To train or not train? That is the question

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Abstract

The health benefits of routine physical exercise are numerous and incontrovertible. But, is it as good as it seems? We are continuing to accumulate evidence that long-term exposure to intense exercise may increase the incidence of certain cardiovascular conditions. We should simultaneously be aware that exercise, like all physiologic stimuli, probably behaves in a typical dose-response fashion in which an individual can reach levels that do more harm than good. Recognizing the patient who exercises excessively may be challenging and requires careful attention to subjective symptomatic reports and temporal exercise patterns. In clinical cardiovascular practice, lessons learned from the science of human performance including the need for dedicated rest and training periodicity may prove applicable to our patients and may be the best defense against the toxicity of excessive exercise.

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The health benefits of routine physical exercise are numerous and incontrovertible. Virtually all common cardiovascular disease processes including atherosclerosis [1], dyslipidemia [2], and hypertension [3], respond favorably to routine aerobic exercise. As such, essentially every professional medical organization involved in clinical practice oversight encourages physicians to prescribe exercise for primary prevention, risk factor modification, and treatment of established disease. Guidelines delineating exercise recommendations are widely available and exercise prescription should be considered a mandatory component of health maintenance and disease treatment for all patients [4,5].

While the cardiovascular medical community has spent a good deal of time developing strategies to motivate the sedentary, there is a fascinating phenomenon evolving at the other end of the exercise spectrum. A rapidly growing segment of the general population is engaging in high levels of physical exercise that far exceed the volume and intensity that have been associated with disease prevention. Intense, high volume exercise training, historically the purview of young, naturally endowed, elite competitors, is now being practiced by men and women of all ages. Evidence of this trend can be found in numerous places with none more compelling than participation rates among recreational sporting events like running road races. Records from the United States indicate a veritable explosion in the number of individuals participating in this activity [6]. There are several explanations for this increase in popularity including mounting awareness of the heath benefits of exercise, simple enjoyment of sport, pleasure derived from the sense of belonging to sporting communities, and fulfillment of competitive and

achievement-based drives. This exciting phenomenon is refreshing and seems poised to offset the record levels of obesity and cardiovascular disease that are crippling the human race. But, is it as good as it seems?

The growth of high-level recreational exercise has been accompanied by emerging data suggesting that exercise may lead to overuse cardiovascular pathology [7,8]. This should not come as a surprise. Exercise, like any physiologic stimulus, is not a binary phenomenon and must be considered as a continuous variable. Quantification of exercise, though somewhat challenging in clinical and research settings, is of paramount importance in the context of any discussion about its risks and benefits. The two basic parameters that define the exercise exposure or "dose," much like the strength and frequency with which we prescribe medications, are intensity and volume. These concepts are familiar to those who work in the cardiac rehabilitation setting but are infrequently used in the clinical assessment of athletic patients. Like all physiologic and biological stimuli, exercise follows a dose-response curve coupling increasing effects with increasing exposure to a point of toxicity at which further increase may do more harm than good. In routine clinical practice, we strive often fruitlessly to motivate our patients to exercise enough to exceed the threshold level at which the benefits of exercise begin to accrue. In contrast, the avid athlete may push exercise to upper end of the curve at which point negative results (i.e., toxicities) begin to develop.

The concept of exercise toxicity, most commonly referred to as overuse injuries or overuse syndromes, is well recognized. Examples include skeletal stress fractures, chronic tendonopathies, and acquired amenorrhea. The notion that the concept of overuse injury can apply the cardiovascular system is only now beginning to crystallize. Reports documenting cardiac fatigue with biochemical evidence of mild cardiac damage following prolonged exercise continue to accumulate [9–11]. While this concept remains incompletely understood, the bulk of available evidence suggests that the myocardium behaves like skeletal muscle in that it can fatigue if worked hard over an extended period of time. Reductions in contractility (systolic function) and lusitropy (diastolic function) of both the right and left ventricle have been documented following completion of marathon runs, long distance cycling events, and triathlons [12,13,11]. Studies with extended follow-up demonstrate normalization of function within hours to days of event completion suggesting that the vast majority of the observed functional deterioration is transient and fully reversible. But, the impact of repetitive bouts of exercise sufficient to induce cardiac fatigue remains uncertain.

Cardiac adaptations to exercise are well documented and have a focus of scientific inquiry for more than 100 years [14]. The constellation of cardiac changes attributable to endurance exercise training include mild eccentric left ventricular (LV) hypertrophy with concomitant right ventricular (RV) dilation, biatrial dilation, and enhanced diastolic function. In aggregate, these findings contribute to stroke volume augmentation and thus increased substrate delivery to peripheral tissue during exercise. This is one of several key adaptive mechanisms that contributes to supra-normal exercise capacity in trained individuals. But can you have too much of a good thing?

We previously conducted a study examining the impact of high volume / high intensity training on left ventricular mechanics, a term used to denote the process of tissue deformation, in a small cohort of competitive rowers [15]. Male collegiate athletes, all with significant prior exercise exposure, were studied with echocardiography before and after a 90-day period of intense team based rowing training. In this setting we saw increases in systolic fiber shortening in all regions of the left ventricle in each of the three cardinal strain vectors (radial, longitudinal, and circumferential) with one notable exception. Specifically, we documented relative functional decrements in systolic circumferential shortening. Though correlation analyses do not establish causality, it was noteworthy that the magnitude of septal dysfunction correlated tightly with the corollary increases in RV dilation. Thus, it appeared that the RV dilation attributed to training was associated with a decrement in LV septal function. Although explanations remain speculative, it seems plausible that exercise-induced RV remodeling leads to functional deterioration, perhaps a form of fatigue, within septal regions comprised of inter-digitating RV and LV fibers.

Although the clinical relevance of this finding remains unknown, several recent studies appear to be of direct relevance. Within the last year, two independent publications have documented cardiac

fibrosis in seasoned endurance athletes. Utilizing magnetic resonance imagine, Wilson and colleagues observed septal "hinge-point" (i.e., region of interface between the right and left ventricles) fibrosis in 4 of 12 veteran athletes (57±6 years of age, 43 ± 6 years of training) and no evidence of scar among control groups of younger athletes and sedentary age matched people [7]. It is noteworthy that the presence fibrosis was associated the number of years of prior exercise training and the number of previously completed competitive endurance racing events. Subsequently, La Gerche et al used multi-modality imaging (echocardiography and magnetic resonance imaging) to examine biventricular structure, function, and fatiguability in accomplished endurance athletes (n = 39) [8]. In this study, small groups of athletes were studied before and after long-distance events including marathon running, cycling, and triathlon. Post-event decrements in cardiac function, particularly of the right ventricle, suggested an element of cardiac fatigue. However, the most notable finding of this study was the presence of cardiac fibrosis in a small subset of these accomplished athletes. Myocardial fibrosis, confined in each case to the interventricular septum, was documented in 5 of these individuals and was associated with greater cumulative prior exercise exposure and more RV dilation. In aggregate, these recent studies suggest that repetitive exercise-induced cardiac fatigue may lead to fibrosis (permanent tissue damage) in a subset of experienced athletes. It must be emphasized that these observational, cross-sectional data do not establish causality and thus the above studies simply establish the fact that certain patterns of cardiac fibrosis have been observed in small, research cohorts. What do these findings mean and how do they impact the clinical care of the endurance athlete?

It is well established that while routine physical exercise reduces the risk of cardiovascular disease, it is not fully protective. This concept is most easily understood when one considers the interplay between exercise and atherosclerotic coronary artery disease. Numerous studies have shown that exercise is an effective treatment for coronary disease and reduces associated event rates. However, individuals with atherosclerosis are more likely to suffer a coronary event during the exercise that contributes to their longevity than during periods of inactivity [16]. This simple but important paradox illustrates the point that while exercise is good for health, it is not completely protective. Our patients need to understand this paradox and must not fall victim to the belief that exercise, even very high levels of it, obviates the need for alternative risk factor reduction strategies. We may now also wish to acknowledge that there can be too much of a good thing in that high amounts exercise may actually increase the likelihood of certain forms of heart disease.

Conclusion

We are continuing to accumulate evidence that long-term exposure to intense exercise may increase the incidence of certain cardiovascular conditions. This concept is increasingly recognized with respect to the atrial tachyarrhythmias, most notably atrial fibrillation which is a common and increasingly recognized problem among aging endurance athletes [17,18]. The recent studies by Wilson and La Gerche suggest that in addition, certain individuals may develop distinct patterns of cardiac fibrosis following years of endurance exercise. Thus, we are now beginning to appreciate that there may be a distinct form of cardiomyopathy attributable to "excessive" exercise. What factors dictate susceptibility to this condition and to what extent a cardiomyopathy of excessive exercise impacts morbidity and mortality remains completely speculative. At the present time, studies defining cardiac damage in athletes should be considered hypothesis generating and should serve as rationale for future study. Specifically, we need carefully designed, longitudinal studies of aging endurance athletes with serial phenotyping, physiologic provocation, careful observation to exclude alternative explanations for non-coronary disease related fibrosis (i.e., myocarditis), and attention to clinically relevant end-points.

While we await this work, it seems prudent to rely on the available data regarding the association between longevity and physical fitness when we are asked the question, "Should I train or not?" The links between living longer, living better, and physical fitness are clear and thus we should encourage high levels of physical exercise for our patients and our communities. It cannot be overemphasized that net risk-benefit ratio strongly favors a high-volume and intensity physically active lifestyle as there is incontrovertible evidence that longevity increases with physical fitness and that aging athletes appear to be less susceptible to disease than sedentary counterparts [19–21]. We should simultaneously be

aware that exercise, like all physiologic stimuli, probably behaves in a typical dose-response fashion in which an individual can reach levels that do more harm than good. Recognizing the patient who exercises excessively may be challenging and requires careful attention to subjective symptomatic reports and temporal exercise patterns. In clinical cardiovascular practice, lessons learned from the science of human performance including the need for dedicated rest and training periodicity may prove applicable to our patients and may be the best defense against the toxicity of excessive exercise. •

References

1. Wannamethee SG, Shaper AG, Walker M (2000) Physical activity and mortality in older men with diagnosed coronary heart disease. Circulation 102: 1358–1363
2. Kraus WE, Houmard JA, Duscha BD et al (2002) Effects of the amount and intensity of exercise on plasma lipoproteins. N Engl J Med 347:1483–1492
3. Duncan JJ, Farr JE, Upton SJ et al (1985) The effects of aerobic exercise on plasma catecholamines and blood pressure in patients with mild essential hypertension. Jama 254:2609–2613
4. Pate RR, Pratt M, Blair SN et al (1995) Physical activity and public health. A recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. Jama 273:402–407
5. Graham I, Atar D, Borch-Johnsen K et al(2007) European guidelines on cardiovascular disease prevention in clinical practice: executive summary: Fourth Joint Task Force of the European Society of Cardiology and Other Societies on Cardiovascular Disease Prevention in Clinical Practice (Constituted by representatives of nine societies and by invited experts). Eur Heart J 28:2375–2414
6. Kim JH, Malhotra R, Chiampas G, d'Hemecourt D et al (2012) Cardiac arrest during long-distance running races. N Engl J Med 366:130–140
7. Wilson M, O'Hanlon R, Prasad S, Deighan A et al (2011) Diverse patterns of myocardial fibrosis in lifelong, veteran endurance athletes. J Appl Physiol 110:1622–1626
8. La Gerche A, Burns AT, Mooney DJ et al (2012) Exercise-induced right ventricular dysfunction and structural remodelling in endurance athletes. Eur Heart J 33:998–1006
9. Neilan TG, Januzzi JL, Lee-Lewandrowski E et al (2006) Myocardial injury and ventricular dysfunction related to training levels among nonelite participants in the Boston marathon. Circulation 114:2325–2333
10. Middleton N, George K, Whyte G, Gaze D, Collinson P, Shave R (2008) Cardiac troponin T release is stimulated by endurance exercise in healthy humans. J Am Coll Cardiol 52:1813–1814
11. Shave R, Baggish A, George K, Wood M et al (2010) Exercise-induced cardiac troponin elevation: evidence, mechanisms, and implications. J Am Coll Cardiol 56:169–176
12. Douglas PS, O'Toole ML, Hiller WD, Hackney K, Reichek N. (1987) Cardiac fatigue after prolonged exercise. Circulation 76:1206–1213
13. Oxborough D, Shave R, Warburton D et al (2011) Dilatation and dysfunction of the right ventricle immediately after ultraendurance exercise: exploratory insights from conventional two-dimensional and speckle tracking echocardiography. Circ Cardiovasc Imaging 4:253–263
14. Henschen S (1899) Skidlauf und Skidwettlauf. Eine medizinische Sportstudie. Mitt Med Klin Upsala 2
15. Baggish AL, Yared K, Wang F et al (2008) The impact of endurance exercise training on left ventricular systolic mechanics. Am J Physiol Heart Circ Physiol 295:H1109–H1116
16. Siscovick DS, Weiss NS, Fletcher RH, Lasky T (1984) The incidence of primary cardiac arrest during vigorous exercise. N Engl J Med 311:874–877
17. Molina L, Mont L, Marrugat J, Berruezo A, Brugada J, Bruguera J, Rebato C, Elosua R (2008) Long-term endurance sport practice increases the incidence of lone atrial fibrillation in men: a follow-up study. Europace 10:618–623
18. Grimsmo J, Grundvold I., Maehlum S, Arnesen H (2010) High prevalence of atrial fibrillation in long-term endurance cross-country skiers: echocardiographic findings and possible predictors--a 28–30 years follow-up study. Eur J Cardiovasc Prev Rehabil 17:100–105
19. Karvonen MJ, Klemola H, Virkajarvi J, Kekkonen A (1974) Longevity of endurance skiers. Med Sci Sports 6:49–51
20. Paffenbarger RS, Jr., Hyde RT, Wing AL, Steinmetz CH (1984) A natural history of athleticism and cardiovascular health. Jama 252:491–495
21. Sesso HD, Paffenbarger RS, Jr., Lee IM (2000) Physical activity and coronary heart disease in men: The Harvard Alumni Health Study. Circulation 102:975–980