



**ARISTOTLE UNIVERSITY OF THESSALONIKI,
GREECE**
SPORTS MEDICINE LABORATORY
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Sports and heart: Is there any “red line?

ASTERIOS DELIGIANNIS

CARDIOLOGIST

PROFESSOR OF SPORTS MEDICINE

No Conflict of Interest





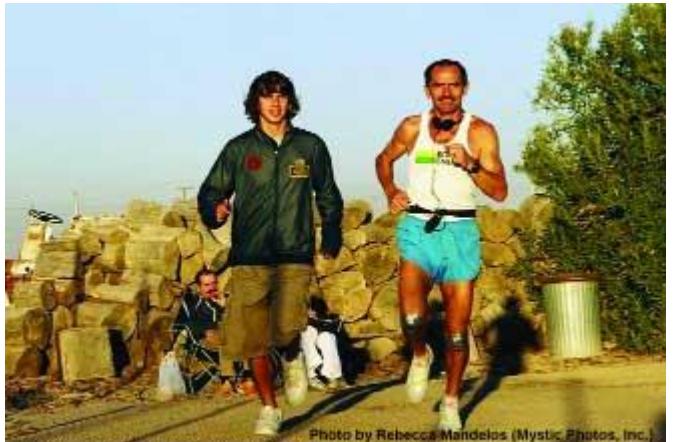


Photo by Rebecca Mandelos (Mystic Photos, Inc.)
Running in the Across the Years 72-hour race in Phoenix, AZ, Kouros spends some time alongside the race director's son, James Bonnett, inspiring a new generation.



Epidemiology

- **College and Professional Athletes**
 - **500,000 participants each year**
- **Competitive Athletics:**
 - **“Several million high school students participate in competitive athletics each year in the United States”.**
- **‘Other’ Organized Sports Participation**
 - **25 million children and young adults**



Med Sci Sports Exerc. 1993 Feb;25(2):237-44.

Increased life expectancy of world class male athletes.

Sarna S¹, Sahi T, Koskenvuo M, Kaprio J.

The increased mean life expectancies were mainly explained by decreased cardiovascular mortality

"In our opinion, physicians, health professionals and general population should not hold the impression that strenuous exercise and/or high-level aerobic competitive sports have deleterious effects, are bad for one's health, and shorten life."

Review Article

Does Physical Activity Increase Life Expectancy? A Review of the Literature

C. D. Reimers,¹ G. Knapp,² and A. K. Reimers³

¹*Klinik für Neurologie, Zentralklinik Bad Berka, Robert-Koch-Allee 9, 99438 Bad Berka, Germany*

²*Fakultät Statistik, Technische Universität Dortmund, 44221 Dortmund, Germany*

³*Sportwissenschaft, Universität Konstanz, Universitätsstraße 10, 78457 Konstanz, Germany*

In conclusion, while regular physical activity increases life expectancy, it remains unclear if high-intensity sports activities further increase life expectancy



Journal of the American College of Cardiology

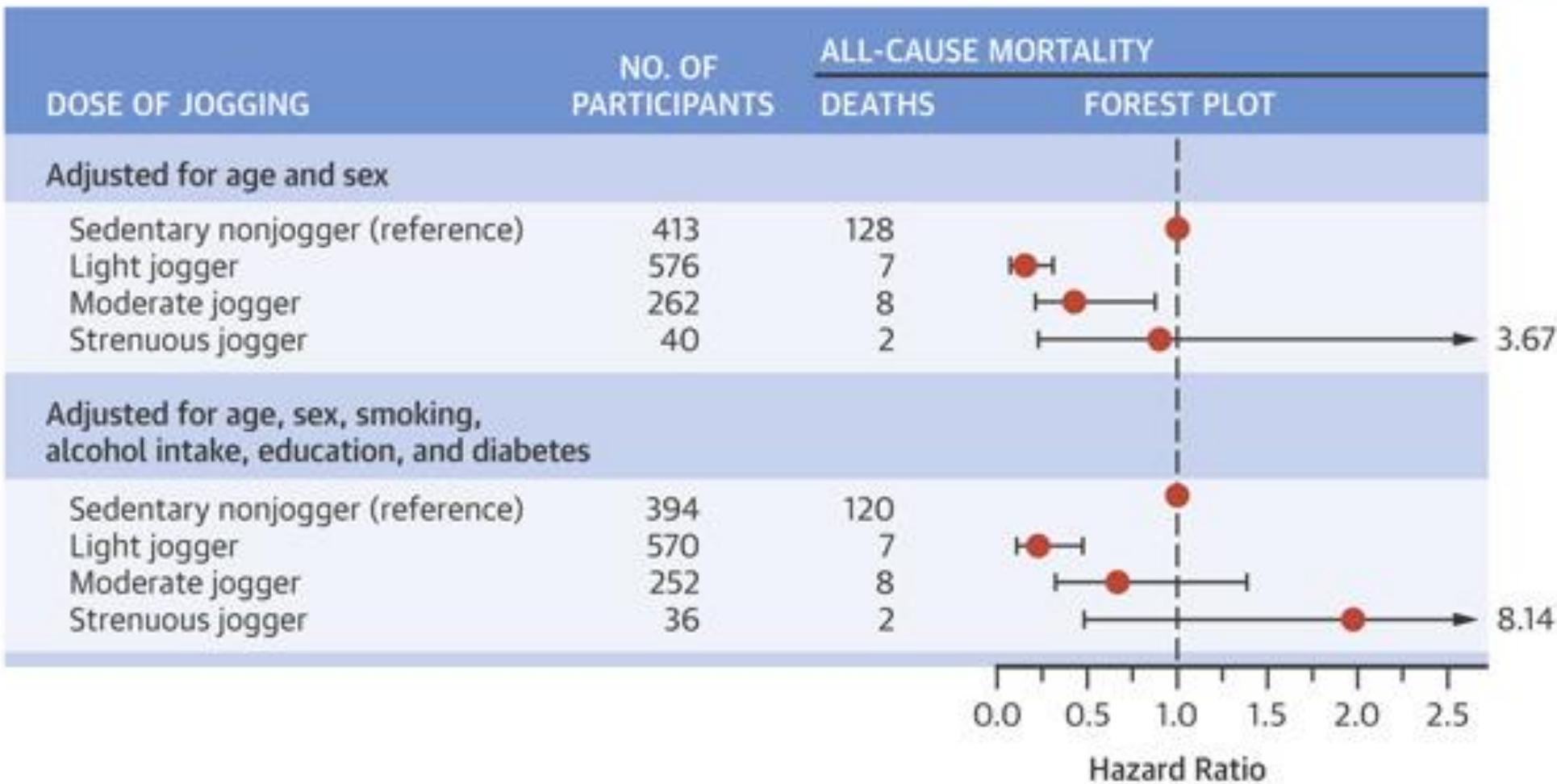
Volume 65, Issue 5, 10 February 2015, Pages 411-419



Original Investigation

Dose of Jogging and Long-Term Mortality: The Copenhagen City Heart Study

Peter Schnohr MD, DMSc *  , James H. O'Keefe MD †, Jacob L. Marott MSc *, Peter Lange MD, DMSc *, ‡, Gorm B. Jensen MD, DMSc *, §



Schnohr, P. et al. J Am Coll Cardiol. 2015; 65(5):411-9.



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November 2017 Volume 92, Issue 11, Pages 1660–1670

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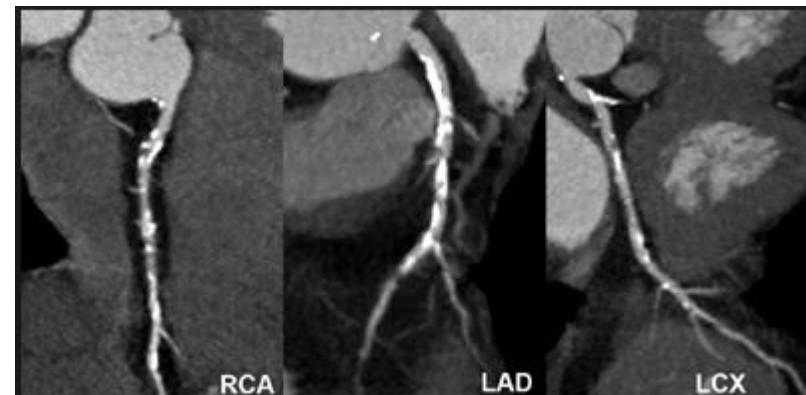
To read this article in full, please review your options for gaining access at the bottom of the page.

25-Year Physical Activity Trajectories and Development of Subclinical Coronary Artery Disease as Measured by Coronary Artery Calcium: The Coronary Artery Risk Development in Young Adults (CARDIA) Study

Deepika R. Laddu, PhD, Jamal S. Rana, MD, PhD , Rosenda Murillo, PhD, Michael E. Sorel, MS, Charles P. Quesenberry Jr., PhD, Norrina B. Allen, PhD, Kelley P. Gabriel, PhD, Mercedes R. Carnethon, PhD, Kiang Liu, PhD, Jared P. Reis, PhD, Donald Lloyd-Jones, MD, ScM, J. Jeffrey Carr, MD, Stephen Sidney, MD, MPH

Conclusion

White individuals who participated in 3 times the recommended physical activity guidelines over 25 years had higher odds of developing coronary subclinical atherosclerosis by middle age. These findings warrant further exploration, especially by race, into possible biological mechanisms for coronary artery calcification risk at very high levels of PA



Coronary and carotid atherosclerosis in asymptomatic male marathon runners

C. Burgstahler¹ | H. Cipowicz¹ | C. Thomas² | C. Schabel² | S. Mangold² |
D. Ketelsen² | C. D. Claussen² | A. M. Niess¹ | I. Tsiflikas² 

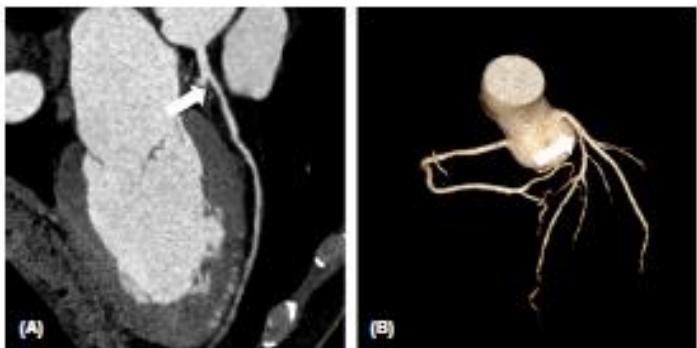


FIGURE 1 Long-distance non-calcified plaque (arrowhead) in segment 6 of LAD (left anterior descending) in a 51-year-old marathon runner without coronary calcification (A, curved MPR (multiplanar reconstruction); B, VRT (volume rendering technique)).



FIGURE 2 Cross-sectional ultrasound image (A) of a common carotid artery plaque in a 55-year-old marathon runner without evidence of coronary atherosclerosis (B, curved MPR (multiplanar reconstruction)) of LAD (left anterior descending), CX (circumflex branch of left coronary artery), and RCA (right coronary artery).



Clinical Update

Exercise and the heart: the good, the bad, and the ugly

Sanjay Sharma^{1*}, Ahmed Merghani¹, and Lluis Mont²

¹Department of Cardiovascular Sciences, St Georges, University of London, Cranmer Terrace, London SW17 0RE, UK; and ²Institut del Torax, Hospital Clinic, Universitat de Barcelona, Barcelona, Spain

Received 11 January 2015; revised 19 February 2015; accepted 4 March 2015

“In parallel with the extraordinary athletic milieu of physical performances previously considered unachievable, there is emerging data indicating that long-standing vigorous exercise may be associated with adverse electrical and structural remodelling in otherwise normal hearts.”

BENEFITS OF EXERCISE

NEUROLOGICAL

Anxiety/depression ↓
Dementia ↓
Cognitive function ↑
Risk of Stroke ↓

ENDOCRINE

Weight ↓
Diabetes ↓
LDL ↓
HDL ↑



CARDIOVASCULAR

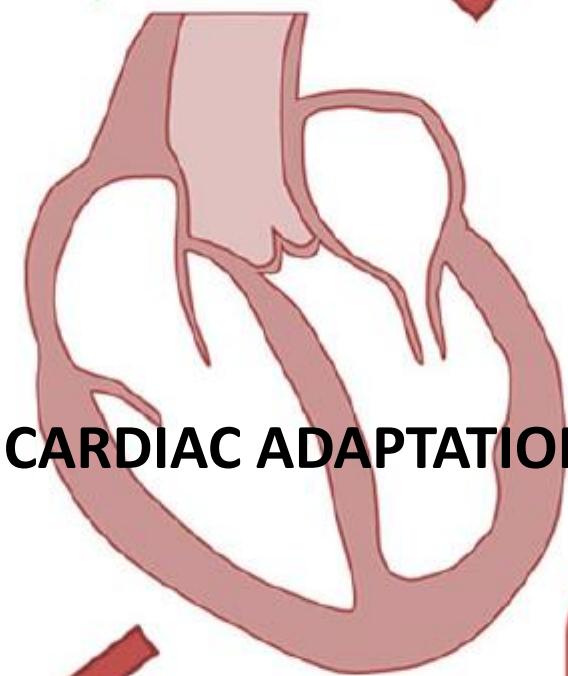
Mortality ↓
CAD ↓
Hypertension ↓
Cardiac Rehab

ONCOLOGICAL

Breast cancer ↓
Prostate cancer ↓
Bowel cancer ↓

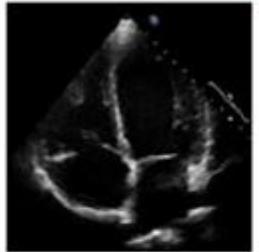
MUSCULOSKELETAL

Osteoporosis ↓
Falls ↓
Disability ↓



CARDIAC ADAPTATIONS

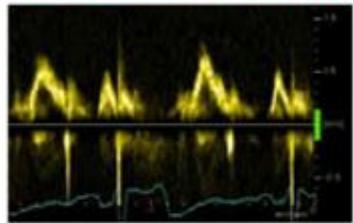
S. Sharma et al, 2015



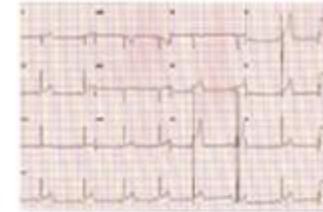
- ↑ LVWT 10-25%
- ↑ LV and RV cavity 15%
- Bi-atrial dilatation

Structural changes

Functional changes



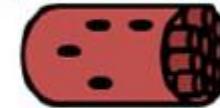
- Functional changes
- ↑ diastolic filling
- $E' > 9 \text{ cm/s}$
- $E/E' < 6$
- $S' > 9$
- ↑ Stroke volume



- Sinus bradycardia
- Sinus arrhythmia
- First degree AV block
- Voltage L VH, and RVH
- Incomplete RBBB
- TWI in V1-V4 in black athletes

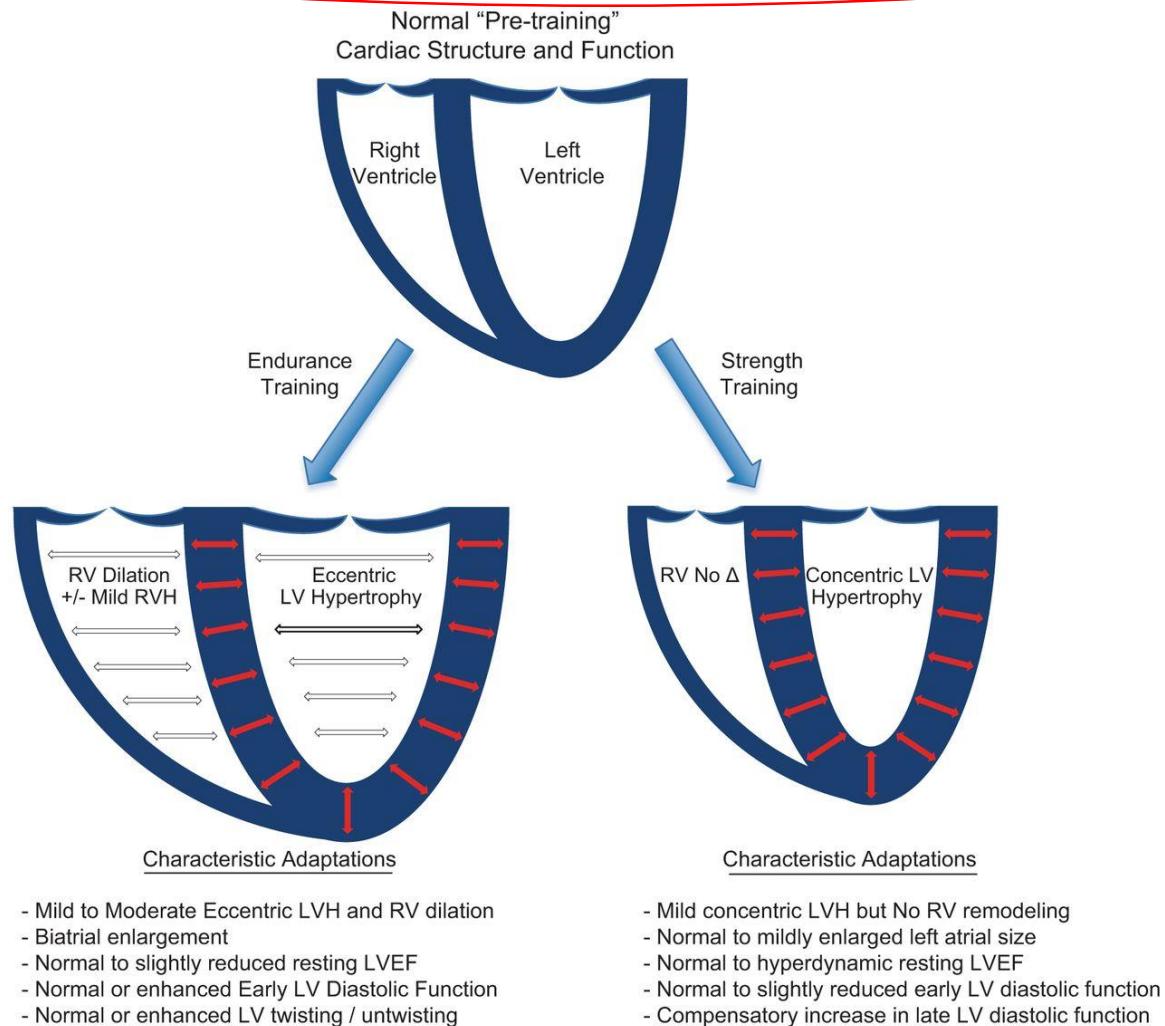
Electrical changes

Peripheral changes



- ↑ skeletal muscle fibres
- ↑ capillary conductance
- ↑ oxidative capacity
- ↑ mitochondrial enzymes
- ↑ O_2 Peak consumption

Summary of exercise-induced remodeling of the left and right ventricle.



Thijs M. H. Eijsvogels et al. Physiol Rev 2016;96:99-125

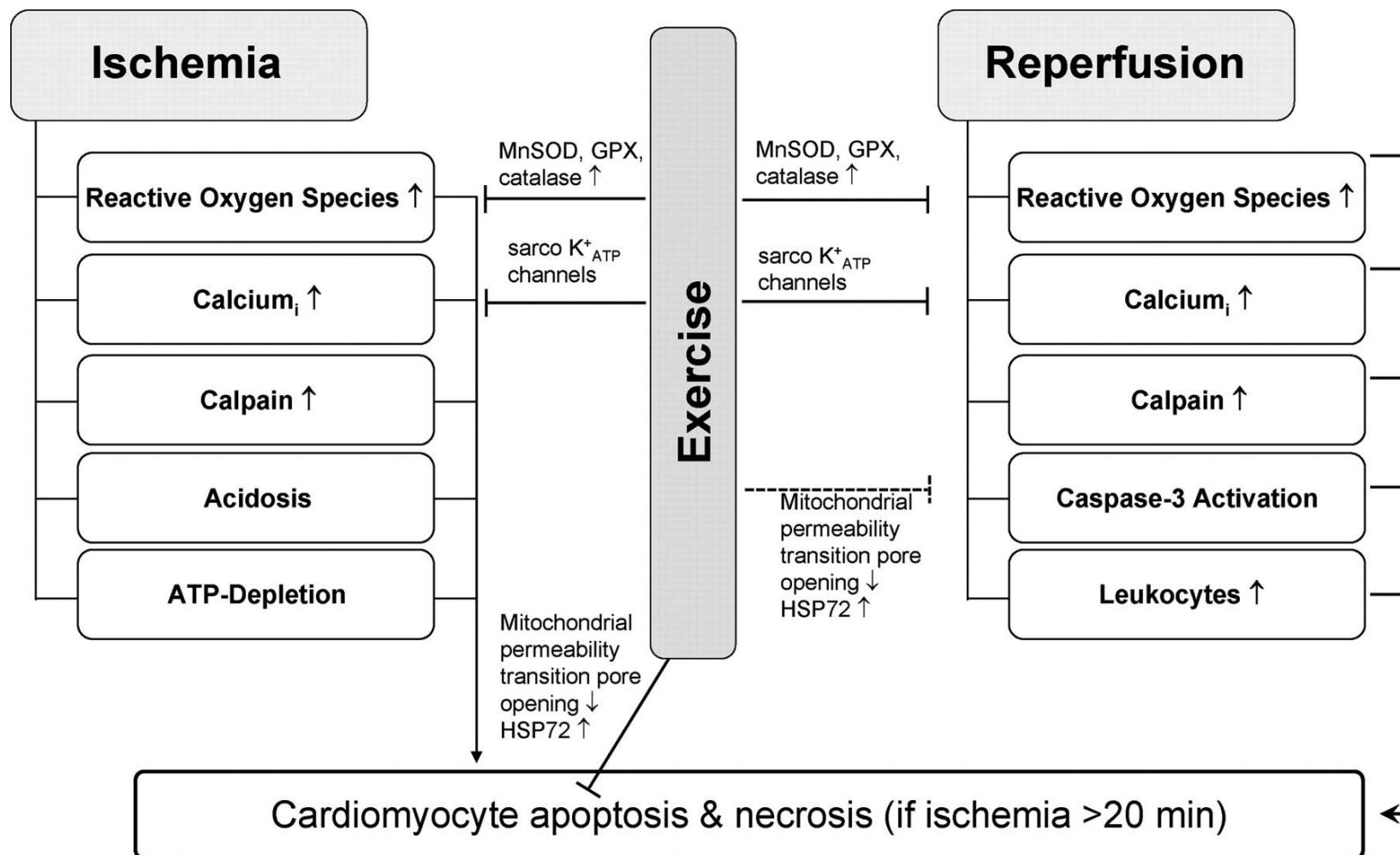
Physiological Reviews

CARDIOVASCULAR BENEFITS OF REGULAR TRAINING

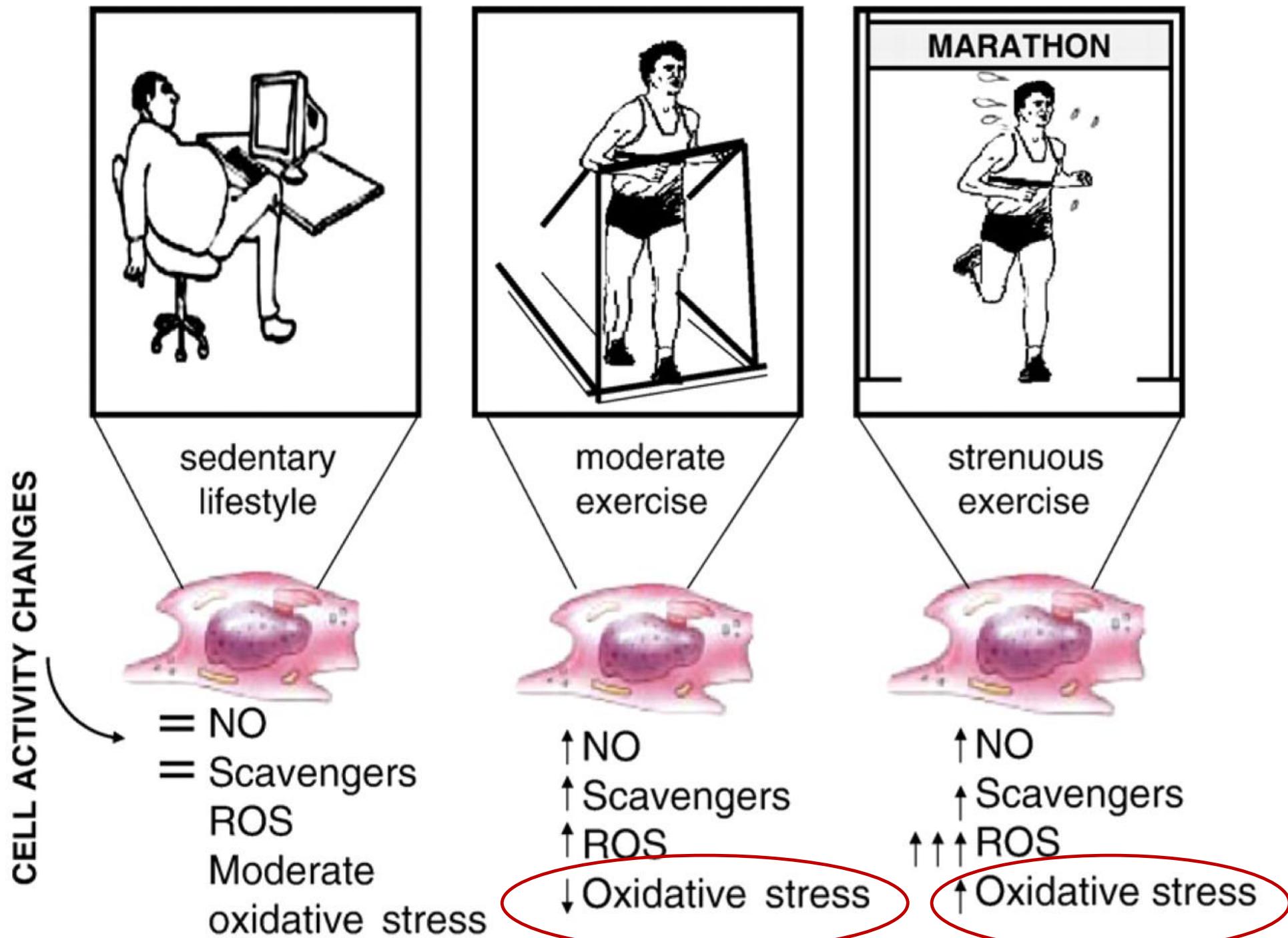
- Endothelial function ↑
- Coronary flow reserve ↑
- Tolerance of myocardial ischemia ↑
- Myocardial capillary density ↑
- Ventricular fibrillation thresholds ↑
- Arterial blood pressure ↓
- Arterial stiffness ↓



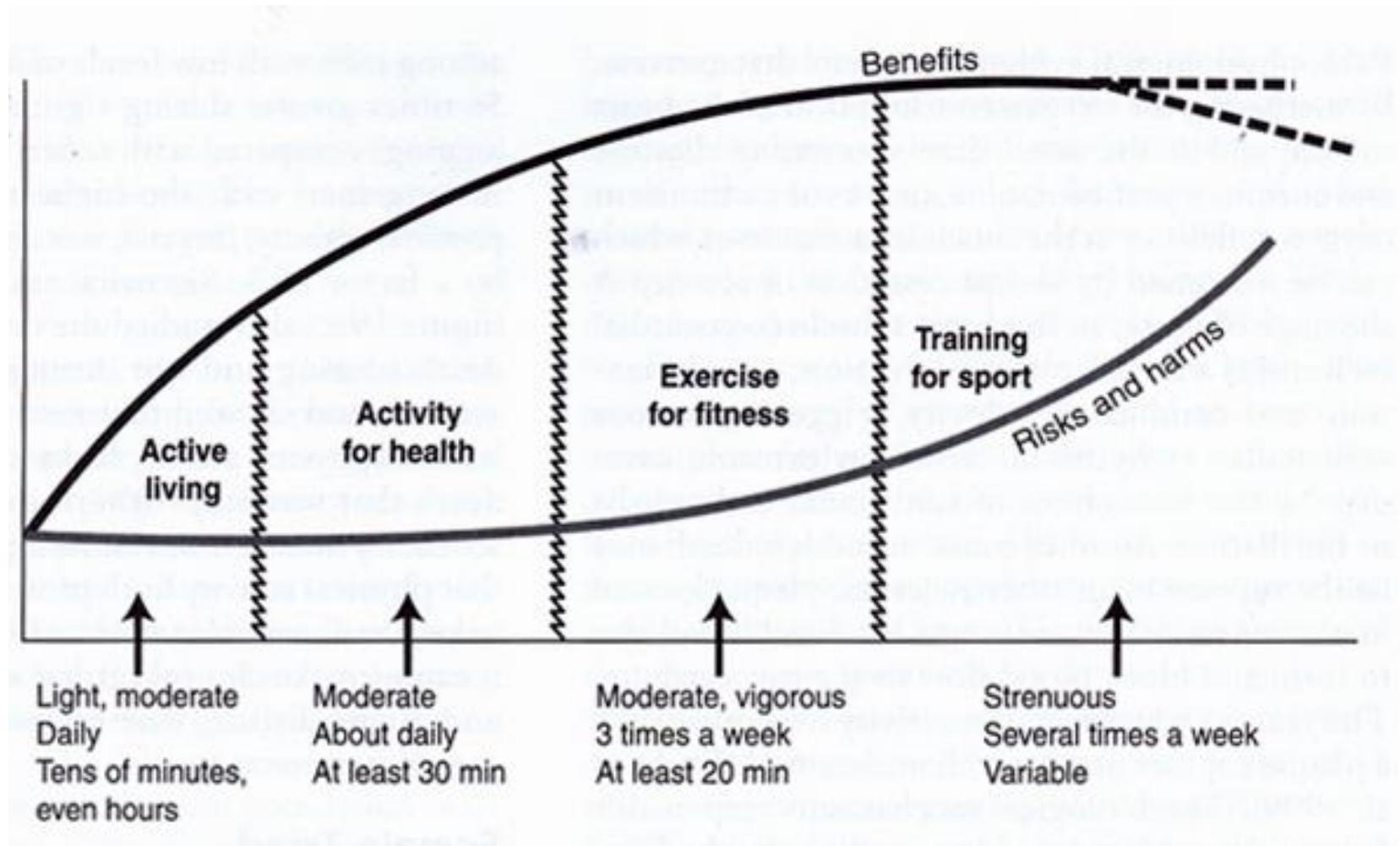
Exercise training has the potential to prevent myocardial damage related to I/R injury.



Stephan Gielen et al. Circulation. 2010;122:1221-1238



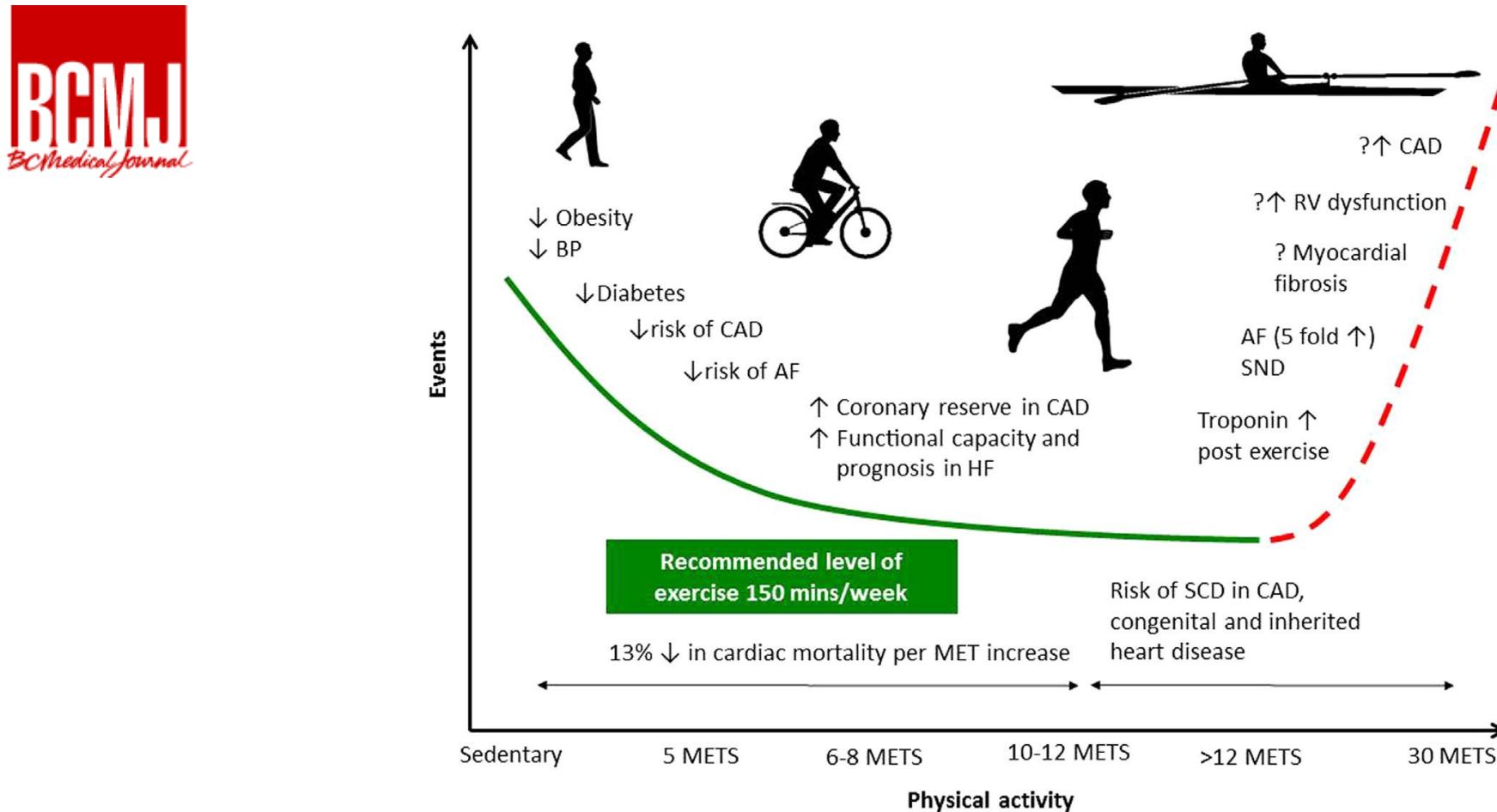
Exercise : From Benefits to Risks



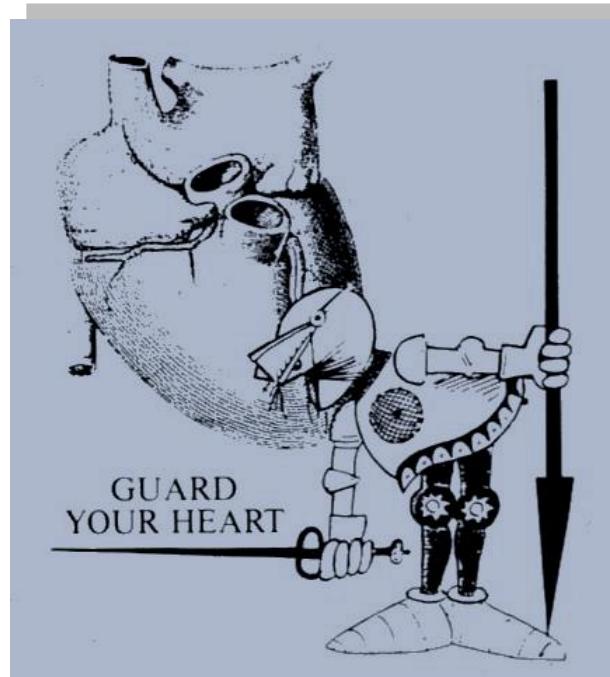
The impact of excessive endurance exercise on the heart

BCMJ, Vol. 58, No. 4, May 2016, page(s) 203-209 Articles

Andrea K.Y. Lee, MD, Barbara N. Morrison, BHK, Saul Isserow, MBBCh, Brett Heilbron, MB ChB, Andrew D. Krahn, MD



“PARADOX” OF EXERCISE



Systematic physical activity is associated with heart health, while intense exercise can lead to sudden death

Barry J. Maron, M.D., 2000

REASONS OF RISKS OF INTENSE EXERCISE

**ACUTE CARDIOVASCULAR RESPONSES AND
CHRONIC ADAPTATIONS**
RISES OF BODY TEMPERATURE
DECREASE OF IMMUNITY
INCREASE OF OXIDATIVE STRESS
EFFECTS OF ACCLIMATIZATION
ENDOCRINE AND METABOLIC DISORDERS
ELECTROLYTIC ABNORMALITIES
MUSCULOSKELETAL INJURIES
OTHERS

The most serious health risks in athletes: Sudden Death !



In partnership with:

Missouri Medicine
The Journal of the Missouri State Medical Association
www.msmma.org



Pheidippides' Last Message: "My Feet Are Killing Me!"

By John C. Hagan III, MD (Former Marathon Runner)



SUDDEN CARDIAC DEATH

Incidence of Sudden Cardiac Death: Organized High School/College Athletes

1:134,000/Year (Male)

(7.47:million/Year)

1:750,000/Year (Female)

(1.33/million/Year)

Air Force Recruits

1:735,000/Year

Marathon Runners

1:50,000 Race Finishers (Mean Age

37yo)

In brief, ~ 300 deaths/year.

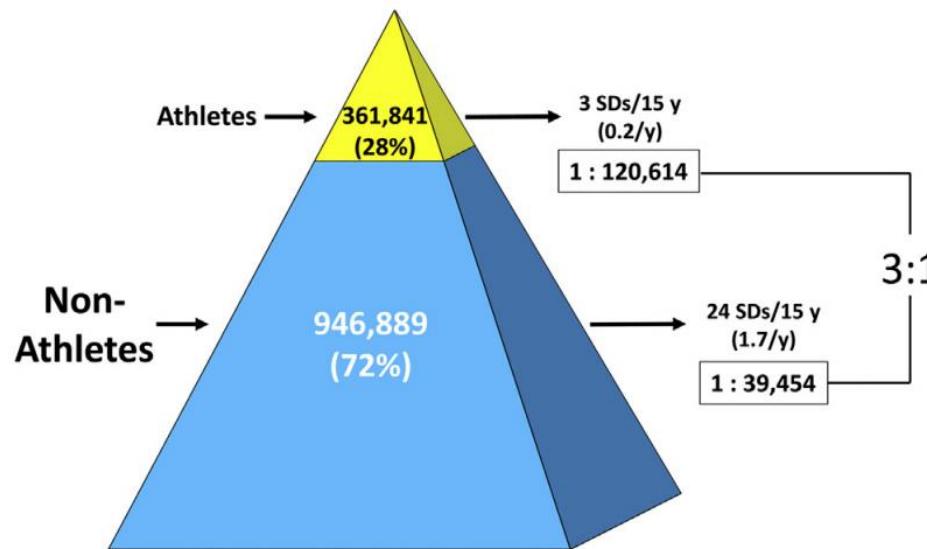
**But the media attention and legal
implications, make these events
standout.**

Comparison of the Frequency of Sudden Cardiovascular Deaths in Young Competitive Athletes Versus Nonathletes: Should We Really Screen Only Athletes?



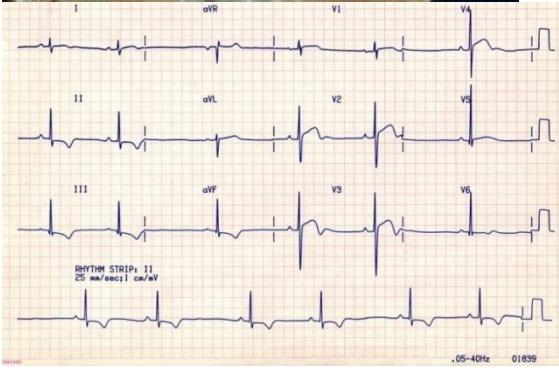
Barry J. Maron, MD^{a,*}, Tammy S. Haas, RN^a, Emily R. Duncanson, MD^b, Ross F. Garberich, MS^a, Andrew M. Baker, MD^c, and Shannon Mackey-Bojack, MD^b

Incidence of SCD in high school and college students

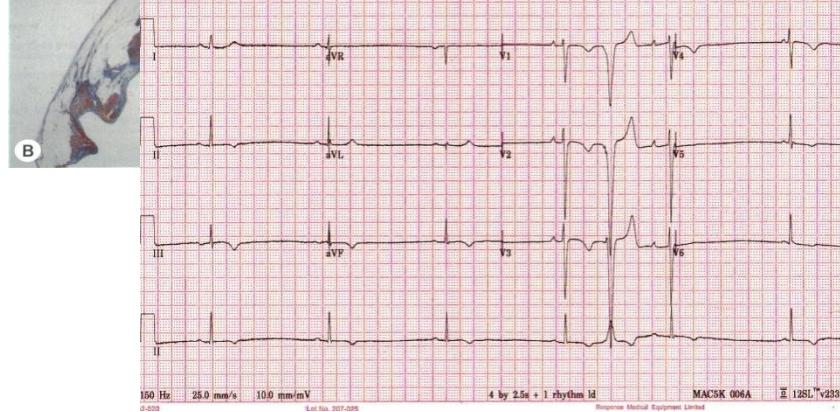


Maron B et al. Am J Cardiol 2016;117:1339-1341

Sudden Cardiac Death in Sport



Hypertrophic
Cardiomyopathy



Arrhythmogenic right
ventricular cardiomyopathy

Incidence, Cause, and Comparative Frequency of Sudden Cardiac Death in National Collegiate Athletic Association Athletes

A Decade in Review

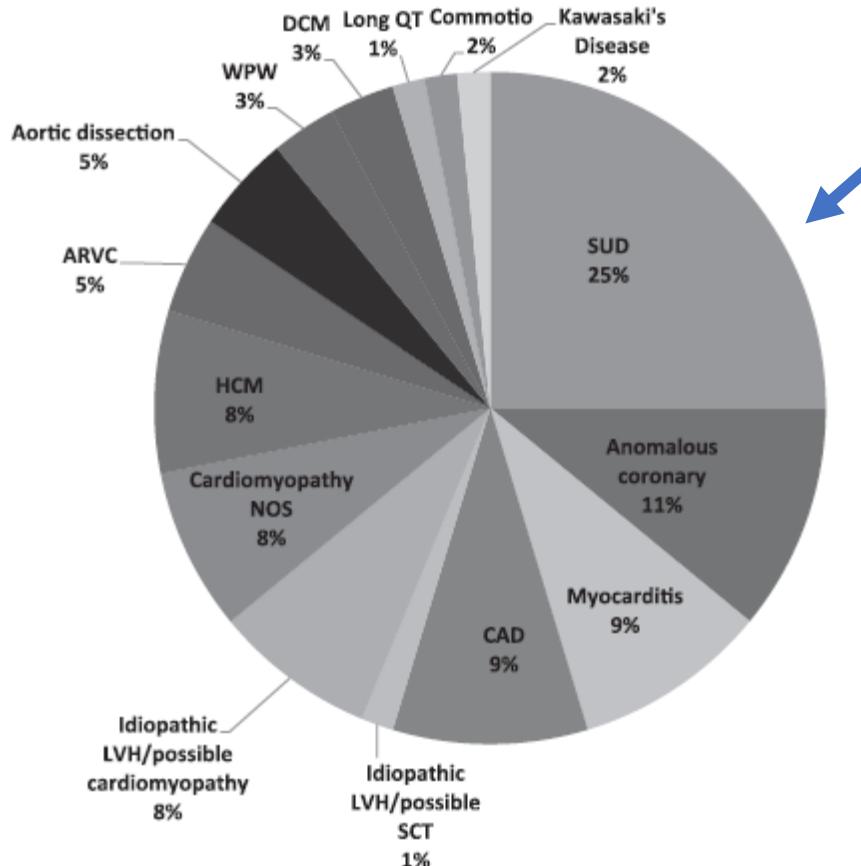
Circulation 2015 Jul 7;132(1):10-9

Kimberly G. Harmon, MD; Irfan M. Asif, MD; Joseph J. Maleszewski, MD;
David S. Owens, MD, MS; Jordan M. Prutkin, MD, MHS; Jack C. Salerno, MD;
Monica L. Zigman, MPH; Rachel Ellenbogen, MS; Ashwin L. Rao, MD;
Michael J. Ackerman, MD, PhD; Jonathan A. Drezner, MD

2003-2013

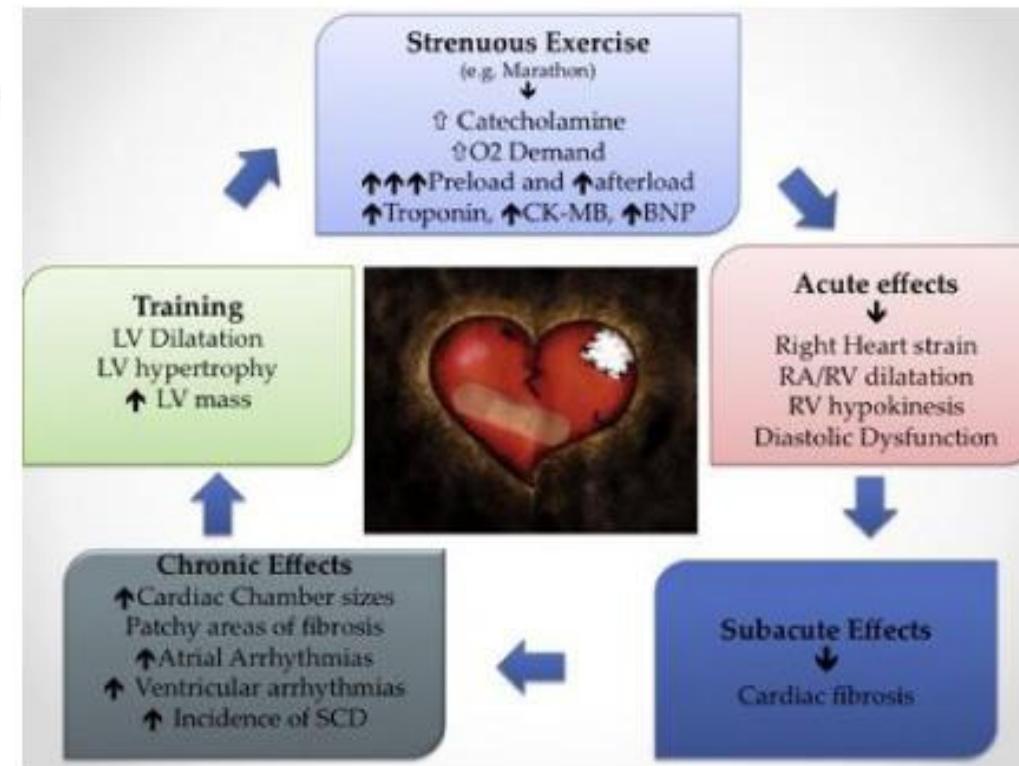
514 deaths

79 sudden cardiac deaths (15%)



25% had a structurally normal heart

Strenuous Exercise: A Harmful Spiral for Cardiac Health ?



RA=Right atrium, RV= Right ventricle, LV= Left ventricle, SCD= Sudden Cardiac Death

The Shift from Pathology to Physiology

HISTORICAL ASPECTS

- **J.E. MORGAN** (**University Oars.** London: Macmillan and Co; 1873)
- He concerned about the possible deleterious effects of the University Boat Race and concluded that there was no evidence for shortened life-expectancy, for tendency to heart-disease or for increased risk of sudden death.
- **R.J. LEE** (**Exercise and Training: Their Effects upon Health.** London: Smith Elder and Co; 1873)
- He suggested that professional sportsmen (specifically pedestrians) sometimes ‘at an early age paid the dearest penalty for their want of prudence’, that is exercising to an extreme and damaging their hearts.

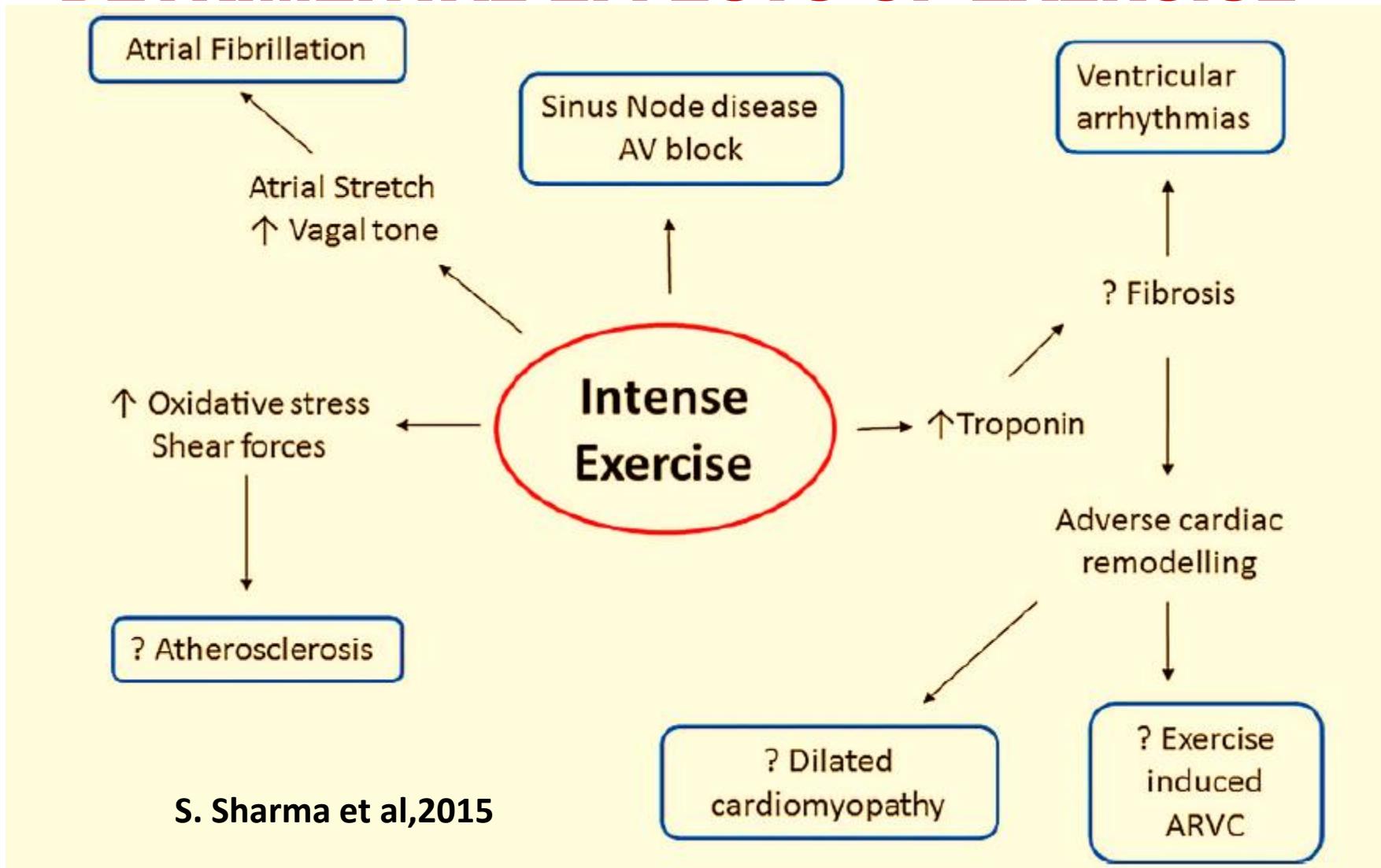
POTENTIAL ADVERSE CARDIOVASCULAR EFFECTS FROM EXCESSIVE ENDURANCE EXERCISE

- 1.The long-term excessive endurance exercise may induce pathologic structural remodeling of the heart and large arteries.**
- 2.Chronic training for and competing in extreme endurance events such as marathons, ultramarathons, ironman distance triathlons, and very long distance bicycle races, can cause transient acute volume overload of the atria and right ventricle, with transient reductions in right ventricular ejection fraction and elevations of cardiac biomarkers, all of which return to normal within 1 week.**

POTENTIAL ADVERSE CARDIOVASCULAR EFFECTS FROM EXCESSIVE ENDURANCE EXERCISE

- 3. Over months to years of repetitive injury, this process, in some individuals, may lead to patchy myocardial fibrosis, particularly in the atria, interventricular septum, and right ventricle, creating a substrate for atrial and ventricular arrhythmias.**
- 4. Additionally, long-term excessive sustained exercise may be associated with coronary artery calcification, diastolic dysfunction, and large-artery wall stiffening.**

DETERRIMENTAL EFFECTS OF EXERCISE



Editorial

Marathon Rat

Myocardial Remodeling in an Animal
Model of Vigorous Endurance Exercise and
Implications for Humans

Paul G.A. Volders

Generally, the risk of primary cardiac arrest is increased among men with infrequent vigorous exercise, whereas, for those exercising habitually, the overall risk is decreased.

“CARDIAC FATIGUE” AFTER PROLONGED STRENUOUS EXERCISE



[J Am Coll Cardiol. 2004 Jul 7;44\(1\):144-9.](#)

Serial left ventricular adaptations in world-class professional cyclists: implications for disease screening and follow-up.

[Abergel E¹, Chatellier G, Hagege AA, Oblak A, Linhart A, Ducardonnet A, Menard J.](#)

CONCLUSIONS: Over one-half of these athletes exhibited unusual LV dilation, along with a reduced LVEF in 11.6% (17 of 147), compatible with the diagnosis of DCM. Increased WT was less common (always <15 mm) and scarce without LV dilation (<1%), eliminating the diagnosis of HCM. Serial examinations showed evidence of further LV dilation along with wall thinning. These results might have important implications for screening in athletes.

Alexiou S, Kouidi E, Fahadidou-Tsilingiroglou A, Karamouzis M, Deligiannis A.

Cardiac function after exhaustive open-sea swimming.

J Sports Med Phys Fitness.

2005;45:98-104.

- **Exhaustive swimming was associated with depressed left ventricular function as suggested by reduced stroke volume, ejection fraction, and left ventricular fractional shortening. In contrast, cardiac index was increased by 31% and total peripheral resistance was increased by 7%.**



Post-exercise left ventricular dysfunction measured after a long-duration cycling event

[Enrique Serrano Ostariz](#) [Marta López Ramón](#), [Daniel Cremades Arroyos](#), [Silvia Izquierdo Álvarez](#),
[BMC Res Notes](#). 2013; 6: 211.

- **Left ventricular systolic and diastolic function was reduced and cardiac biomarkers were increased after the cycling event, but the mechanisms behind such outcomes remain unclear.**

Pre and post-race data (mean \pm standard deviation)

	Pre-race	Post-race	P
LVIDd (mm)	51.2 \pm 4.0	47.7 \pm 4.6	0.000
LVIDs (mm)	29.3 \pm 4.1	30.4 \pm 4.3	0.018
LVPWd (mm)	9.6 \pm 1.1	9.6 \pm 0.9	0.80
IVSDd (mm)	9.6 \pm 1.1	9.5 \pm 1.0	0.58
LVEF (%)	67.7 \pm 8.2	59.7 \pm 9.4	0.000
E/A	1.6 \pm 0.4	1.1 \pm 0.3	0.000
cTnI ($\mu\text{g L}^{-1}$)	0.006 \pm 0.015	0.056 \pm 0.059	0.000
NT-proBNP (ng L^{-1})	27 \pm 16	189 \pm 111	0.000
HR (per min)	58 \pm 8	86 \pm 11	0.000
BPS (mmHg)	125 \pm 12	110 \pm 12	0.000

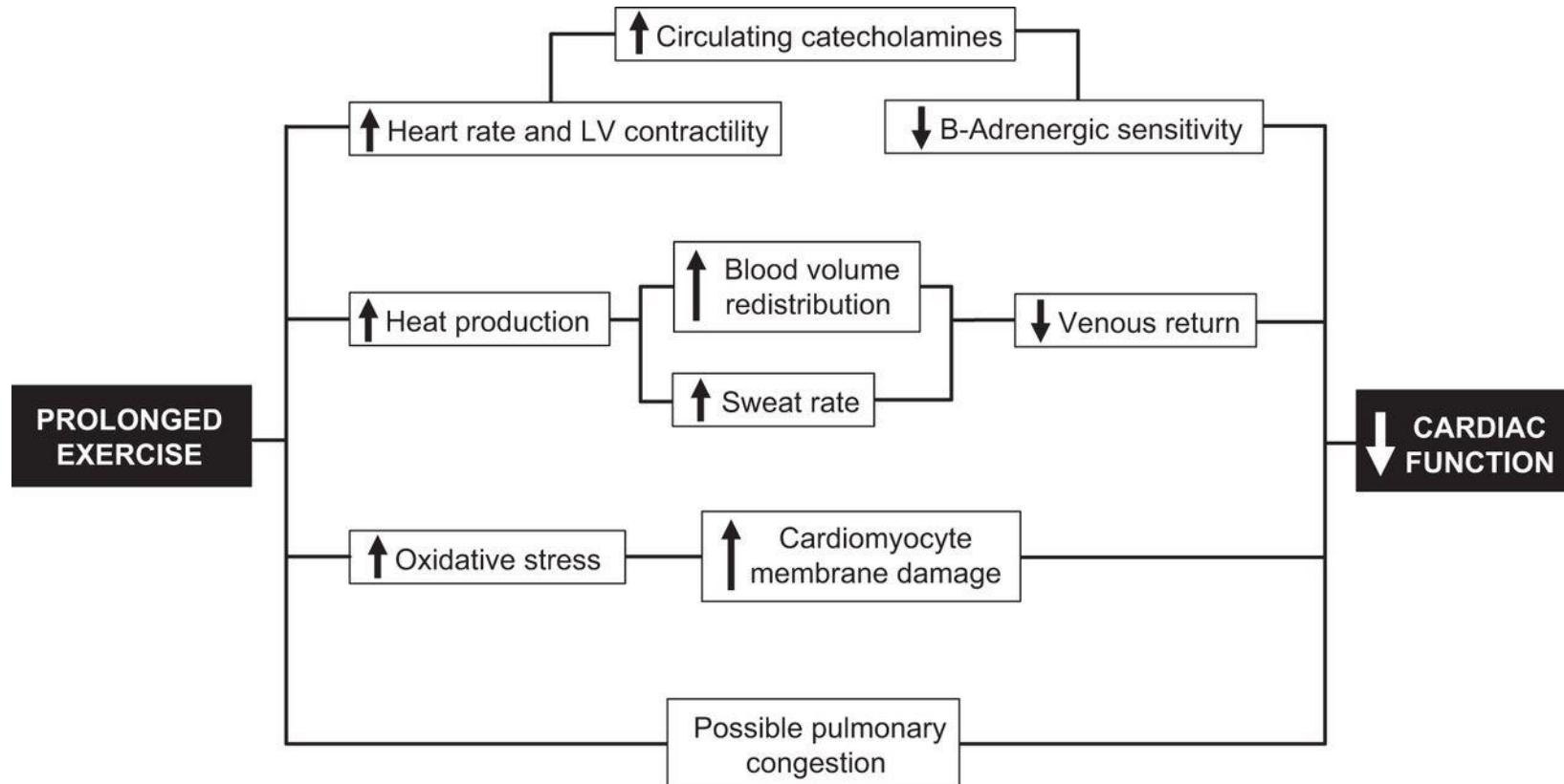
Med Sci Sports Exerc. 2000 Jun;32(6):1067-72.

Cardiac fatigue following prolonged endurance exercise of differing distances.

Whyte GP, George K, Sharma S, Lumley S, Gates P, Prasad K, McKenna WJ.

- **Ironman and half-Ironman competition resulted in reversible abnormalities in resting left ventricular diastolic and systolic function.**
- **Results suggest that myocardial damage may be, in part, responsible for cardiac dysfunction, although the mechanisms responsible for this cardiac damage remain to be fully elucidated.**

Schematic representation of potential mechanisms for impaired cardiac function after prolonged exercise. [Adapted from Shave et al.]



Thijs M. H. Eijsvogels et al. Physiol Rev 2016;96:99-125

Physiological Reviews

[Physiol Rep.](#) 2017 Jun; 5(11): e13297.

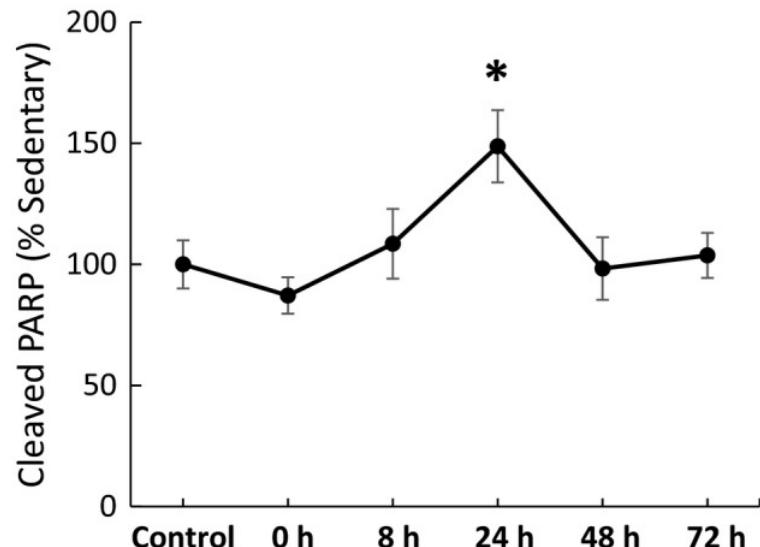
Published online 2017 Jun 2. doi: [10.14814/phy2.13297](https://doi.org/10.14814/phy2.13297)

PMCID: PMC5471436

Myocardial apoptosis and mesenchymal stem cells with acute exercise

Maria F. Arisi,¹ Erica N. Chirico,² Roxanne Sebeny,¹ Geetha Muthukumaran,¹ Anbin Mu,³ Bart C. De Jonghe,¹ Kenneth B. Margulies,² and Joseph R. Libonati¹ 

[Author information](#) ► [Article notes](#) ► [Copyright and License information](#) ►



Our results suggest acute moderate-intensity aerobic treadmill running in exercise-naïve rodents induces temporal cardiomyocyte death due to plasma-borne factors, namely, catecholaminergic stress.

[J Clin Invest.](#) 2005 Aug;115(8):2108-18.

Disruption of coordinated cardiac hypertrophy and angiogenesis contributes to the transition to heart failure.

[Shiojima I](#), [Sato K](#), [Izumiya Y](#), [Schickofer S](#),
[Ito M](#), [Liao R](#), [Colucci WS](#), [Walsh K](#)

Both heart size and cardiac function are angiogenesis dependent, and disruption of coordinated tissue growth and angiogenesis in the heart contributes to the progression from adaptive cardiac hypertrophy to heart failure.

[Appl Physiol Nutr Metab.](#) 2009 Feb;34(1):33-9.

A comparison of Doppler, tissue Doppler imaging, and strain rate imaging in the assessment of postexercise left ventricular function.

[Shave R](#), [George K](#), [Whyte G](#), [Middleton N](#), [Hart E](#), [Artis N](#), [Oxborough D](#).

- A varied response was observed for measures of LV systolic and diastolic function following completion of the marathon (mean +/- SD): EF, 63 +/- 6 vs. 63 +/- 7% ($p > 0.01$); E:A, 1.70 +/- 0.37 vs. 1.17 +/- 0.37; E':A', 2.36 +/- 0.79 vs. 1.60 +/- 0.57 ($p < 0.01$); mean longitudinal epsilon(TDI), 19.1 +/- 5.1 vs. 17.5 +/- 4.2% ($p < 0.01$); mean longitudinal diastolic SR(TDI), 1.81 +/- 0.54 vs. 1.58 +/- 0.51 x s(-1) ($p < 0.01$); mean longitudinal systolic SR(2D), 0.73 +/- 0.21 vs. 0.97 +/- 0.22 x s(-1) ($p < 0.01$); mean longitudinal diastolic SR(2D), 0.94 +/- 0.34 vs. 1.01 +/- 0.23 x s(-1) ($p > 0.01$); mean radial systolic SR(2D), 1.20 +/- 0.15 vs. 1.45 +/- 0.32 x s(-1) ($p < 0.01$); mean radial diastolic SR(2D), 1.19 +/- 0.25 vs. 1.29 +/- 0.41 x s(-1) ($p > 0.01$); mean circumferential systolic SR(2D), -1.09 +/- 0.16 vs. -1.24 +/- 0.18 x s(-1) ($p < 0.01$); and mean circumferential diastolic SR(2D), -1.27 +/- 0.28 vs. -1.22 +/- 0.31 x s(-1) ($p > 0.01$).
- Marathon running promotes a varied echocardiographic response, with some functional parameters showing no change, some increasing, and some decreasing postexercise.

Different Effects of Prolonged Exercise on the Right and Left Ventricles

PAMELA S. DOUGLAS, MD, FACC,* MARY L. O'TOOLE, PhD,†
W. DOUGLAS B. HILLER, MD,‡ NATHANIEL REICHEK, MD, FACC*

Philadelphia, Pennsylvania and Memphis, Tennessee

To examine the functional consequences of the greater increase in right ventricular work with exercise, the effects of prolonged exercise on the right and left heart chambers were compared in 41 athletes before, at the finish (13 min) and after recovery (28 h) from the Hawaii Ironman Triathlon (3.9 km swim, 180.2 km bike ride, 42.2 km run). Two-dimensional and Doppler echocardiograms were analyzed for left and right atrial and ventricular areas at end-diastole and end-systole, right and left ventricular inflow velocities and mitral and tricuspid regurgitation.

After exercise, left ventricular and left and right atrial sizes were reduced, whereas right ventricular size increased (diastole: 21.4 to 24.2 cm²; systole: 15.8 to 18.2 cm²; p < 0.01). The emptying fraction of all chambers was unchanged. Left but not right ventricular inflow showed an

increase in peak velocity of rapid filling, whereas both atrial systolic velocities increased (26 to 38 cm/s tricuspid; 38 to 54 cm/s mitral; both p < 0.01). Overall, the right ventricular early to atrial velocity ratio was reduced after exercise (1.56 to 1.17; p < 0.05) and the left ventricular pattern was unchanged. The prevalence of tricuspid regurgitation was statistically unchanged (86% to 52%), although that of mitral regurgitation was greatly reduced (76% to 0%). Changes in all variables returned toward prerace values during recovery.

Thus, in highly trained athletes, prolonged exercise causes differing responses of the right and left ventricles. These differences may be due to changes in right ventricular function, shape or compliance.

(J Am Coll Cardiol 1990;15:14-9)

[Eur Heart J.](#) 2012 Apr;33(8):998-1006

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

[La Gerche A](#), [Burns AT](#), [Mooney DJ](#), [Inder WJ](#), [Taylor AJ](#), [Bogaert J](#), [Macisaac AI](#), [Heidbüchel H](#), [Prior DL](#).

- Forty athletes were studied at baseline, immediately following an endurance race (3-11 h duration) and 1-week post-race. Right ventricular ejection fraction decreased with increasing race duration ($r = -0.501$, $P < 0.0001$) and VO_2max ($r = -0.359$, $P = 0.011$). Right ventricular function mostly recovered by 1 week.

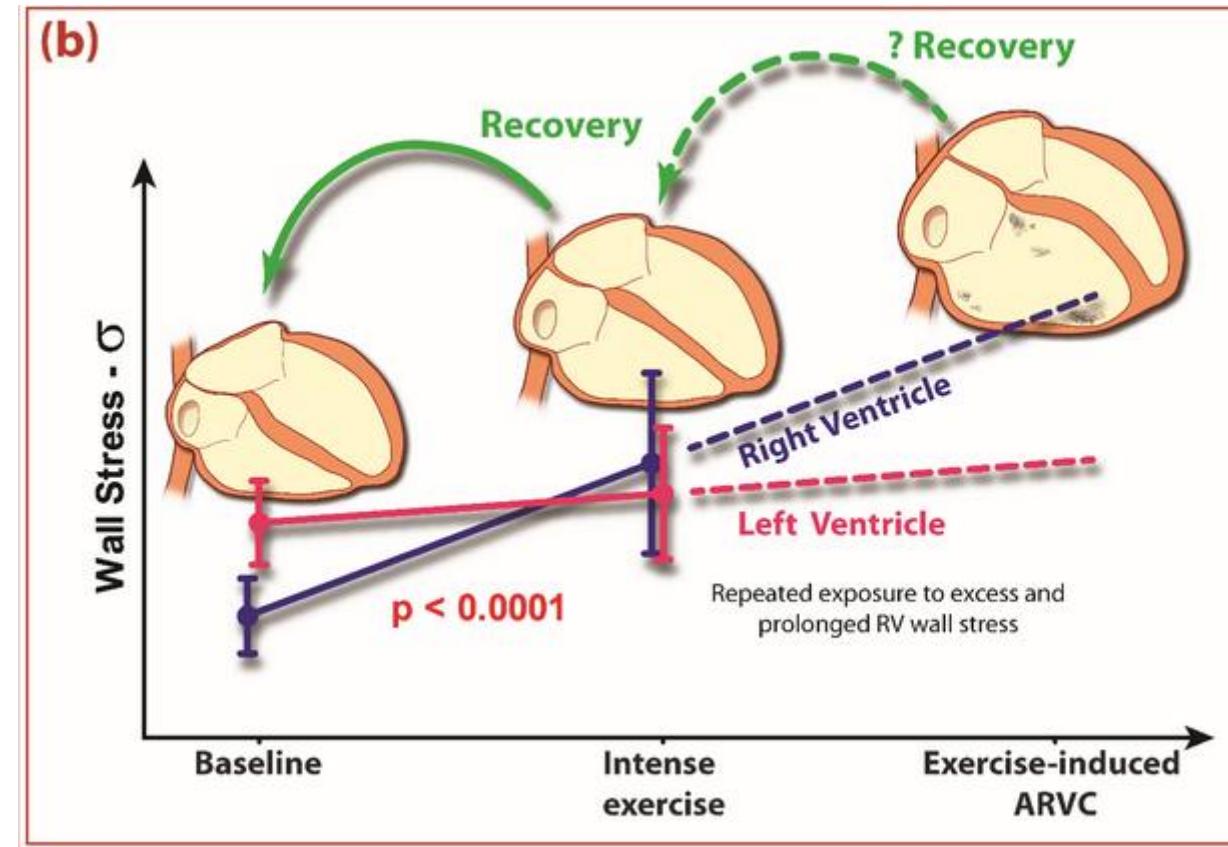
Intense endurance exercise causes acute dysfunction of the RV, but not the LV.

~~Although short-term recovery appears complete, chronic structural changes and reduced RV function are evident in some of the most practiced athletes, the long-term clinical significance of which warrants further study.~~



Mysteries of the Right Ventricle in Sports and Exercise

Nov 05, 2015 | Expert Analysis



As compared with the LV, relative increases in wall stress are greater for the RV during intense exercise, the result of which is healthy cardiac remodelling with a very slight RV dominance, which diminishes with de-training. Repeated bouts of excessive and prolonged RV wall stress may result in cumulative RV damage, which may predispose to arrhythmias. The degree to which this adverse remodelling may recover with de-training is unclear.

The Response of the Pulmonary Circulation and Right Ventricle to Exercise: Exercise-Induced Right Ventricular Dysfunction and Structural Remodeling in Endurance Athletes

André La Gerche, Timothy Roberts, Guido Claessen

Both echocardiographic and invasive studies are consistent in demonstrating that pulmonary arterial pressures increase progressively with exercise intensity, such that the harder one exercises, the greater the load on the RV.

“the RV should be considered a potential Achilles' heel of the exercising heart !”

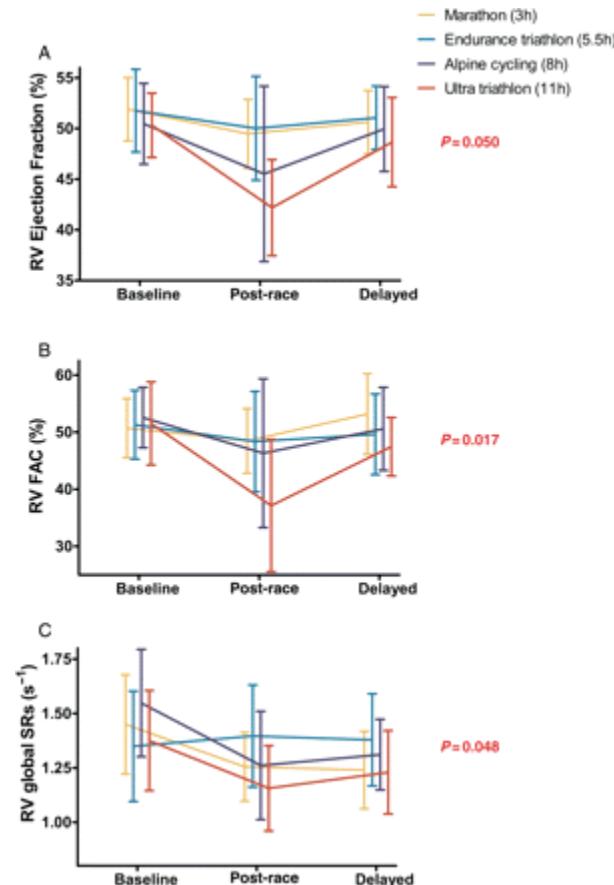


Exercise-induced right ventricular dysfunction and structural remodelling in endurance athletes FREE

André La Gerche , Andrew T. Burns, Don J. Mooney, Warrick J. Inder, Andrew J. Taylor, Jan Bogaert, Andrew I. MacIsaac, Hein Heidbüchel, David L. Prior

European Heart Journal, Volume 33, Issue 8, 1 April 2012, Pages 998–1006, <https://doi.org/>

Duration-dependent increase in right ventricular dysfunction. Ejection fraction (A), fractional area change (B), and systolic strain rate (C) decreased in the post-race setting. There was a significant interaction between event type and time point (*P*-value) with a greater reduction in function in those completing the longest event (ultra-triathlon).





JACC

JOURNAL OF THE AMERICAN COLLEGE OF CARDIOLOGY

1997 Aug;30(2):468-73.

Transient right but not left ventricular dysfunction after strenuous exercise at high altitude.

Dávila-Román VG, Guest TM, Tuteur PG, Rowe WJ, Ladenson JH, Jaffe AS.

In trained athletes, strenuous exercise at high altitude did not result in LV damage. However, wheezing, reversible pulmonary hypertension and RV dysfunction occurred in a third of those completing the race.

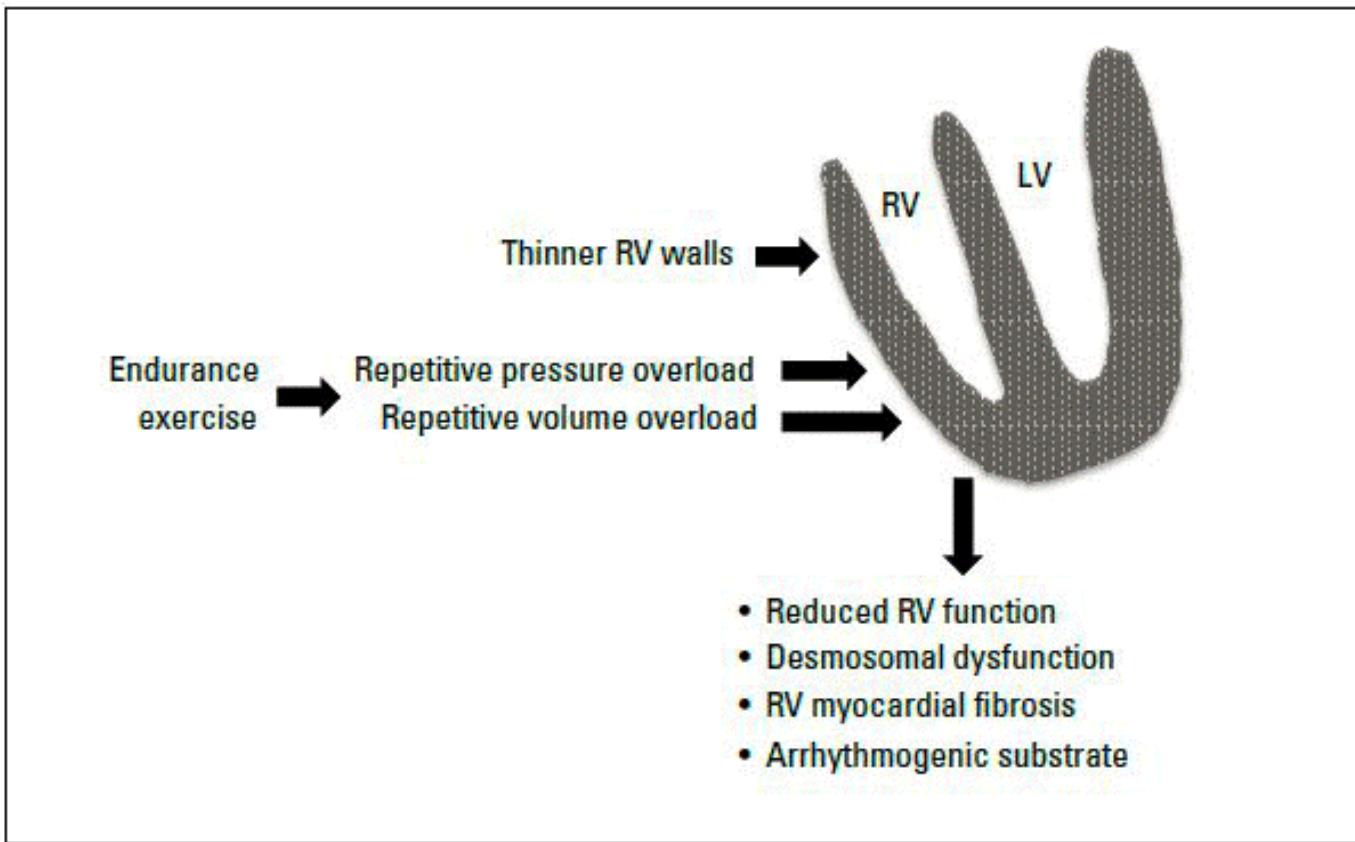
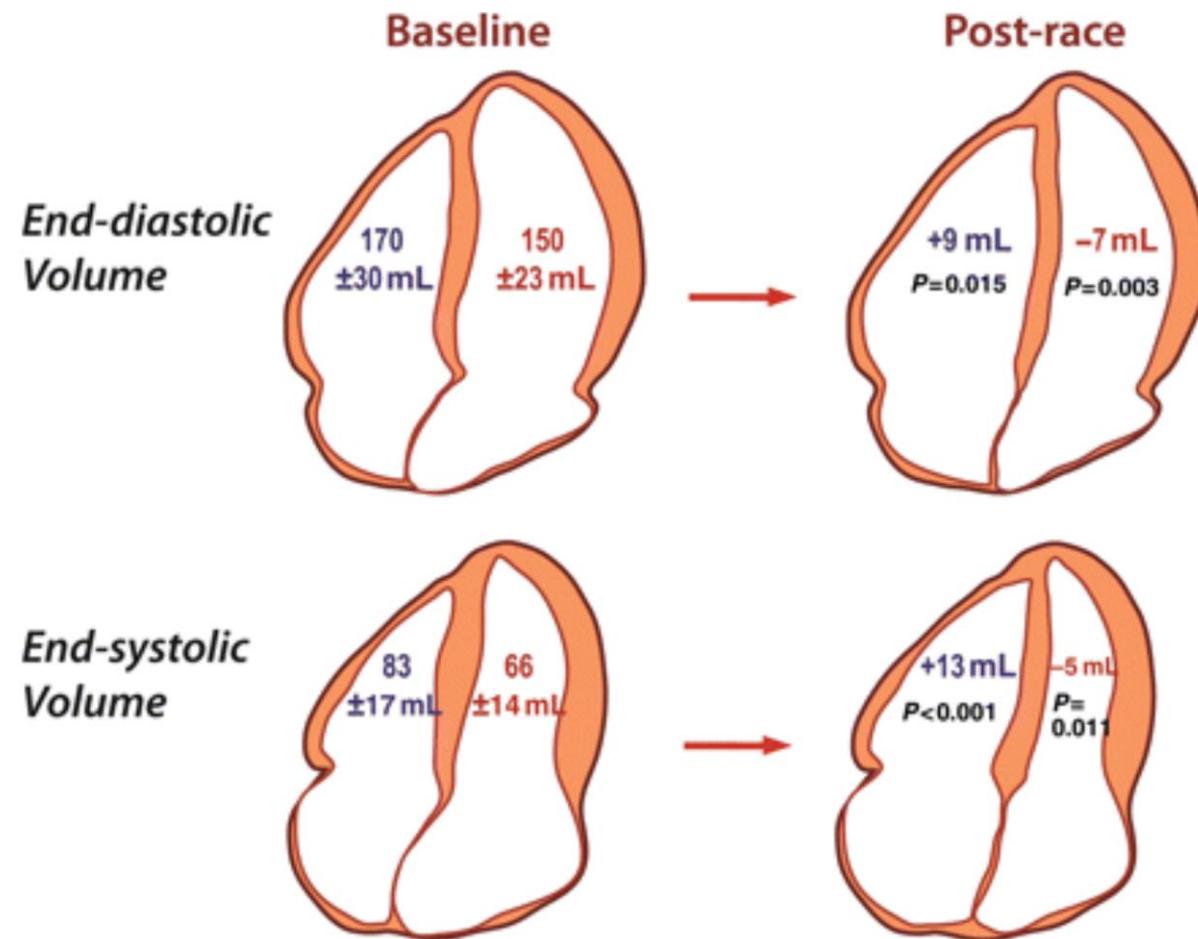


Figure 2. Effects of repetitive endurance exercise on the right ventricle (RV) compared with the left ventricle (LV).

Factors that contribute to the possibly reversible exercise-induced RV changes include pressure and volume overload.



From: Exercise-induced right ventricular dysfunction and structural remodelling in endurance athletes
Eur Heart J. 2011;33(8):998-1006. doi:10.1093/eurheartj/ehr397
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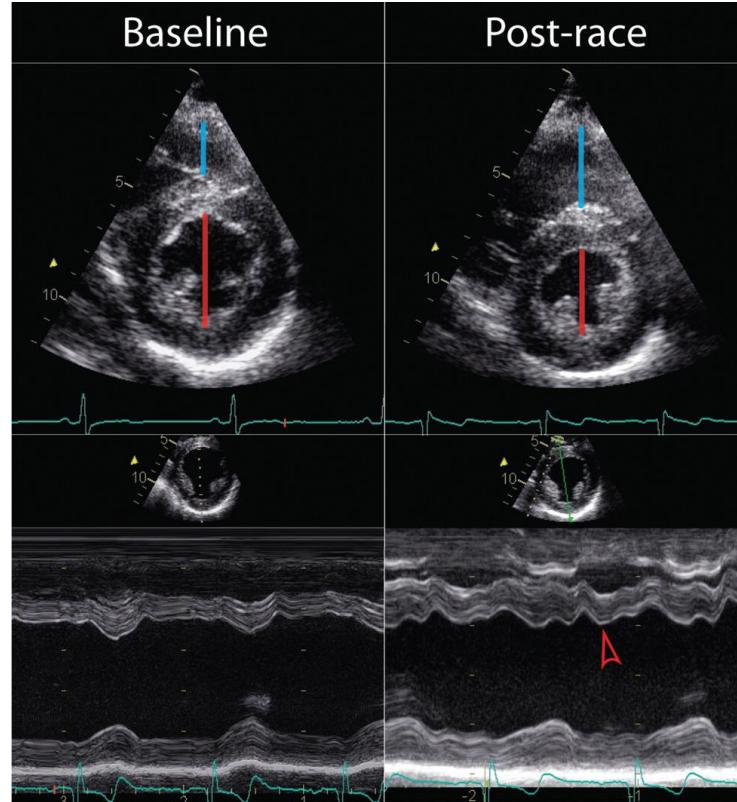


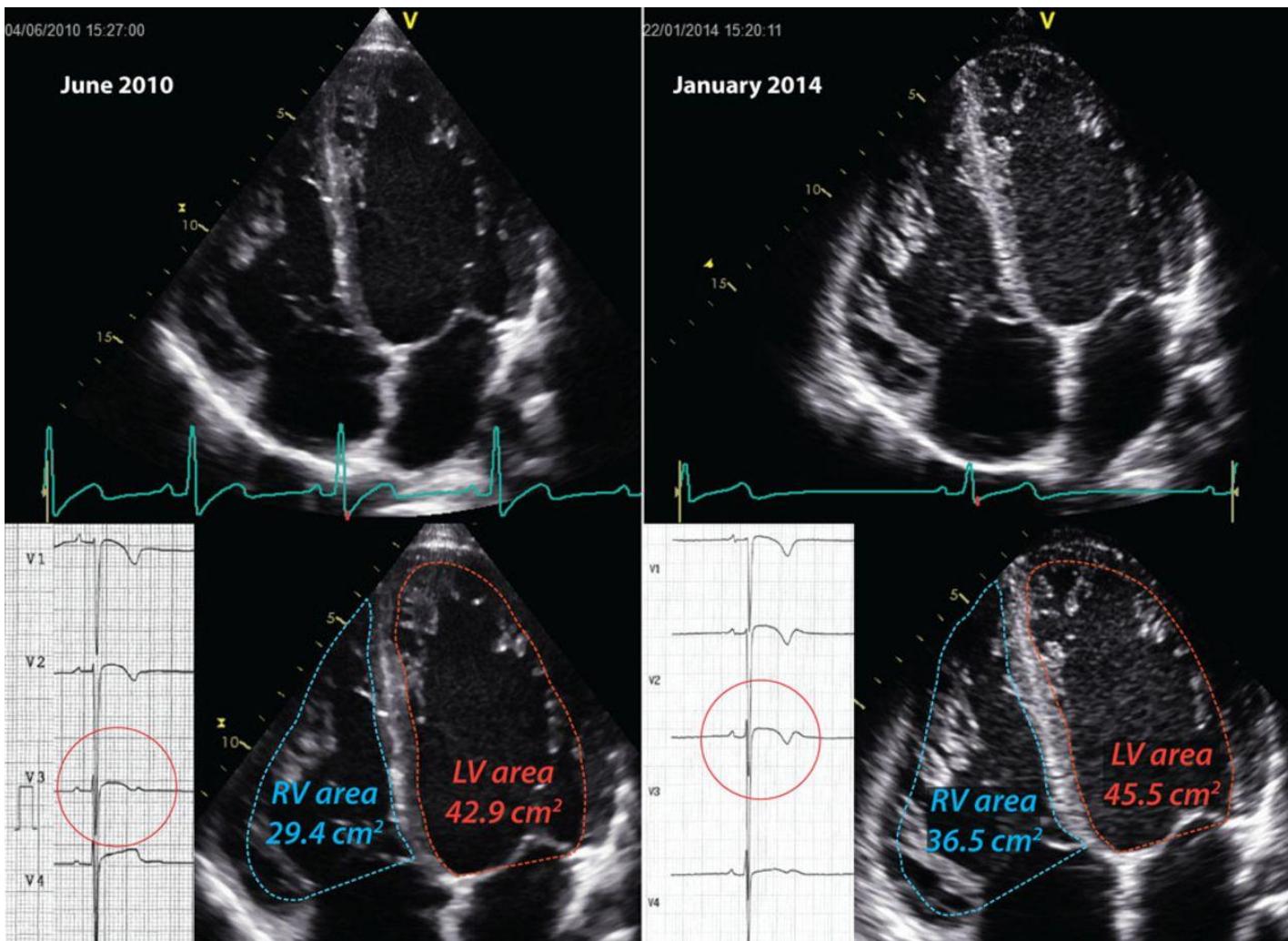
Figure 3. Acute right ventricular dilation and dysfunction following ultraendurance exercise causing early diastolic septal shift toward the left ventricle. Compared with baseline, right ventricular (RV) dimensions (blue line) are increased and left ventricular (LV) dimensions (red line) reduced immediately following an ultraendurance triathlon. The dilation and delayed contraction of the RV results in septal shift toward the LV in early diastole (red arrow) such that RV dysfunction impairs early LV filling. This would suggest that overall cardiac output may be predominantly limited by reduced RV function in the endurance exercise setting.

Published in: André La Gerche; Timothy Roberts; Guido Claessen; *Pulm Circ* 4, 407-416.

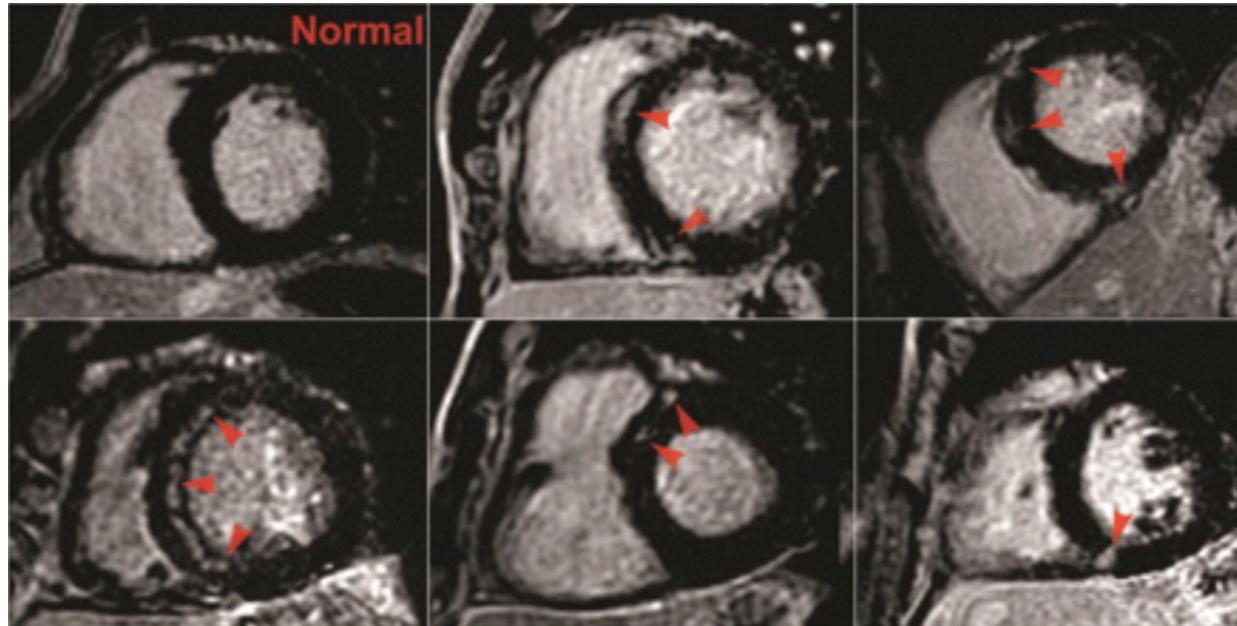
DOI: 10.1086/677355

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Relative increase in right ventricular dimensions in an elite cyclist with arrhythmias.



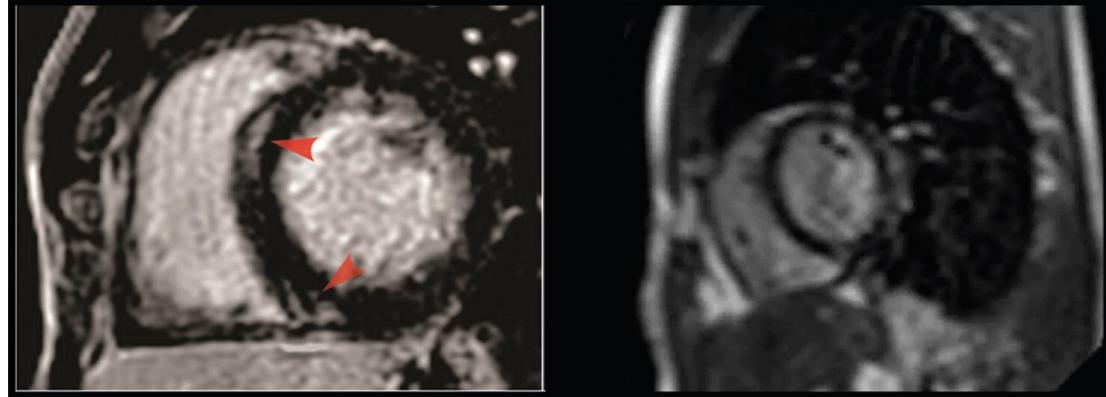
La Gerche A, Heidbuchel H. Circulation 2014;130:992-1002



Delayed gadolinium enhancement in five athletes. Images of five athletes in whom focal delayed gadolinium enhancement (DGE) was identified in the interventricular septum (indicated with arrows) when compared with an athlete with a normal study (top left)

From: Exercise-induced right ventricular dysfunction and structural remodelling in endurance athletes
Eur Heart J. 2011;33(8):998-1006. doi:10.1093/eurheartj/ehr397
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Using exercise CMR to investigate potential mechanisms of exercise-induced fibrosis



Septal delayed gadolinium enhancement (DGE) in an endurance athlete—potential mechanism. In this ostensibly healthy endurance athlete, discreet patches of DGE are noted in the inferior and mid-interventricular septum in a pattern reminiscent of pathologies that result in increased right ventricle loading. This video (available online) demonstrates a short-axis real-time cine acquisition of the same region during intense exercise. The video has been slowed to reveal considerable septal shift with the mechanical hinge points seeming to approximate the regions of DGE. Thus, it may be hypothesized that focal fibrosis results from repeated mechanical stress induced by strenuous exercise. CMR: cardiac magnetic resonance.

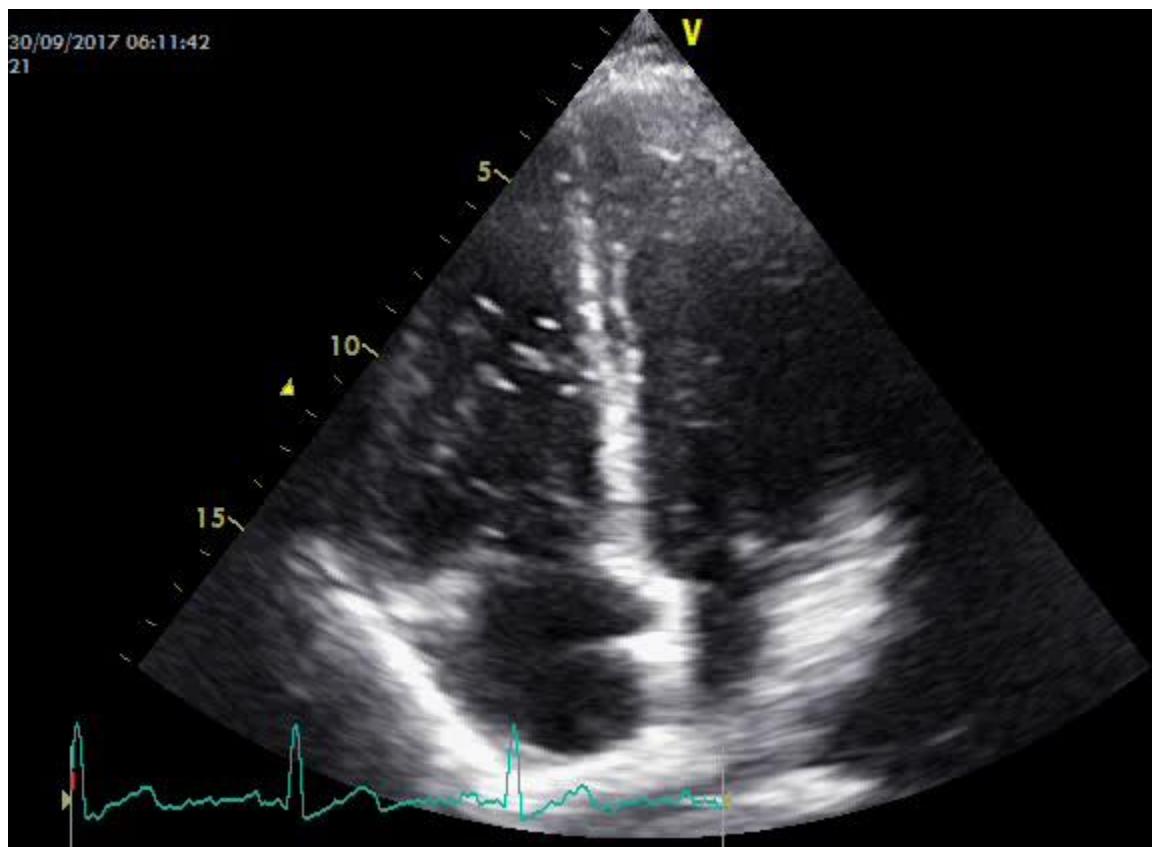
Published in: André La Gerche; Timothy Roberts; Guido Claessen; *Pulm Circ* 4, 407-416.

DOI: 10.1086/677355

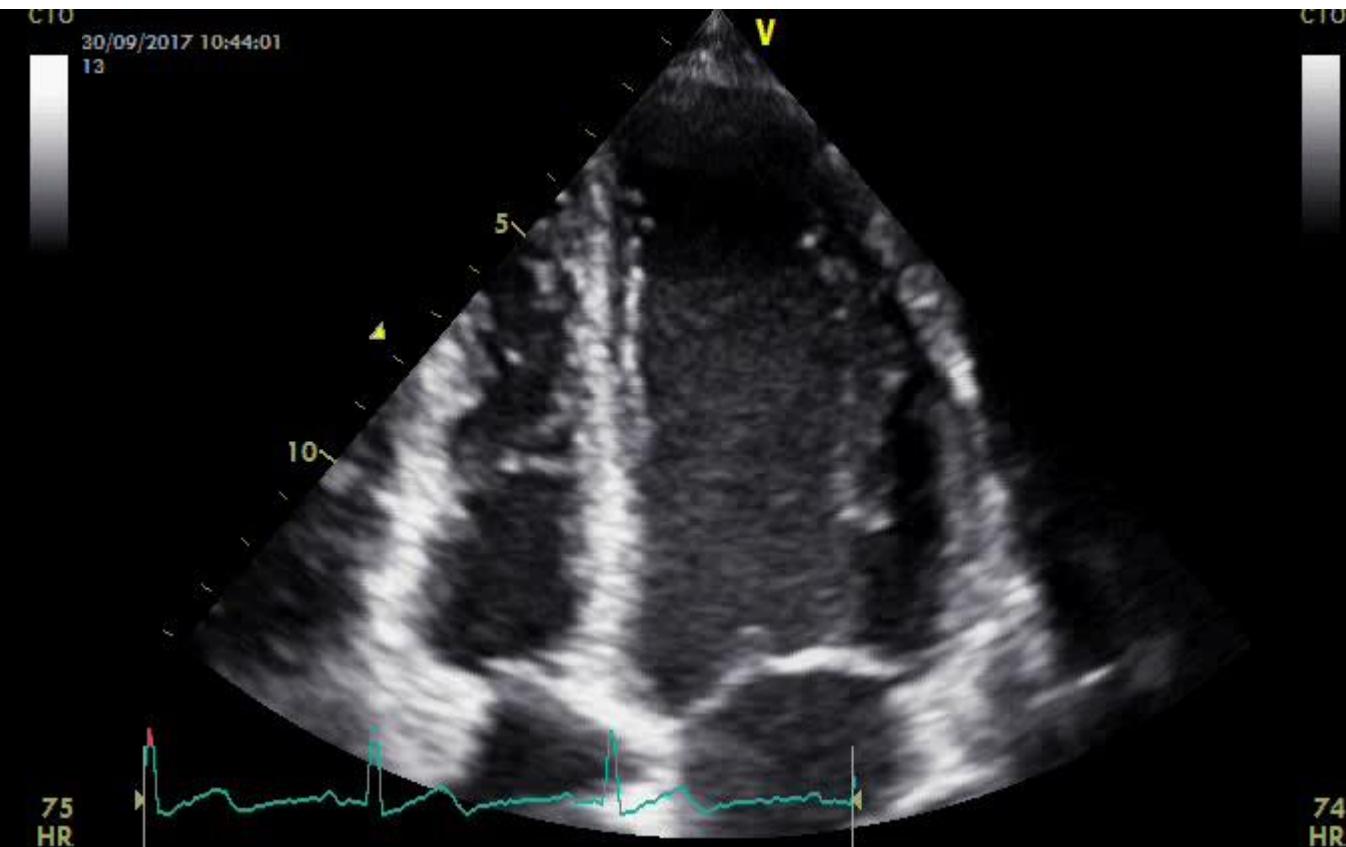
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ECHO IMAGES OF CARDIAC VENTRICLES FROM THE 1st FINISHER OF THE SPARTATHLON

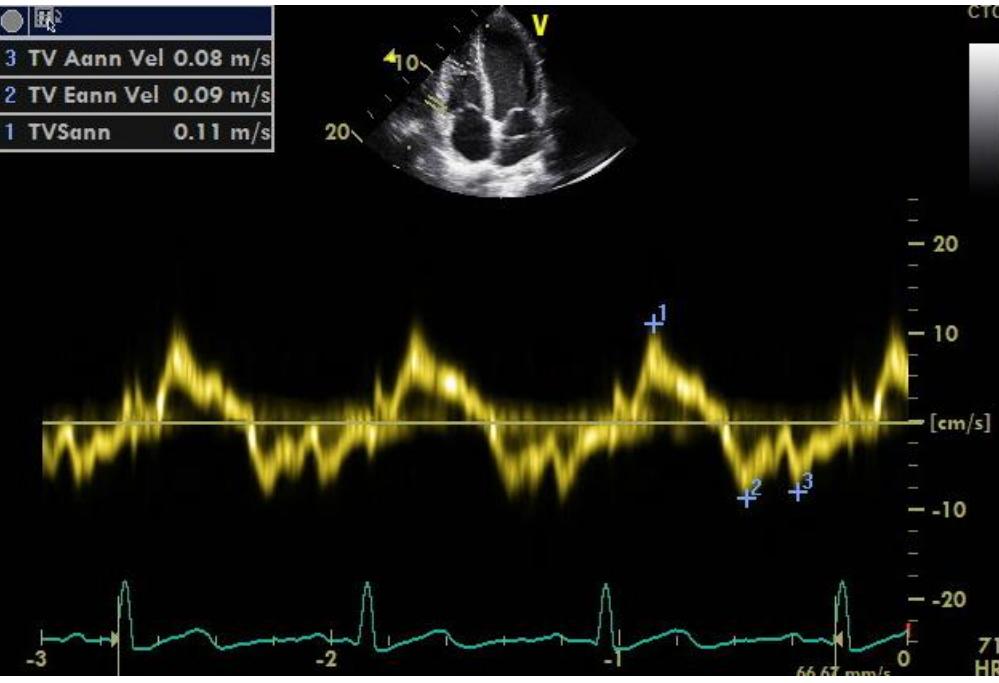
IMMEDIATELY AFTER FINISH



5 HOURS AFTER



PULSED TISSUE DOPPLER OF RV FROM THE 1st FINISHER



Accelerated cardiac remodeling in desmoplakin transgenic mice in response to endurance exercise is associated with perturbed Wnt/β-catenin signaling

Ruben Martherus, Rahul Jain, Ken Takagi, Uzmee Mendsaikhan, Subat Turdi, Hanna Osinska, Jeanne F. James, Kristen Kramer, Enkhsaikhan Purevjav, Jeffrey A. Towbin

American Journal of Physiology - Heart and Circulatory Physiology Published 15 January 2016 Vol. 310 no. 2, H174-H187

- The purpose of this study was to investigate how chronic endurance exercise contributes to desmoplakin (DSP) mutation-induced AVC pathogenesis.
- The data suggest that endurance exercise accelerates AVC pathogenesis in Tg-DSP^{R2834H} mice and this event is associated with perturbed AKT1 and GSK3-β signaling.

“Exercise” Right Ventricular Cardiomyopathy



Introduction

Intense sports activity leads to the adaptation of cardiac structure and function, the so-called athlete's heart. These changes usually comprise a balanced dilation and hypertrophy of all four cardiac chambers. However, research over the last 20 years has indicated that changes in the right ventricle (RV) do not always parallel those of the left ventricle (LV), both in the short term (i.e. immediately after an endurance sports event) and in the long term. In 2003, we presented the hypothesis that intense endurance activities may lead to cardiac alterations due to a particularly high strain on the RV [10]. Exercise RV stress may lead to minor cell damage. Over time, repetitive injury may lead to changes that resemble right, or less often left, ventricular cardiomyopathy, even in the absence of underlying demonstrable genetic abnormalities. The syndrome of “exercise-induced arrhythmogenic RV cardiomyopathy” (ARVC) may have been overlooked and under-recognized in prior series. This text will describe the pathophysiological findings that corroborate our hypothesis of exercise-induced ARVC.

Ventricular arrhythmias in athletes are rare, but often of RV origin

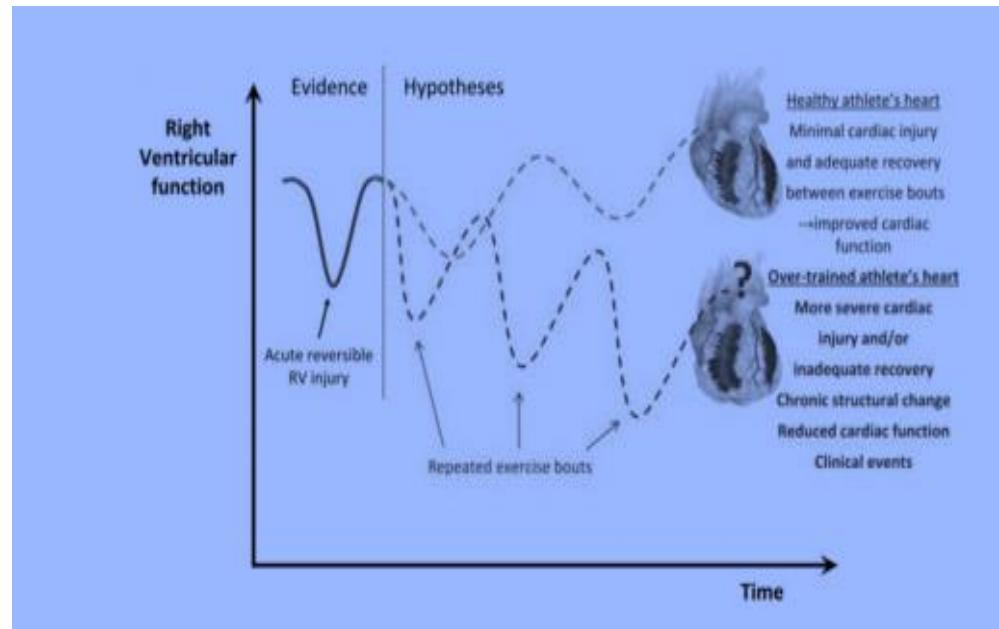
Ventricular arrhythmias in athletes are rare, but by nature they may be life-threatening. Physical activity is associated with a

2.3 times increased risk for sudden death [1]. The classic concept is that arrhythmic events are due to underlying (structural or electrical) heart disease, on which physical activity acts as a trigger for the initiation of arrhythmias. A multitude of underlying cardiovascular conditions have been shown to predispose an athlete to exercise-related sudden death at multiple stages [2, 8]. When evaluating 46 high-level endurance athletes (performing ≥1–2 h of sports per week for more than 3 years; 80% competition; 80% cyclists who were referred to us for evaluation of asymptomatic symptoms that could be attributed to arrhythmic arrhythmias [2]), we were intrigued by the fact that the great majority of these ventricular arrhythmias, 86%, had an RV origin. This was not expected based on the known predisposing pathologies, which statistically would have a much higher probability of inducing arrhythmias of LV origin. Nevertheless, the arrhythmic outcome was not benign; after a median follow-up of 4.7 years, 10 out of 46 athletes had a major rhythm disorder, of which nine were (albeit) sudden deaths (all cyclists; a mean of 3 years after presentation). Most were inducible during an electrophysiological study pertaining to an alleged RV structural substrate. Although the athletes presented with RV arrhythmias, overt structural findings of ARVC were less frequently present than seen in familial forms. When combining major and minor criteria of the original ARVC diagnostic framework, we found that 59% had manifest ARVC and an ad-

ditional 30% had probable ARVC. Nevertheless, only one of the 46 cases had a familial history. We therefore started to question how far the structural adaptation of the athlete's RV, especially under extreme endurance load, had developed into RV degeneration and arrhythmogenicity [8].

As mentioned, 80% of the athletes whom we evaluated with this syndrome were high-level (often competitive) cyclists or triathlon athletes [8, 10]. We have observed the same presentation in rowers, marathoners and swimmers. Long-distance runners are notably scarce in our series, although this sport is very popular in our region. Therefore, the RV effects seem to be linked to high-intensity endurance activities, particularly those that are of long duration and combine endurance and power. We suspect that other sports with a similar physiological load, e.g. cross-country skiing, may also be associated with an increase in RV arrhythmias, but this sporting demographic is not part of our experience. Early reports from the Nordic regions, however, seem to suggest that the syndrome is also observed in this type of athlete.

On the other hand, the incidence of (acquired) RV arrhythmogenicity seems to be relatively small. Deaf moral estimates exist because of the lack of good registries. There is both uncertainty about the numerator (complete registry of cases?) and denominator (population of intense endurance athletes at risk?). Rough estimates based on the inferred pattern for our



Endurance Exercise: Is it Proarrhythmic?

David M. Gilligan



TABLE 2. POTENTIAL PROARRHYTHMIC EFFECTS OF EXTREME ENDURANCE EXERCISE.

Increased incidence of atrial fibrillation

Sinus bradycardia and conduction impairment

Increased ventricular ectopy

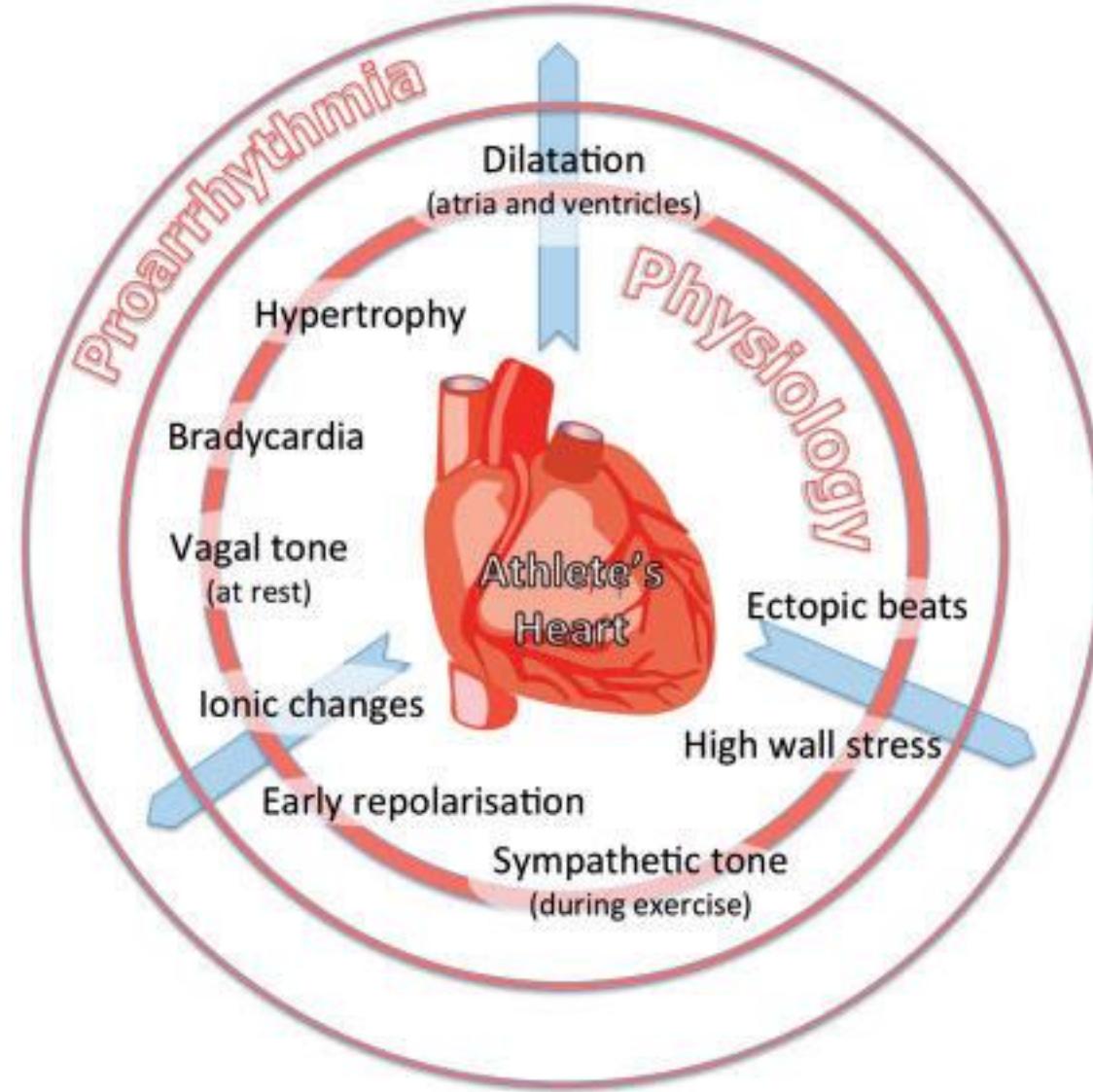
Increased risk of ventricular tachycardia/fibrillation



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

Kasper Andersen^{1*}, Bahman Farahmand^{2,3}, Anders Ahlbom², Claes Held¹, Sverker Ljunghall¹, Karl Michaëlsson⁴, and Johan Sundström¹

- 52,755 Skiers in 90 km (56 miles) Vasaloppet 1989–98 followed to Dec 2005
- Most Races – More Afib (HR 1.29; CI 1.04–1.61)
- Fastest – More Afib (1.20; CI 0.93–1.55)



INTENSE EXERCISE AND CARDIAC ARRHYTHMIAS

Heart block

PPM in 3% athletes
vs. 0% non-athletes

*Balderberger et al EHJ 2007
*Stein et al JACC 2002

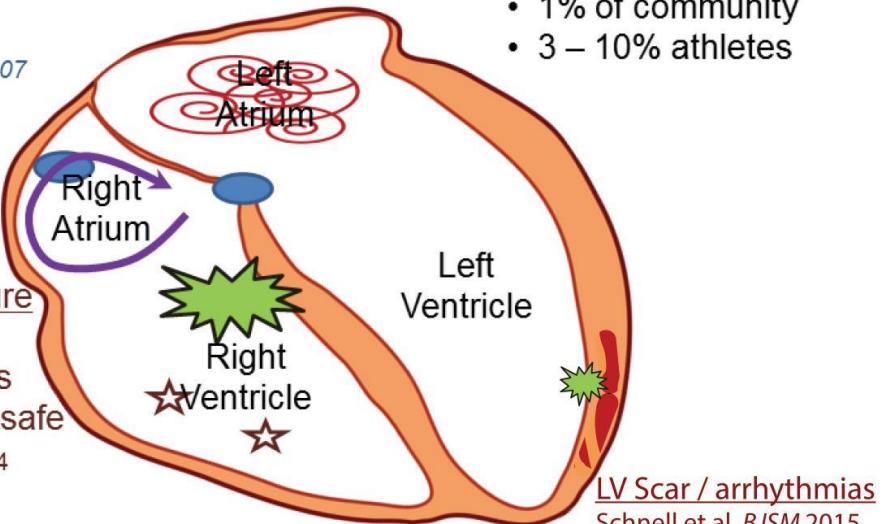
Ventricular premature
beats (ectopics)
Common in athletes
Usually completely safe

*Biffi JACC 2004

Atrial flutter

- 31% vs. 8% are endurance athletes

*Claessen, La Gerche,
Heidbuchel Heart 2011



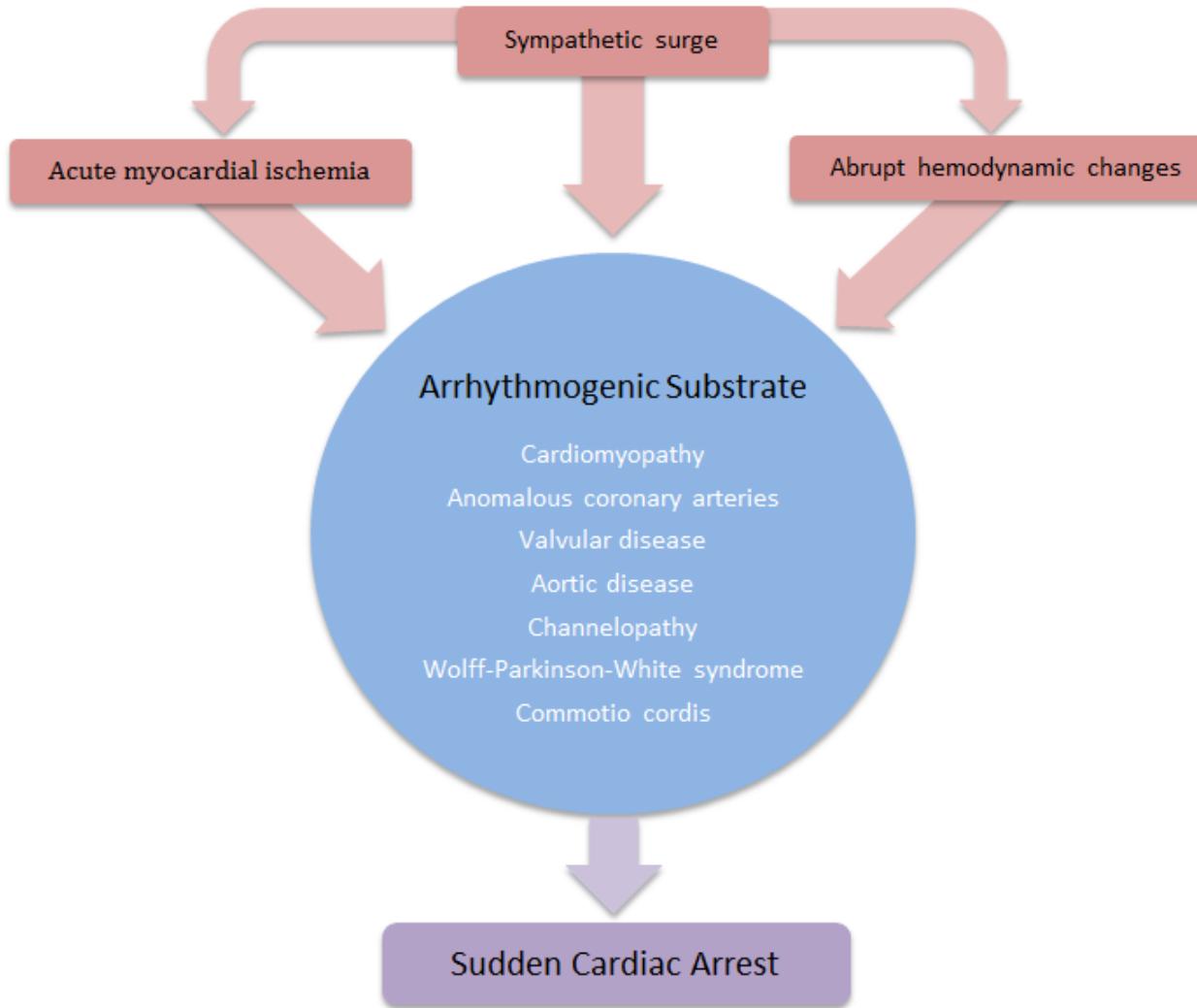
Atrial fibrillation

- 1% of community
- 3 – 10% athletes

Ventricular tachycardia

Rare. Potentially serious
Most often of RV origin
??? More common in athletes

*Heidbuchel, Hoogsteen et al. EHJ 2003



Lee D, Chung E. JACC 2016

<http://www.acc.org/latest-in-cardiology/articles/2016/06/28/07/06/>



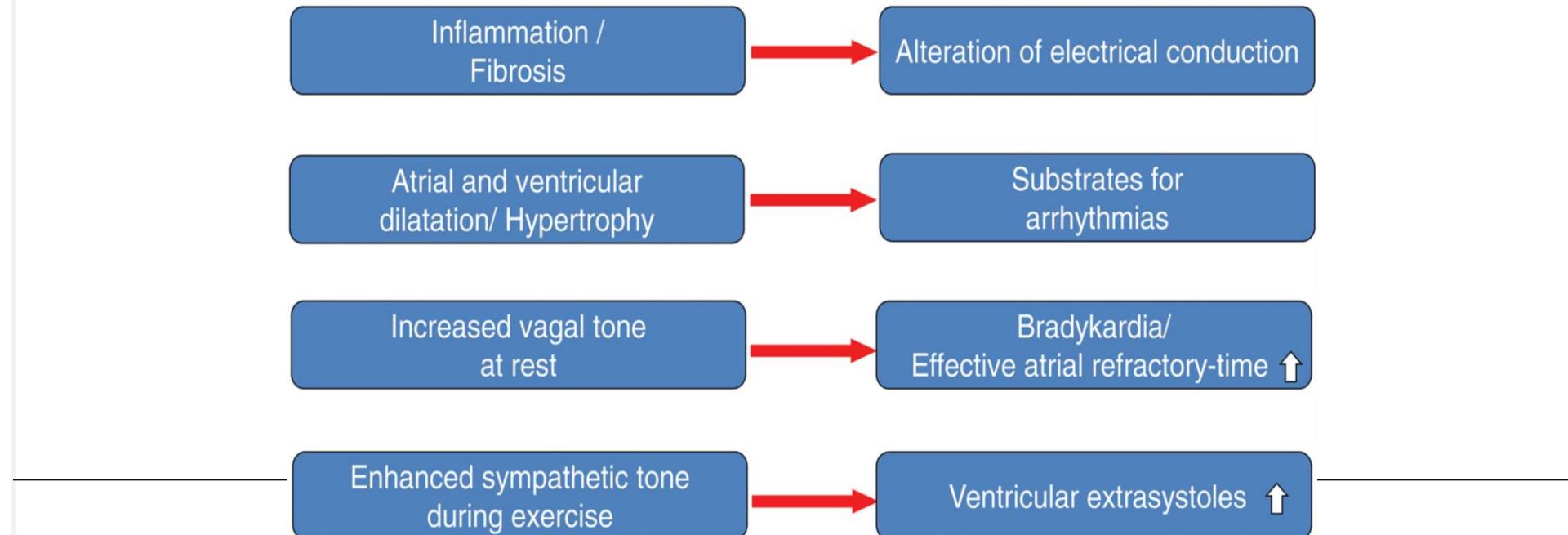
Endurance sport practice as a risk factor for atrial fibrillation

Lluís Mont Roberto Elosua Josep Brugada

EP Europace, Volume 11, Issue 1, 1 January 2009, Pages 11–17

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Mechanisms of arrhythmias

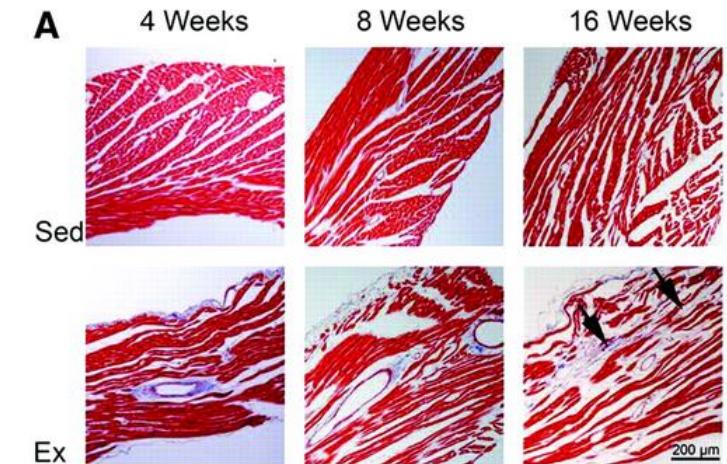
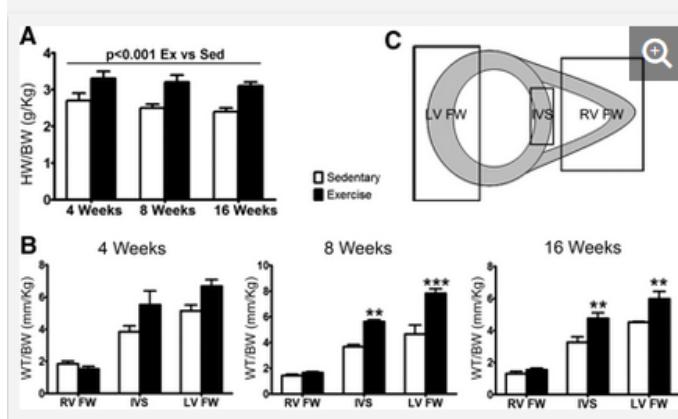


**From: Marathon run: cardiovascular adaptation and cardiovascular risk
Eur Heart J. 2014;35(44):3091-3098.**

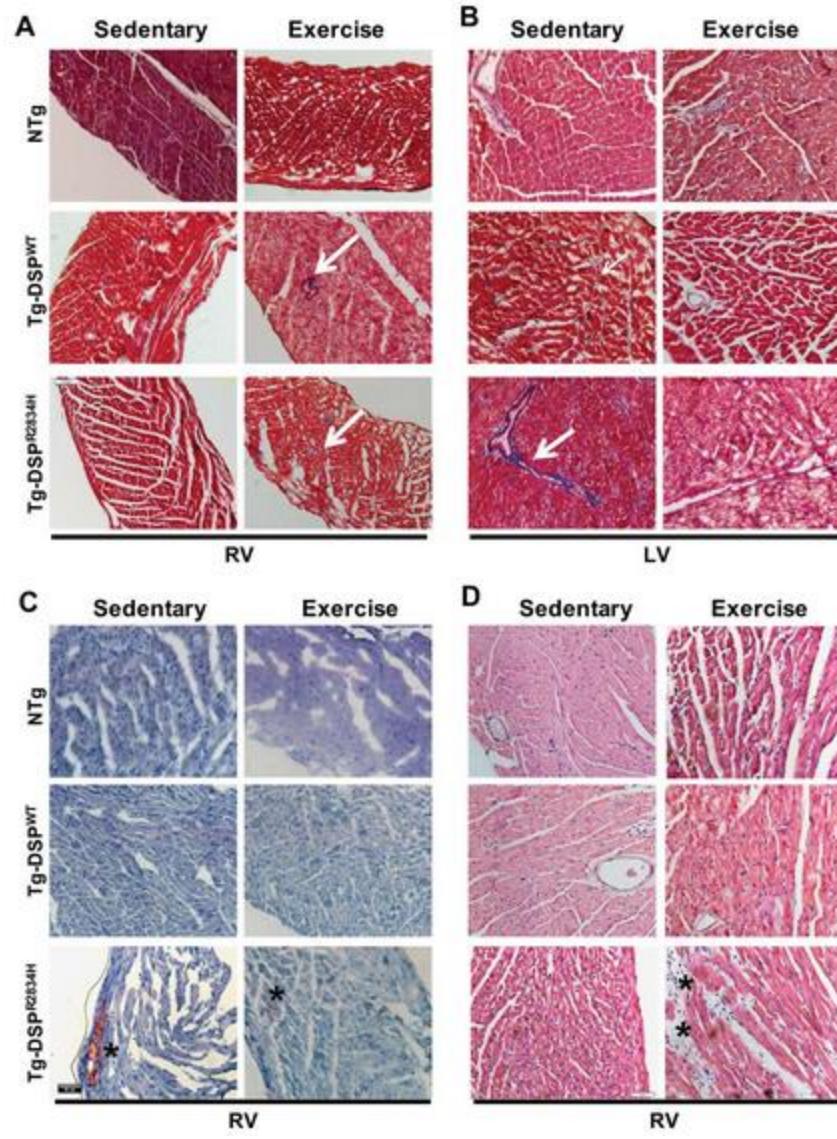
Original Articles

Cardiac Arrhythmogenic Remodeling in a Rat Model of Long-Term Intensive Exercise Training

Begoña Benito, Gemma Gay-Jordi, Anna Serrano-Mollar, Eduard Guasch, Yanfen Shi, Jean-Claude Tardif, Josep Brugada, Stanley Nattel, Lluis Mont



In this animal model, we documented cardiac fibrosis after long-term intensive exercise training, together with changes in ventricular function and increased arrhythmia inducibility. If our findings are confirmed in humans, the results would support the notion that long-term vigorous endurance exercise training may in some cases promote adverse remodeling and produce a substrate for cardiac arrhythmias.



Curr Sports Med Rep. 2013 Mar-Apr;12(2):63-9.

Can intense endurance exercise cause myocardial damage and fibrosis?

La Gerche A

Extreme exercise has been associated with biochemical and functional evidence of acute damage, and some recent imaging techniques raise the possibility of small areas of myocardial scar. Moreover, some arrhythmias appear to be more prevalent amongst endurance athletes.

[Eur Heart J.](#) 2015 Aug 7;36(30):1998-2010.

Exercise-induced right ventricular dysfunction is associated with ventricular arrhythmias in endurance athletes.

[La Gerche A](#), [Claessen G](#), [Dymarkowski S](#), [Voigt JU](#), [De Buck F](#), [Vanhees L](#), [Droogne W](#),
[Van Cleemput J](#), [Claus P](#), [Heidbuchel H](#).

- Among athletes with normal cardiac function at rest, exercise testing reveals RV contractile dysfunction among athletes with RV arrhythmias. RV stress testing shows promise as a non-invasive means of risk-stratifying athletes.

[Eur Heart J.](#) 2015 Aug 7;36(30):1955-7.

Arrhythmogenic right ventricular remodelling in endurance athletes: Pandora's box or Achilles' heel?

[Zaidi A, Sharma S.](#)

- Extreme exercise has been associated with biochemical and functional evidence of acute damage, and some recent imaging techniques raise the possibility of small areas of myocardial scar. Moreover, some arrhythmias appear to be more prevalent amongst endurance athletes. Only large prospective trials will enable us to really assess the health benefits and risks of regular intense endurance sports.

Circulation: Cardiovascular Imaging

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EDITORIAL

Endurance Exercise and Myocardial Fibrosis

Let Us Keep the Risk in Perspective

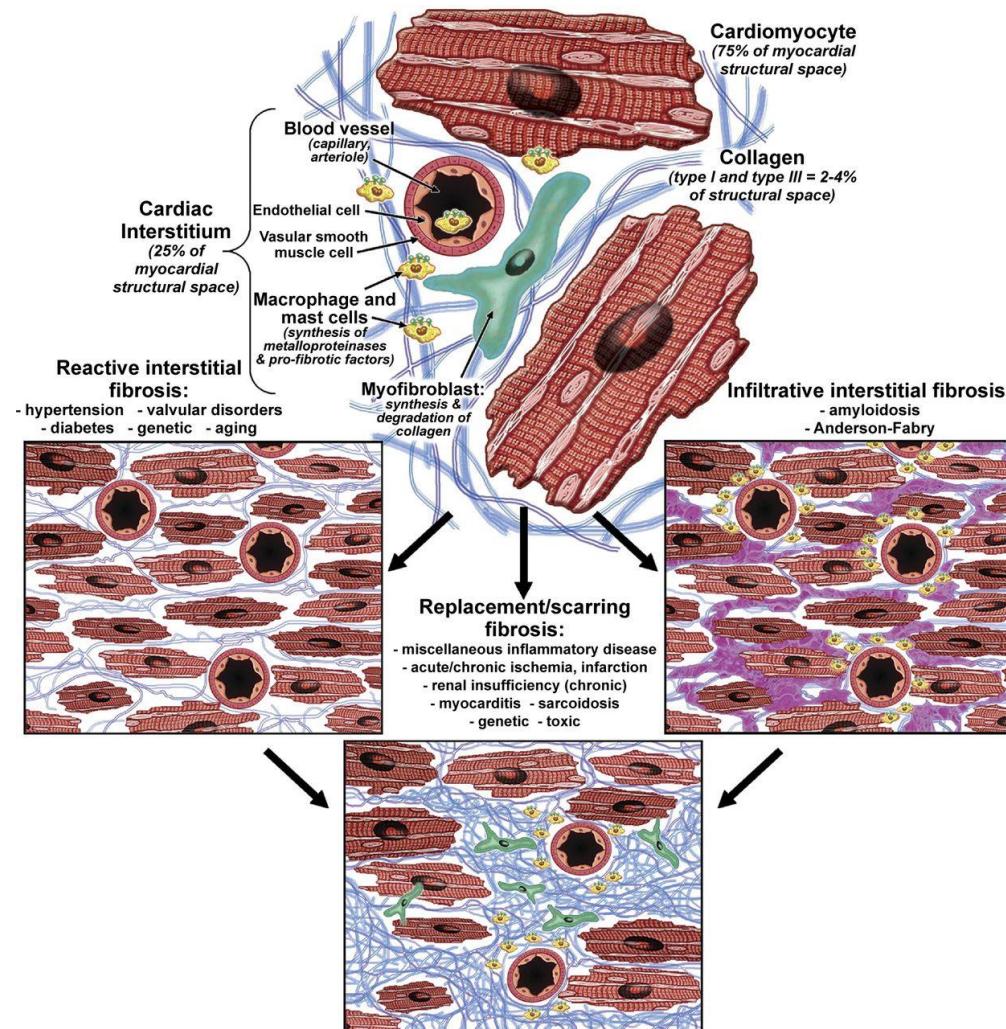
Rob Shave, David Oxborough

DOI <https://doi.org/10.1161/CIRCIMAGING.116.005730>

Circulation: Cardiovascular Imaging. 2016;9:e005730

Originally published November 10, 2016

Pathophysiology of myocardial fibrosis. [From Mewton et al.]



Thijs M. H. Eijsvogels et al. Physiol Rev 2016;96:99-125

Physiological Reviews

Mayo Clin Proc. 2016 Nov;91(11):1617-1631

Myocardial Fibrosis in Athletes.

van de Schoor FR, Aengevaeren VL, Hopman MT, Oxborough DL, George KP, Thompson PD, Eijsvogels TM

- Myocardial fibrosis in athletes was predominantly identified in the intraventricular septum and where the right ventricle joins the septum. Although the underlying mechanisms are unknown, we summarize the evidence for genetic predisposition, silent myocarditis, pulmonary artery pressure overload, and prolonged exercise-induced repetitive micro-injury as contributors to the development of MF in athletes.

EVIDENCE OF MYOCARDIAL FIBROSIS IN ATHLETES

Radiology. 2009 Apr;251(1):50-7. doi: 10.1148/radiol.2511081118.

Myocardial late gadolinium enhancement: prevalence, pattern, and prognostic relevance in marathon runners.

Breuckmann F¹, Möhlenkamp S, Nassenstein K, Lehmann N, Ladd S, Schermund A, Sievers B, Schlosser T, Jöckel KH, Heusch G, Erbel R, Barkhausen J.

Author information

Abstract

PURPOSE: To prospectively analyze the myocardial distribution of late gadolinium enhancement (LGE) with delayed-enhancement cardiac magnetic resonance (MR) imaging, to compare the prevalence of this distribution in nonprofessional male marathon runners with that in asymptomatic control subjects, and to examine the prognostic role of LGE.

MATERIALS AND METHODS: Institutional review board and ethics committee approval were obtained for this study, and all subjects provided written informed consent. Two-dimensional inversion-recovery segmented k-space gradient-echo MR sequences were performed after administration of a gadolinium-containing contrast agent in 102 ostensibly healthy male runners aged 50-72 years who had completed at least five marathons during the past 3 years and in 102 age-matched control subjects. Predominantly subendocardial regions of LGE typical of myocardial infarction (hereafter, coronary artery disease [CAD] pattern) were distinguished from a predominantly midmyocardial patchy pattern of LGE (hereafter, non-CAD pattern). Marathon runners with LGE underwent repeat cardiac MR imaging and additional adenosine perfusion imaging. Runners were followed up for a mean of 21 months +/- 3 (standard deviation) after initial presentation. The chi(2), Fisher exact, and McNemar exact tests were used for comparisons. Event-free survival rates were estimated with the Kaplan-Meier method, and overall group differences were evaluated with log-rank statistics.

RESULTS: Of the 102 runners, five had a CAD pattern of LGE, and seven had a non-CAD pattern of LGE. The CAD pattern of LGE was located in the territory of the left anterior descending coronary artery more frequently than was the non-CAD pattern ($P = .0027$, Fisher exact test). The prevalence of LGE in runners was higher than that in age-matched control subjects (12% vs 4%; $P = .077$, McNemar exact test). The event-free survival rate was lower in runners with myocardial LGE than in those without myocardial LGE ($P < .0001$, log-rank test).

CONCLUSION: Ostensibly healthy marathon runners have an unexpectedly high rate of myocardial LGE, and this may have diagnostic and prognostic relevance.

MYOCARDIAL FIBROSIS AFTER PROLONGED AND INTENSE EXERCISE

J Appl Physiol 110: 1622–1626, 2011.
First published February 17, 2011; doi:10.1152/japplphysiol.01280.2010.

Diverse patterns of myocardial fibrosis in lifelong, veteran endurance athletes

M. Wilson,¹ R. O'Hanlon,^{2,3} S. Prasad,² A. Deighan,⁴ P. MacMillan,⁵ D. Oxborough,⁶ R. Godfrey,⁷ G. Smith,² A. Maccira,⁸ S. Sharma,⁹ K. George,¹⁰ and G. Whyte¹⁰

N=12 Lifelong Endurance Athletes
Aged 50 to 67 Years
6Athletes Had LGE (1 Probable MI)
LGE Was Associated With:
Years of Training
Number of Marathons
Ultra Endurance Marathons

February 17, 2011; doi:10.1152/japplphysiol.01280.2010.—This study examined the cardiac structure and function of a unique cohort of documented lifelong, competitive endurance veteran athletes (>50 yr). Twelve lifelong veteran male endurance athletes [mean \pm SD (range) age: 56 \pm 6 yr (50–67)], 20 age-matched veteran controls (60 \pm 5 yr, (52–69), and 17 younger male endurance athletes [31 \pm 5 yr (26–40)] without significant comorbidities underwent cardiac magnetic resonance (CMR) imaging to assess cardiac morphology and function, as well as CMR imaging with late gadolinium enhancement (LGEd) to assess myocardial fibrosis. Lifelong veteran athletes had smaller left (LV) and right ventricular (RV) end-diastolic and end-systolic volumes ($P < 0.05$), but maintained LV and RV systolic function compared with young athletes. However, veteran athletes had a significantly larger absolute and indexed LV and RV end-diastolic and systolic volumes, intraventricular septum thickness during diastole, posterior wall thickness during diastole, and LV and RV stroke volumes ($P < 0.05$), together with significantly reduced LV and RV ejection fractions ($P < 0.05$), compared with veteran controls. In six (50%) of the veteran athletes, LGEd of CMR indicated the presence of myocardial fibrosis (4 veteran athletes with LGE of nonspecific cause, 1 probable previous myocarditis, and 1 probable previous silent myocardial infarction). There was no LGE in the age-matched veteran controls or young athletes. The prevalence of LGE in veteran athletes was not associated with age, height, weight, or body surface area ($P > 0.05$), but was significantly associated with the number of years spent training ($P < 0.001$), number of competitive marathons ($P < 0.001$), and ultraendurance (>50 miles) marathons ($P < 0.007$) completed. An unexpectedly high prevalence of myocardial fibrosis (50%) was observed in healthy, asymptomatic, lifelong veteran male athletes, compared with zero cases in age-matched veteran controls and young athletes. These data suggest a link between lifelong endurance exercise and myocardial fibrosis that requires further investigation.

Missouri Medicine

The Journal of the Missouri State Medical Association

www.mssma.org

March/April 2014

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

Marathons In the Long Run Not Heart Healthy

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*

- Total Plaque Volume (200 vs. 126 mm³, p < 0.01)
- Calcified Plaque Volume (84 vs. 44 mm³, p < 0.0001)
- Non - Calcified Plaque Volume (116 vs. 82 mm³, p = 0.04)
- Were Greater in the Athletes

Effect of Prolonged Exercise on Parathyroid Hormone

2 Hrs Moderate-Intensity Cycling
20 Competitive Male Cyclists
Aged 22–45 Yrs

Table 2 Changes in serum PTH, calcium, and hematocrit in response to the 2-hour exercise bout, with and without correction for hemoconcentration (mean \pm SD)

	Before exercise	After exercise uncorrected	After exercise corrected
Serum PTH (pg/mL)	40.6 \pm 15.6	72.8 \pm 24.6*	69.5 \pm 25.5*
Serum calcium (mg/dL)	9.3 \pm 0.3	9.6 \pm 0.3*	8.8 \pm 0.5†
Hematocrit (%)	45.5 \pm 2.6	47.3 \pm 2.2*	

Prolonged exercise should be considered alongside typical symptoms of acute myocardial infarction when evaluating increases in cardiac troponin T

R E Shave, G P Whyte, K George, D C Gaze, P O Collinson

Heart 2005;91:1219–1220. doi: 10.1136/heart.2004.046052

72 runners (22-63 yrs), London Marathon 2002/3

All pre race troponins < 0.01.
>0.05 = ABN

78% > 0.01 µg/l

58% >0.03 µg/l

36% >0.05 µg/l

11% >0.1 µg/l

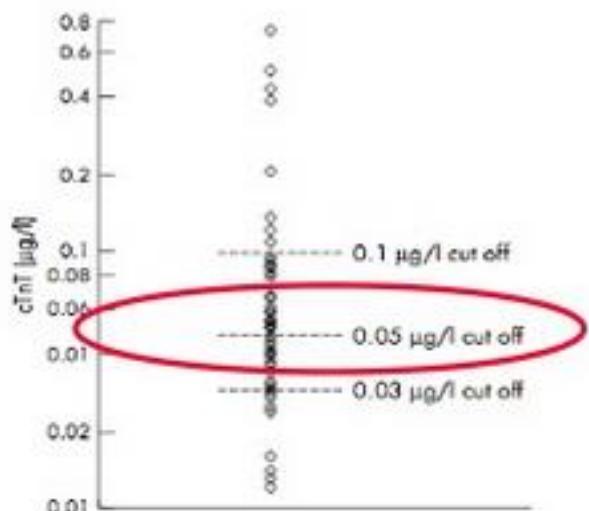
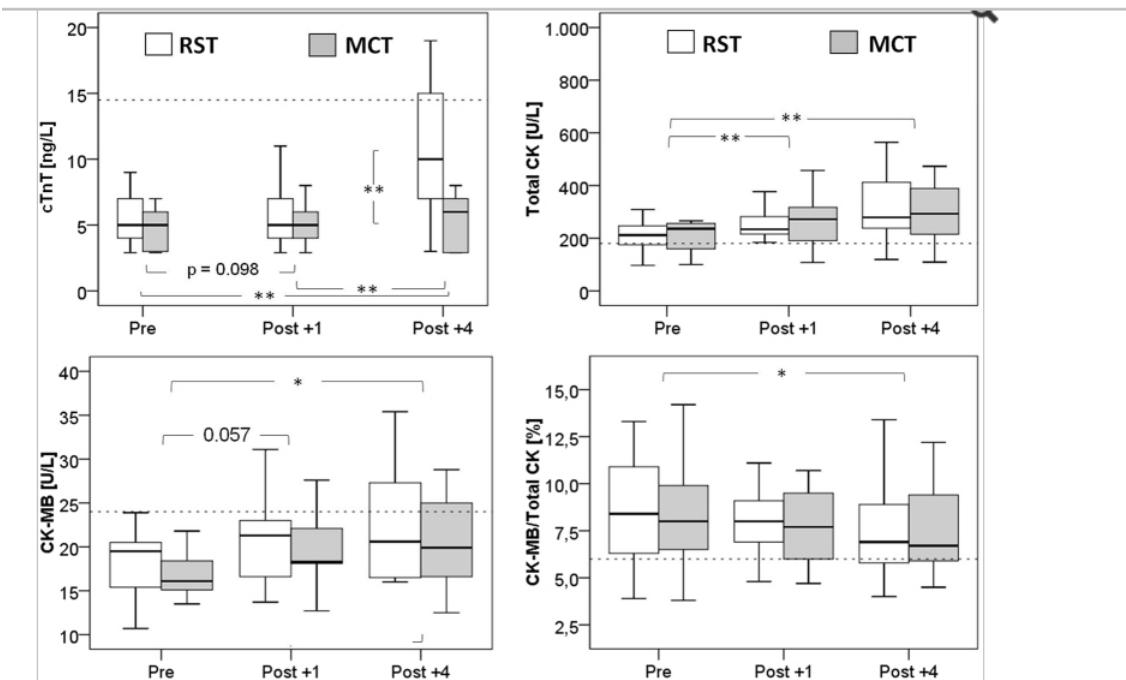


Figure 1 Positive cardiac troponin T (cTnT) samples after the London marathon [scale is log plotted because of the data spread].

Cardiac troponin T and echocardiographic dimensions after repeated sprint vs. moderate intensity continuous exercise in healthy young males

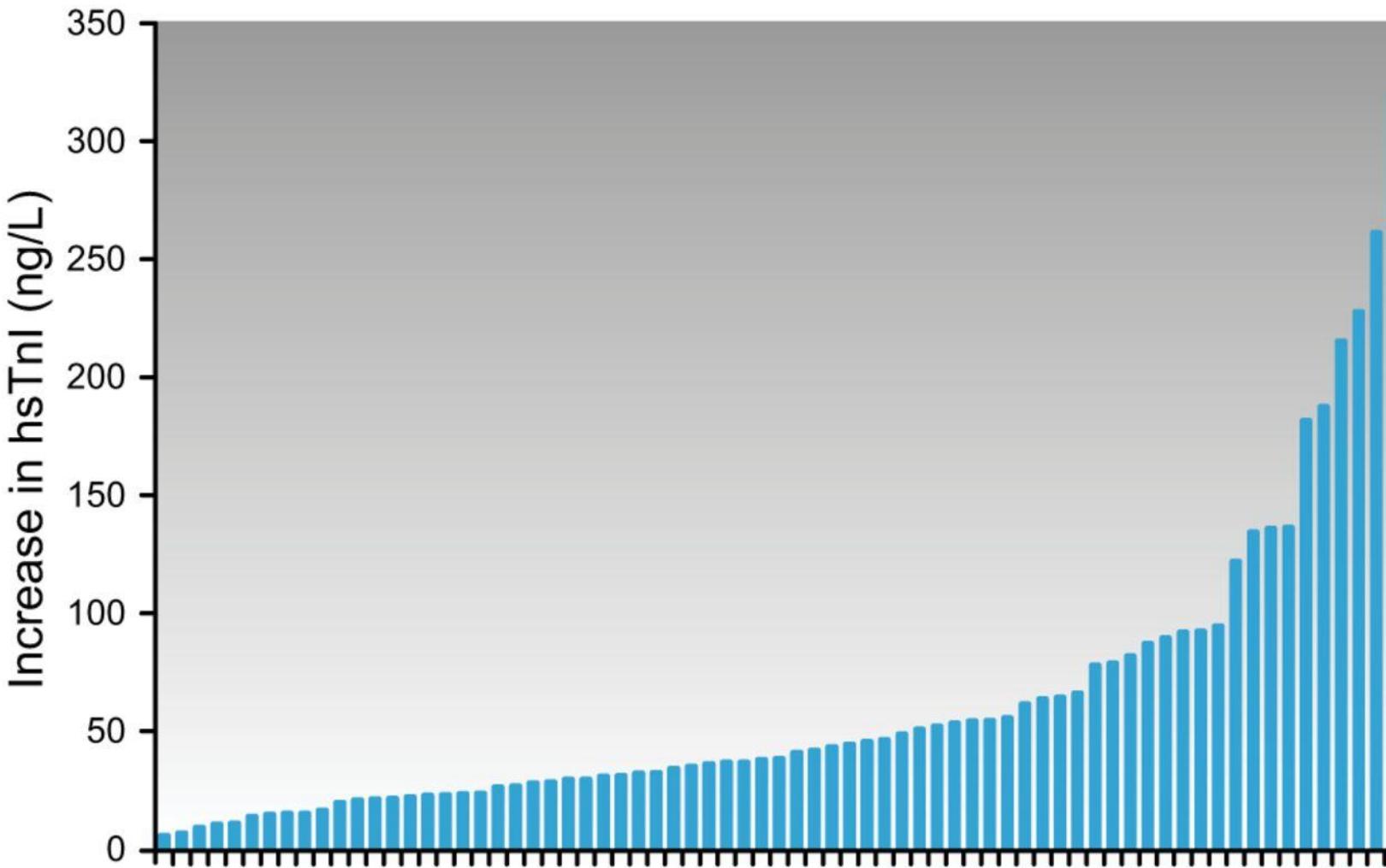
Matthias Weippert,^{*} Dimitar Divchev,^{*} Paul Schmidt, Hannes Gettel, Antina Neugebauer, Kristin Behrens



Biomarker concentration before (PRE), 1 (POST + 1) and 4 hours (POST + 4) after cessation of the different exercise protocols; ^{*} p < 0.05, ^{**} p < 0.001, dotted line = reference limit; MCT, moderate intensity continuous training; RST, repeated sprint interval training; cTnT, cardiac troponin T; CK, creatine kinase; CK-MB, CK muscle-brain isoform; %CK-MB, ratio of CK-MB: total CK in %. N = 13

	Baseline	0.5 h after RST	0.5 h after MCT	p
	Mean ± SD	Mean ± SD	Mean ± SD	
LVEDD [mm]	53.6 ± 2.8	53.8 ± 2.0	52.7 ± 2.3	0.658
FS [%]	37.6 ± 4.1	40.6 ± 2.8	37.5 ± 5.5	0.471
LVEF [%]	64.3 ± 9.9	65.3 ± 9.8	65.9 ± 4.0	0.647
RVEDD [mm]	38.2 ± 2.7	37.6 ± 2.1	37.4 ± 2.7	0.838
AoV _{max} [cm/sec]	178.7 ± 15.7	176.9 ± 14.1	176.8 ± 17.9	0.767
PVV _{max} [cm/sec]	111.6 ± 20.3	106.8 ± 13.0	102.8 ± 13.7	0.007
E [cm/sec]	83.7 ± 20.8	70.4 ± 16.6	85.3 ± 10.5	0.470
E' [cm/sec]	19.1 ± 3.8	18.5 ± 4.1	18.2 ± 3.5	0.624
E/E'	4.4 ± 1.0	3.9 ± 0.7	4.8 ± 0.8	0.661
mPCWP [mmHg]	7.4 ± 1.2	6.7 ± 0.8	7.9 ± 1.0	0.673
PA _{mean} [mmHg]	9.6 ± 1.1	8.8 ± 0.9	9.9 ± 1.1	0.167
PA _{dia} [mmHg]	7.6 ± 1.0	6.6 ± 0.8	5.3 ± 10.5	0.193
TAPSE [mm]	24.8 ± 1.6	22.3 ± 2.4	23.7 ± 1.4	0.753
TASV [cm/sec]	15.7 ± 2.3	13.5 ± 2.8	14.6 ± 1.6	0.646

**Exercise-induced increases in high-sensitive cardiac troponin I (hsTnI) levels in participants in
the 2011 Boston marathon (n = 71).**



Thijs M. H. Eijsvogels et al. Physiol Rev 2016;96:99-125

Physiological Reviews

CORRESPONDENCE

**Research
Correspondence**

**Cardiac Troponin T Release Is Stimulated
by Endurance Exercise in Healthy Humans**

- 9 Trained Men
- Ran a Treadmill Marathon
- TnT Measured Before & q 30 Minutes
- All Increased TnT Between 1-2 Hours
- All Normal Within 1 Hour of Finish
- All But 1 Had A Second Late Peak

Explanation For Elevated “Cardiac Enzymes”

- Runners Are Constantly Injuring Skeletal Muscle
- Which is Repaired Using Satellite Cells
- Satellite Cells are Pleuripotential & Can Make “Cardiac Enzymes”
- Injured Satellite Cells Release CKMB After the Race
- Case Dismissed !!!!

Position Paper

ESC Study Group of Sports Cardiology Position Paper on adverse cardiovascular effects of doping in athletes

Asterios Deligiannis^a, Hans Björnstad^b, Francois Carre^c, Hein Heidbüchel^d, Evangelia Kouidi^a, Nicole M. Panhuyzen-Goedkoop^e, Fabio Pigozzi^f, Wilhelm Schänzer^g and Luc Vanhees^h on behalf of the ESC Study Group of Sports Cardiology



Int J Legal Med (2007) 121: 48–53
DOI 10.1007/s00414-005-0055-9

CASE REPORT

Vittorio Fineschi · Irene Riezzo · Fabio Centini ·
Enrico Silingardi · Manuela Licata ·
Giovanni Beduschi · Steven B. Karch

Sudden cardiac death during anabolic steroid abuse: morphologic and toxicologic findings in two fatal cases of bodybuilders

Received: 6 January 2005 / Accepted: 26 September 2005 / Published online: 15 November 2005
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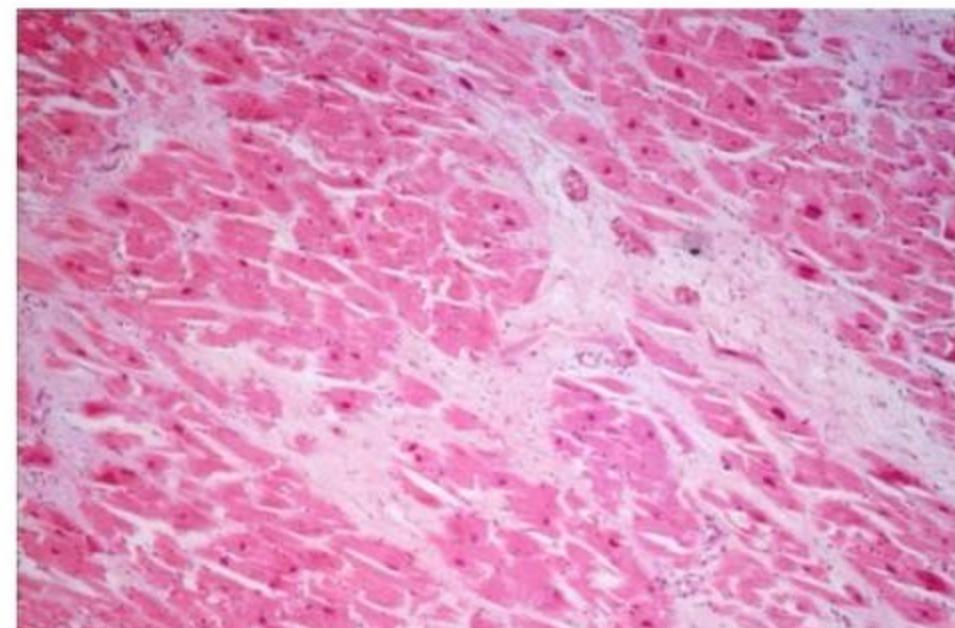
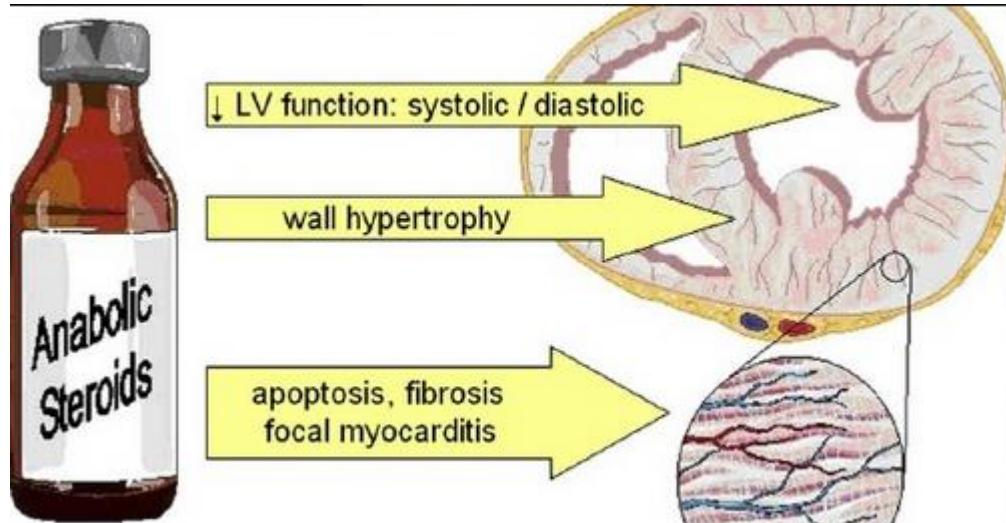


Fig. 2 Scar fibrosis (H & E ×100)

An overview of potential deleterious cardiac effects of the performance of acute and chronic endurance exercise.

Can lifelong endurance exercise hurt the heart?



Acute cardiovascular risks

- ↑ risk for sudden cardiac death
- ↑ risk for acute myocardial infarction
- ↓ ventricular function of the heart

Evidence of acute myocardial injury

- ↑ CK and CK-MB concentrations
- ↑ cardiac troponin concentrations
- ↑ BNP and NT-proBNP concentrations

Cardiac remodeling

- ↑ dimensions of right and left ventricle
- ↑ dimensions of right and left atria
- ↑ wall thickness

Potential cardiac maladaptations

- = / ↓ Carotid intima media thickening
- ↑ ↓ Coronary artery calcification
- ↑ prevalence of myocardial fibrosis
- ↑ risk for atrial fibrillation
- ↑ risk for bradycardia
- ↑ aortic diameter
- ↑ progression of ARVC

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THE RED LINE: The exercise intensity, exercise endurance, time and type of exercise are all variables that cause different effects within the body systems of each athlete.
But, other reasons ?

Symposium: Exercise and the Heart

Exercise and the Heart: Can You Have Too Much of a Good Thing?

KEITH GEORGE¹, ROB SHAVE², DARREN WARBURTON³, JÜRGEN SCHARHAG⁴, and GREG WHYTE¹

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**'The right amount of
nourishment and
exercise, not too
much, not too little, is
the safest way to
health'**

*HIPPOCRATES (460 -377 BC)
Father of Medicine
Contemporary of Pheidippides*