MASSACHUSETTS GENERAL HOSPITAL
HEART CENTER

Cardiovascular Performance Program

Sports Cardiology 2021:

Past Progress and Future Challenges

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Disclosures

Cardiovascular Performance Program

Athletic Affiliations:

Funding Sources:

- National Institutes of Health
- American Heart Association
- American Society of Echocardiography
- Department of Defense
- National Football League Player's Association

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The Athlete in CV Practice

- 32 y.o. male professional marathoner
- Fatigue >> acute pleuritic chest pain
- Evaluation concludes viral myopericarditis
- Rx: NSAIDs and activity restriction

The Athlete in CV Practice

- 3 months later, back to training, Emails his doc:

"Doc: all is well. Feeling good. Think the NSAIDs did the trick. Hoping to PR at my next race."

- MD with prompt reply:

"Racing again sounds fine. However, I would prefer you take your medication orally, not PR."

Sports Cardiology: *Then*

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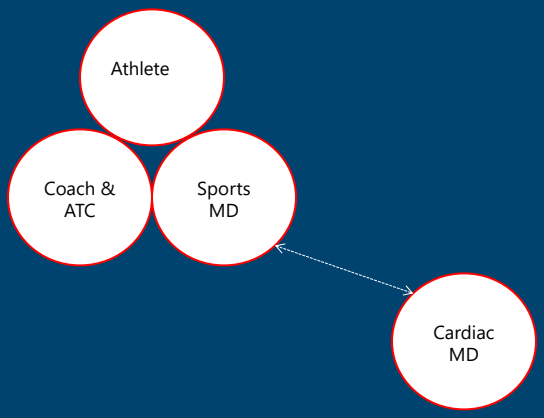
The Old Schema



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Sports Cardiology 2020

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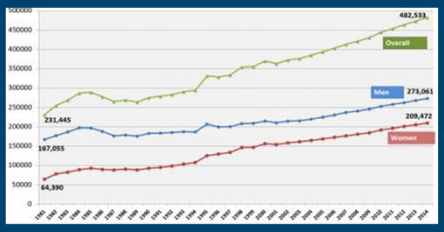
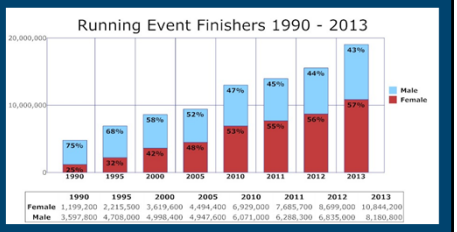
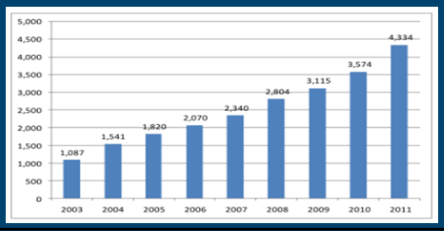
NCAA Athletes



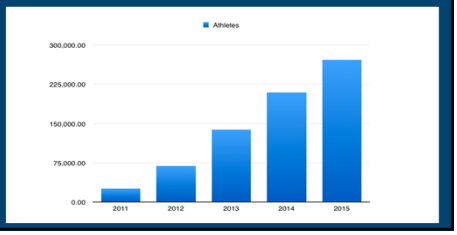
USA Road Race Finishers



US Triathlon Events



"Cross Fit" Athletes



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Sports Cardiology: *Now*

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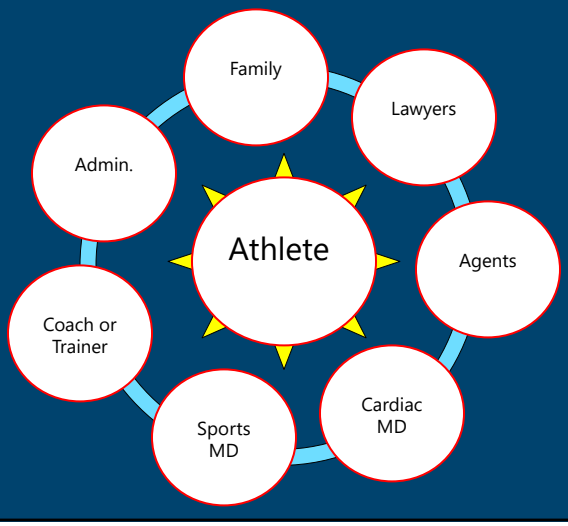
The New Schema



SPORTS & EXERCISE CARDIOLOGY
Member Section
American College of Cardiology

Advisory Board "2011"

Membership 4000 +

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Sports Cardiology: *Now*

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Our Mission

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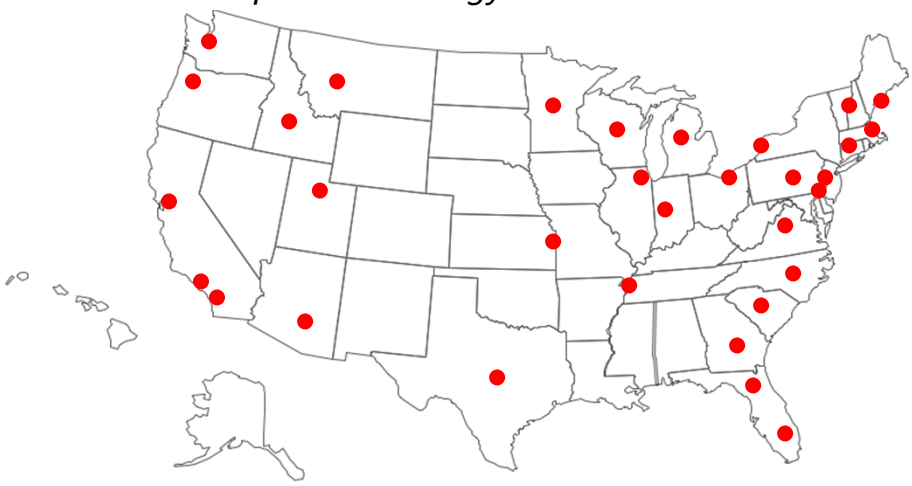
United States Sport Cardiology Resources: 2020



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Sports Cardiology 2020

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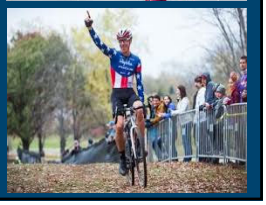
Where have we come from?
&
Where do we need to go?

Eligibility for Young Athletes:
Paternalism Is Over

Care for Masters Athletes:
From Opinion to Data



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No IA Data....What to do?

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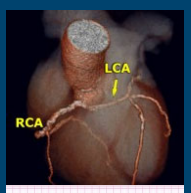

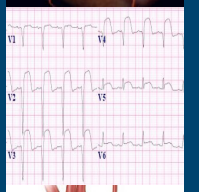

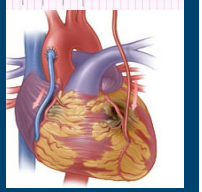

JACC Vol. 6, No. 6
December 1985:1189–90

1189

16th BETHESDA CONFERENCE: CARDIOVASCULAR ABNORMALITIES IN THE ATHLETE: RECOMMENDATIONS REGARDING ELIGIBILITY FOR COMPETITION

Jere H. Mitchell, MD, FACC, Co-Chairman, Barry J. Maron, MD, FACC, Co-Chairman, Stephen E. Epstein, MD, FACC, Co-Chairman

Introduction

BARRY J. MARON, MD, FACC, STEPHEN E. EPSTEIN, MD, FACC, JERE H. MITCHELL, MD, FACC

The focus of the 16th Bethesda Conference is the athlete with an underlying primary cardiovascular abnormality, and its goal is to arrive at consensus opinions for prudent recommendations regarding the eligibi competition. Before the convening formal guidelines were available fo recommendations to athletes were l tuition of the individual physician. jectives, we have tried to determin abnormalities (as well as their se athlete at risk for sudden death, lif cular alterations or disease progres

For the purposes of this Confer the *competitive* athlete as one who nized team or individual sport that petition against others as a central c premium on excellence and achiev orous training in a systematic fash facet of competitive activity is that able to use proper judgment in dete tricate himself or herself from the ce that become necessary. For example cardiovascular distress, such as ligh chest discomfort occurring during c ficult to reliably distinguish from n the physical activity itself; furtherm immediately terminate the physical need to do so is perceived, because stances and pressures of organized a have considered the athlete to be "c of age; he or she could participate (less than 12 years old), interscholastic, intercollegiate, professional or masters (greater than 40 years old) levels. The recommendations of this Conference were not formulated with the purely recreational athlete in mind; they also do not pertain to participation in cardiac rehabilitation programs.

Bowling
Cricket
Curling
Golf
Riflery

is helped formulate some of our work- gh we recognize that these definitions many individuals may participate in in a truly competitive fashion. We that the risk of sudden death in any cardiovascular disease is low; fur- of athletes who die prematurely every l, particularly when the large number ing in sports is considered. Although dete may not be a major public health nevertheless that such catastrophes have n the public consciousness and on the l profession. In addition to the prob- the recommendations of this Confer- nd with the impact that athletic com- n the symptomatic and functional cardiovascular diseases.

ons contained in this report must be First, there are few definitive data hat strenuous exertion predisposes to ose would not have occurred or the awal from competitive athletics will is possible that our recommendations e some individuals to withdraw from This, of course, would be extremely hletes may potentially derive consid- and confidence, as well as physical activities. The primary impetus and justification for this Conference, however, is the general perception, largely intuitive, that individuals with certain organic or structural heart diseases are at increased risk of

0735-1097/85/$3.50

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Bethesda Updates: Key Points

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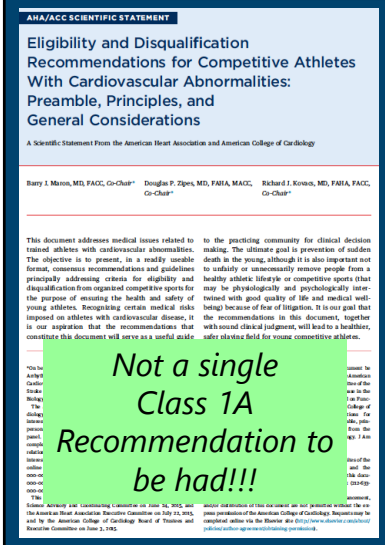
AHA/ACC SCIENTIFIC STATEMENT

Eligibility and Disqualification Recommendations for Competitive Athletes With Cardiovascular Abnormalities: Preamble, Principles, and General Considerations

A Scientific Statement From the American Heart Association and American College of Cardiology

Barry J. Maron, MD, FACC, *Co-Chair*; Douglas P. Zipes, MD, FAHA, MACC, *Co-Chair*; Richard J. Kovacs, MD, FAHA, FACC, *Co-Chair*

This document addresses medical issues related to trained athletes with cardiovascular abnormalities. The objective is to present, in a readily useable format, consensus recommendations and guidelines principally addressing criteria for eligibility and disqualification from organized competitive sports for the purpose of ensuring the health and safety of young athletes. Recognizing certain medical risks imposed on athletes with cardiovascular disease, it is our aspiration that the recommendations that constitute this document will serve as a useful guide to the practicing community for clinical decision making. The ultimate goal is prevention of sudden death in the young, although it is also important not to unfairly or unnecessarily remove people from a healthy athletic lifestyle or competitive sports (that may be physiologically and psychologically intertwined with good quality of life and medical well-being) because of fear of litigation. It is our goal that the recommendations in this document, together with sound clinical judgment, will lead to a healthier, safer playing field for young competitive athletes.

Not a single Class 1A Recommendation to be had!!!

- *15 "Task Force" documents*
- *Topics & Diseases*
- *Disqualification & Restriction (not mgmt.)*
- *Geared to "competitive athletes"*
  (HS, College, Professional)
- *Conflicting Views:*
  - *"Limited Control" populations*
  - *Newly added – Class 2a & 2b Recs*
  - *"It may be reasonable to play…."*

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Bethesda Updates: Key Points

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Class II Recommendations: *Clinical Progress*

412 Total, Class IIa (n=39) & Class IIb (n=47)

Full participation: Asxs R-AOCA with "-" testing (IIa)

Full participation: Post-ICD implantation (IIb)

Full participation: LVNC with normal LVFx (IIb)

Full participation: Revascularized CAD, norm LVFx, no Isch/Elect. (IIb)

Full participation: Cardiac transplant recipients with normal LVFx (IIa)

Full participation: Asxs LQTS following optimal rx. (IIb)

THESE RECOMMENDATIONS NECESSITATE A PROCESS

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Class 2 Recs....What to do?

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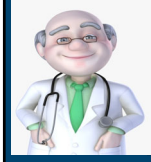
Paternalism



The Decision Making Control Spectrum

Autonomy

Shared Decision Making

Paternalism:

Docs know best

Clearance is our job

The process is simple

Why make it complex?

Autonomy:

Docs don't always know

Patients are individuals

Medical vs. Non-medical

Because it is complex

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Class 2 Recs....What to do?

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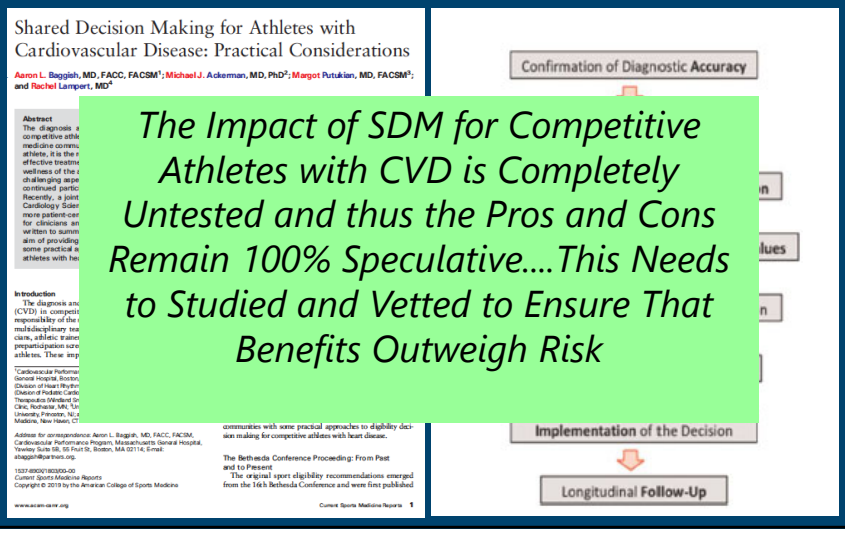
So what do you mean consideration? ..... Shared Decision Making


Shared Decision Making for Athletes with Cardiovascular Disease: Practical Considerations

Aaron L. Baggish, MD, FACC, FACSM[1]; Michael J. Ackerman, MD, PhD[2]; Margot Putukian, MD, FACSM[3]; and Rachel Lampert, MD[4]

Abstract

Introduction

The Bethesda Conference Proceeding: From Past and to Present

The original sport eligibility recommendations emerged from the 16th Bethesda Conference and were first published

Confirmation of Diagnostic Accuracy

Implementation of the Decision

Longitudinal Follow-Up

The Impact of SDM for Competitive Athletes with CVD is Completely Untested and thus the Pros and Cons Remain 100% Speculative....This Needs to Studied and Vetted to Ensure That Benefits Outweigh Risk

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COVID-19 Cardiac

Cardiovascular Performance Program

The "ORCCA" Registry

Outcomes Registry for Cardiac Conditions in Athletes

Aaron Baggish
Nathanial Moulson
Bradley Petek

Jonathan Drezner
Kimberly Harmon

Stephanie Kliethermes
(AMSSM CRN, U of Wisc.)

Manesh Patel (Duke)
Mariell Jessup (AHA)

Members of the ORCCA Study Group:
Steering Committee:
Irfan M. Asif, MD, Aaron L. Baggish, MD, James Borchers, MD, Jonathan A. Drezner, MD, Katherine M. Edenfield, MD, Michael S. Emery, MD, MS, Kyle Goerl, MD, Brian Hainline, MD, Kimberly G. Harmon, MD, Jonathan H. Kim, MD, MSc, Stephanie Kliethermes, PhD, William E. Kraus, MD, Rachel Lampert, MD, Matthew Leiszler, MD, Benjamin D. Levine, MD, Matthew W. Martinez, MD, Nathaniel Moulson, MD, Francis G. O'Connor, MD, MPH, Manesh R. Patel, MD, Bradley J. Petek, MD, Dermot Phelan, MD, Lawrence D Rink, MD, Herman A Taylor, MD, MPH.

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COVID-19 Cardiac

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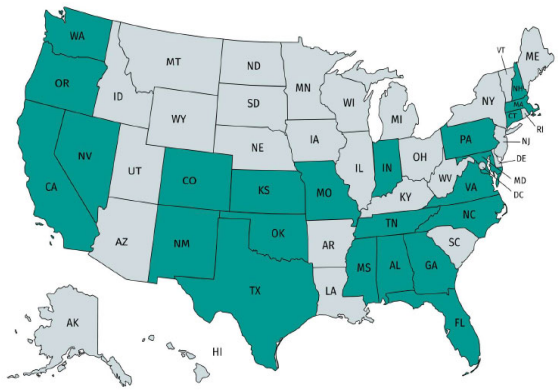
42 Colleges/Universities • 23 States • 14 Athletic Conferences



Median Length of Follow-up = 113 days IQR [90,146]

Circulation, April 2021

Total Athletes Tested for SARS-CoV-2 (n=19,378)

SARS-CoV-2 Positive Athletes (n=3384) **17.5%**

No cardiac evaluation per school preference (n=74)

Planned Cardiac Evaluation (n=3310) **17.1%**

Incomplete Evaluation
- Athlete quit/cut (n=10)
- Testing not started (n=98)
- Missing data from incomplete school submission (n=184)

Final Cohort with Cardiac Evaluation (n=3018) 15.6% **15.6%**

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COVID-19 Cardiac

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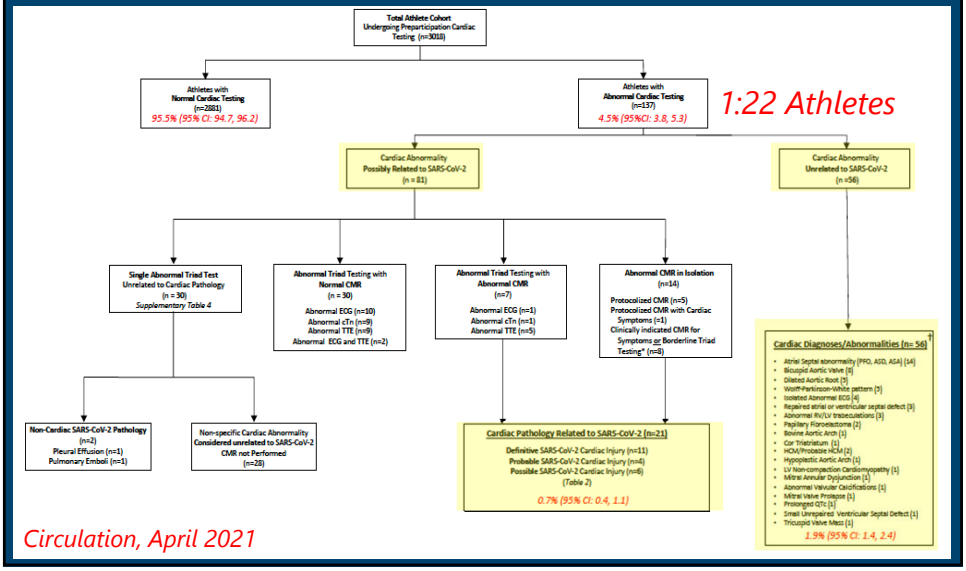

1:22 Athletes

Circulation, April 2021

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CV Care: Masters Athletes

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Men and women >35 years of age who exercise vigorously >5 hours weekly with emphasis on "goals"…

https://www.massgeneral.org/heartcenter/news/multimedia.aspx?id=1101

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Too Much Exercise??

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THE WALL STREET JOURNAL.

SPORTS | November 27, 2012, 8:02 p.m. ET

One Running Shoe in the Grave

New Studies on Older Endurance Athletes Suggest the Fittest Reap Few Health Benefits

Running can take a toll on the heart that essentially eliminates the benefits of exercise. "Running too far, too fast and for too many years may speed one's progress toward the finish line of life," concludes an editorial to be published next month in the British journal Heart.

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Current Controversies

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Re-Eval Old Epi

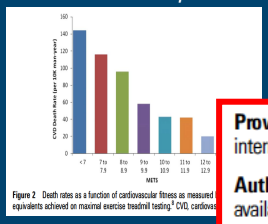


Figure 2 Death rates as a function of cardiovascular fitness as measured equivalents achieved on maximal exercise treadmill testing[1] CVD, cardiova

Run for your life … at a comfortable speed and not too far

James H O'Keefe,[1,2] Carl J Lavie[3,4]

During the Greco-Persian War in 490 BCE, Phidippides, a 40-year-old herald messenger (professional running-courier) ran the 26 miles from a battlefield near Marathon, Greece, into Athens carrying momentous news of Greek victory. Upon arriving at the Acropolis, he proclaimed: 'I rejoice, we have won!' and then immediately collapsed and died.[1] Fast-forward about 2500 years to an era when the

longevity improvements conferred by moderate physical activity. Thus, even though chronic extreme exercise may not kill you, it may erase many of the health advantages of regular moderate exercise.

Indeed, regular vigorous exercise is probably the single best step a person can take to ensure robust CV health. In a study of 416 000 adults followed for a mean of 8 years, 40–50 min per day of vigorous

measured by peak performance on a treadmill, show a curvilinear relationship whereby improvements from unfit to moderately fit confer dramatic reductions in mortality and mortality (figure 2).[4] However, fitness levels above 12 metabolic equivalents do not seem to translate into additional gains in CV health and longevity. Thus, if one is training to be able to run at speeds above 7.5 miles per hour, this is being done for some reason other than further improvements in life expectancy.

CV DAMAGE FROM EXCESSIVE EXERCISE

High-intensity exercise sessions lasting beyond 1–2 h cause acute volume overload of the atria and right ventricle (RV),

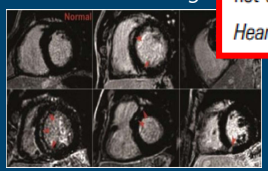
Provenance and peer review Commissioned; internally peer reviewed.

Author note A video presentation on this topic is available on the internet: You Tube, TEDx Talk, James O'Keefe, Run for your life… at a comfortable pace and not too far.

Heart 0;**0**:1–4. doi:10.1136/heartjnl-2012-302886

Phidippides CMP

Cardiac Damage



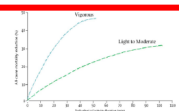
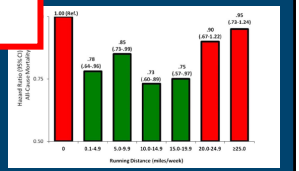


Figure 1 Duration of daily exercise and reduction in long-term all-cause mortality.[1]

Emerging Epi



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The central controversy

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But, for the most part, where there is lots of smoke there's usually at least a little fire….

Does too much exercise over too much time hurt your heart (*and affect morbidity and mortality*) and if so, what does this pathology look like?

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The central controversy

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Why are we talking about this topic now?

- Emerging Epidemiologic Data with a focus on the high ends of fitness
- Anecdotal Observations of Physically Fit Athletes Dying, Perhaps due to Sport-induced CV Disease
- Observational data describing CV pathology among athletes and athletic patients

A syndrome of cardiovascular abnormalities (perhaps caused by) chronic exposure to high volume exercise

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Exercise Dose - Response

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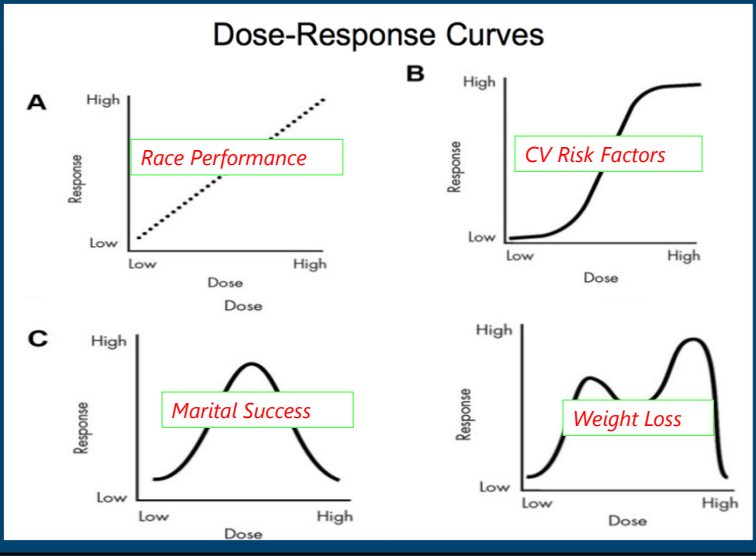

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Too Much Exercise??

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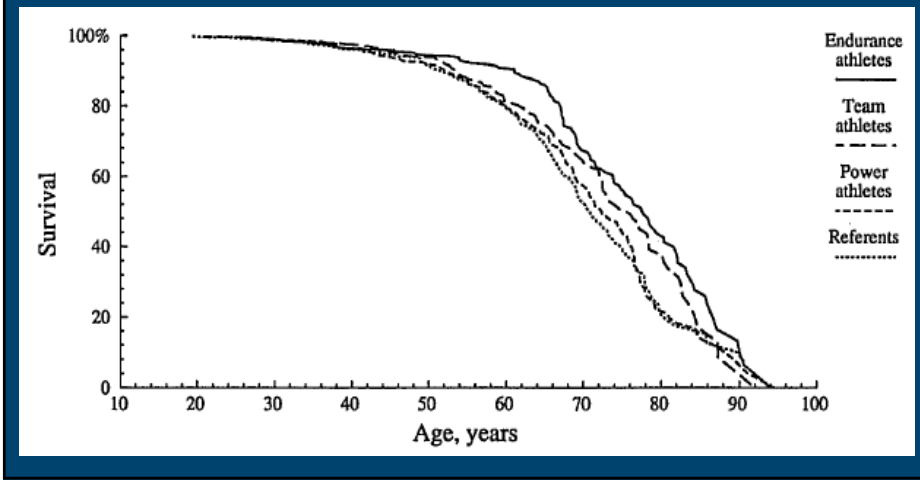
Increased life expectancy of world class male athletes

SEPPO SARNA, TIMO SAHI, MARKKU KOSKENVUO, and JAAKKO KAPRIO



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Too Much Exercise??

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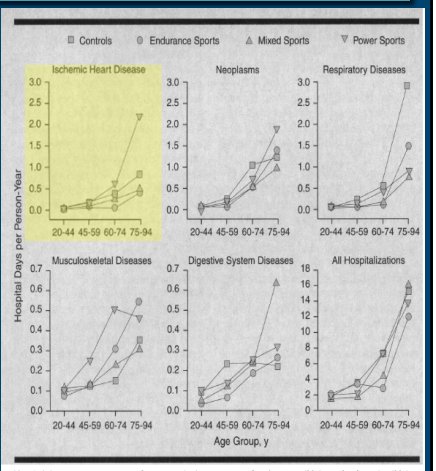
Hospital Care in Later Life Among Former World-Class Finnish Athletes

Urho M. Kujala, MD; Seppo Sarna, PhD; Jaakko Kaprio, MD; Markku Koskenvuo, MD

Table 5.—Rate Ratios (RRs) Adjusted for Age Group and Occupational Group and Their 95% Confidence Intervals for Hospital Days per Year for Different Disease Catergories Among Former Athletes Compared With Controls*

Disease Category	Endurance Sports	Mixed Sports	Power Sports
Diseases of the circulatory system	0.55 (0.53-0.57)	0.90 (0.88-0.93)	0.72 (0.70-0.73)
Ischemic heart disease	0.29 (0.27-0.33)	0.50 (0.48-0.53)	1.34 (1.29-1.40)
Neoplasms	0.51 (0.48-0.54)	0.55 (0.52-0.57)	0.68 (0.66-0.71)
Diseases of the respiratory system	0.41 (0.38-0.44)	0.36 (0.34-0.38)	0.61 (0.59-0.64)
Diseases of the musculoskeletal system and connective tissue	1.44 (1.35-1.55)	1.34 (1.27-1.41)	2.05 (1.95-2.15)
Diseases of the digestive system	0.50 (0.46-0.54)	0.71 (0.67-0.75)	0.74 (0.70-0.78)

*$P<.001$ for all comparisons with controls.



Hospital days per person-years of exposure in 4 age groups of endurance athletes, mixed sports athletes, power sports athletes, and controls for all reasons and for selected diagnostic categories. Hospitalization data from January 1, 1970, through December 31, 1990, for Finnish elite athletes from 1920 to 1965 and controls. For overall statistical differences between groups, see Table 5.

JAMA. 1996;276:216-220

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Too Much Exercise??

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MARATHON RUNNING AND IMMUNITY TO ATHEROSCLEROSIS

Thomas J. Bassler

Department of Pathology
Centinela Hospital
Inglewood, California 90307

Atherosclerosis (ASCVD) is a specific term limited to the lipid-related arterial lesion that accounts for half of the adult deaths in the urban centers of advanced countries. Many factors, the so-called "benefits" of civilization, have been associated with this increased incidence of ASCVD. These include: refined tobacco use,[1] lack of exercise,[2] and a rich diet. The urban diet is suspect because it lacks food "fiber,"[3] unsaturated fats,[4] ascorbic acid,[5] and tocopherols.[6]

Immunity to atherosclerosis has been reported in the Masai warriors who herd cattle on foot[7] and the Tarahumara Indians who take part in ceremonial runs.[8] These populations are primitive. They lack the refined foods found in our urban diet. Their tobacco is coarse and in short supply. It has a harsh smoke that is difficult to inhale. They cover a great deal of ground on foot routinely, averaging 20 kilometers and burning 1,200 kcals each day.

Marathon runners (42-km men) have much in common with these primitive populations.[9] They avoid inhaling tobacco smoke. They cover a great deal of ground on foot, often in the range of 50 to 100 kilometers a week; and their diet contains conspicuous amounts of unsaturated fats, food fiber, and the vitamins C and E. At autopsy, their coronary arteries have been described as "enlarged" and "widely patent."[10] Mann observed this same enlargement in the coronary arteries of Masai warriors and attributed it to their high degree of fitness.[11] Marathon running is "addictive."[12] Few of the 42-km men have discontinued their sport. Thus, in marathoning, we have a "low-risk" life-style with high adherence. Also, the marathon races, themselves, give us a clear record of who the 42-km men are, and their level of fitness at the time of the race. Therefore, the American Medical Joggers Association (AMJA) considers these runners to be the ideal population to study the effects of their multifactoral life-style in the prevention of a high-incidence multifactoral disease, atherosclerosis.

Methods

Reports of deaths in marathon runners are reviewed, world-wide. An excess of 200 reports have been received during the past 10 years. Many are duplicates. Most were below the 42-km threshold.

Results

To date, there have been no reports of fatal ASCVD, histologically proven, among 42-km men.

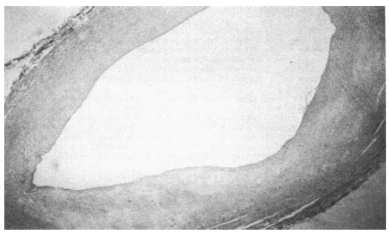

FIGURE 3. Marathoner: Coronary artery with characteristic widely patent lumen and minimal ASCVD. (H & E stain; × 10.)

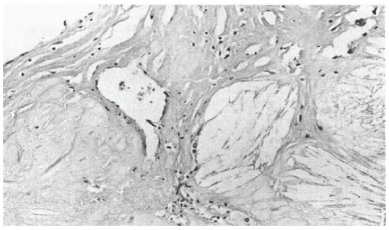

FIGURE 4. Same vessel as shown in FIGURE 3, at higher magnification: Mild inflammatory reaction to lipids in plaque. (H & E stain; × 245.)

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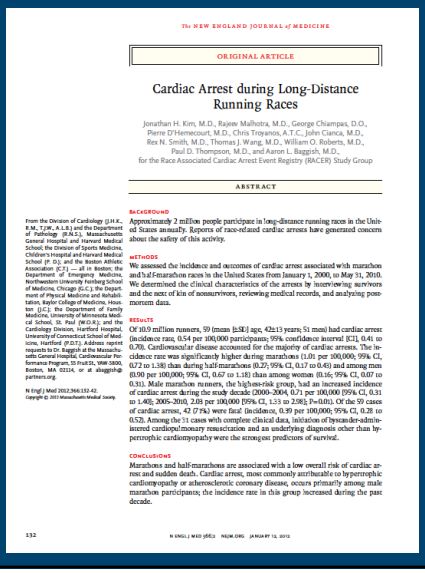
Too Much Exercise??

Cardiovascular Performance Program



The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Cardiac Arrest during Long-Distance Running Races

Jonathan H. Kim, M.D., Rajeev Malhotra, M.D., George Chiampas, D.O., Pierre D'Hemecourt, M.D., Chris Troyanos, A.T.C., John Cianca, M.D., Rex N. Smith, M.D., Thomas J. Wang, M.D., William O. Roberts, M.D., Paul D. Thompson, M.D., and Aaron L. Baggish, M.D., for the Race Associated Cardiac Arrest Event Registry (RACER) Study Group

ABSTRACT

BACKGROUND

Approximately 2 million people participate in long-distance running races in the United States annually. Reports of race-related cardiac arrests have generated concern about the safety of this activity.

METHODS

We assessed the incidence and outcomes of cardiac arrest associated with marathon and half-marathon races in the United States from January 1, 2000, to May 31, 2010. We determined the clinical characteristics of the arrests by interviewing survivors and the next of kin of nonsurvivors, reviewing medical records, and analyzing postmortem data.

RESULTS

Of 10.9 million runners, 59 (mean [±SE] age, 42±13 years; 51 men) had cardiac arrest (incidence rate, 0.54 per 100,000 participants; 95% confidence interval [CI], 0.41 to 0.70). Cardiovascular disease accounted for the majority of cardiac arrests. The incidence rate was significantly higher during marathons (1.01 per 100,000; 95% CI, 0.72 to 1.38) than during half-marathons (0.27; 95% CI, 0.17 to 0.45) and among men (0.90 per 100,000; 95% CI, 0.67 to 1.18) than among women (0.16; 95% CI, 0.07 to 0.31). Male marathon runners, the highest-risk group, had an increased incidence of cardiac arrest during the study decade (2000–2004, 0.71 per 100,000 [95% CI, 0.31 to 1.40]; 2005–2010, 2.05 per 100,000 [95% CI, 1.33 to 2.98]; P=0.01). Of the 59 cases of cardiac arrest, 42 (71%) were fatal (incidence, 0.39 per 100,000; 95% CI, 0.28 to 0.52). Among the 31 cases with complete clinical data, initiation of bystander-administered cardiopulmonary resuscitation and an underlying diagnosis other than hypertrophic cardiomyopathy were the strongest predictors of survival.

CONCLUSIONS

Marathons and half-marathons are associated with a low overall risk of cardiac arrest and sudden death. Cardiac arrest, most commonly attributable to hypertrophic cardiomyopathy or atherosclerotic coronary disease, occurs primarily among male marathon participants; the incidence rate in this group increased during the past decade.

From the Division of Cardiology (J.H.K., R.M., T.J.W., A.L.B.) and the Department of Pathology (R.N.S.), Massachusetts General Hospital and Harvard Medical School; the Division of Sports Medicine, Children's Hospital and Harvard Medical School (P. D.); and the Boston Athletic Association (C.T.) — all in Boston; the Department of Emergency Medicine, Northwestern University Feinberg School of Medicine, Chicago (G.C.); the Department of Physical Medicine and Rehabilitation, Baylor College of Medicine, Houston (J.C.); the Department of Medicine, University of Minnesota Medical School, St. Paul (W.O.R.); and the Cardiology Division, Hartford Hospital, University of Connecticut School of Medicine, Hartford (P.D.T.). Address reprint requests to Dr. Baggish at the Massachusetts General Hospital, Cardiovascular Performance Program, 55 Fruit St., YAW-5800, Boston, MA 02114, or at abaggish@partners.org.

N Engl J Med 2012;366:132-42.

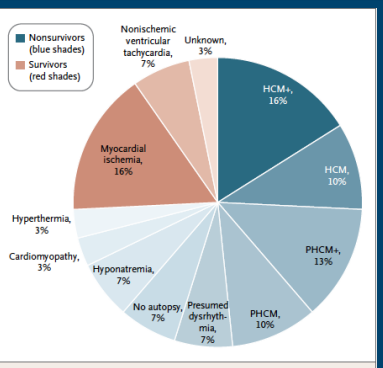


Figure 2. Causes of Cardiac Arrest among Nonsurvivors and Survivors.
HCM denotes hypertrophic cardiomyopathy; HCM+ denotes HCM and additional diagnoses, including coronary artery disease (in 2 persons), myocarditis (in 2), and bicuspid aortic-valve and coronary anomaly (in 1). PHCM denotes possible hypertrophic cardiomyopathy. PHCM+ denotes PHCM and additional diagnoses, including coronary artery disease (in 1 person), accessory atrioventricular nodal bypass tract (in 1), hyperthermia (in 1), and bicuspid aortic-valve and coronary anomaly (in 1). One nonsurvivor with hyponatremia was also found to have myxomatous valvular disease of the tricuspid, mitral, and aortic valves. Data include arrhythmogenic right ventricular cardiomyopathy (in 1 person). Because of rounding, percentages do not add up to 100.

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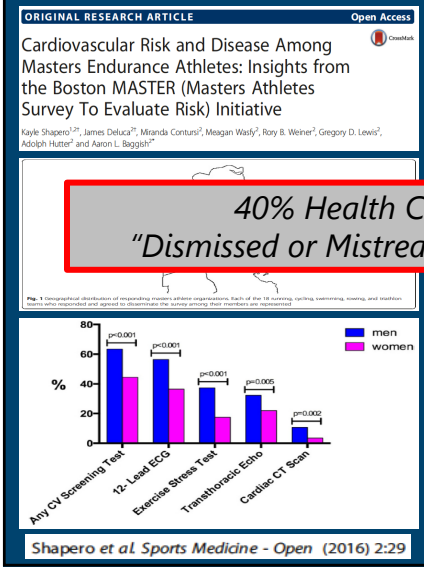
Athletes as a Patient Population

Cardiovascular Performance Program



ORIGINAL RESEARCH ARTICLE Open Access

Cardiovascular Risk and Disease Among Masters Endurance Athletes: Insights from the Boston MASTER (Masters Athletes Survey To Evaluate Risk) Initiative

Kayle Shapero[1,2*], James Deluca[2*], Miranda Contursi[2], Meagan Wasfy[2], Rory B. Weiner[2], Gregory D. Lewis[2], Adolph Hutter[2] and Aaron L. Baggish[2*]

Shapero et al. Sports Medicine - Open (2016) 2:29

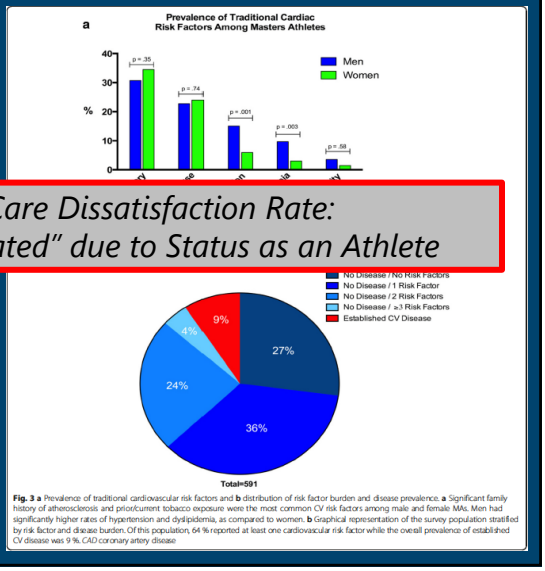


Fig. 3 a Prevalence of traditional cardiovascular risk factors and **b** distribution of risk factor burden and disease prevalence. **a** Significant family history of atherosclerosis and prior/current tobacco exposure were the most common CV risk factors among male and female MAs. Men had significantly higher rates of hypertension and dyslipidemia, as compared to women. **b** Graphical representation of the survey population stratified by risk factor and disease burden. Of this population, 64 % reported at least one cardiovascular risk factor while the overall prevalence of established CV disease was 9 %. *CAD* coronary artery disease

40% Health Care Dissatisfaction Rate:
"Dismissed or Mistreated" due to Status as an Athlete

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Key CV Controversies

Cardiovascular Performance Program

1.) Do endurance sports cause arrhythmia?

2.) Do endurance sports cause cardiomyopathy?

3.) Do endurance sports cause aortopathy?

4.) Do endurance sports cause coronary disease?

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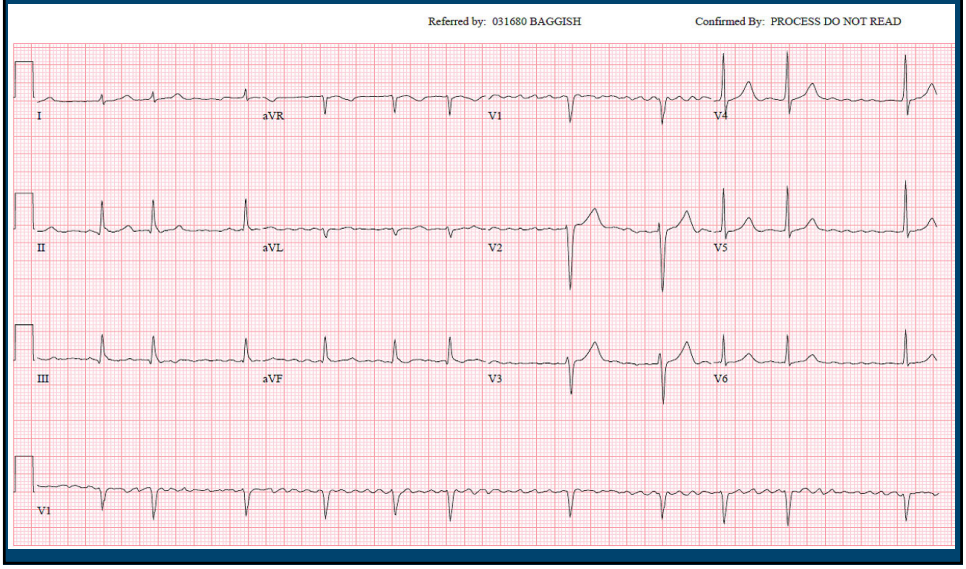
52 y.o. marathoner with decreased ex tolerance

Cardiovascular Performance Program



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Arrhythmia: AFib

1st Author	Reference	Subjects	Primary Finding
Karjalainen	BMJ 1998	Runners (n=100)	5.5 (1.3–24.4)
Mont	EHJ 2002	Endurance (n=70)	71% with lone AF
Elosua	Int J Card 2006	Endurance (n=51)	2.87 (1.39–7.05)
Heidbuchel	Int J Card 2006	Endurance (n=53)	1.81 (1.10-2.98)
Molina	Europace 2008	Runnners (n=39)	8.8 (1.26-61.3)
Baldesberger	EHJ 2008	Cyclist (n=67)	10% AF in athletes
Mont	Europace 2008	Endurance (n=48)	7.31 (2.33-22.9)
Aizer	Am J Card 2009	PHS Database (n=16,921)	1.20 with ≥ 7 days/week ex.*
Claessen	Heart 2011	Non-sel. Flutter RFA (n=58)	"Sportsmen" 50% of Lone Afl pop.
Andersen	EHJ 2013	Nordic Skiers (n=52,755)	1.29 (1.04-1.61)*

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Arrhythmia: A-Fib.

Risk of arrhythmias in 52 755 long-distance cross-country skiers: a cohort study

Kasper Andersen[1*], Bahman Farahmand[2,3], Anders Ahlbom[2], Claes Held[1], Sverker Ljunghall[1], Karl Michaëlsson[4], and Johan Sundström[1]

- Retrospective design
- Sweden -- Vasaloppet (90K)
- >50,000 participants
- 1989-99 >> Dec. 2005
- Incidence of arrhythmia events
- Race time / # of races > Arrhyth.
- AF the dominant rhythm issue

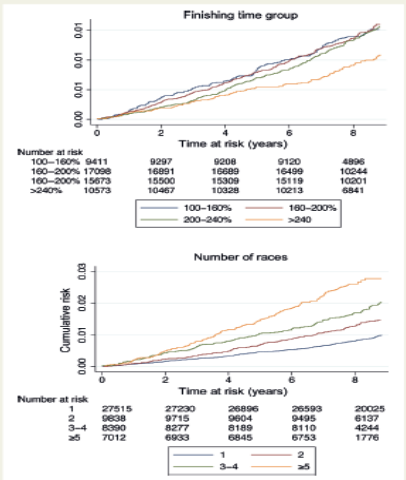

Figure 1 Cumulative incidence of any arrhythmia by finishing time group in per cent of winning time and number of completed races during the period 1989–98.

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Arrhythmia: A-Fib.

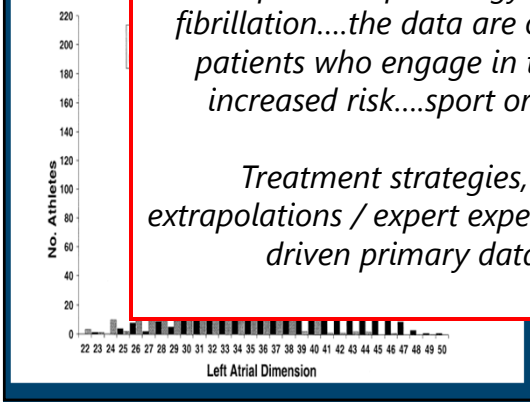
Cardiovascular Performance Program

Prevalence and Clinical Significance of Left Atrial Remodeling in Competitive Athletes

Antonio Pelliccia, MD,* Barry J. Maron, MD,† Fernando M. Di Paolo, MD,* Alessandro Biffi, MD,* Filippo M. Quattrini, MD,* Cataldo Pisicchio, MD,* Alessandra Roselli, MD,* Stefano Caselli, MD,* Franco Culasso, PhD‡

Rome, Italy; and Minneapolis, Minnesota

Pelliccia et al.
Left Atrial Remodeling in Co



The strongest association between endurance sport and pathology lies with atrial fibrillation….the data are clear that our aging patients who engage in these sports are at increased risk….sport or lifestyle choices?

Treatment strategies, at present, are extrapolations / expert experience. More outcome driven primary data are needed.

MECHANISMs of ATHLETIC AF

- *LA Dilation / Remodeling*

ion

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Rx: A-fib

Cardiovascular Performance Program

But wait a minute….We've got this Afib thing figured out!

Rate control vs. Rhythm Maintenance

The New England Journal of Medicine

A COMPARISON OF RATE CONTROL AND RHYTHM CONTROL IN PATIENTS WITH ATRIAL FIBRILLATION

"Rate control ruined my life"

Clotting vs. Bleeding

Arrhythmia/Electrophysiology

Selecting Patients With Atrial Fibrillation for Anticoagulation
Stroke Risk Stratification in Patients Taking Aspirin

TABLE 3. Validation of Stratification Schemes for Primary Prevention of Stroke in 2014 Participants Prescribed Aspirin

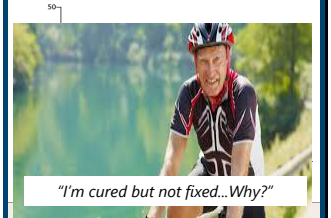
Scheme	Strokes Per 100 Patient-Years, Stratified by Risk		
	Low	Moderate	High
AFI	0.9 (0.3–2.3; n=235)	1.7 (1.1–2.5; n=781)	3.5 (2.7–4.5; n=998)
SPAF	1.1 (0.7–1.8; n=668)	2.7 (1.8–4.0; n=462)	3.6 (2.7–4.7; n=884)

"Were these scores made for people like me?"

Anti-arrhythmics vs. Catheters

The NEW ENGLAND JOURNAL of MEDICINE

Radiofrequency Ablation as Initial Therapy in Paroxysmal Atrial Fibrillation



"I'm cured but not fixed…Why?"

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Key CV Controversies

Cardiovascular Performance Program

1.) Do endurance sports cause arrhythmia? ✔

2.) Do endurance sports cause cardiomyopathy? ✔

3.) Do endurance sports cause aortopathy?

4.) Do endurance sports cause coronary disease?

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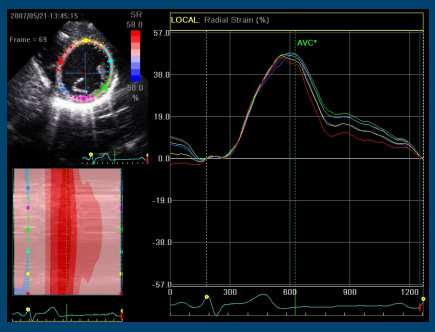
Cardiomyopathy

Cardiovascular Performance Program

The impact of endurance exercise training on left ventricular systolic mechanics

Aaron L. Baggish,[1] Kibar Yared,[1] Francis Wang,[2] Rory B. Weiner,[1] Adolph M. Hutter Jr.,[1] Michael H. Picard,[1] and Malissa J. Wood[1]
[1]Division of Cardiology, Massachusetts General Hospital, Boston, Massachusetts; and [2]University Health Services, Harvard University, Cambridge, Massachusetts

Submitted 15 April 2008; accepted in final form 7 July 2008



The Harvard Athlete Initiative

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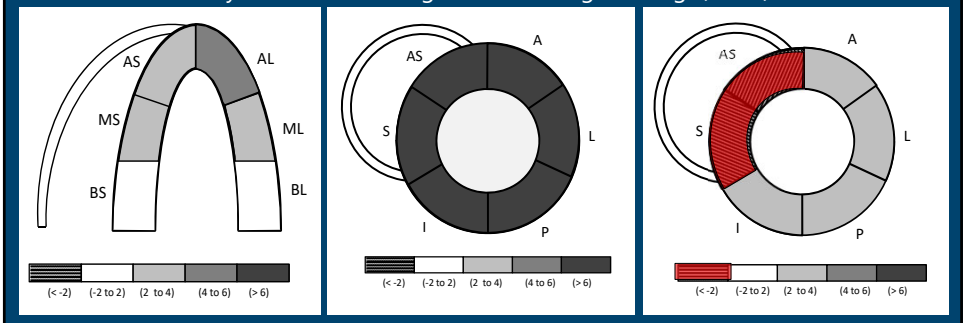
Cardiomyopathy

The impact of endurance exercise training on left ventricular systolic mechanics

Am J Physiol Heart Circ Physiol 2008

LV Systolic Strain Changes with Rowing Training (n=20)



Longitudinal ~ ⇧20-25%

Radial ~ ⇧50-60%

Circumferential ~ ⇩20-25%

Normal to Supranormal Strain….Focal Septal Dysfunction Fatigue??

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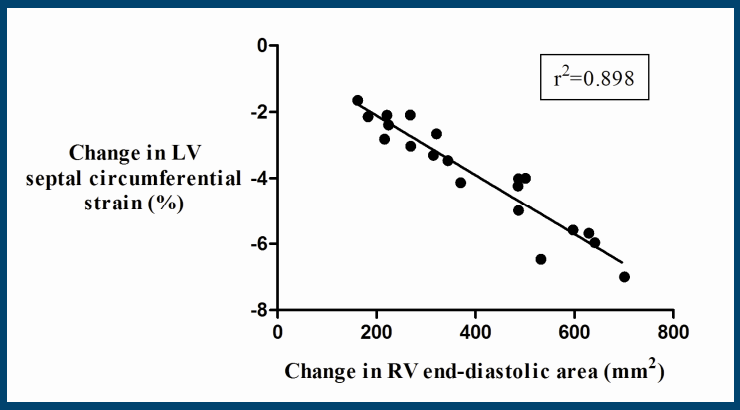
Cardiomyopathy

The Ventricular Interdependence of Physiologic Remodeling



Am J Physiol Heart Circ Physiol 2008;295:1109

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Cardiomyopathy

Cardiovascular Performance Program

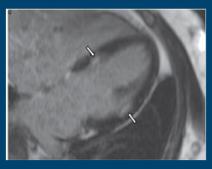
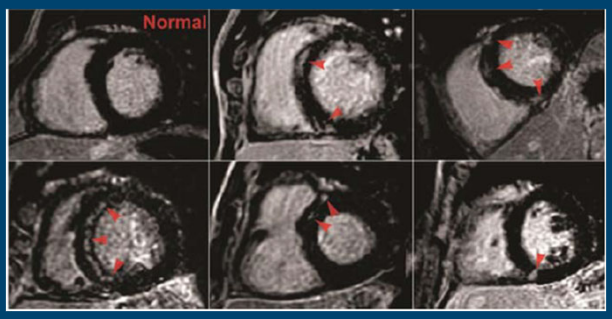


Table 3. *Location and extent of LGE in veteran athletes*

Participant No.	Age, yr	Percentage of Total LGE Mass, g	LGE Pattern	Perfusion Defect	Interpretation	Location
1	67	18.9	CAD	Yes	Probable dual infarction	Septal and lateral wall
2	50	8	Non-CAD	No	Probable myocarditis	Epicardial lateral wall
3	66	3	Non-CAD	No	Nonspecific	Basal and midinsertion point
4	60	3	Non-CAD	No	Nonspecific	Inferior insertion point mid and apical
5	50	1	Non-CAD	No	Nonspecific	Insertion point inferior mid/apical
6	51	1	Non-CAD	No	Nonspecific	Inferior insertion point

Whyte et al. JAP 2011



LaGerche et al. EHJ 2011

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Cardiomyopathy

Cardiovascular Performance Program

Exercise-induced right ventricular dysfunction and structural remodelling in endurance athletes

André La Gerche[1,2*], Andrew T. Burns[3], Don J. Mooney[3], Warrick J. Inder[1], Andrew J. Taylor[4], Jan Bogaert[5], Andrew I. MacIsaac[3], Hein Heidbüchel[2], and David L. Prior[1,3]

Table 1 Baseline demographic and functional measures according to the endurance event completed

	Overall	Marathon run	Endurance triathlon[a]	Alpine cycling	Ultra triathlon[a]	P-value
Number of athletes	40	7	11	9	13	
Race distance (km)		42.2	1.9/90/21.1	207	3.8/180/42.2	
Race completion time		2 h 59 min ± 30 min	5 h 24 min ± 25 min	8 h 5 min ± 42 min	10 h 52 min ± 1 h 16 min	
Ambient temperature (°C)		16–20	18–31	24–34	17–28	
Age (years)	37 ± 8	38 ± 3	33 ± 7	44 ± 9	34 ± 8	0.014
Male (%)	90	86	91	78	100	0.378
BMI (kg/m^2)	23.6 ± 1.9	22.3 ± 1.6	24.0 ± 2.1	23.9 ± 2.1	23.5 ± 1.3	0.306
% of predicted VO_2max	146 ± 18	142 ± 8	141 ± 20	154 ± 20	148 ± 18	0.36
Training (years)	10 ± 9	13 ± 8	6 ± 5	12 ± 14	11 ± 9	0.277
Training (h/week)	16.3 ± 5.1	14 ± 6	14 ± 3	13 ± 4	21 ± 5	<0.0001

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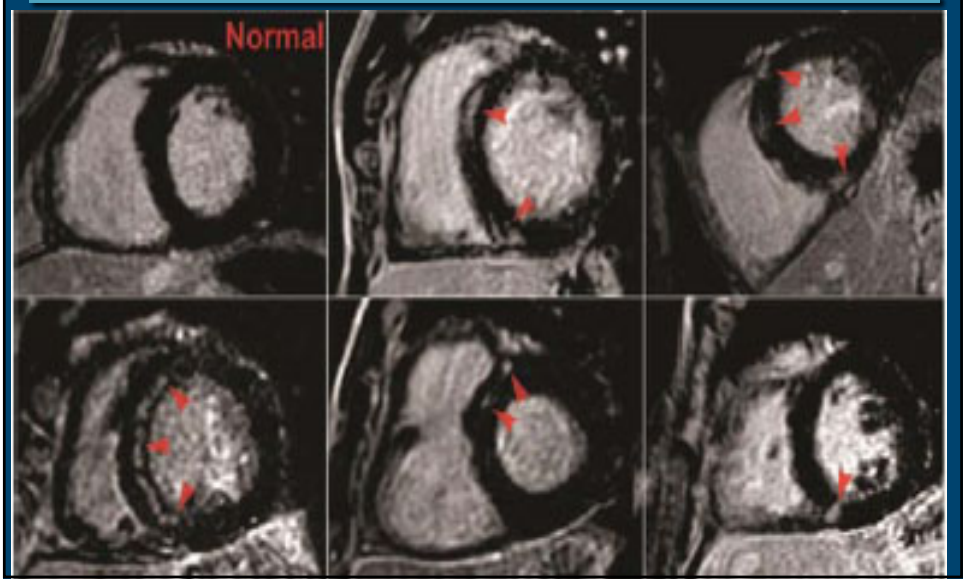
Cardiomyopathy

Exercise-induced right ventricular dysfunction and structural remodelling in endurance athletes

André La Gerche[1,2*], Andrew T. Burns[3], Don J. Mooney[3], Warrick J. Inder[1], Andrew J. Taylor[4], Jan Bogaert[5], Andrew I. MacIsaac[3], Hein Heidbüchel[2], and David L. Prior[1,3]



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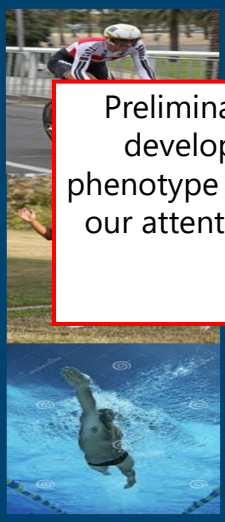
Exercise Cardiomyopathy ?

Cardiovascular Performance Program



Chronic Extreme Volume & Intensity

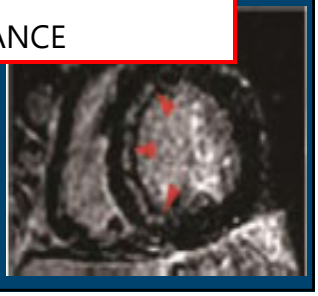
A Theoretical Pathogenic

Preliminary data suggests that some athletes may develop a patchy fibrosis (?cardiomyopathy)…a phenotype of uncertain clinical relevance that deserves our attention and further outcomes-based study….at this point:
NO CLEAR CLINICAL RELEVANCE

(Genetics)

+

Secondary Process (Drugs, Infection, Disease)



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Key CV Controversies

Cardiovascular Performance Program

1.) Do endurance sports cause arrhythmia? ✔

2.) Do endurance sports cause cardiomyopathy? ✔

3.) Do endurance sports cause aortopathy? ✔

4.) Do endurance sports cause coronary disease?

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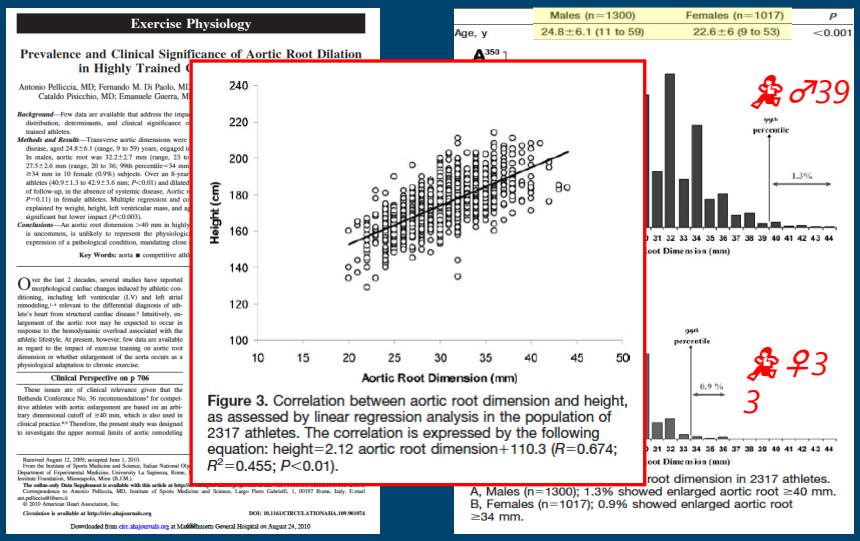
Aortopathy

Cardiovascular Performance Program



Exercise Physiology

Prevalence and Clinical Significance of Aortic Root Dilation in Highly Trained

Antonio Pelliccia, MD; Fernando M. Di Paolo, MD; Cataldo Pisicchio, MD; Emanuele Guerra, MD

Clinical Perspective on p 706

Downloaded from circ.ahajournals.org at Massachusetts General Hospital on August 24, 2010

Figure 3. Correlation between aortic root dimension and height, as assessed by linear regression analysis in the population of 2317 athletes. The correlation is expressed by the following equation: height=2.12 aortic root dimension+110.3 (R=0.674; R^2=0.455; P<0.01).

root dimension in 2317 athletes.
A, Males (n=1300); 1.3% showed enlarged aortic root ≥40 mm.
B, Females (n=1017); 0.9% showed enlarged aortic root ≥34 mm.

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Aortopathy

Cardiovascular Performance Program

Table 3. Results of Base Case and Subgroup Analyses of Aortic Root in Athletes Compared With Controls at the Aortic Valve Annulus

Study-Level Factor	No. of Subjects	Meta-Regression Analysis: Adjusted Difference at Aortic Valve Annulus, mm (95% CI)
Base case analysis		
Participant type		
Athlete	2104	1.6 (0.2 to 3.0)*
Control	493	Reference
Gender		
Male	830	4.1 (0.4 to 7.8)*
Mixed/NA	1316	−2.6 (−7.3 to 2.1)
Female	451	Reference
Study location		
Non-US	2281	−1.5 (−4.0 to 1.0)
US	316	Reference
Subgroup analysis		
Training type		
Endurance athletes	799	2.2 (0.4 to 4.0)*
Strength athletes	425	1.6 (−0.4 to 3.5)
Control subjects	463	Reference

CI indicates confidence interval; NA, not available.
*$P<0.05$.

Background—The aorta is exp… …t the aorta is larger in athletes is not clear. We performed a… …whethere athletes demonstrate increased aortic root dimensi…

Methods and Results—We se… …ugust 12, 2012, for English-language studies reporting t… …ndently extracted athlete and study characteristics. A multi… …ion analyses. We identified 71 studies reporting aortic root d… …s met our criteria by reporting aortic root dimensions at the… …580). Athletes were compared directly with controls (n=727… …root diameter measured at the sinuses of Valsalva was 3.2 m… …whereas aortic root size at the aortic valve annulus was 1.6…

Conclusions—Elite athletes hav… …ses of Valsalva and aortic valve annulus, but this difference i… …etes should know that marked aortic root dilatation likely re… …ion to exercise. (***Circulation.*** **2013;127:791-798.)**

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Aortopathy

Cardiovascular Performance Program

JAMA Cardiology | Original Investigation

Association of Ascending Aortic Dilatation and Long-term Endurance Exercise Among Older Masters-Level Athletes

Timothy W. Churchill, MD; Erich Groesinger, MS; Jonathan H. Kim, MD; Garrett Loomer, MS; J. Sawalla Guseh, MD; Meagan M. Wasfy, MD; Eric M. Isselbacher, MD, MHCDS; Gregory D. Lewis, MD; Rory B. Weiner, MD; Christian Schmied, MD; Aaron L. Baggish, MD

IMPORTANCE Aortic dilatation is frequently encountered in clinical practice among aging endurance athletes, but the distribution of aortic sizes in this population is unknown. It is additionally uncertain whether this may represent aortic adaptation to long-term exercise, similar to the well-established process of ventricular remodeling.

OBJECTIVE To assess the prevalence of aortic dilatation among long-term masters-level male and female athletes with about 2 decades of exercise exposure.

DESIGN, SETTING, AND PARTICIPANTS This cross-sectional study evaluated aortic size in veteran endurance athletes. Masters-level rowers and runners aged 50 to 75 years were enrolled from competitive athletic events across the United States from February to October 2018. Analysis began January 2019.

EXPOSURES Long-term endurance exercise.

MAIN OUTCOMES AND MEASURES The primary outcome was aortic size at the sinuses of Valsalva and the ascending aorta, measured using transthoracic echocardiography in accordance with contemporary guidelines. Aortic dimensions were compared with age, sex, and body size–adjusted predictions from published nomograms, and z scores were calculated where applicable.

RESULTS Among 442 athletes (mean [SD] age, 61 [6] years; 267 men [60%]; 228 rowers [52%]; 214 runners [48%]), clinically relevant aortic dilatation, defined by a diameter at sinuses of Valsalva or ascending aorta of 40 mm or larger, was found in 21% (n = 94) of all participants (83 men [31%] and 11 women [6%]). When compared with published nomograms, the distribution of measured aortic size displayed a rightward shift with a rightward tail (all $P < .001$). Overall, 105 individuals (24%) had at least 1 z score of 2 or more, indicating an aortic measurement greater than 2 SDs above the population mean. In multivariate models adjusting for age, sex, body size, hypertension, and statin use, both elite competitor status (rowing participation in world championships or Olympics or marathon time under 2 hours and 45 minutes) and sport type (rowing) were independently associated with aortic size.

CONCLUSIONS AND RELEVANCE Clinically relevant aortic dilatation is common among aging endurance athletes, raising the possibility of vascular remodeling in response to long-term exercise. Longitudinal follow-up is warranted to establish corollary clinical outcomes in this population.

Author Audio Interview
Supplemental content

Author Affiliations: Cardiovascular Performance Program, Massachusetts General Hospital, Boston (Churchill, Groesinger, Loomer, Guseh, Wasfy, Lewis, Weiner, Baggish); Echocardiography Laboratory, Massachusetts General Hospital, Boston (Churchill, Wasfy, Isselbacher, Weiner, Baggish); Division of Cardiology, Emory Clinical Cardiovascular Research Institute, Atlanta, Georgia (Kim); Thoracic Aortic Center, Massachusetts General Hospital, Boston (Isselbacher); University Heart Center, Zurich, Switzerland (Schmied).

Corresponding Author: Aaron L. Baggish, MD, Cardiovascular Performance Program, Massachusetts General Hospital, 55 Fruit St, Yawkey Sta 5B, Boston, MA 02114 (abaggish@partners.org).

JAMA Cardiol. doi:10.1001/jamacardio.2020.0054
Published online February 26, 2020.

Table 1. Participant Data and Select Echocardiographic Parameters

Characteristic	Mean (SD)			
	Male		Female	
	Rowers (n = 114)	Runners (n = 153)	Rowers (n = 114)	Runners (n = 61)
sAge, y	63 (6)	59 (6)	62 (7)	59 (6)
Height, in	72 (3)	70 (2)	66 (2)	65 (3)
Weight, kg	87 (13)	74 (8)	67 (10)	57 (8)
BMI	25 (3)	24 (2)	23 (3)	21 (2)
Blood pressure, mm Hg				
Systolic	125 (15)	132 (15)	119 (13)	122 (14)
Diastolic	75 (9)	78 (9)	71 (9)	75 (10)
Medical history, No. (%)				
Coronary artery disease	6 (5)	6 (4)	1 (1)	1 (2)
Hypertension	24 (21)	19 (12)	7 (6)	5 (8)
Hyperlipidemia	26 (23)	31 (20)	19 (17)	8 (13)
Diabetes mellitus	3 (3)	2 (1)	1 (1)	2 (3)
Smoking (current or former)	18 (16)	19 (12)	26 (23)	16 (26)
Atrial fibrillation	10 (9)	2 (1)	4 (4)	0
Medications				
No medications	50 (44)	104 (68)	41 (37)	31 (51)
Antihypertensive medication	19 (17)	9 (6)	4 (4)	2 (3)
Statin	25 (22)	11 (7)	3 (3)	1 (2)
Antiplatelet/anticoagulant	8 (7)	7 (5)	6 (5)	0
Athletic history				
Cumulative years training	26 (11)	24 (11)	20 (8)	22 (9)
Annual training volume, h	244 (96)	243 (98)	239 (96)	237 (77)
No. of marathons[a]	6 (9)	32 (31)	7 (13)	33 (54)
Marathon time (personal best), min	NA	196 (23)	NA	222 (21)
Select echocardiographic parameters				
Left ventricular volume index, end-diastole, mL/m^2	66 (14)	75 (13)	55 (12)	64 (11)
Left ventricular mass index, g/m^2	75 (11)	78 (12)	68 (9)	73 (11)
Lateral e', cm/s	7.9 (2.3)	9.4 (2.2)	8.5 (2.0)	9.0 (1.9)
Medial e', cm/s	6.5 (2.0)	7.2 (1.6)	7.0 (1.6)	7.3 (1.5)

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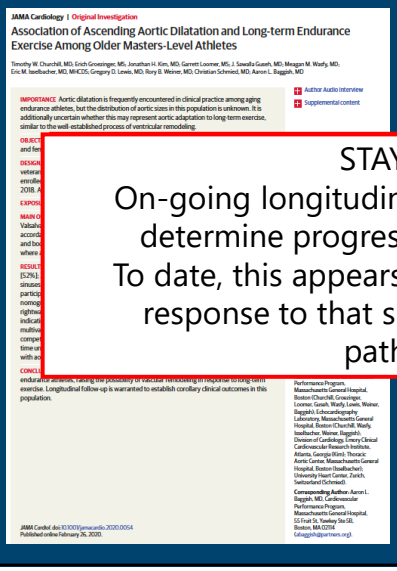
Aortopathy


JAMA Cardiology | Original Investigation

Association of Ascending Aortic Dilatation and Long-term Endurance Exercise Among Older Masters-Level Athletes

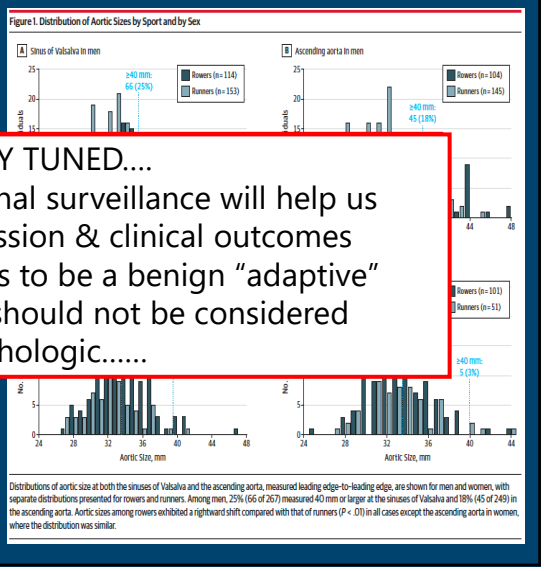


Figure 1. Distribution of Aortic Sizes by Sport and by Sex

A Sinus of Valsalva in men

B Ascending aorta in men

Rowers (n = 114)
Runners (n = 153)
≥40 mm: 66 (25%)
Rowers (n = 104)
Runners (n = 145)
≥40 mm: 45 (18%)
Rowers (n = 101)
Runners (n = 51)
≥40 mm: 5 (3%)
No. of Individuals
Aortic Size, mm

Distributions of aortic size at both the sinuses of Valsalva and the ascending aorta, measured leading edge-to-leading edge, are shown for men and women, with separate distributions presented for rowers and runners. Among men, 25% (66 of 267) measured 40 mm or larger at the sinuses of Valsalva and 18% (45 of 249) in the ascending aorta. Aortic sizes among rowers exhibited a rightward shift compared with that of runners ($P < .01$) in all cases except the ascending aorta in women, where the distribution was similar.

STAY TUNED….
On-going longitudinal surveillance will help us determine progression & clinical outcomes
To date, this appears to be a benign "adaptive" response to that should not be considered pathologic……

Key CV Controversies

1.) Do endurance sports cause arrhythmia? ✔

2.) Do endurance sports cause cardiomyopathy? ✔

3.) Do endurance sports cause aortopathy? ✔

4.) Do endurance sports cause coronary disease? ✔

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CV Risk Factors with Exercise

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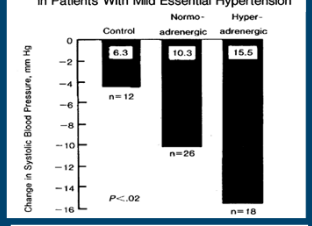
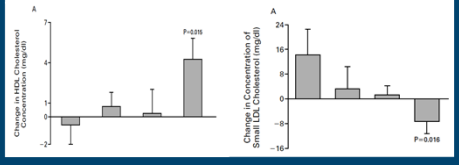
JAMA 1985

The Effects of Aerobic Exercise on Plasma Catecholamines and Blood Pressure in Patients With Mild Essential Hypertension



NEJM 2002

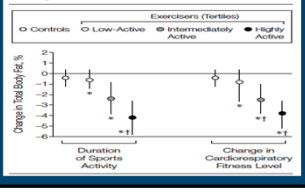
EFFECTS OF THE AMOUNT AND INTENSITY OF EXERCISE ON PLASMA LIPOPROTEINS



JAMA 2003

Effect of Exercise on Total and Intra-abdominal Body Fat in Postmenopausal Women
A Randomized Controlled Trial

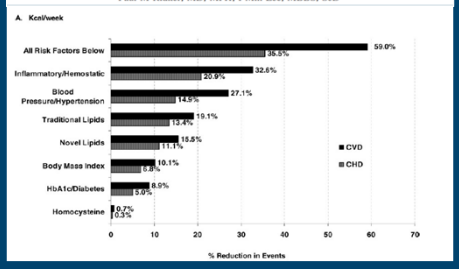
Figure 2. Percentage Change in Total Body Fat at 12 Months by Tertile of Duration and Change in Fitness Level



Circulation 2007

Physical Activity and Reduced Risk of Cardiovascular Events
Potential Mediating Mechanisms

Samia Mora, MD, MHS; Nancy Cook, ScD; Julie E. Buring, ScD; Paul M Ridker, MD, MPH; I-Min Lee, MBBS, ScD



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CAD: Observational Data

Cardiovascular Performance Program

Running: the risk of coronary events†

Prevalence and prognostic relevance of coronary atherosclerosis in marathon runners

108 M (>50 y.)
At least 5 marathons

Table 2 Distribution of coronary artery calcification (CAC) me

	Participants of the Heinz Nixdorf Recall Study		
	Marathon runners (group I)	Age-matched controls (8:1) (group II)	Con risk
$\log_2$(CAC + 1) (mean ± SD)	4.1 ± 3.6	4.9 ± 3.3	3.8 ±
CAC (Q1/median/Q3)	0/36/217	3/38/187	0/12
zero CAC (%)	28.7	18.4	31.5
CAC >75th percentile (%)	25.0	24.2	14.8
CAC 0 to <10	40.74	34.61	48.6
CAC 10 to <100	23.15	29.05	29.6
CAC 100 to <400	23.15	22.80	13.4
CAC ≥400	12.96	13.54	8.33

Comparisons in continuous or binary measures adjusted for matching factors (age for group I/g

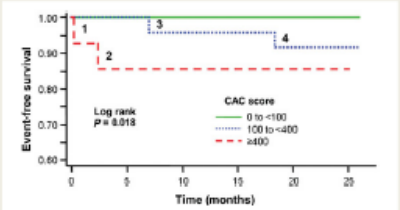

Figure 1 Kaplan–Meier estimates of event-free survival by extent of coronary artery calcification (CAC). No marathon runners with CAC <100 experienced a coronary event, while 8% and 14.3% of those with CAC 100 to <400 and ≥400, respectively, required revascularization during follow-up. Using Cox regression analysis, hazard ratios for a two-fold increase in $\log_2$(CAC+1) were: hazard ratio = 1.51, 95% confidence interval = 0.97–2.36, $P = 0.07$. The numbers pertain to the subjects with events in *Table 3*.

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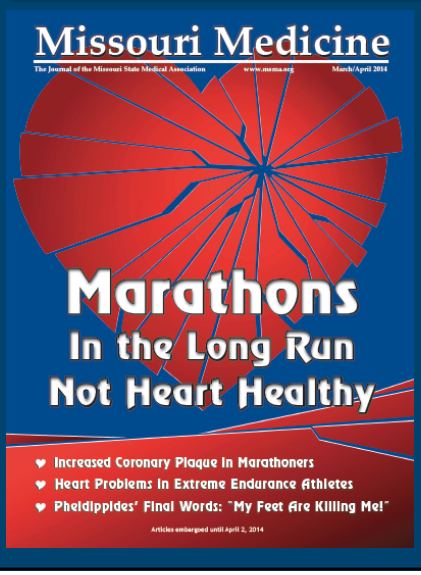
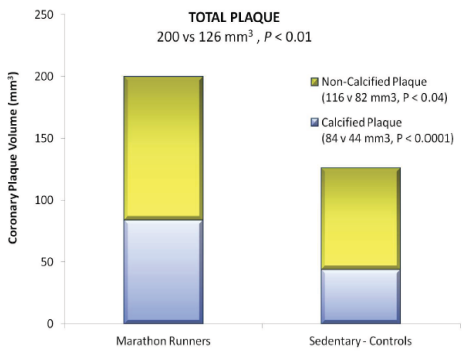
CAD: Observational Data



Increased Coronary Artery Plaque Volume Among Male Marathon Runners

by Robert S. Schwartz, MD, Stacia Merkel Kraus, MPH, Jonathan G. Schwartz, MD, Kelly K. Wickstrom, BS, Gretchen Peichel, RN, Ross F. Garberich, MS, John R. Lesser, MD, Stephen N. Oesterle, MD, Thomas Knickelbine, MD, Kevin M. Harris, MD, Sue Duval, PhD, William O. Roberts, MD & James H. O'Keefe, MD

Figure 1
Marathoners had significantly more total coronary plaque volume, non-calcified plaque volume and calcified plaque volume compared to control subjects.



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CAD: Observational Data

Interpretation Options:

1.) High levels of exercise causes atherosclerosis (CAD)

2.) Traditional CAD risk factors causal/ underappreciated

3.) CAD driven by unknown/unmeasured risk factors

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CAD: Observational Data

Running: the risk of coronary events[†]

Prevalence and prognostic relevance of coronary atherosclerosis in marathon runners

Table 4 Risk factors and test results of participants with events during follow-up

Risk factors/test results at baseline	Subjects with an event during follow-up				Normal range[a]
	1	2	3	4	
Age (years)	66	64	55	62	
BMI (kg/m^2)	22.5	24.6	22.0	22.0	<25
Systolic blood pressure (mmHg)	110/61	105/67[b]	153/96	138/82	<120/80
History of hypertension	No	Yes	No	Yes	No
Total cholesterol (mg/dL)	344	201	233	240	<240
LDL cholesterol (mg/dL)	170	116	98	131	<160
HDL cholesterol (mg/dL)	109	60	100	65	>40
Smoking status	Former	Never	Former	Former	Never
10-year F…					…er the
CAC sco…					…AC
CAC per…					…AC
Myocardi…					
Resting h…					
Weekly MET	4241	4806	8296	5054	
Marathons completed (no.)	14	22	65	140	
Findings on invasive angiography	Three-VD	Two-VD	Myocardial bridge/ One-VD	Three-VD	
Type of event	VT during exercise, stent	Stent/ CABG	Stent	VT during exercise, CABG	

Do you really think it was the running that caused these men's coronary disease??

Note that details on subject 2 have previously been reported.[11]
LGE, late gadolinium enhancement; VD, vessel disease; b.p.m., beats per minute; VT, ventricular tachycardia; CABG, coronary artery bypass graft; HDL, high-density lipoprotein; LDL, low-density lipoprotein; CAC, coronary artery calcium; MET, metabolic equivalent; no., number.
[a]For asymptomatic males without known CAD.
[b]On 5 mg Ramipril (Sanofi Aventis, Frankfurt, Germany) once daily.

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CAD: Observational Data

Increased Coronary Artery Plaque Volume Among Male Marathon Runners

by Robert S. Schwartz, MD, Stacia Merkel Kraus, MPH, Jonathan G. Schwartz, MD, Kelly K. Wickstrom, BS, Gretchen Peichel, RN, Ross F. Garberich, MS, John R. Lesser, MD, Stephen N. Oesterle, MD, Thomas Knickelbine, MD, Kevin M. Harris, MD, Sue Duval, PhD, William O. Roberts, MD & James H. O'Keefe, MD

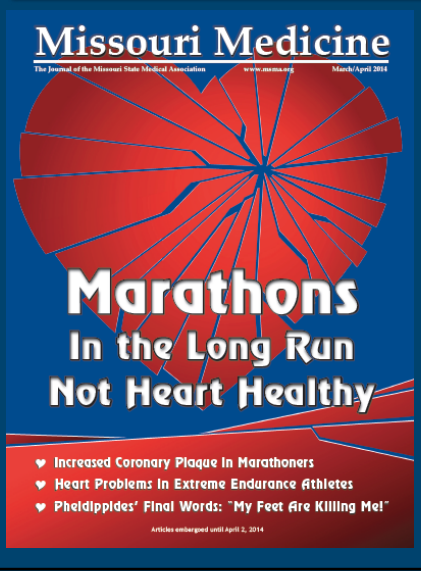

Table 1
Demographic Characteristics of Subjects

	Men		
	Sedentary	Marathon	
Age, …			
Systo…			
Diasto…			
Heart…			
Heigh…			
Weigh…			
BMI, …			
Hyper…			
Hyper…			
Diabe…			
Histor…			
Creati…			
Total …			
HDL, …			
LDL, …			
Triglycerides, mg/dl*	130.80 ± 63.00	83.36 ± 38.58	NS

Values presented are mean ± SD or n (%), p values from Fisher's Exact Test/T-test/Wilcoxon test for non-normal data
*Indicates failure of the normality assumptions based on Shapiro-Wilk test
†BMI-body mass index, ‡HDL-high-density lipoprotein, §LDL-low-density lipoprotein, ||CAD-coronary artery disease

Missing Risk Issues:

1.) Diet (Fit vs. Healthy)

2.) "Care-free" 20's-30's

3.) Family Genes

Without controlling for these, can we say anything about causality?

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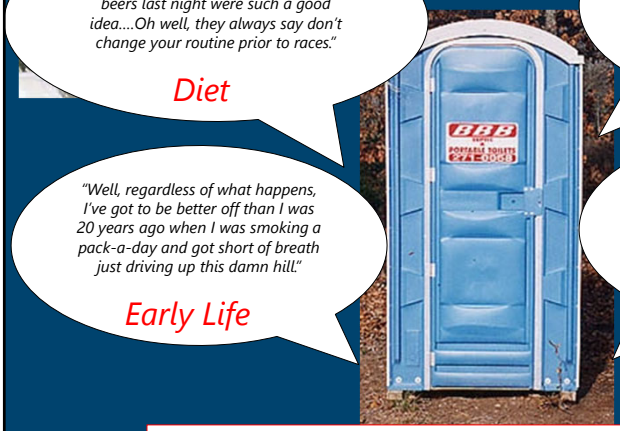
CAD in Athletes: Causality?

Cardiovascular Performance Program



"Man, not so sure that second plate of chili nachos and the extra couple of beers last night were such a good idea....Oh well, they always say don't change your routine prior to races."

Diet

"Just wish my old man could see me now. He died at age 45 from a massive heart attack. Glad I got my wake up call. Bad stuff runs in my family"

Genes

"Well, regardless of what happens, I've got to be better off than I was 20 years ago when I was smoking a pack-a-day and got short of breath just driving up this damn hill."

Early Life

"Cough, cough, cough....If this race wasn't only once a year I'd be home in bed. I feel like shit. Oh well, this little climb will help me clear out the junk."

Viral Influence

Is running really the main causal issue??

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CAD: Observational Data

Cardiovascular Performance Program

ORIGINAL RESEARCH ARTICLE

Prevalence of Subclinical Coronary Artery Disease in Masters Endurance Athletes With a Low Atherosclerotic Risk Profile

42(A) vs. 22(C) %

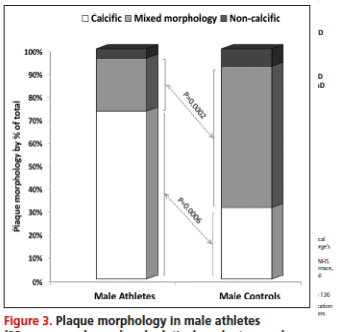
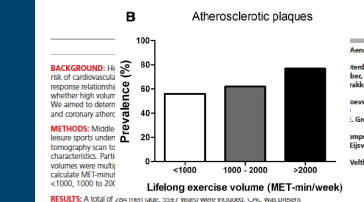
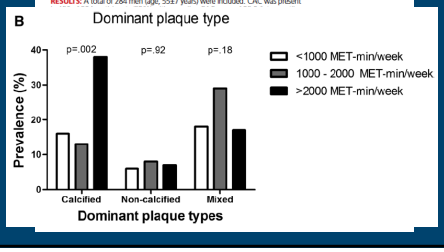


Figure 3. Plaque morphology in male athletes (99 coronary plaques) and relatively sedentary males (26 coronary plaques).

ORIGINAL RESEARCH ARTICLE

Relationship Between Lifelong Exercise Volume and Coronary Atherosclerosis in Athletes





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Elevated CAC Score

EDITORIAL

Coronary Artery Calcification Among Endurance Athletes
"Hearts of Stone"

Articles, see p 126 and p 138

Aaron L. Baggish, MD
Benjamin D. Levine, MD

...cause you'll never break, never break, never break, this heart of stone.
—Mick Jagger and Keith Richards, The Rolling Stones, Heart of Stone, 1964

Routine moderate-intensity exercise reduces incident cardiovascular disease and increases longevity. The complex mechanisms by which exercise promotes favorable cardiovascular health outcomes include attenuation of traditional atherosclerotic risk factors including dyslipidemia, hypertension, central adiposity, and glucose intolerance. As such, current physical activity guidelines recommend either 150 minutes of moderate-intensity exercise or 75 minutes of higher-intensity exercise weekly.[1] This recommendation is justified by a broad epidemiological and exercise intervention literature base and represents the current standard of cardiovascular care for all patients.[2]

However, the dose-response relationship between exercise and health outcomes, in particular, at exercise doses that exceed current recommendations, remains incompletely understood. For example, highly fit individuals have reduced risk of sudden cardiac death[3] and development of heart failure,[4] with little evidence of a plateau at high levels of fitness.[5] Competitive athletes, people who in engage in very high levels of exercise over many years and in some cases a lifetime, typically live longer than sedentary and normally active people,[6] and develop beneficial adaptive cardiovascular traits including physiological myocardial remodeling[7,8] and increased myocardial[9,10] and vascular compliance. Conversely, athletes and highly active people are not immune to cardiovascular disease. Sudden cardiac death certainly occurs among this population, and atherosclerotic coronary disease is the most common etiology of death during sport among those >35 years of age.[11,12] Recent data have unexpectedly raised the possibility that long-term, high-volume endurance exercise may actually accelerate rather than reduce coronary atherosclerosis.[13] This controversial hypothesis has been passionately debated and remains a key issue of uncertainty in the clinical care of athletic patients.

In this issue of *Circulation*, 2 studies present data examining coronary artery morphology among middle-aged endurance athletes.[14,15] Merghani et al[15] recruited 152 competitive cyclists and runners (age=55±9 years) and a normally active, age-matched control cohort without a previous diagnosis of coronary artery disease, a family history of premature coronary artery disease (<40 years), diabetes mellitus, hypertension, hypercholesterolemia, and active or prior tobacco smoking. Coronary computerized tomography angiography revealed that the majority of athletes (60%) and control participants (63%) had no demonstrable coronary artery calcification (CAC). However, male athletes had a higher prevalence of atherosclerotic plaque with luminal irregularity (44% versus 22% in controls) with plaques composed predominantly of calcium. Among athletes, a CAC score

The opinions expressed in this article are not necessarily those of the editors or of the American Heart Association.

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Key Words: Editorials ■ athletes ■ cardiomegaly, exercise-induced ■ coronary angiography ■ coronary artery calcium

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Circulation. 2017;136:149–151. DOI: 10.1161/CIRCULATIONAHA.117.028750 July 11, 2017 149

Sedentary CAC ≠ Athletic CAC

Mechanistic Evolution of CAC in Athletes?

↑ Fitness

↑ Longevity + ↑ CAC

Prognostic Significance of CAC in Athletes?

Traditional CRF vs. ↑ CAC

Therapeutic Implications of CAC in Athletes?

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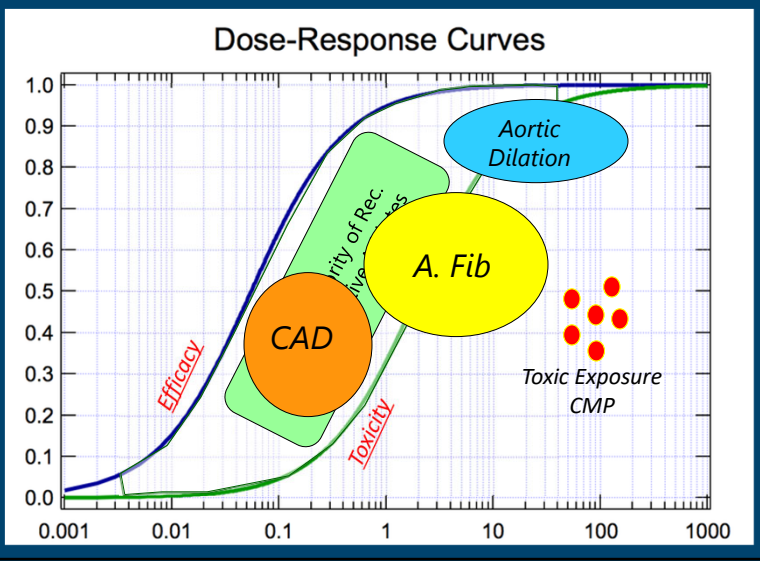
My Synthesis of the Controversy



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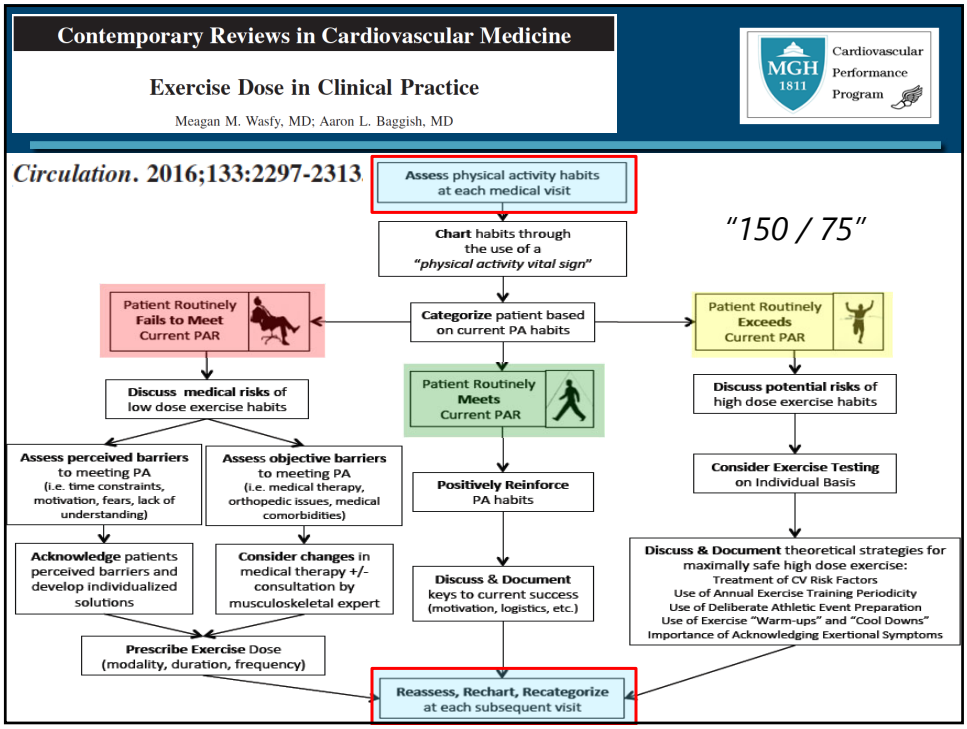
Contemporary Reviews in Cardiovascular Medicine
Exercise Dose in Clinical Practice
Meagan M. Wasfy, MD; Aaron L. Baggish, MD
MGH 1811
Cardiovascular Performance Program
Circulation. 2016;133:2297-2313
Assess physical activity habits at each medical visit
Chart habits through the use of a "physical activity vital sign"
"150 / 75"
Categorize patient based on current PA habits
Patient Routinely Fails to Meet Current PAR
Patient Routinely Meets Current PAR
Patient Routinely Exceeds Current PAR
Discuss medical risks of low dose exercise habits
Discuss potential risks of high dose exercise habits
Assess perceived barriers to meeting PA (i.e. time constraints, motivation, fears, lack of understanding)
Assess objective barriers to meeting PA (i.e. medical therapy, orthopedic issues, medical comorbidities)
Positively Reinforce PA habits
Consider Exercise Testing on Individual Basis
Acknowledge patients perceived barriers and develop individualized solutions
Consider changes in medical therapy +/- consultation by musculoskeletal expert
Discuss & Document keys to current success (motivation, logistics, etc.)
Discuss & Document theoretical strategies for maximally safe high dose exercise:
Treatment of CV Risk Factors
Use of Annual Exercise Training Periodicity
Use of Deliberate Athletic Event Preparation
Use of Exercise "Warm-ups" and "Cool Downs"
Importance of Acknowledging Exertional Symptoms
Prescribe Exercise Dose (modality, duration, frequency)
Reassess, Rechart, Recategorize at each subsequent visit

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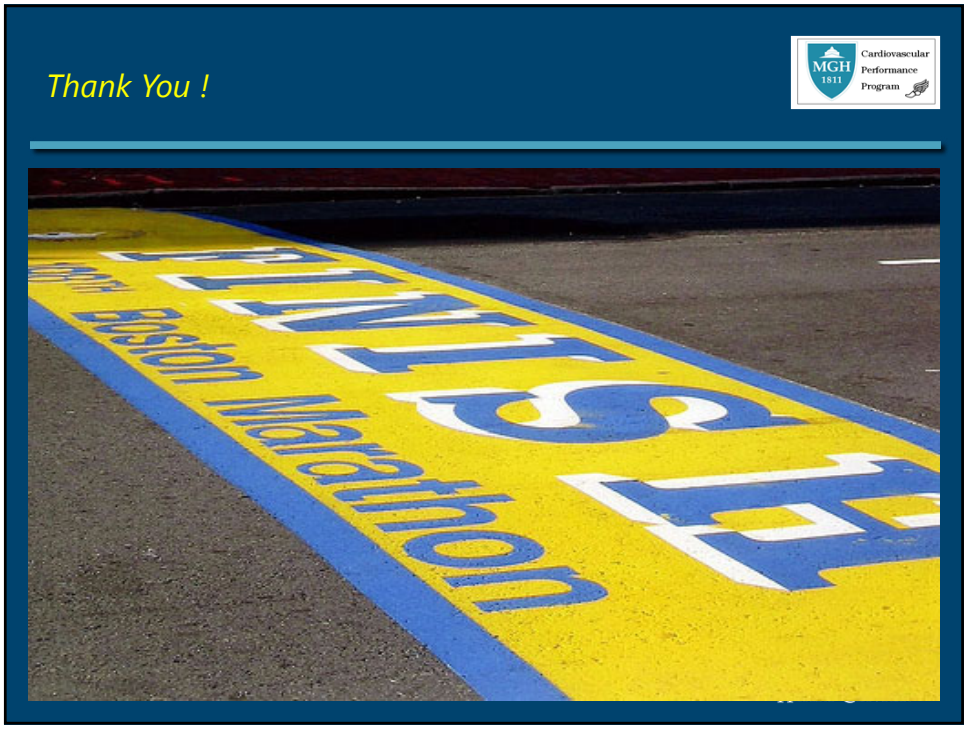
Thank You !
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