



Endurance Exercise – Is It Worth It?

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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]

References

1. Wannamethee, SG, et al. Changes in physical activity, mortality, and incidence of coronary heart disease in older men. *The Lancet*, Vol. 351, May 30, 1998, pp. 1603-08
2. Lee, IM, et al. Physical activity and coronary heart disease in women: is “no pain, no gain” passé? *JAMA*, Vol. 285, March 21, pp. 1447-54
3. Haskell, WL, et al. Physical activity and public health. Updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. *Circulation*, Vol. 116, August 28, 2007
4. Nelson, ME, et al. Physical activity and public health in older adults. Updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. *Circulation*, Vol. 116, August 28, 2007
5. Hood, S and Northcote, R.J. Cardiac assessment of veteran endurance athletes: a 12-year follow up study. *British Journal of Sports Medicine*, Vol. 33, 1999, pp. 239-43
6. Jensen-Urstad, K, et al. High prevalence of arrhythmias in elderly male athletes with a lifelong history of regular strenuous exercise. *Heart*, Vol. 79, 1998, pp. 161-64
7. Baldesberger, S, et al. Sinus node disease and arrhythmias in the long-term follow-up of former professional cyclists. *European Heart Journal*, Vol. 29, 2008, pp. 71-78
8. Karjalainen, J, et al. Lone atrial fibrillation in vigorously exercising middle aged men. *British Medical Journal*, Vol. 316, June 13, 1998, pp. 1784-85
9. Mont, L, et al. Long-lasting sport practice and lone atrial fibrillation. *European Heart Journal*, Vol. 23, March 2002, pp. 477-82
10. Elosua, R, et al. Sport practice and the risk of lone atrial fibrillation. *International Journal of Cardiology*, Vol. 108, No. 3, April 14, 2006, pp. 332-27
11. Mont, L, et al. Physical activity, height, and left atrial size are independent risk factors for lone atrial fibrillation in middle-aged healthy individuals. *Europace*, Vol. 10, 2008, pp. 15-20
12. Pelliccia, A, et al. Prevalence and clinical significance of left atrial remodeling in competitive athletes. *Journal of the American College of Cardiology*, Vol. 46, No. 4, August 16, 2005, pp. 690-96
13. Steinacker, J.M., et al. Training of junior rowers before world championships: effects on performance, mood state and selected hormonal and metabolic responses. *Journal of Sports Medicine and Physical Fitness*, Vol. 40, December 2000, pp. 327-35
14. Hackney, A.C. Endurance exercise training and reproductive endocrine dysfunction in men: alterations in the hypothalamic-pituitary-testicular axis. *Curr Pharm Des*, Vol. 7, March 2001, pp. 261-73
15. Matsuo, S., et al. Cardiac sympathetic dysfunction in an athlete’s heart detected by 123I-metaiodobenzylguanidine scintigraphy. *Japanese Circ J*, Vol. 65, May 2001, pp. 371-4

16. Hautala, A., et al. Changes in cardiac autonomic regulation after prolonged maximal exercise. *Clin Physiol*, Vol. 21, March 2001, pp. 238-45
17. Margeli, A, et al. Dramatic elevations of interleukin-6 and acute-phase reactants in athletes participating in the ultradistance foot race Spartathlon. *Journal of Clinical Endocrinology & Metabolism*, Vol. 90, No. 7, July 2005, pp. 3914-18
18. Swanson, DR. Atrial fibrillation in athletes: implicit literature-based connections suggest that overtraining and subsequent inflammation may be a contributory mechanism. *Medical Hypotheses*, Vol. 66, 2006, pp. 1085-92
19. Frustaci, A, et al. Histological substrate of atrial biopsies in patients with lone atrial fibrillation. *Circulation*, Vol. 96, August 19, 1997, pp. 1180-84
20. Friman, G and Wesslen, L. Special feature for the Olympics: effects of exercise on the immune system. *Immunol Cel Biol*, Vol. 78, No. 5, October 2000, pp. 510-22
21. Coumel, P. Atrial fibrillation: one more sporting inconvenience? *European Heart Journal*, Vol. 23, No. 6, March 2002, pp. 431-33
22. Obel, OA and Davidson, C. Arrhythmias in an athlete: the effect of de-training. *Postgraduate Medical Journal*, Vol. 81, January 2005, pp. 62-64
23. Mujika, I and Padilla, S. Cardiorespiratory and metabolic characteristics of detraining in humans. *Medicine and Science in Sports and Exercise*, Vol. 33, March 2001, pp. 413-21
24. Heidbuchel, H, et al. An endurance sport is a risk factor for atrial fibrillation after ablation for atrial flutter. *International Journal of Cardiology*, Vol. 107, No. 1, February 8, 2006, pp. 67-72
25. Furlanello, F, et al. Radiofrequency catheter ablation of atrial fibrillation in athletes referred for disabling symptoms preventing usual training schedule and sport competition. *Journal of Cardiovascular Electrophysiology*, published online, February 8, 2008
26. Lampert, R. Atrial fibrillation in athletes: toward more effective therapy and better understanding. *Journal of Cardiovascular Electrophysiology*, published online, March 21, 2008
27. Ragozzino, MW, et al. Self-cardioversion of paroxysmal lone atrial fibrillation with exercise. *New England Journal of Medicine*, Vol. 347, No. 25, December 19, 2002, pp. 2085-86
28. Larsen, HR. Lone Atrial Fibrillation: Towards A Cure. IHN, Victoria, BC, Canada, 2002, p. ii

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