40:00-2:00:01 15.0
2:00:00-1:36:01 23.0
1:36:00-1:20:01 31.0
1:20:00-1:04:01 39.0
1:04:00-53:21 47.0
53:20-45:44 55.0
Under 45:43 63.0

9.0 miles

Over 3:00:01 8.0
3:00:00-2:15:01 17.0
2:15:00-1:48:01 26.0
1:48:00-1:30:01 35.0
1:30:00-1:12:01 44.0
1:12:00-1:00:01 53.0
1:00:00-51:27 62.0
Under 51:26 71.0

10.0 miles

Over 3:20:01 9.0
3:20:00-2:30:01 19.0
2:30:00-2:00:01 29.0
2:00:00-1:40:01 39.0
1:40:00-1:20:01 49.0
1:20:00-1:06:41 59.0
1:06:40-57:10 69.0
Under 57:09 79.0

Outdoor Cycling

2.0 miles

Time/Point Value
Over 12:01 0
12:00-8:01 0.5
8:00-6:01 1.5
Under 6:00 2.5

3.0 miles

Over 18:01 0
18:00-12:01 1.5

12:00-9:01 3.0
Under 9:00 4.5

4.0 miles
Over 24:01 0
24:00-16:01 2.5
16:00-12:01 4.5
Under 12:00 6.5

5.0 miles
Over 30:01 2.0
30:00-20:01 3.5
20:00-15:01 6.0
Under 15:00 8.5

6.0 miles
Over 36:01 2.7
36:00-24:01 4.5
24:00-18:01 7.5
Under 18:00 10.5

7.0 miles
Over 42:01 3.4
42:00-28:01 5.5
28:00-21:01 9.0
Under 21:00 12.5

8.0 miles
Over 48:01 4.1
48:00-32:01 6.5
32:00-24:01 10.5
Under 24:00 14.5

9.0 miles
Over 54:01 4.8
54:00-36:01 7.5
36:00-27:01 12.0
Under 27:00 16.5

10.0 miles
Over 1:00:01 5.5
1:00:00-40:01 8.5
40:00-30:01 13.5
Under 30:00 18.5

11.0 miles
Over 1:06:01 6.2
1:06:00-44:01 9.5
44:00-33:01 15.0
Under 33:00 20.5

12.0 miles
Over 1:12:01 6.9
1:12:00-48:01 10.5
48:00-36:01 16.5
Under 36:00 22.5

13.0 miles
Over 1:18:01 7.6
1:18:00-52:01 11.5
52:00-39:01 18.0
Under 39:00 24.5

14.0 miles
Over 1:24:01 8.3
1:24:00-56:01 12.5
56:00-42:01 19.5
Under 42:00 26.5

15.0 miles
Over 1:30:01 9.0
1:30:00-1:00:01 13.5
1:00:00-45:01 21.0
Under 45:00 28.5

20.0 miles
Over 2:00:01 12.5
2:00:00-1:20:01 18.5
1:20:00-1:00:01 28.5
Under 1:00:00 38.5

Swimming

200 yards
Time Point Value
Over 6:41 0
6:40-5:01 1.25
5:00-3:21 1.67
Under 3:20 2.5

400 yards
Over 13:21 0
13:20-10:01 2.5
10:00-6:41 3.33
Under 6:40 5.0

600 yards
Over 20:01 0
20:00-15:01 3.75
15:00-10:01 5.0
Under 10:00 7.5

800 yards Over 26:41 0
26:40-20:01 6.0
20:00-13:21 7.67
Under 13:20 11.0

1000 yards
Over 33:21 0
33:20-25:01 8.25
25:00-16:41 10.33
Under 16:40 14.5

1200 yards
Over 40:01 0
40:00-30:01 10.5
30:00-20:01 13.0
Under 20:00 18.0

1400 yards
Over 46:41 0
46:40-35:01 12.75
35:00-23:21 15.67
Under 23:20 21.5

1600 yards
Over 53:21 0
53:20-40:01 15.0
40:00-26:41 18.33
Under 26:40 25.0

Rope Skipping

Time 70-90 rpm 90-110 rpm 110-130 rpm
5:00 1.5 2.0 2.5
10:00 3.0 4.0 5.0
15:00 5.5 7.0 8.5
20:00 8.0 10.0 12.0
25:00 10.5 13.0 15.5
30:00 13.0 16.0 19.0

Handball/Racquetball/Squash/Basketball/Soccer/Hockey/Lacrosse

0.75 points per 5 minutes; do not count breaks and timeouts.

Golf (no motorized cart)

1.5 points per 9 holes

Tennis/Badminton (Doubles)

0.38 points per 15 minutes

Tennis/Badminton (Singles)

0.33 points per 5 minutes

Waterskiing and Downhill Snow Skiing

0.5 points for every 5 minutes of actual skiing

Cross Country Skiing

1.5 points for every 5 minutes of skiing

Ice or Roller Skating

1.13 points for every 15 minutes, for speed skating triple the point value

Volleyball

0.33 points for every 5 minutes

Fencing

1 point for every 10 minutes

Football

0.5 points for every 5 minutes of actual play

Wrestling/Boxing

2 points for every 5 minutes

Circuit Weight Training

0.84 points for every 5 minutes

Super Circuit Weight Training

1.3 points for every 5 minutes

Mini-trampoline

1.25 points for every 5 minutes

Aerobic Dance and Other Exercise Programs Conducted to Music

1 point for every 5 minutes

Schwinn Air-Dyne Ergometer (using arms and legs)

Work Load/Time-min

...../5 10 15 20 25 30 35 40

1.5 0.7 1.4 2.1 2.8 3.5 4.2 4.9 5.6

2.0 1.0 2.1 3.3 4.2 5.2 6.3 7.4 8.4

2.5 1.4 2.9 4.4 5.8 7.2 8.7 10.2 11.6

3.0 1.9 3.9 5.8 7.8 9.8 11.7 13.6 15.6

3.5 2.5 5.0 7.5 10.0 12.5 15.0 17.5 20.0

4.0 3.2 6.3 9.4 12.6 15.8 18.9 22.0 25.2

4.5 3.8 7.7 11.6 15.4 19.2 23.1 27.0 30.8

5.0 4.6 9.3 14.0 18.6 23.2 27.9 32.6 37.2

If you do not find an activity that is applicable to your workout, aerobics points can be estimated by taking total caloric expenditure and dividing by 20.

Cooper's point system is based on ml's oxygen/kg/min, so 1 point = 7 ml's/kg/min

Author: [Jackie](mailto:---.neo.res.rr.com) (---.neo.res.rr.com)

Date: 05-27-08 06:31

George – listed below are some of the oxidative stress links from my files but I don't find the exercise connection... those may be in a couple of the exercise physiology links I have yet to check. I'll continue looking.

I did find this clip from the paper by David VonWagoner, PhD researcher at CCF/CWR..

(From Molecular Basis by Von Wagoner)

Oxidative and Inflammatory Mechanisms in AF

Several studies have clearly identified high rate activity and myocyte Ca²⁺ overload as a crucial early participant in the cellular remodeling process. However, despite significant efforts, the subcellular mechanisms by which CA²⁺ overload elicits electrophysiologic remodeling are still unclear. Possible mechanisms include activation of cellular proteases (e.g. calpains), activation of CA²⁺ dependent kinases or phosphatases, and increased intracellular oxidative stress, resulting from altered mitochondrial function and/or inflammatory mechanisms.

Although all of these mechanisms and perhaps several others likely are involved in the atrial remodeling processes, we have focused attention on the pathways related to oxidant injury and inflammatory signaling. We found that increased

protein tyrosine nitration was evident in atrial tissues from patients in permanent AF, which is highly suggestive of increased peroxynitrate formation (due to the avid interaction of nitric oxide and superoxide anion).

Interestingly the extent of protein nitration was correlated with creatine kinase activity (which supplies myofibrillar ATP) and myosin isoform switching. Thus oxidative stress may underlie structural and contractile changes in the fibrillating atria.

And this clip from some Q&A portions from <http://www.coloradohealthsite.org/index.html>

Not specific to oxidative stress, but more on exercise with heart complications:

1.Question: I'm a 57 year old cyclist, who was recently diagnosed with idiopathic cardiomyopathy, after having 4 SVT episodes (and much diagnosis). Each SVT episode occurred early on during an exercise session. I would estimate the onset, of SVT, to be at 120-132BPM. Ablation therapy was attempted, but any part of my heart, touched, triggered tachycardia. I've been on Coreg 3.125 for 2.5 weeks. My EF is 42%. My arteries are clean, and my cholesterol routinely tests at 140-160, w/35% HDL. I experienced no other symptoms, and have tons of energy. My cardiologist reduced my weight training to 50% (of weight previously lifted) and my heart rate ceiling to 110-120BPM, and I was told "not to strain" or "get out of breath" to keep pressure off my thin heart wall. Is it important to lower training volume, as well? I was working out, about, 15 hours/wk. w/one long session of 3-5hours. Is there any hope for recovery and/or improvement?

Obviously, I'll do anything I can, to improve my situation, and I'm seeking as much information as possible. Thank you.

Answer: I've been running for the last 33 years so I can truly understand your profound frustration for the predicament in which you find yourself. There are two issues to be discussed. The first question is whether all reasonable causes for the cardiomyopathy have been investigated. I'm going to assume the workup has been thorough especially since you have already been through an ablation attempt. It also sounds like further attempts would be futile. If we assume no other cause has been found, then the second issue is how do we get you better. The first thing you have got in your head is to be patient.

Whatever happened to your heart may be reversible, at least to some degree. I have seen dramatic improvement in heart function over a period of 4-6 months once appropriate therapy has been started. You have just begun on the Coreg and you have a long way to go in terms of increasing the dose over the next 2-3 months. This class of medicine can truly do amazing things, but you have got to be patient.

It usually takes months to see real improvement. While I'm on the subject you might ask your cardiologist about using an ACE inhibitor. There is substantial data to suggest one of these might also help, but your heart is not quite bad enough to say you have to be on it.

I bet your cardiologist thinks the beta blocker alone is enough, and he is probably correct. I cannot promise your heart will completely recover not matter what you do, but you must give it every chance.

You are getting very sound advice as far as how to exercise, but I would recommend even more drastic changes.

Let me put it to you in these terms. If you broke your leg, but you couldn't put a cast on it, would you be working the leg very hard? You obviously would not or the bone would have no chance to heal correctly. Otherwise you know you might never walk, much less run or bike, again. And you also know bones don't heal overnight. Well I suspect you see where I'm going with this analogy. Back off and given your wounded heart every chance to come back. Quit thinking in terms of maintaining any sort of training condition (at the possible expense of not letting your heart recover).

All you need to do is keep "tuned up" until the heart gets better. Quite simply I would tell you to stop all exercise for 8-12 weeks. In other words, let time heal. Then I would start you off working out at 1/4 speed. This means you would start exercising with your old routine decreased 3/4 (time, distance, speed, and weight). In other words, start back real easy. I would then gradually bring you back to a reasonable routine over 3-6 months depending on how the heart is doing (with serial echo studies).

You should know I might not ever recommend you go back to the intensity of training you are used to (this is clearly true if your heart does not come back to 100% which is definitely possible).

You are not helping your overall health with such rigorous, prolonged training (even if your heart were normal). All evidence demonstrates that moderate intensity exercise for about 1 hour, 5-6 days out of 7, is optimal for overall (and cardiovascular) health. Furthermore weight training to build bulk does not help you long term. You just want to lift enough to maintain strength, tone, and flexibility.

As far as aerobic conditioning is concerned I would recommend you limit your very strenuous exercise to 30-60 minutes daily. You can still ride distance as long as you pace yourself in a reasonable fashion. As a comparison I usually run 20-30 minutes each day which is fairly intense running, but it does not wear me out, and my knees are still good for another 20 years (hopefully). But if I had pushed myself like you are, there is no way I would still be able to run. (And I fully intend to keep running until I check out.) So back off on the intensity and enjoy yourself.

Finally you need to know the Coreg will probably not let you exercise like you used to. It slows the heart (this is dose related) which usually means peak performance and endurance will significantly decrease (which, as I have said, is exactly what I want for your heart). Good luck.

Randall Carter Marsh, M.D., Division of Cardiology, North Colorado Medical Center, Greeley, Colorado May 2003
[http://www.coloradohealthsite.org/chnqna.html?Heart%20Disease?Heart%](http://www.coloradohealthsite.org/chnqna.html?Heart%20Disease?Heart%20Disease)

Dysrhythmias?

1. Question: Hello: I am a 54 year old otherwise healthy male suffering from paroxysmal idiopathic atrial fibrillation. I have had this condition since about 1987, when I got a thorough workup by a cardiologist. This gentleman suggested that while bothersome, my condition was not dangerous in and of itself, and suggested ASA as a preventive measure, to guard against emboli formation during an attack.

An episode would usually begin in the middle of the night, and would autoconvert to sinus rhythm by about 10am the following morning. I was jogging about 15 miles a week at the time, and stopped running in 1985, for reasons unrelated to my afibs.

This year, I have been trying to get back into running, and was progressing quite well, until I suffered a bout of my afibs again earlier this month. The difference, this time, is that my heart did not autoconvert to sinus rhythm, and after 26 hours, I admitted myself to hospital, where I was electroconverted. The cardiologist I was referred to has prescribed a beta blocker for me, Monorcor (bisoprolol fumarate). I suppose my question to you is: does this medication affect my capacity for exercise? Since beginning the drug regimen, I seem, at times, to lack stamina while jogging, and occasionally must slow to a walk, as I am out of breath. If this med is at fault, is there any alternative treatment that would not affect my ability to maximally exert myself during exercise?

I have a call in to my cardiologist on this matter, but he has not gotten back to me yet. I have since discovered he is on vacation, hence my query to the forum. Thanks very much.

Answer: The bisoprolol is causing all of your symptoms. It is from a class of medicine called beta blockers. These drugs will slow your heart rate at rest and during exercise so you simply cannot run normally while on the medicine. They will also cause shortness of breath with exercise and can cause general fatigue.

There are alternatives in terms of trying to keep the rhythm normal, but they come at a price and that price is potential toxicity. It gets even more complicated by the recent release of information that trying to maintain normal rhythm long term with drugs is not necessarily the best approach for all patients with intermittent AF.

This is complicated and you will need to sit down with your cardiologist and go over the options. I will commonly spend 30 minutes or more with someone in your situation talking about various drugs and discussing risks of various therapies.

Finally I would suggest you read the primer at this web site on Atrial Fib. It will give you a good working knowledge of this frustrating and very common rhythm problem.

Randall Carter Marsh, M.D., Division of Cardiology, North Colorado Medical Center, Greeley, Colorado December 2002

Other links on oxidative stress:

<http://www.ncbi.nlm.nih.gov/pubmed/12821281>

<http://www.ncbi.nlm.nih.gov/pubmed/15042451?dopt=Abstract&holding=f1000.f1000m.isrctn>

http://www.humankinetics.com/products/showexcerpt.cfm?excerpt_id=3146

<http://www.ajcn.org/cgi/content/full/72/2/647S>

<http://www.abcbodybuilding.com/freeradicals.php>

<http://www.westonaprice.org/moderndiseases/oxidativestress.html>

<http://www.gsdl.com/assessments/oxidativestress/>

<http://www.oxidative-stress.com/en/os/info2.php>

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Date: 06-01-08 20:35

IMHO, at the very least, afibbers should avoid overtraining.

In this link, previously referenced by Dick on his 5/9/08 post

<http://sportsmedicine.about.com/cs/overtraining/a/aa062499a.htm> is a section on measuring overtraining-

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There are several ways you can objectively measure some signs of overtraining. One is by documenting your heart rates over time. Track your aerobic heart rate at a specific exercise intensities and speed throughout your training and write it down. If your pace starts to slow, your resting heart rate increases and you experience other symptoms, you may heading into overtraining syndrome.

You can also track your resting heart rate each morning. Any marked increase from the norm may indicated that you aren't fully recovered.

Another way to test recover to use something called the orthostatic heart rate test, developed by Heikki Rusko while working with cross country skiers. To obtain this measurement:

1. Lay down and rest comfortably for 10 minutes the same time each day (morning is best).
2. At the end of 10 minutes, record your heart rate in beats per minute.
3. Then stand up
4. After 15 seconds, take a second heart rate in beats per minute.
5. After 90 seconds, take a third heart rate in beats per minute.
6. After 120 seconds, take a fourth heart rate in beats per minute.

Well rested athletes will show a consistent heart rate between measurements, but Rusko found a marked increase (10 beats/minutes or more) in the 120 second-post-standing measurement of athletes on the verge of overtraining. Such a

change may indicate that you have not recovered from a previous workout, are fatigued, or otherwise stressed and it may be helpful to reduce training or rest another day before performing another workout.

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Dick also said, "Read this book for a method of breathing that may reduce the stress of physical training:
Body, Mind, and Sport : The Mind-Body Guide to Lifelong Health, Fitness, and Your Personal Best by John Douillard"

I've previously referenced this book and want to expand on it a bit.

Douillard does advocate a breathing technique - Ujjayi Pranayama on the exhale. However his exercise technique is more involved. Briefly, all exercises are done using the Ujjayi Pranayama technique on the exhale. All breathing is done through the nose (if you have to open your mouth, you're exercising too hard). In addition, prior to starting, several rounds of the yogic Sun Salutation exercises are performed (with the breathing). Then warm up, resting, listening and performance exercise phases, all at specified heart rates (varying by age & etc). I'm convinced that it would be difficult to overtrain using this technique. In addition, it would seem that oxidative damage would be minimized.

At one time, Douillard was a triathlete, suffering from overtraining. One physician from India suggested he could train as much as he wanted, as long as he could remain in a meditative state during meditation and not fall asleep. Douillard had to reduce his training volume by half to abide by this. After several months, his competitive standing went from being in the top 10% to in the top 10 of pro triathletes at that time.

He went on to develop the training system he describes in "Body, Mind, and Sport." It is a very low stress way to exercise. I would recommend it to all who want to do more than the bare minimum of "getting off the couch" in their exercise programs.

Author: [Jackie](mailto:---.neo.res.rr.com) (---.neo.res.rr.com)
Date: 06-02-08 09:42

I have submitted a similar post to this on the regular BB, but wanted to post this segment separately so it is preserved here.

I've included only the segment referencing exercise; there are other topics as well – see the main BB for details.

ATRIAL FIBRILLATION

New risk factors for atrial fibrillation: causes of 'not-so-lone atrial fibrillation'

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Abstract

Atrial fibrillation (AF) is a prevalent arrhythmia in patients with cardiovascular disease. The classical risk factors for developing AF include hypertension, valvular disease, (ischaemic) cardiomyopathy, diabetes mellitus, and thyroid disease. In some patients with AF, no underlying (cardiovascular) pathology is present and the aetiology remains unknown. This condition is known as lone AF. However, in recent years, other factors playing a role in the genesis of AF have gained attention, including obesity, sleep apnoea, alcohol abuse and other intoxications, excessive sports practice, latent hypertension, genetic factors, and inflammation. In this review, we address these 'new risk factors' (i.e. as opposed to the classical risk factors) and the mechanisms by which they lead to AF.

Introduction

Atrial fibrillation (AF) may be caused by many cardiac and non-cardiac conditions, including hypertension, valvular disease (in particular, of the mitral valve), (ischaemic) cardiomyopathy, diabetes mellitus, and thyroid disease.¹ The

vast majority of patients with AF suffers from one or more of these conditions and is >60 years of age.

However, a subset of patients with AF is <60 years and routine evaluation, including physical examination, laboratory tests including thyroid function, echocardiography, and exercise stress testing, does not reveal any abnormalities. These patients are considered to suffer from 'lone' AF, i.e. AF without an underlying (cardiovascular) disorder.² There is increasing evidence that, from a pathophysiological point of view, the underlying mechanism of lone AF is different than that of AF in the setting of underlying disease. The latter is 'substrate related', i.e. due to diseased and dilated atria with stretch and fibrosis. In contrast, lone AF is probably more related to electrophysiological phenomena (triggers) in structurally normal atria. This explains why patients with real lone AF have a normal life expectancy when compared with individuals without arrhythmia (Figure 1), a low risk for stroke, and why paroxysmal lone AF does not often progress to persistent or permanent AF.^{3,4}

In contrast, AF in the setting of underlying cardiac pathology usually progresses from paroxysmal to persistent and permanent AF together with the progression of the underlying cardiac substrate and is associated with an increased incidence of stroke.

Although having a benign course, regular follow-up of patients with lone AF is warranted. In time, risk factors such as hypertension, heart failure, diabetes, and peripheral vascular disease may develop, changing prognosis. Obviously, the patient also ages. The incidence of stroke is strongly related with the presence or development of these risk factors³ and age^{5,6} in patients with lone AF. In addition, prognosis in patients with lone AF has been shown to be strongly related to the presence or development of left atrial dilatation,⁷ which might suggest the formation of an atrial structural substrate.

Furthermore, clinicians should ask themselves when AF is truly lone. Underlying hypertension is often not recognized anymore after institution of rate-control therapy by β -blockers or calcium channel blocking agents. Long-term data from the Mayo Clinic revealed that only 2% of the total population of patients with AF really have lone AF.³ Apart from the (cardiovascular) conditions traditionally known to be related to AF, other factors may also be involved in the pathogenesis of arrhythmia, i.e. being risk factors for AF. In what follows, we discuss some of these risk factors and the underlying mechanisms by which these conditions may contribute to the development of, apparently lone, AF. These underlying factors also may alter the prognosis. Adequate treatment or reduction of these risk factors possibly may reduce the prevalence of AF and improve prognosis.

Sports and exercise

Although regular exercise is well known to reduce cardiovascular morbidity,³⁶ excessive (endurance) sport practice is associated with a higher prevalence of AF. Athletes may present with any arrhythmia,³⁷ but AF is a most usual cause when an athlete suffers from palpitations.³⁸ Arrhythmia may occur both at rest (vagal conditions) and during exercise.

Mont et al. determined the proportion of individuals engaging in frequent and long-term sports activity in patients with lone AF. In a group of 1160 consecutive patients, 70 individuals had lone AF and were <65 years. Of these 70 patients, 32 had engaged in long-term sport practice, defined as at least 3 h a week for at least 2 years. Surprisingly, these were all men. In 57% of the sportsmen with AF, the paroxysms started in vagal situations (in rest, after exercise, or eating), compared with only 18% in the non-sport male patients with lone AF. Of all men with lone AF in this study, 63% had been participating in sports; this percentage is significantly higher than that of males in the general population who practice sports at a similar intensity (15%).³⁹ In a case-control study, the same group reported the current sport activity to be associated with a three times higher prevalence of lone AF and a five times higher prevalence of vagal lone AF when compared with controls.⁴⁰ Of note, these associations were observed when the cumulative time of lifetime sports practice exceeded 1500 h. Recently, these authors additionally demonstrated a relation between lone AF and cumulative work-related physical activity.⁴¹

Karjalainen et al. evaluated the presence of AF in 228 veteran male orienteers (cross-country runners) and compared this with the prevalence of AF in a matched control group (n = 212). The mean age was 47.5 years in the orienteer group and 49.6 years in the controls. Lone AF was diagnosed in 12 orienteers (5.3%) vs. 2 control subjects (0.9%), whereas there was no difference in the prevalence of AF in the presence of risk factors.⁴² Heidbuchel et al.⁴³ assessed the influence of sports activity on the risk of AF after the ablation of atrial flutter. Of 137 patients undergoing ablation of the right atria isthmus because of atrial flutter, 31 (23%) participated in endurance sports on a regular basis.

A history of competitive sports practice was associated with an elevated risk of developing AF [multivariate HR 1.81 (1.10–2.98, P = 0.02)] after the ablation. In addition, continuation of endurance sports (19 patients) after the ablation showed a trend towards an increased risk of AF [multivariate HR 1.68 (0.92–3.06), P = 0.08].

There are several mechanisms by which sports may induce AF. First, sporting results in enlargement of the cardiac chambers and an increase in the left ventricular mass and left atrial diameter as an adaptation mechanism. One could speculate whether these adaptations are associated with the development of fibrosis or electrophysiological remodelling in the atria. Pelliccia et al.⁴⁴ determined the left atrial dimension in 1777 competitive athletes and found a dimension of 40 mm in 20% of these individuals. Nevertheless, the prevalence of AF was only 0.3% in this group. Another factor favouring AF in sportsmen is the increased vagal tone, resulting in bradycardia and shortening of the atrial refractory period. Furthermore, during exercise, hypovolaemia may develop because of fluid loss, resulting in altered pressures leading to an increased vulnerability to AF.⁴⁵ Finally, changes in electrolytes due to sweating can cause changes in atrial electrophysiology.

A possible link between sports and AF is the use or abuse of anabolic steroids. There is anecdotal evidence of sportsmen developing AF after the use of these drugs.^{46, 47} The mechanism by which anabolic steroids cause AF is largely unknown but changes in the autonomic function and the baro-reflex have been demonstrated in animal models.^{48, 49}

Conclusion

In most patients, AF develops from a substrate that is the common final pathway of different underlying cardiovascular disorders. The process of atrial remodelling leading to this substrate already commences a long time before the first episode of AF occurs. Therefore, treating the underlying disease is the first step in trying to prevent AF and reduce AF burden once the first paroxysms appear in these patients.

In a significant subset of patients, no underlying cardiovascular disease is present and these individuals are considered to suffer from lone AF. However, there may be other conditions present in such patients that predispose to AF. Many of these factors are life style-related, such as drinking, excessive sports practice, or obesity, possibly resulting in sleep apnoea. Previously, socio-economic factors have also been identified to influence AF incidence.⁸¹ Hypertension may be still occult while already damaging the atrial tissue. At present, it is unknown whether these factors result in a substrate comparable to that of AF in the setting of underlying heart disease or these factors are solely leading to electrophysiological changes that trigger AF without concomitant structural changes of the atria. Consequently, one can only speculate whether these forms of 'not-so-lone' AF carry an increased risk for thrombo-embolic complications and other adverse events. Therefore, all patients with these forms of AF should be offered follow-up and the development of risk factors for thrombo-embolic events, left atrial diameter, and/or symptoms should be monitored. Finally, little is known about the effect of lifestyle modification on AF burden in these patients. Future research is warranted to address these issues.

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