

Exercise and the heart: Caring for athletes and sports participants

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From the outside.....

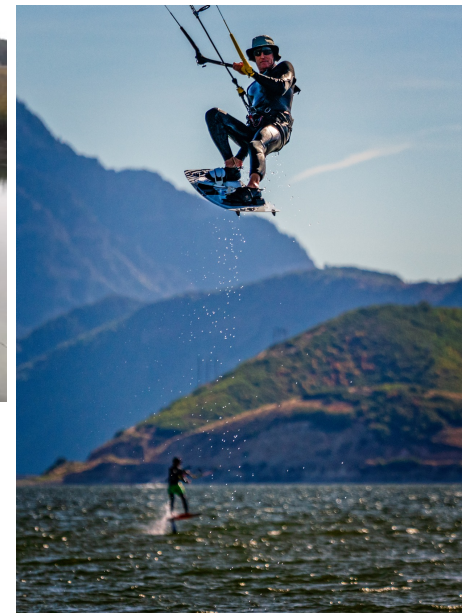


From the inside.....



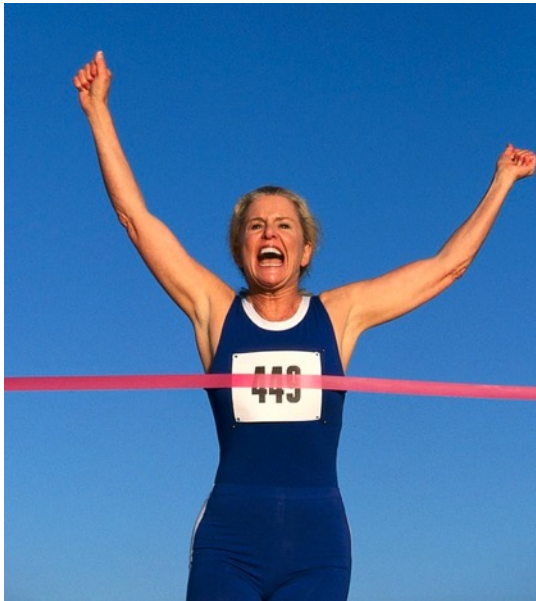
No financial conflicts or disclosures

However, I have a bias....



The problem

- Exercise *is* REALLY good for you
 - Don't over diagnose heart disease in athletes
 - Don't be too restrictive about allowing participation
- Exercise *can be* dangerous (rarely)
 - Don't miss real heart disease in athletes



The first marathon and...

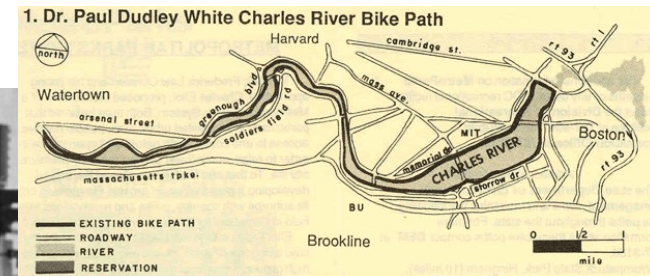
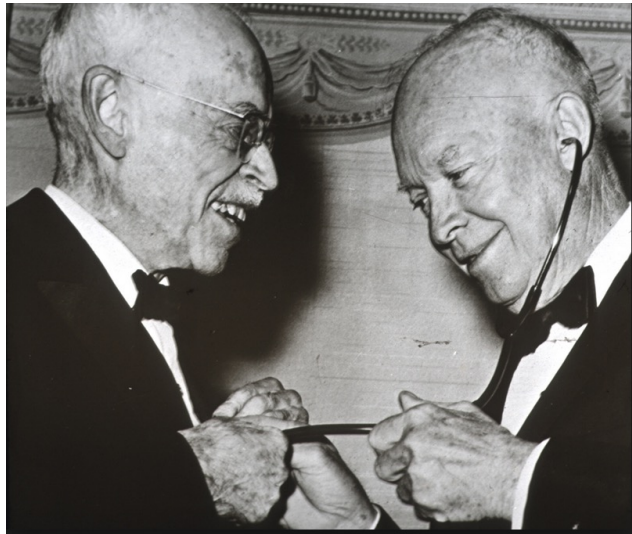


Pheidippides as he gave word of the Greek victory over Persia at the Battle of Marathon to the people of Athens. Luc-Olivier Merson, 1869

...the first sports related sudden death

Paul Dudley White

Following the Eisenhower case, White wrote an article on preventing heart attacks, which appeared on the front page of *The New York Times* and other newspapers. The article was said to have reached an audience of over 50 million readers. White presented a philosophy for a healthy way of life that included three main elements: optimism, regular physical activity and work. He was a staunch proponent of vigorous exercise (bicycling was his favorite), which was somewhat contrary to the medical opinion at the time that physical exertion could damage the heart.



Goals

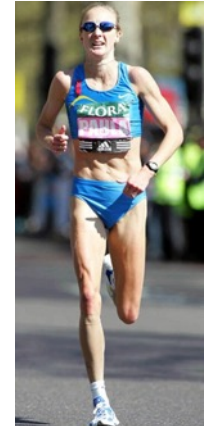
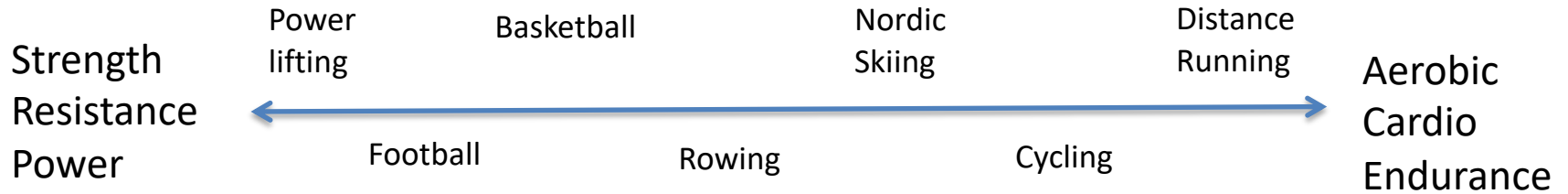
- Know the expected changes in cardiac structure and function in athletes
- Recognize that there is considerable overlap between normal findings in athletes and patients with heart disease
- There are *NO* definitive echo findings that exclude or confirm all worrisome conditions
- Make rational, informed, shared decisions about risks and benefits of sports/exercise in people with suspected or known heart disease
- Review return to play after covid infection
- Understand controversy over whether “too much” exercise has detrimental CV effects

Exercise-induced cardiac remodeling

- *Key point 1: The degree of cardiac chamber enlargement is related to the type, duration and intensity of training and is typically reversible upon detraining. Sex, race and age all may influence the degree of cardiac adaptation in trained individuals.*

*Athletes may have large body size that needs to be taken into account

Type of exercise



Thicker
walls

Mainly left ventricle

Chamber
dilation

All 4 chambers and
major vessels

Key point 2: Athletes often have symmetrical enlargement of all 4 chambers of the heart. There may also be increased left ventricular wall thickness, most often with normal LV geometry or eccentric hypertrophy.

Case 1

58 y.o. male lifelong recreational athlete, predominantly aerobic (running, cycling, rowing, nordic skiing, weights, etc)

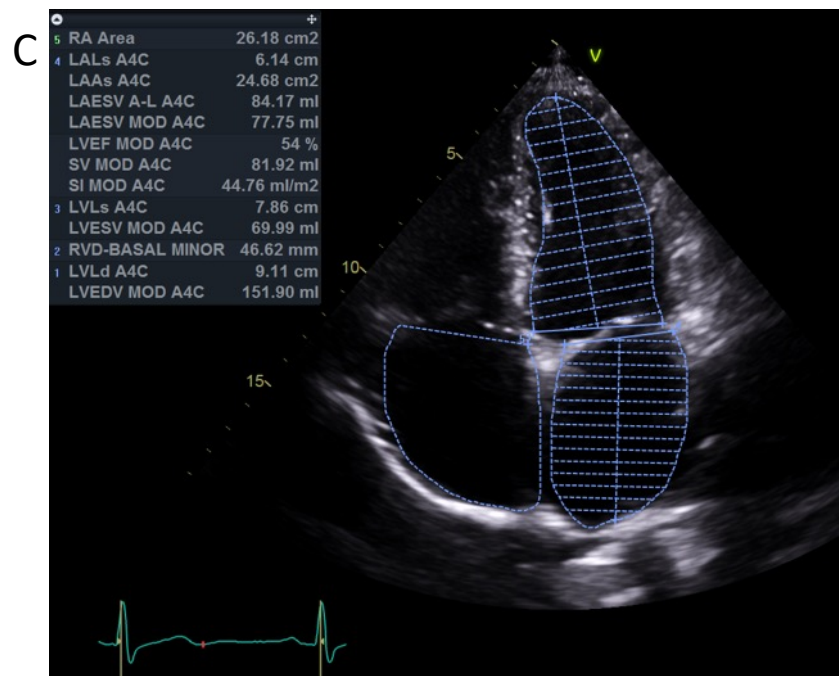
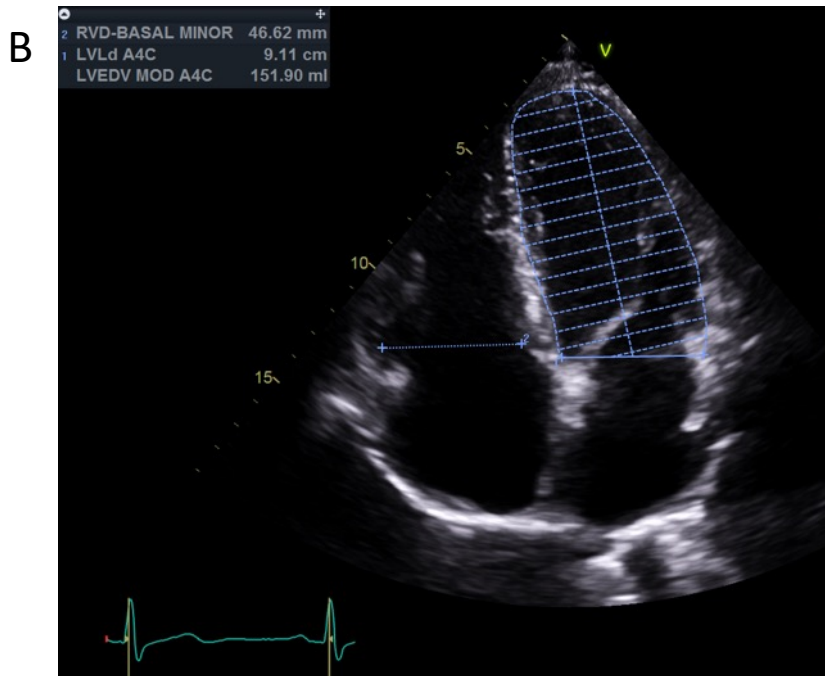
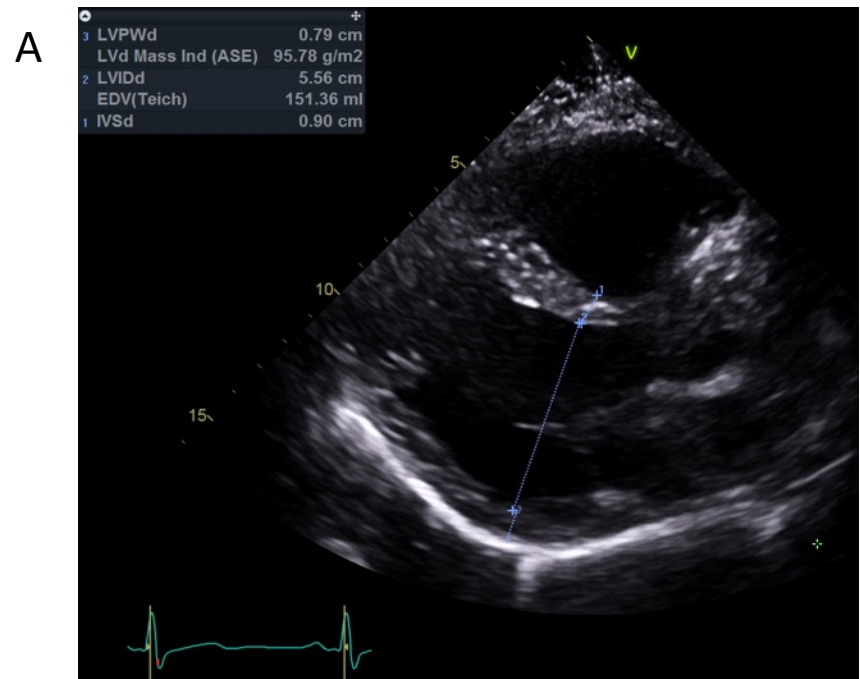
5' 9", 150 lbs, BMI 23

12-15 hours per week moderate to high intensity for > 30 years

Not as fast as he used to be. Concern for possible cardiac condition?

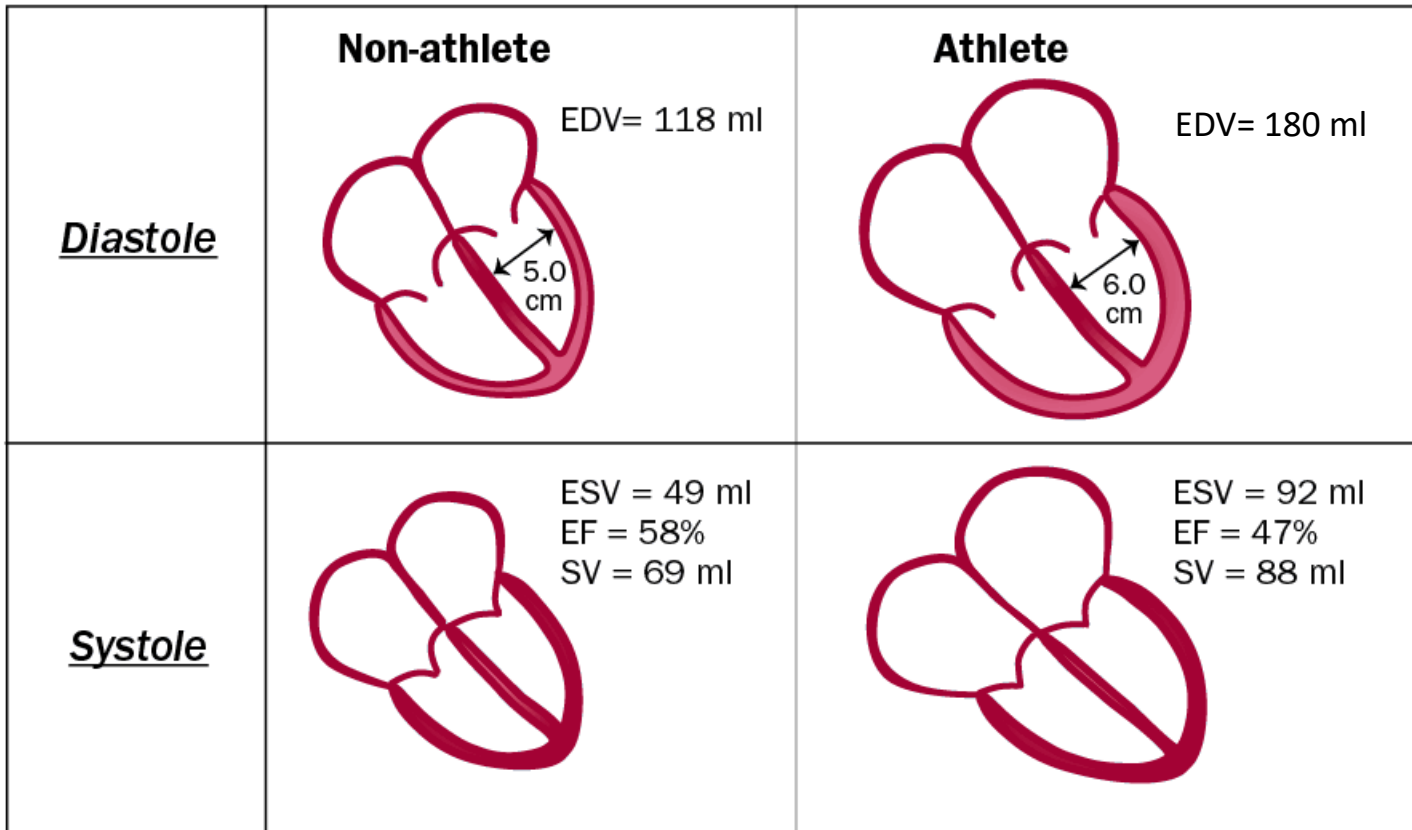
Case 1, Figure 1.

- A. Parasternal long axis view. Calculated **end-diastolic volume = 151 ml** (83.8 ml/m^2).
- B. Apical 4 chamber view. Calculated end-diastolic volume = 151 ml. RV basal diameter 4.6 cm.
- C. Apical 4 chamber view (systole). Calculated LV ejection fraction = **54%**. **LA volume = 84 ml** (44 ml/m^2). RA = 47 ml/m^2 .



Key point 3: The resting left ventricular ejection fraction is usually normal, but may be mildly reduced in healthy athletes. This is due to the presence of cavity dilation, which allows for a normal stroke volume at a lower ejection fraction.

	Rest					
	LVID (cm)	EDV (ml)	HR (bpm)	CO (L/min)	SV (ml)	EF (%)
Non-athlete	5.0	118	70	4.8	69	58
Athlete	6.0	180	55	4.8	88	47



Exercise, Diet, and the Heart

Serial Left Ventricular Adaptations in World-Class Professional Cyclists

Implications for Disease Screening and Follow-Up

Eric Abergel, MD,* Gilles Chatellier, MD,† Albert A. Hagege, MD, PhD,* Agnes Oblak, MD,*
Ales Linhart, MD,* Alain Ducardonnet, MD,‡ Joël Menard, MD, PhD§

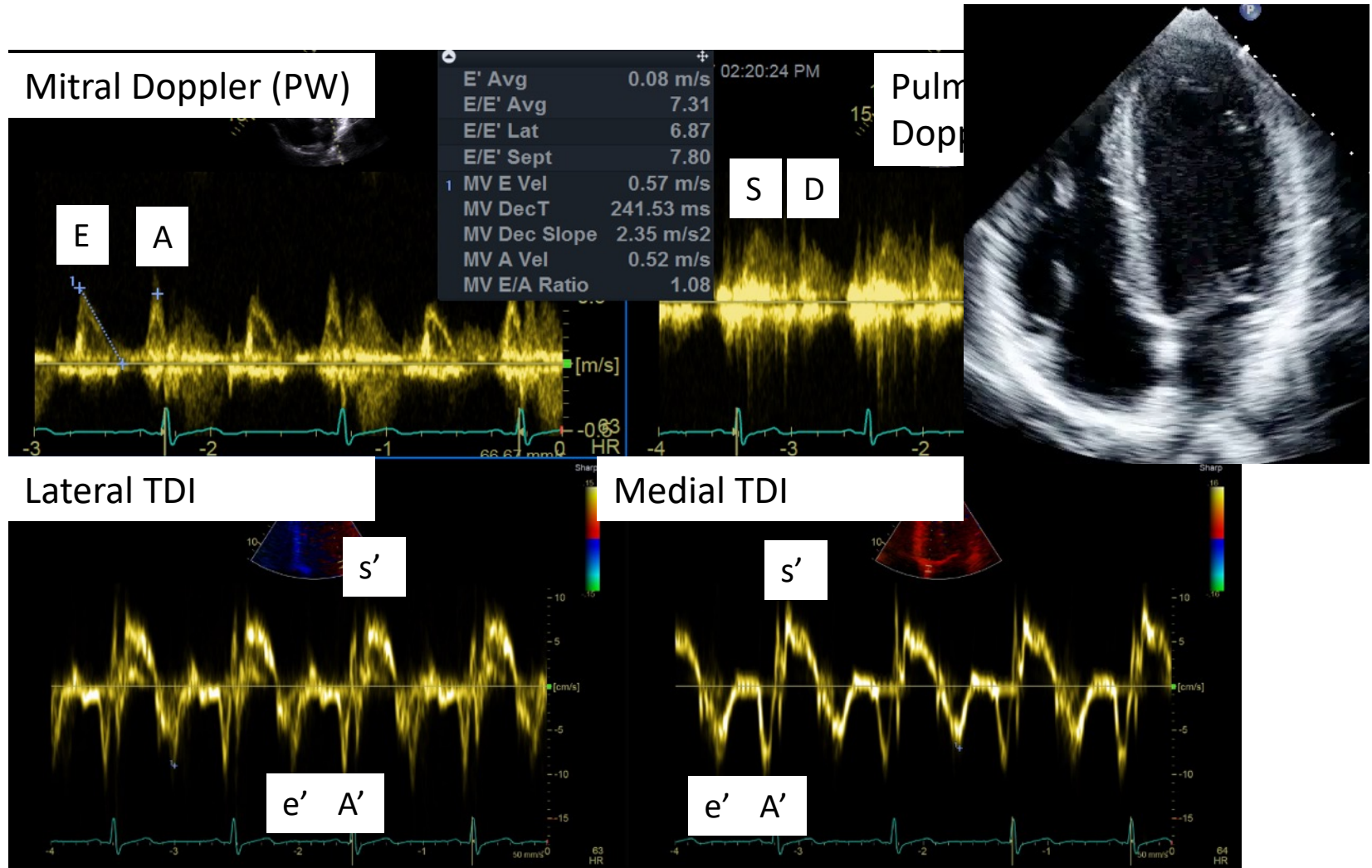
Paris, France

280 former Tour de France cyclists

- **50% had LVIDd > 6.0 cm**
- **11% had EF < 52%**

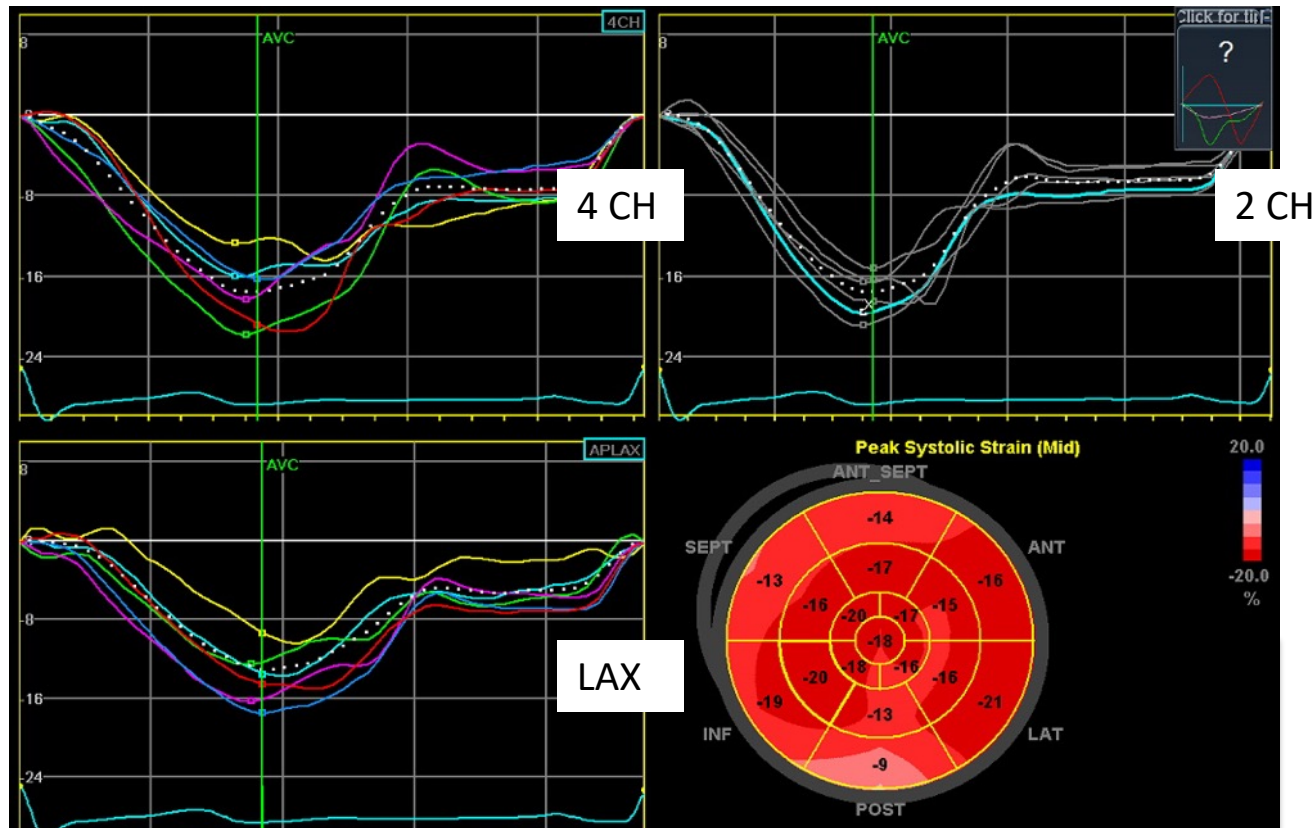
Key point 4: Measures of resting left ventricular function based on tissue Doppler velocities are generally in the normal range, but strain may be lower in athletes than untrained subjects (for the same reason that EF is low).

Case 1, 58 y.o. lifelong athlete concerned about decline in athletic performance



Lateral e' 10 cm/s, Septal e' 7 cm/s. E/e' 7.3. Normal for age. Supports diagnosis of athletes heart.

Case 1, 58 y.o. lifelong athlete concerned about decline in athletic performance



Global Longitudinal Strain -17%

Strain = (resting length – contracted length) / resting length

If amount of shortening is the same, but resting length is longer (dilated LV), then strain will be lower. Same reasoning as the low EF in athletes.

Patterns of Left Ventricular Longitudinal Strain and Strain Rate in Olympic Athletes

Stefano Caselli, MD, PhD, FASE, Dalma Montesanti, MD, Camillo Autore, MD, FESC, Fernando M. Di Paolo, MD, Cataldo Pisicchio, MD, Maria Rosaria Squeo, MD, Beatrice Musumeci, MD, Antonio Spataro, MD, Natesa G. Pandian, MD, and Antonio Pelliccia, MD, FESC, *Rome, Italy; and Boston, Massachusetts*

JASE 2015;28:245-53

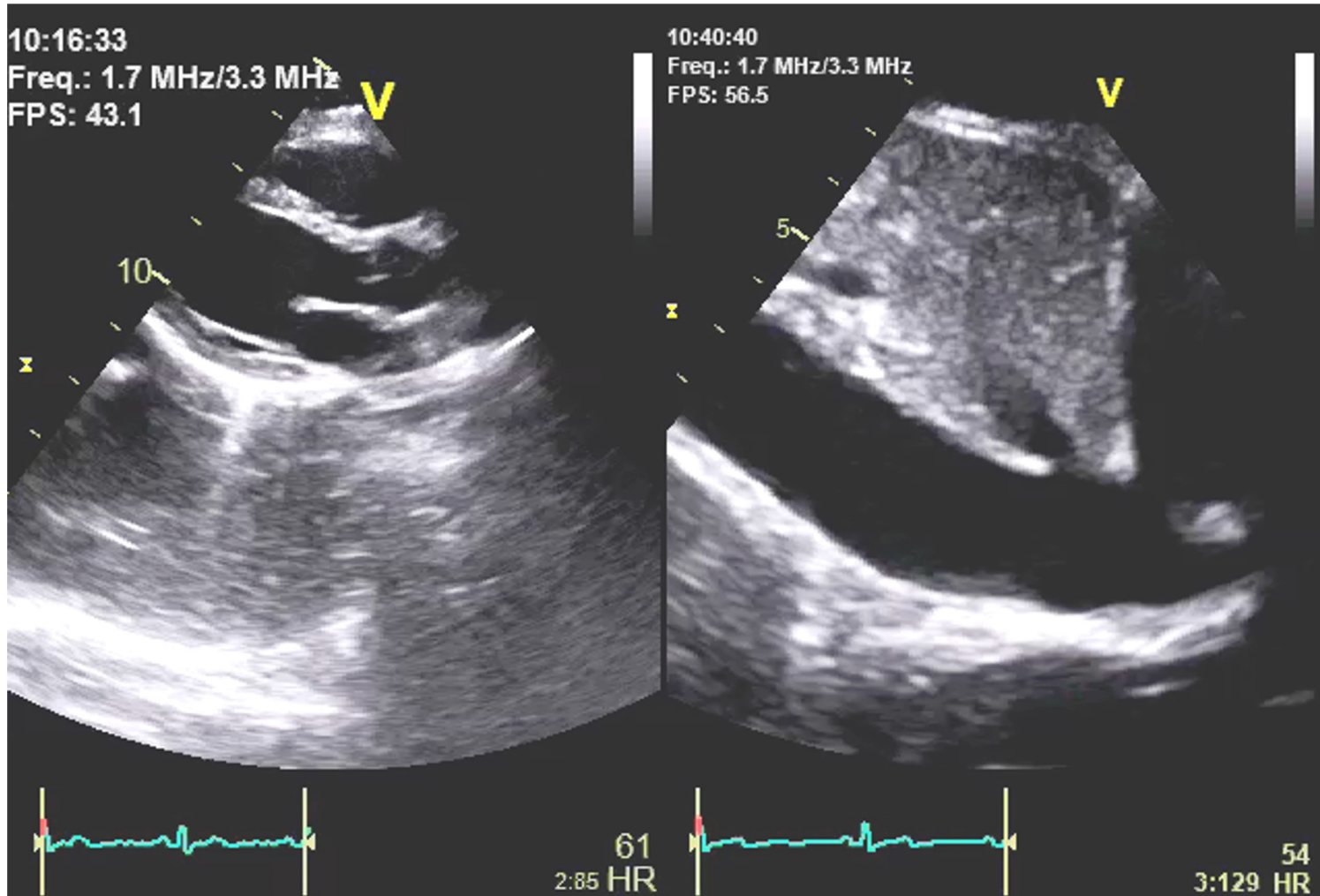
200 Olympic athletes (various disciplines), 50 controls

Global longitudinal strain was normal (-15 to -22%) in athletes, although *lower than controls* (-18.1% vs. -19.4%)

Exercise and veins...



“My legs look tired.” – Pawal Poljanski (Tour de France rider)



Key point 5: The inferior may vena cava may be dilated and relatively noncollapsible in trained athletes, even though central venous pressure is normal.

Unexpected death during sports in young athletes (< 35 years)



Reggie Lewis 1987-1993

NBA star

Collapsed during playoff game

HCM vs. neurocardiogenic syncope

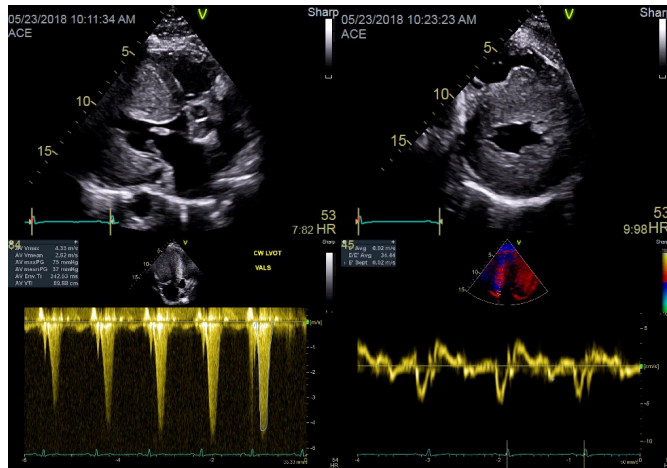
Public controversy between treating cardiologists

Died after collapsing while shooting baskets (noncompetitive)

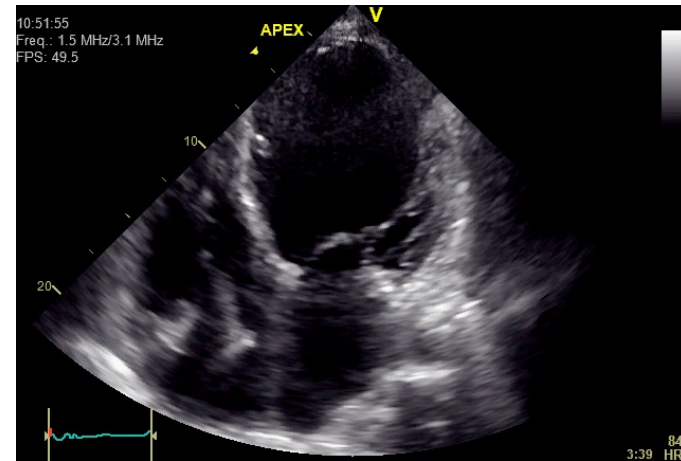
Cause of death?

Key point 7: Distinction between athlete's heart and various pathological conditions, particularly, hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy and dilated cardiomyopathy can be challenging (esp mild forms). Use of tissue Doppler and strain imaging along with MRI may be helpful in distinguishing between these entities. Unfortunately, there are no absolute imaging criteria for ruling in or ruling out these diseases.

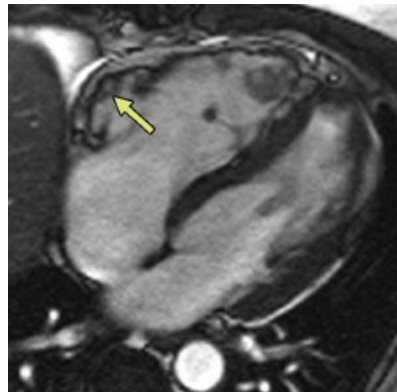
HCM



DCM

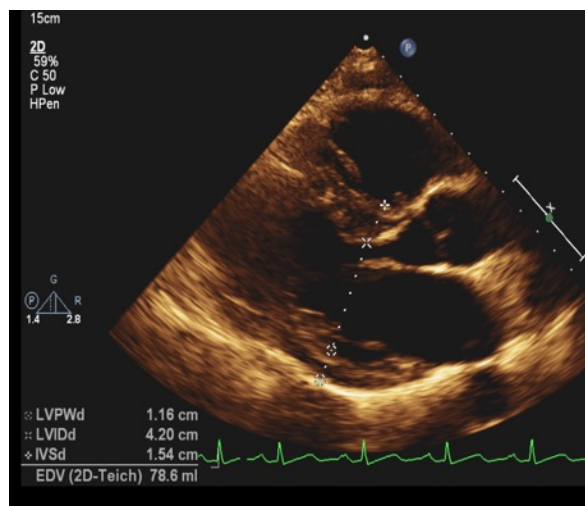


ARVC

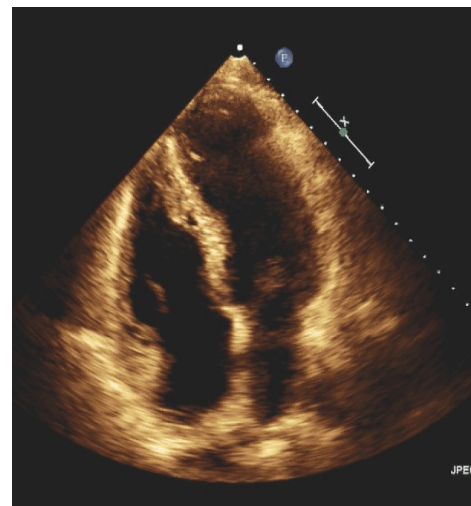


Case 4: 35 y.o. competitive triathlete and ultradistance runner with family history of HCM (father diagnosed after having syncope at end of Boston marathon, age 56)

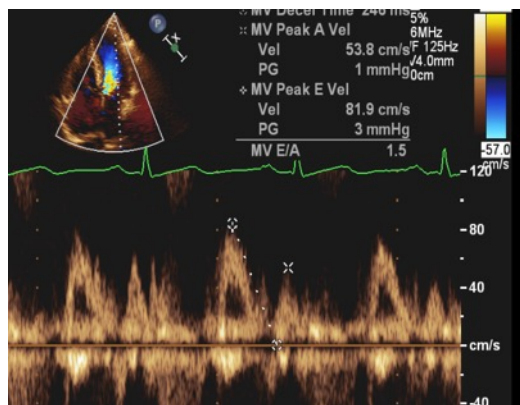
Parasternal long axis view



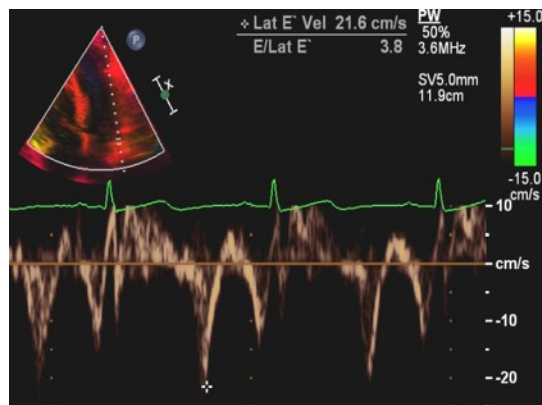
Video 1: apical 4 chamber view



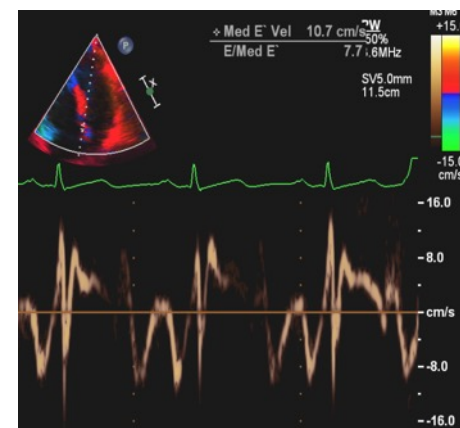
Mildly increased septal thickness (1.4-1.5 cm). No SAM or LVOT obstruction



A) Mitral Doppler



B) Lateral TDI

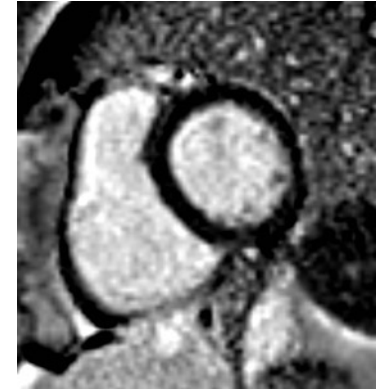
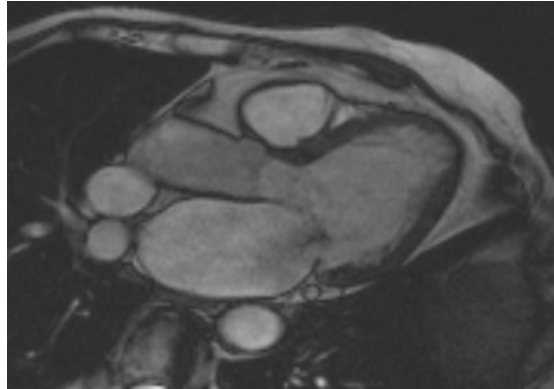


C) Medial TDI

Case 4: 35 y.o. competitive triathlete and ultradistance runner with family history of HCM

cMRI: LV wall thickness 14 mm

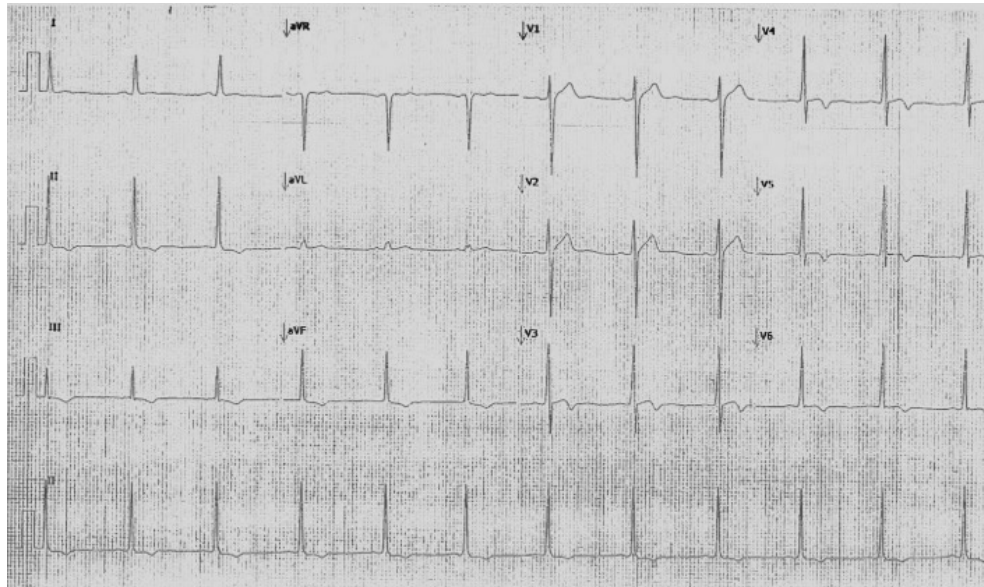
No late gadolinium enhancement



- Genotyped father: Myosin binding protein C mutation (pathogenic)
- Genotyped patient: Myosin binding protein C mutation (heterozygous)
- Can he continue to compete or exercise? Does he need an ICD?
- Recommend retiring from competitive racing. Continued moderate exercise, but concerned about long (remote) runs and swimming where risk of arrhythmias probably higher. Need long term monitoring due to late onset of phenotype in father.

18 y.o. Division 1 College Football player

- Syncopal episode after a game senior year of HS (dehydration)
- 6'3", 360 lbs AA male
- Defensive tackle (lineman)
- Hx HTN (not taking meds), asthma
- Denies CP, SOB, exercise limitations, palpitations, + snoring
- FH negative for cardiomyopathy or sudden death
- Exam unremarkable except for BP 160/86



Refined Seattle ECG criteria for black and white athletes

Refined Criteria Training Related Normal Variants *Not Warranting Further Investigation**

- Sinus bradycardia
- First-degree AV block
- Incomplete RBBB
- Early repolarisation
- Isolated QRS voltage criteria for LVH

Refined Criteria Borderline Variants *Potentially Warranting Further Investigation*

- Left atrial enlargement
- Right atrial enlargement
- Left axis deviation
- Right axis deviation
- Right ventricular hypertrophy
- TWI up to V4 in BA[†]

Refined Criteria Training Unrelated Changes *Warranting Further Investigation*

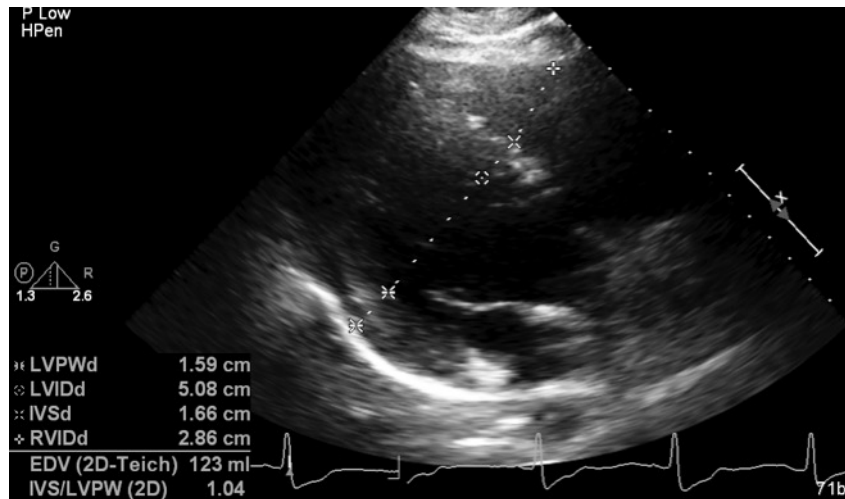
- ST-segment depression
- Pathological Q-waves
- Ventricular pre-excitation
- TWI beyond V1 in WAs
beyond V4 in BAs
- Complete LBBB or RBBB
- QTc ≥ 470 ms in males
 ≥ 480 ms in females
- Brugada-like ER
- Atrial or vent. arrhythmias
- ≥ 2 PVCs per 10 sec tracing

*If present in ISOLATION**

If TWO OR MORE present

Refined criteria increased specificity from 40% 84% in BA's
Sensitivity for HCM reportedly 98%

Echocardiogram



EF 60%

LV mass 374 g, LVMI 129 g/m², RWT 0.65

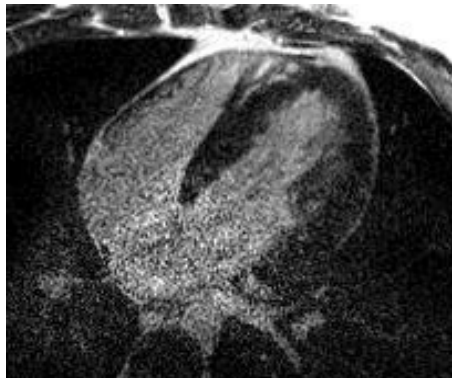
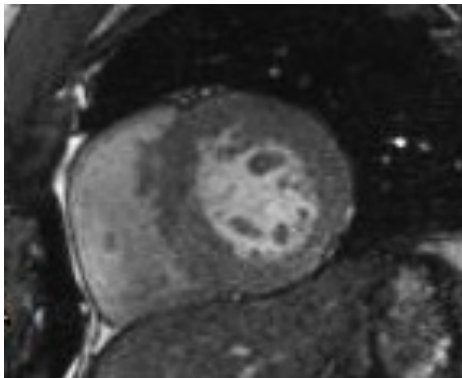
Cardiac MRI

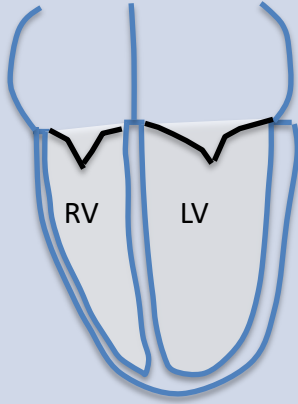
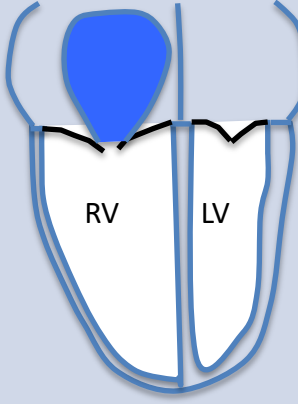
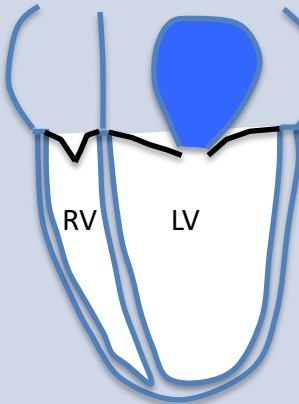
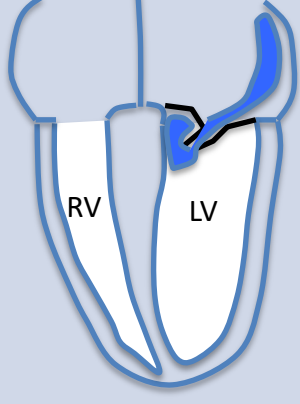
SSFP SAX LV

Wall thickness 15 mm

Post Gadolinium IR sequence

No delayed enhancement

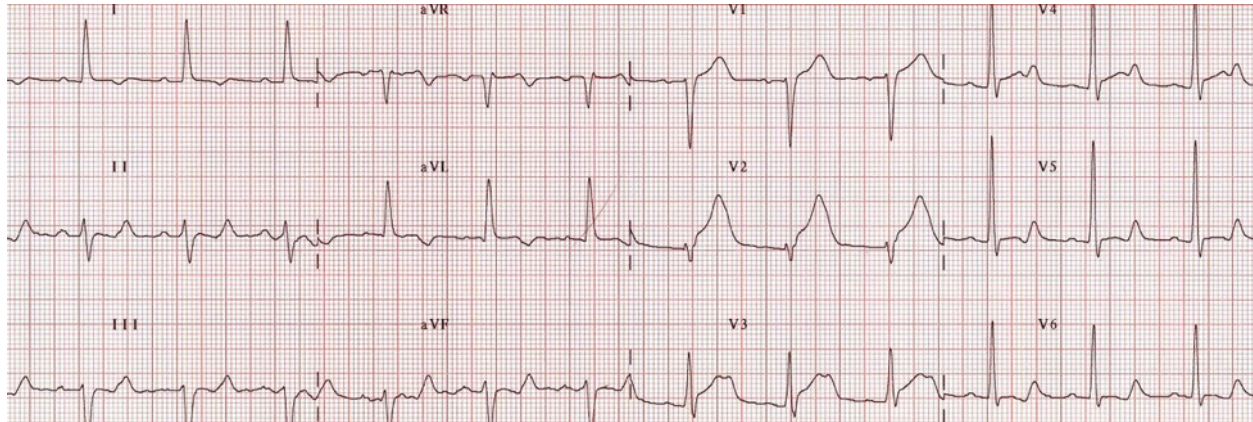


	EICR	ARVC	DCM or ICM	HCM
				
Echo findings	<p>Symmetrical 4 chamber enlargement (usually mild)</p> <p>LV wall thickness < 14 mm</p> <p>Normal or slightly reduced LV EF (> 45%)</p> <p>Normal RV function</p> <p>Normal tissue Doppler velocities</p> <p>Normal GLS</p> <p>PASP < 40 mmHg</p>	<p>RV disproportionately enlarged</p> <p>RV global dysfunction</p> <p>RV regional dysfunction</p> <p>RV aneurysms</p> <p>PASP < 40 mmHg</p>	<p>≥ Moderate LVE (6.0 cm)</p> <p>≥ Moderate LAE</p> <p>LV disproportionately enlarged</p> <p>LV dysfunction (EF < 45%)</p> <p>LV regional dysfunction</p> <p>Decreased tissue Doppler velocities</p> <p>Decreased GLS</p> <p>Significant MR</p> <p>PASP > 40 mmHg</p>	<p>LV wall thickness > 14 mm</p> <p>Asymmetric hypertrophy (especially septal)</p> <p>Systolic anterior motion of mitral valve</p> <p>≥ Moderate LAE</p> <p>Decreased tissue Doppler velocities</p> <p>Decreased GLS</p> <p>Significant MR</p> <p>PASP > 40 mmHg</p>
Other	<p>History of aerobic exercise training</p> <p>Good exercise capacity</p> <p>ECG (bradycardia, incomplete RBBB, voltage criteria LVH, early repolarization, TWI V2-4)</p>	<p>Family history</p> <p>Syncope</p> <p>ECG (TWI V1-3, epsilon wave)</p>	<p>Family history</p> <p>Coronary Risk Factors</p> <p>Chest pain</p> <p>ECG (Q waves, ischemic changes)</p> <p>LGE on MRI</p>	<p>Family history</p> <p>ECG (LVH, deep TWI, narrow Q waves lateral leads)</p> <p>LGE on MRI</p>

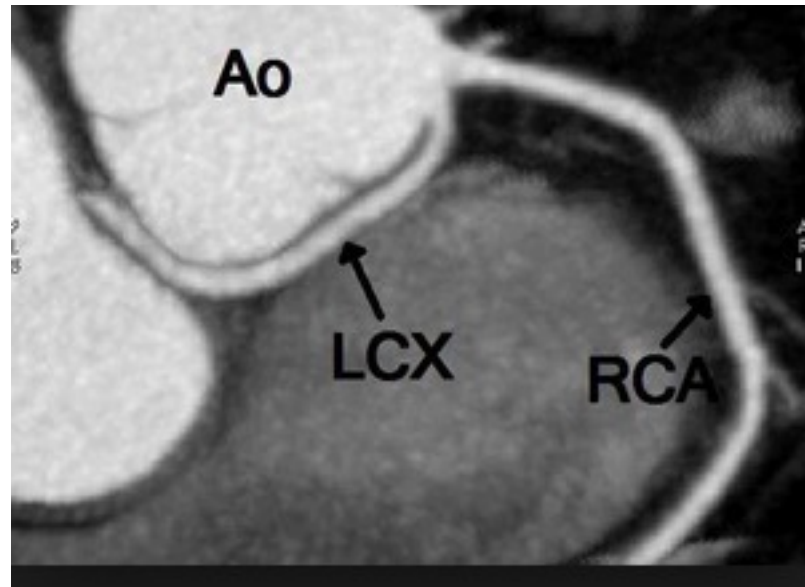
EICR = Exercise induced cardiac remodeling; ARVC = arrhythmogenic right ventricular cardiomyopathy; DCM = dilated cardiomyopathy; ICM = ischemic cardiomyopathy; HCM = hypertrophic cardiomyopathy; LVE = left ventricular enlargement; LAE = left atrial enlargement; GLS = global longitudinal strain; TWI = T wave inversions; LGE = late gadolinium enhancement; MR = mitral regurgitation;

Things we can't diagnose with echo

Long QT



Anomalous
coronary artery



Return-to-Play for Athletes With Long QT Syndrome or Genetic Heart Diseases Predisposing to Sudden Death

Original Investigation

Kathryn E. Tobert, J. Martijn Bos, Ramin Garmany, and Michael J. Ackerman

J Am Coll Cardiol. Jul 27, 2021. Epublished DOI: 10.1016/j.jacc.2021.04.026

Editorial Comment: [Toward a Long and Happy Life of a Patient With Genetic Heart Disease*](#)

There were 672 athletes with GHD overall including 494 athletes with LQTS (231 female athletes [46.8%]; mean age at diagnosis 14.8 ± 10.5 years; mean follow-up 4.2 ± 4.8 years) who were given return-to-play approval. Overall, 79 of 494 athletes with LQTS (16.0%) were symptomatic before diagnosis, and 58 (11.7%) had an implantable cardioverter-defibrillator. ***In 2,056 combined years of follow-up, there was no GHD–sports associated mortality.***

Instead, 29 patients (5.9%) had ≥ 1 nonlethal, LQTS-associated breakthrough cardiac event. Of those, 15 (3.0%) were athletes at the time of the breakthrough cardiac event, with ***3 (0.6%) experiencing a sports-related breakthrough cardiac event, and 12 (2.4%) a non–sports-related event. Overall, the event rate was 1.16 nonlethal events per 100 athlete-years of follow-up.***

Conclusions

This 20-year single center experience ***challenges the status quo of disqualification*** for all athletes with LQTS and provides additional observational evidence, albeit from a single center, in support of ***the more contemporary SDM approaches*** to this complex issue.

For both men and women, sports participation protect against hopelessness and suicidality.

ORIGINAL ARTICLE

Safety of Sports for Young Patients With Implantable Cardioverter-Defibrillators

Long-Term Results of the Multinational ICD Sports Registry

Thirty-five (27%) athletes received at least 1 shock with overall 49 shocks. There were 29 appropriate shocks occurring in 18 individuals, of which, *6 appropriate shocks in 4 individuals occurred during competition or practice*

there were no serious adverse sequelae. Lead malfunction rates were similar to previously reported in unselected pediatric ICD populations

129 young athletes (mean age 16) participating in competitive or dangerous sports (12)

LQT 49, HCM 30, CHD 16

ORIGINAL ARTICLE

Sudden Cardiac Arrest during Participation in Competitive Sports

Cameron H. Landry, M.D., Katherine S. Allan, Ph.D.,
Kim A. Connelly, M.B., B.S., Ph.D., Kris Cunningham, M.D., Ph.D.,
Laurie J. Morrison, M.D., and Paul Dorian, M.D., for the Rescu Investigators*

Rescu Epistry cardiac arrest data base (Canada) 2009-2014

All out of hospital arrests during sports age 12-45

Adjudication based on multiple sources

18.5 million person years of observation, 74 sudden deaths

16 competitive sports, 58 noncompetitive sports

0.76 per 100,000 athlete years

43.8% survival to discharge

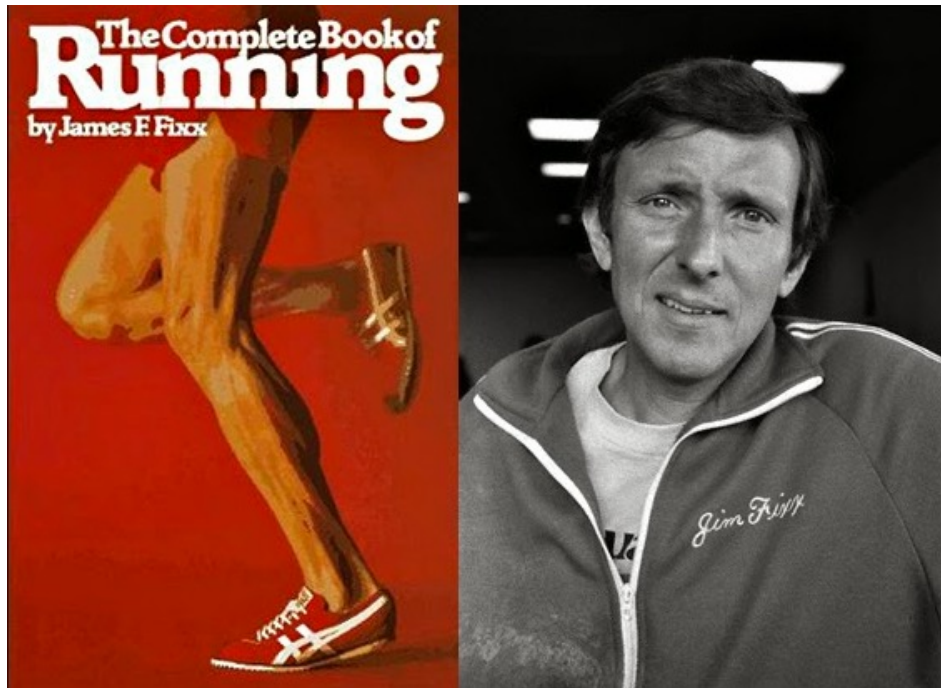
Competitive athletes: 2 HCM, none due to ARVD

3 cases potentially identifiable with pre-participation screening

Table 4. Causes of Sudden Cardiac Arrest among Competitive and Noncompetitive Athletes, According to Age Group.

Variable	Age Group			
	12–17 yr	18–34 yr	35–45 yr	All
Competitive				
No. of athletes	4	9	3	16
Percent of athletes who survived	50.0	44.4	33.3	43.8
Diagnosis				
Ischemic*	0	0	3	3
Primary arrhythmic	0	6	0	6
Structural†‡	2	3	0	5
Commotio cordis	2	0	0	2
Noncompetitive				
No. of athletes	9	18	31	58
Percent of athletes who survived	66.7	50.0	35.5	44.8
Diagnosis				
Ischemic*	0	5	21	26
Primary arrhythmic	4	5	0	9
Unknown	2	2	0	4
Structural‡§	3	6	8	17
Other¶	0	0	2	2

Unexpected death during sports in older athlete (> 35 years)



Jim Fixx 1932-1984

Started running age 35, 214 lbs, 2 ppd smoker

Father MI age 35

Died of MI age 52

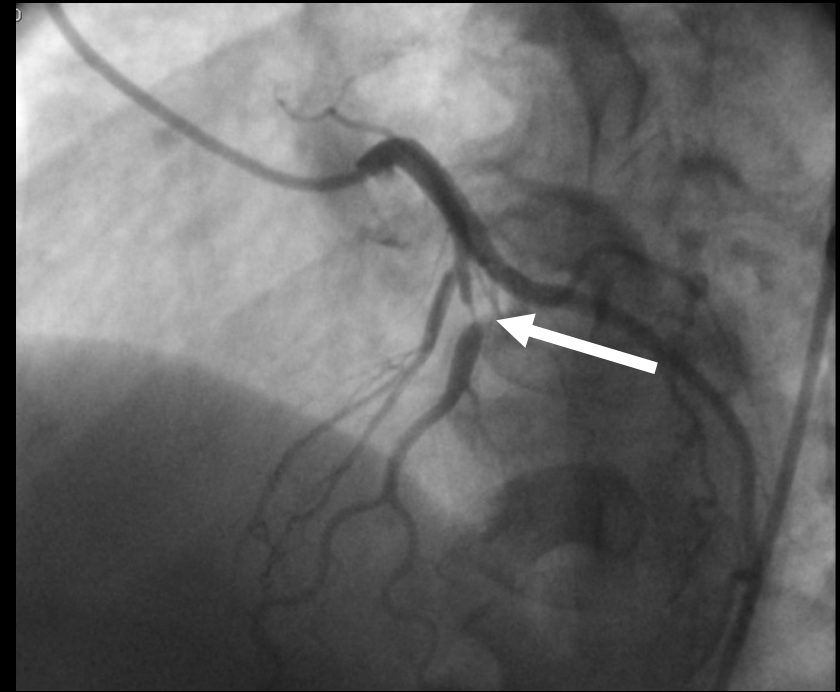
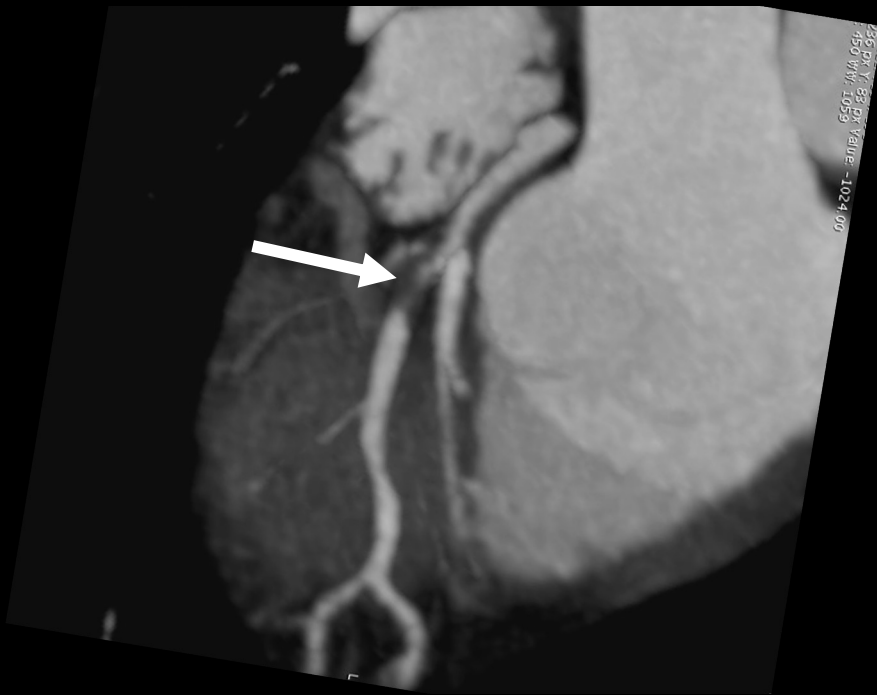
63 year old man

- Active, healthy
- Exercises daily (jogs ~ 3 miles)
- Family history of coronary disease (Father died in his 50' s)
- Referred for exercise stress test
- 13 minutes of treadmill exercise (good)
- No chest pain during the test
- ECG minimal changes, “not meeting criteria for ischemia”

“Widowmaker” blockage in the left anterior artery

CT Scan

Angiogram



DES to LAD, home next day

Pearl: Athletes hide CAD well. High exercise capacity. Low symptoms. Lack of ECG changes. Be concerned in setting of *strong* family history. The notion that 10 METs excludes CAD is not true

Sheldon E. Litwin, M.D.

63 y.o. recreational triathlete with known cardiac disease

- Hx of bicuspid aortic valve
- Bioprosthetic AVR and aortic root replacement (emergent) ~ 12 years ago with perioperative MI (embolic)
- Clinically stable without CP, DOE, edema
- Exercises daily for up to 2 hours (no change)
- Enters olympic distance triathlons, but no longer “competitive”

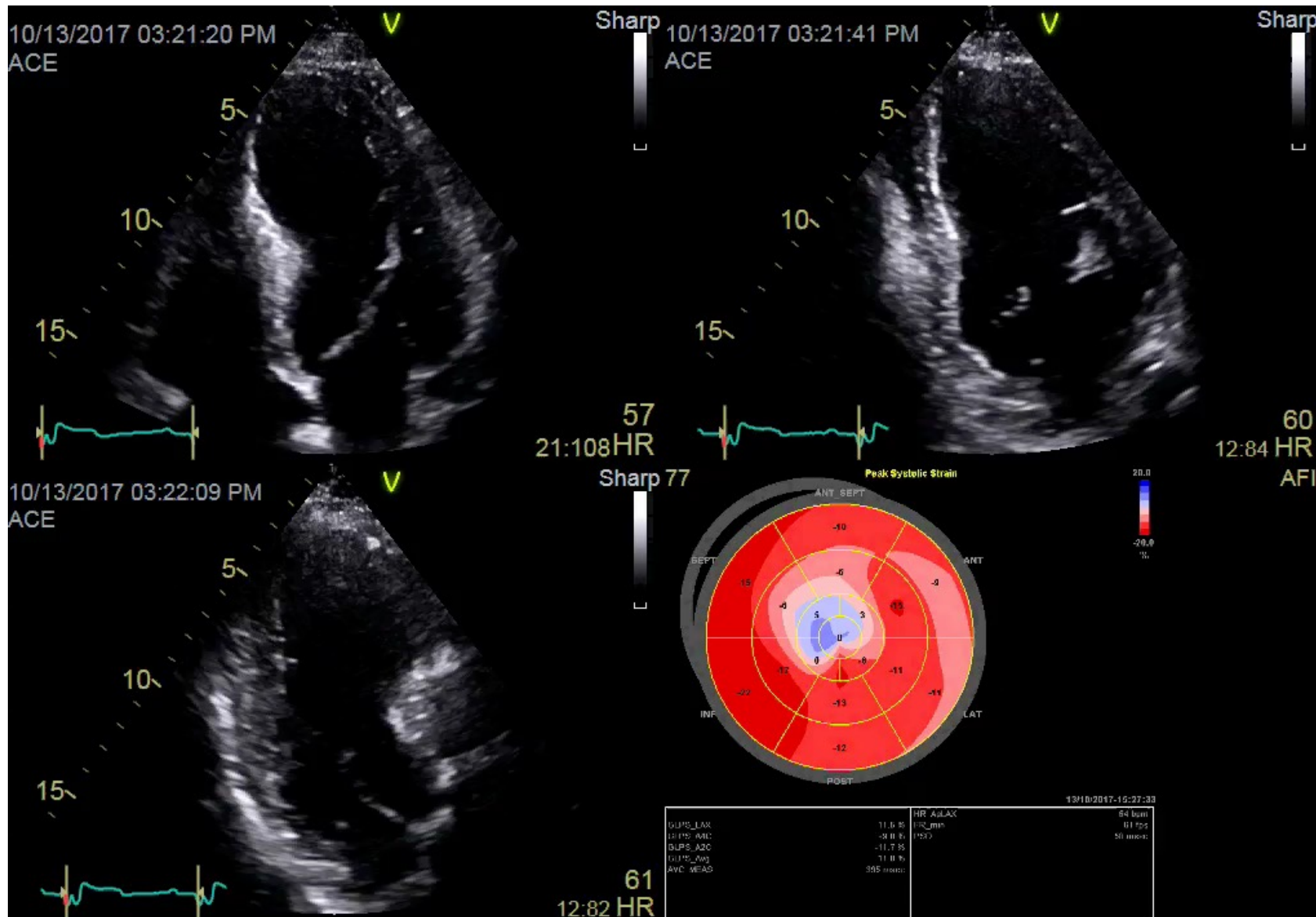
AHA/ACC Scientific Statement

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; on behalf of the American Heart Association
Electrocardiography and Arrhythmias Committee of the Council on Clinical Cardiology, Council on
Cardiovascular Disease in the Young, Council on Cardiovascular and Stroke Nursing, Council on
Functional Genomics and Translational Biology, and the American College of Cardiology

The most recent update published in 2015, uses the standard recommendation classification system and includes a large number of Class IIa and IIb recommendations with wording such as “*may be reasonable*” or “*may be considered*,” providing sports cardiologists and their patients an opportunity to engage in **shared decision making** in situations with uncertain risk and limited outcomes data. This approach represents *an important departure from prior versions of this document, which gave a dichotomous yes/no recommendation for sports eligibility*.

Case 3: 63 y.o. recreational athlete with prosthetic aortic valve

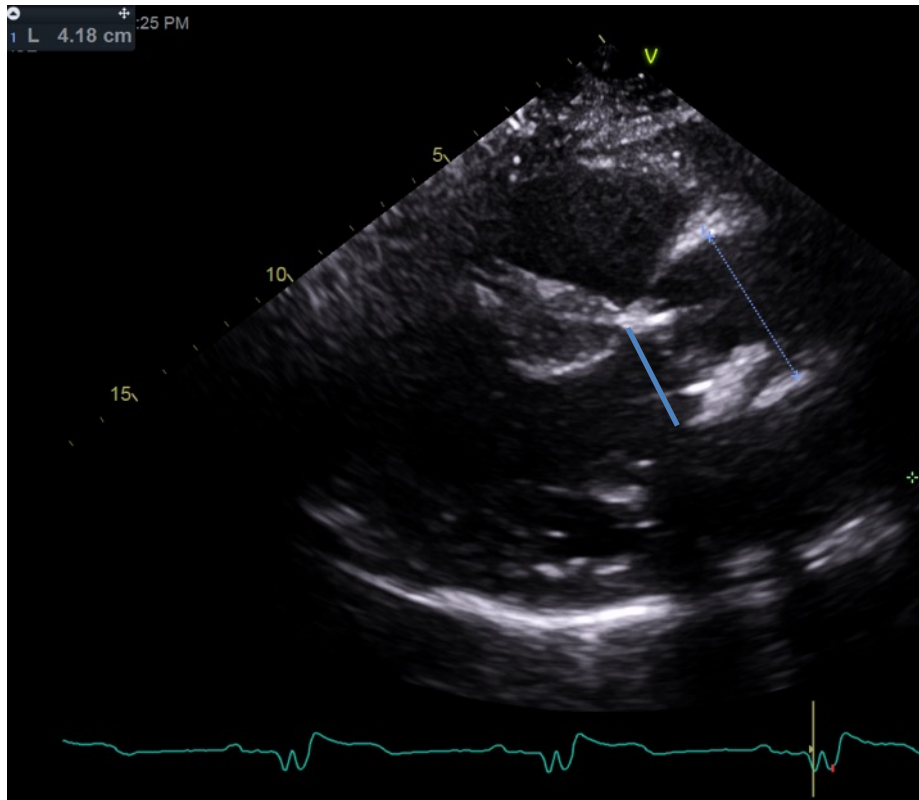


Case 3: Figure 1. 64 y.o. recreational athlete with bioprosthetic aortic valve

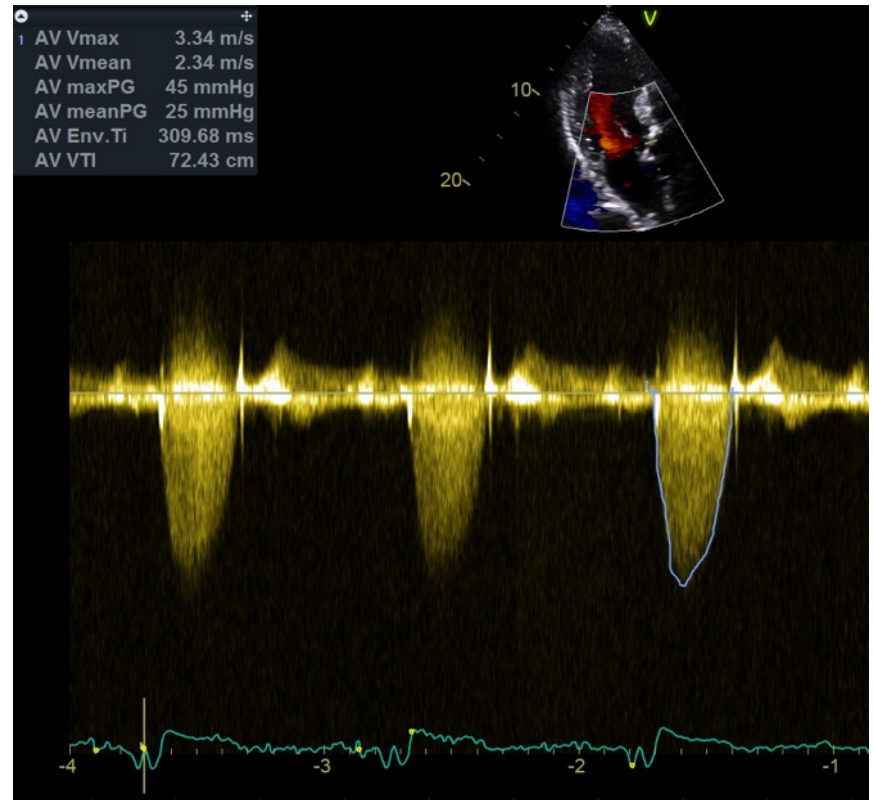
Parasternal Long Axis View

LVOT dimension 2.3 cm

Aortic root 4.2 cm



Continuous Wave Doppler across aortic valve (mean gradient 25 mmHg)



Eligibility and Disqualification Recommendations for Competitive Athletes With Cardiovascular Abnormalities: Task Force 8: Coronary Artery Disease

JACC 2015, 66:2406-11

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

It is *reasonable* for athletes with aortic or mitral bioprosthetic valves, not taking anticoagulant agents, who have normal valvular function and normal LV function to participate in *low-intensity and some moderate-intensity competitive sports* (classes IA, IB, IC, and IIA) (Class IIa; Level of Evidence C).

Athletes with moderate AS (stage B) can participate in *low and moderate static or low and moderate dynamic competitive sports* (classes IA, IB, and IIA) *if exercise tolerance testing to at least the level of activity achieved in competition and the training regimen demonstrates satisfactory exercise capacity without symptoms, ST-segment depression, or ventricular tachyarrhythmias, and with a normal blood pressure response* (Class IIa; Level of Evidence C).

It is reasonable for patients with clinically manifest ASCAD to participate in all competitive activities if their resting left ventricular ejection fraction is >50%, they are asymptomatic, and they have *no inducible ischemia or electrical instability* (Class IIb; Level of Evidence C).

The risk

- Running on treadmill
- Witnessed cardiac arrest
- Paramedics => ROSC (epinephrine)
- Cooling protocol
- Hemodynamically stable after initial need for pressors
- Oxygenating well (ventilator, 40% FIO₂)
- Making urine with normal kidney function
- Echo unchanged
- Irreversible anoxic brain injury



Eddie Merckx “The Cannibal”
Arguably greatest cyclist of all time
Tour de France 5 x winner
Giro d’Italia 7 x winner
Vuelta a Espana 1 x winner
19 classic race victories
World Champion 3 x
World record 1 hour distance

- During 1968 Giro d’Italia a leading cardiologist was invited to do ECG’s on some of the riders
- Dr. Lavazerro was “shocked” by Merckx’s ECG – looked like he was in the middle of a heart attack
- “Lavezzaro returned to his home in Turin and fully expected to hear news every day over the next fortnight of a Merckx collapse during race.”
- Family history of multiple men dying suddenly at young ages
- Likely has HCM
- Would have been banned from competition today
- Still alive (age 73)



Effect of Moderate-Intensity Exercise Training on Peak Oxygen Consumption in Patients With Hypertrophic Cardiomyopathy

A Randomized Clinical Trial

Sara Saberi, MD, MS; Matthew Wheeler, MD, PhD; Jennifer Bragg-Gresham, MS, PhD; Whitney Hornsby, PhD; Prachi P. Agarwal, MD, MS; Anil Attili, MD; Maryann Concannon, MSW; Annika M. Dries, BA; Yael Shmargad, BS; Heidi Salisbury, RN, MSN, CNS; Suwen Kumar, MBBS; Jonathan J. Herrera, MS; Jonathan Myers, PhD; Adam S. Helms, MD, MS; Euan A. Ashley, FRCP, DPhil; Sharlene M. Day, MD

RCT 136 patients with HCM
16 weeks moderate-intensity exercise
training vs. control (30% w ICD's)

+1.35 ml/kg/min

no occurrences of sustained ventricular arrhythmia, sudden cardiac arrest, appropriate defibrillator shock, or death in either group



“...the RESET-HCM by Saberi et al does not address whether exercise is safe for patients with hypertrophic cardiomyopathy. Assessing safety would require a much larger study powered to clinical events, a longer duration of follow-up, and a higher-risk population.”

Competitive sports in patients with HCM

...only a small subset of patients with HCM will experience SCD. In fact, the vast majority of patients with HCM live a normal life span with non-HCM etiology accounting for the majority of SCD mortality in these patients...

Snir et al. Am J Physiol Heart Circ Physiol 320: H2101–H2111, 2021

Maron et al. J Am Coll Cardiol 42: 882–888, 2003

Gimeno et al. Eur Heart J 30: 2599–2605, 2009

Association of Obesity With Adverse Long-term Outcomes in Hypertrophic Cardiomyopathy

Carlo Fumagalli et al. for the SHARE Investigators

JAMA Cardiol. 2020;5(1):65-72. doi:10.1001/jamacardio.2019.4268

N=3200, 70-80% overweight or obese

Figure 1. Prevalence of Left Ventricular Outflow Tract Obstruction at Baseline by Body Mass Index (BMI) Class

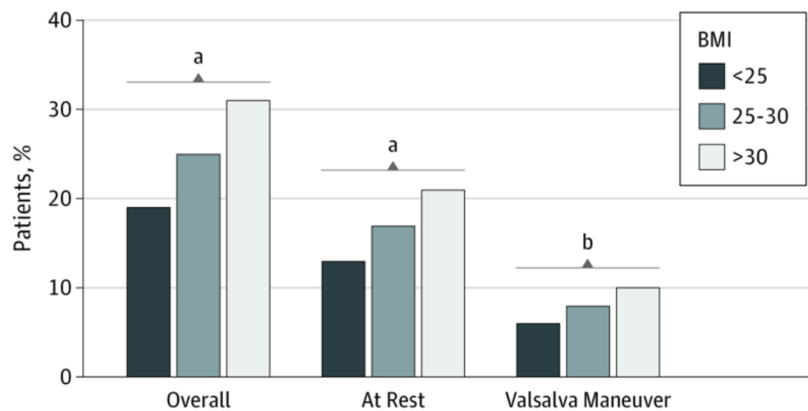
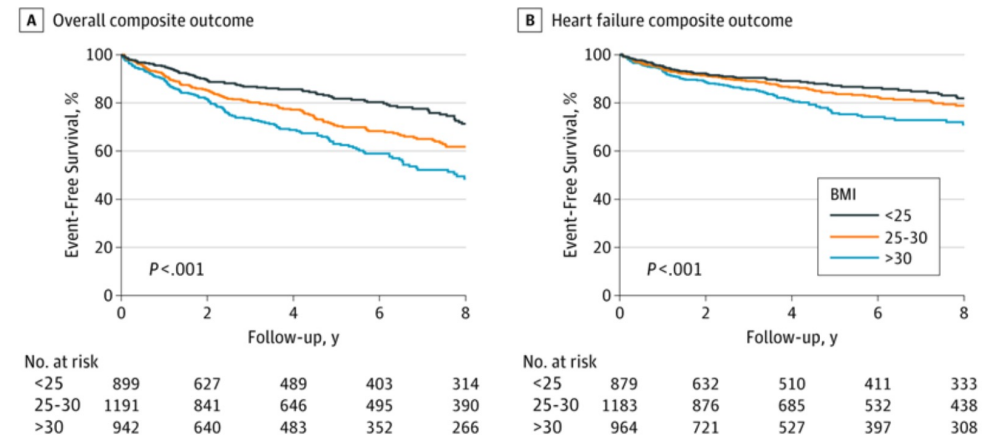


Figure 2. Kaplan-Meier Analysis of the Overall Composite and Heart Failure Composite Outcomes



a body mass index > 30 was independently associated with atrial fibrillation, disease progression, and heart failure onset irrespective of age, sex, outflow tract obstruction, and genotype

Exercise at the Extremes

The Amount of Exercise to Reduce Cardiovascular Events



Thijs M.H. Eijssvogels, PhD,*† Silvana Molossi, MD, PhD,‡ Duck-chul Lee, PhD,§ Michael S. Emery, MD,|| Paul D. Thompson, MD¶

ABSTRACT

Habitual physical activity and regular exercise training improve cardiovascular health and longevity. A physically active lifestyle is, therefore, a key aspect of primary and secondary prevention strategies. An appropriate volume and intensity are essential to maximally benefit from exercise interventions. This document summarizes available evidence on the relationship between the exercise volume and risk reductions in cardiovascular morbidity and mortality. Furthermore, the risks and benefits of moderate- versus high-intensity exercise interventions are compared. Findings are presented for the general population and cardiac patients eligible for cardiac rehabilitation. Finally, the controversy of excessive volumes of exercise in the athletic population is discussed. (J Am Coll Cardiol 2016;67:316-29)

FIGURE 1 The Curvilinear Relationship Between Physical Activity and Cardiovascular Risk

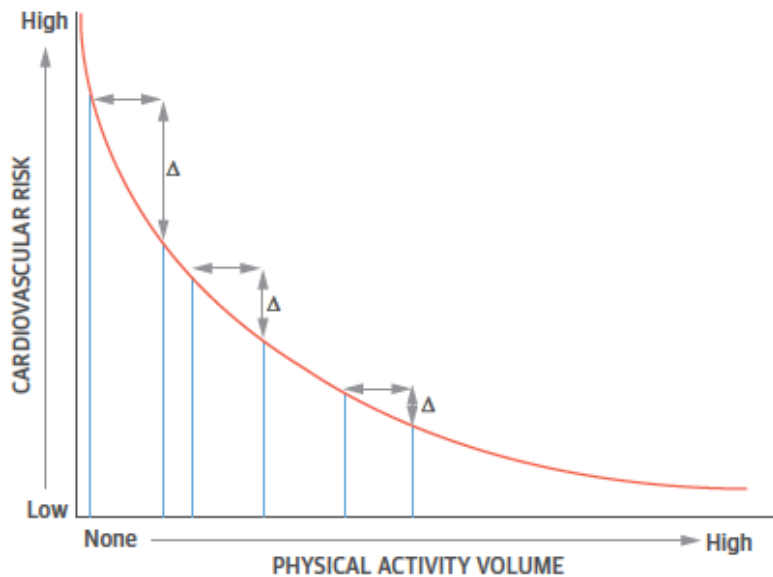
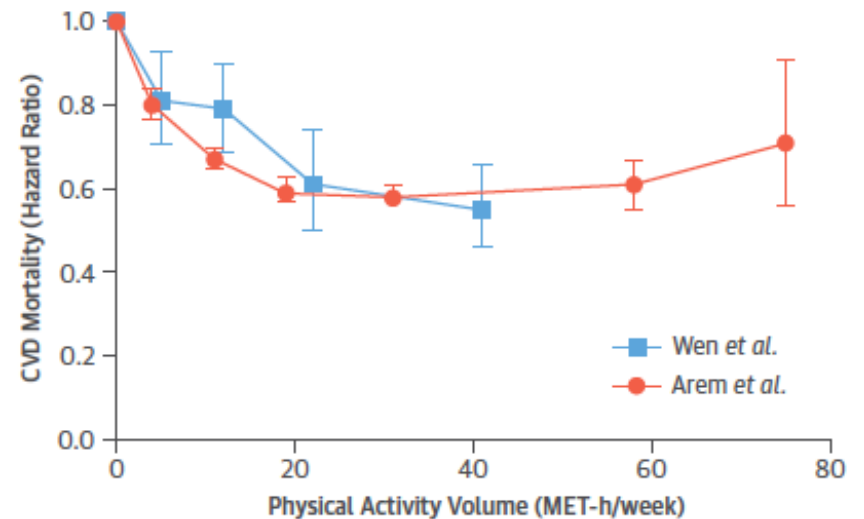
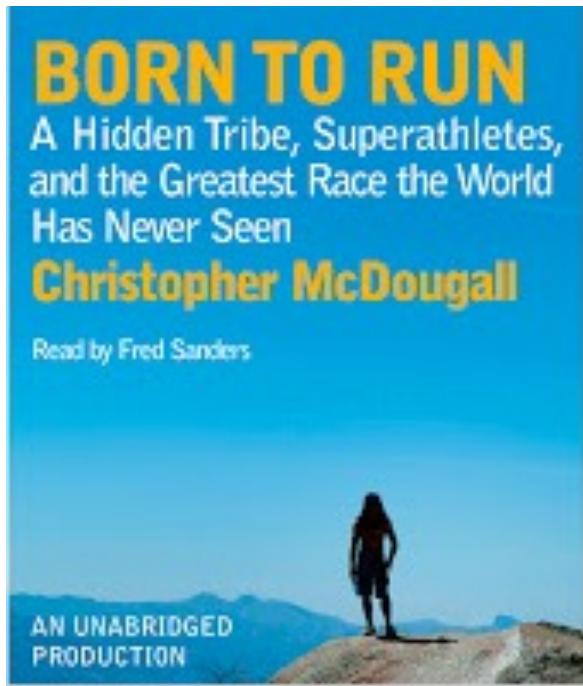


FIGURE 2 The Dose-Response Curve of Physical Activity and Cardiovascular Mortality



Vigorous activity transiently increases the risk of acute events, but reduces overall risk



Micah True “Caballo Blanco”

Ran ~ 170 miles/week for many years (~25-30 hours/week)

Died during a trail run in the Gila wilderness area at age 58

Autopsy showed unspecified cardiomyopathy

The Goldilocks Zone for Exercise: Not Too Little, Not Too Much



by James H. O'Keefe, MD, Evan L. O'Keefe, MS & Carl J. Lavie, MD



“...middle-aged and older individuals engaging in *excessive strenuous endurance exercise appear to be at increased risk* for a variety of adverse cardiovascular effects including atrial fibrillation, myocardial fibrosis and coronary atherosclerosis.”

“...moderate doses of physical activity significantly reduce long-term risks for both total mortality and CV mortality, however, *at very high doses of chronic strenuous exercise much of the protection...is lost.*”

“...150 minutes per week of moderate-intensity aerobic exercise or 75 minutes per week of vigorous-intensity aerobic activity, but not more than 4-5 hours per week of vigorous (heart-pounding, sweat-producing) exercise, especially those over 45 years of age.”

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

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Participants who completed Vasaloppet (90 km cross country ski race) 1989-98
Followed through national registries until 2005

919 experienced arrhythmia (1.7%)

Adjusted HR for *number of races* (> 5 vs. 1) = 1.3 (any arrhythmia), 1.29 (afib), 2.1 (brady)
Faster finishing times also had higher risk. HF 1.3 for 100-160% of winning time vs. > 240%

AF incidence in Framingham study (men aged 55-64) = 31 per 10,000 person years at risk
This study, AF = 49 per 10,000 PYAR's

Efficacy of circumferential pulmonary vein ablation of atrial fibrillation in endurance athletes

Naiara Calvo[†], Lluís Mont^{*†}, David Tamborero, Antonio Berruezo, Graziana Viola, Eduard Guasch, Mercè Nadal, David Andreu, Barbara Vidal, Marta Sitges, and Josep Brugada

- 182 consecutive PVI patients (81% men; 59% lone AF, LA diameter 42+/-6 mm)
- Endurance athletes (n=42) engaged in > 3 hr/week high-intensity exercise
- Mean age 48 (athletes) vs. 52 (controls)
- *Freedom from arrhythmia no different between groups (59% athletes vs. 48% controls after 1 procedure, 1 year)*
- Only predictors of recurrence were LA size and longstanding AF

Risk of arrhythmias in athletes appears to be almost exclusively in men

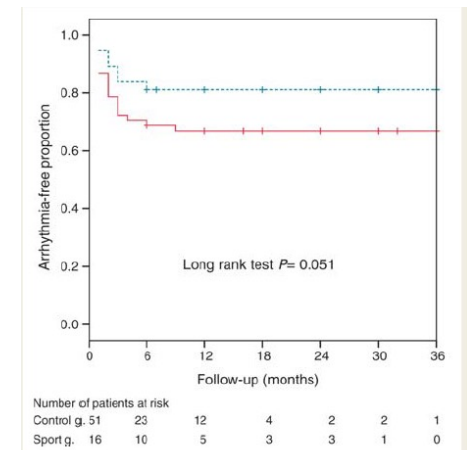


Figure 3 Kaplan–Meier curves for long-term freedom from recurrent arrhythmias after repeated ablation procedures in the lone AF sport group (dashed line) and the control group (solid line).

Mortality amongst participants in Vasaloppet: a classical long-distance ski race in Sweden

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49,219 men; 24,403 women

Standardized mortality ratios (SMR) calculated from national death registry

-adjusted for age and calendar year

410 deaths compared to 850 expected (**SMR 0.48**, CI 0.44-0.53)

Lowest SMR in older participants and those who participated in several races

Decreased mortality for cancer, circulatory diseases, injuries, suicide and poisoning

Physical training vs. related lifestyle factors vs. genetics (or combination)



Mortality of French participants in the Tour de France (1947–2012)

Eloi Marijon^{1,2,3,4*}, Muriel Tafflet^{1,2,5}, Juliana Antero-Jacquemin^{1,5}, Nour El Helou^{1,5,6}, Geoffroy Berthelot^{1,5}, David S. Celermajer⁷, Wulfran Bougouin^{1,2,4}, Nicolas Combes⁸, Olivier Hermine^{1,9,12,13}, Jean-Philippe Empana^{1,2}, Grégoire Rey¹⁰, Jean-François Toussaint^{1,5,11†}, and Xavier Jouven^{1,2,3,4†}

768 cyclists who participated at least once in TDF

268 deaths by 2012 (26%); median duration of f/u 37 years

Neoplasms and CVD main causes of death

Standardized mortality ratio (SMR = 0.59, 41% reduction) compared to French males

SMR for CVD 0.67 and for neoplasms 0.56

Row for Your Life: A Century of Mortality Follow-Up of French Olympic Rowers

Juliana Antero-Jacquemin^{1,2*}, François Deni Desgorces^{1,2}, Frédéric Dor¹, Adrien Sedeaud¹, Amal Haïda¹, Philippe LeVan^{3,4}, Jean-François Toussaint^{1,2,5}

203 French olympic rowers (1912 – 2013)

Causes of death from national registry

Overall cohort: 42% reduction in mortality (**SMR 0.58**)

1912-1936: 37% reduction in mortality (SMR 0.63)

1948-2012: 60% reduction in mortality (SMR 0.40)

Mortality due to CVD significantly reduced (SMR 0.41)



Summary

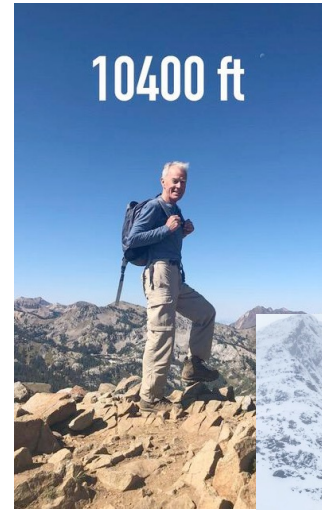
- Things that may be normal in athletes
 - 4 chamber enlargement
 - LVH
 - Low normal or mildly reduced LV EF
 - Mildly elevated pulmonary artery pressure
 - Dilated IVC
 - Low normal strain
 - Abnormal ECG
 - Coronary calcium (older athletes)
- Refer for additional imaging testing when needed
- Inherited diseases main risk for younger athletes, CAD main risk for older athletes
- Shared decision making for patients with known disease (including covid)
- Exercise and high levels of physical activity are strongly protective
 - Very high, chronic levels of exercise *may* have deleterious effects, especially arrhythmias

If you keep on moving, you never grow old

Carl Kjeldsberg, M.D.



Snowbird Hill Climb circa 1998



Harry Hill, M.D.

