

Shared Decision Making in Athletes – Has the Data Changed risk?"

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HCM Medical Director
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I have **no** commercial interests nor financial relationships with any manufacturer of any product or class of products that are discussed in this educational activity, or which is a commercial supporter of this activity.



Case Presentation

17 y.o elite Caucasian Female
American Soccer midfielder

Asymptomatic.

No prior syncope, CV limitations.

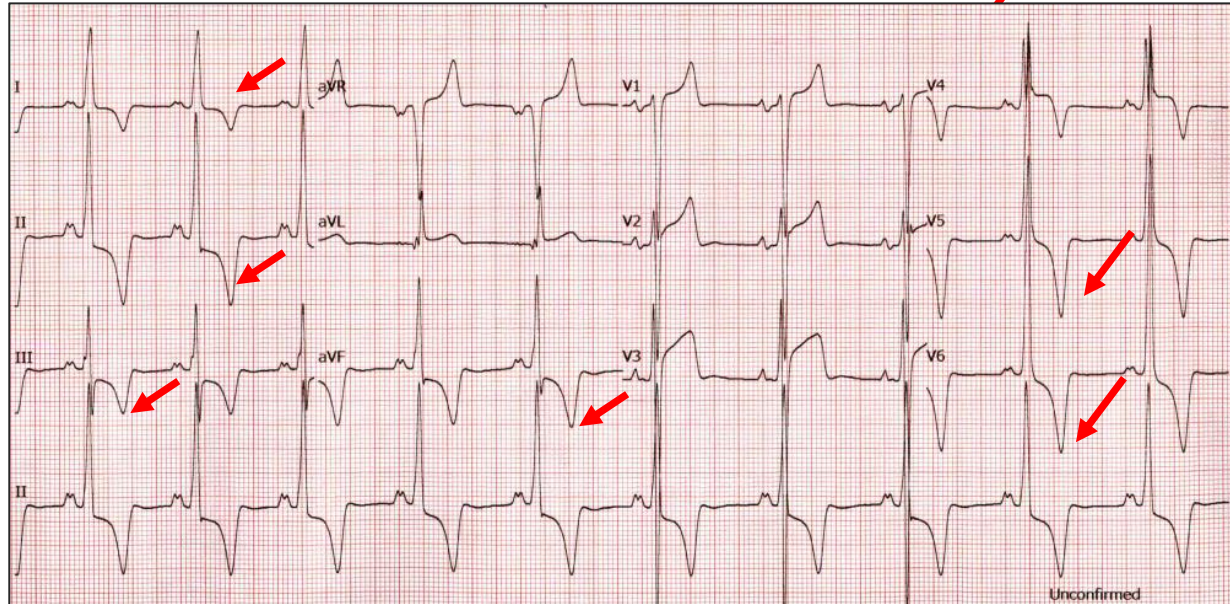
No family hx of cardiomyopathy or sudden cardiac arrest.

Preparticipation screening evaluation including ECG.

ECG was abnormal.



Electrocardiogram



Sinus bradycardia
Normal QTc
TWI II, III, aVF, V3-V6

No prior ECG

Referred for ECHO

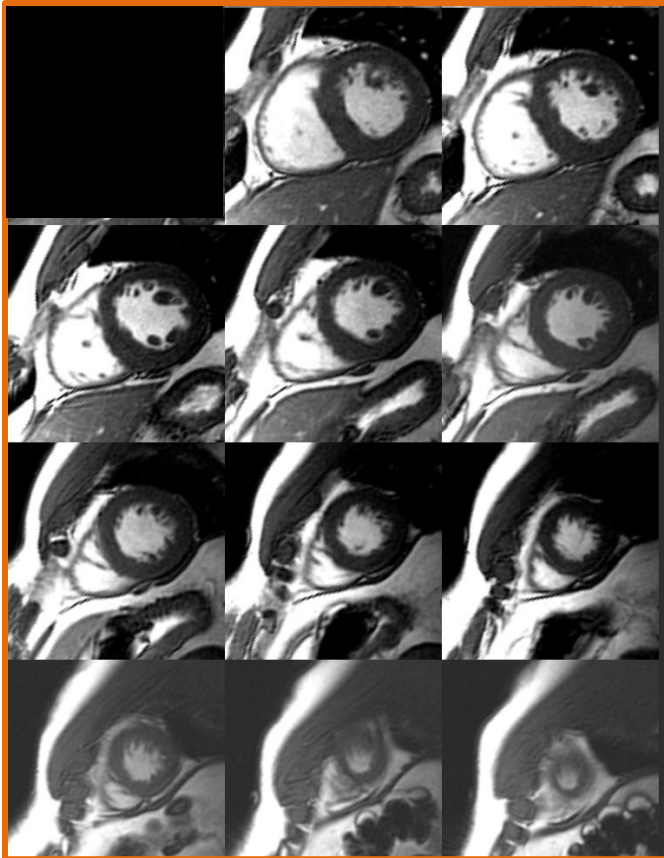


ECHO

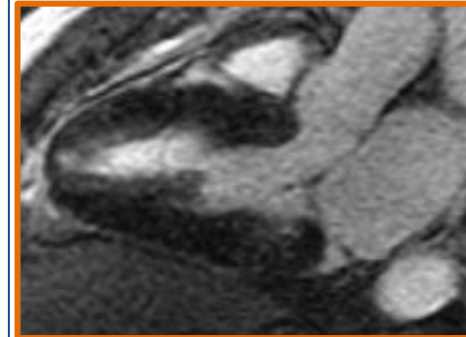
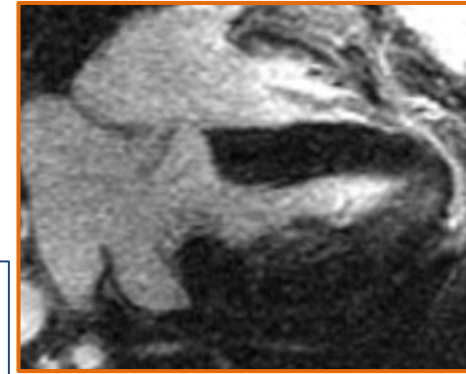
- Normal biventricular size and function.
- LVEF = 65%.
- Normal wall motion. No valve disease: SAM or MVP. Abnormal diastolic function
- 17mm septal thickness.
- **Exercise Echo** – 16minutes + sprints; no arrhythmias or obstruction, Normal HR and BP response
- **48hr Ambulatory monitor**: Rare PVCs



CMR



- LVEF = 75%.
- No SAM
- 18mm septum
- No LGE/scar



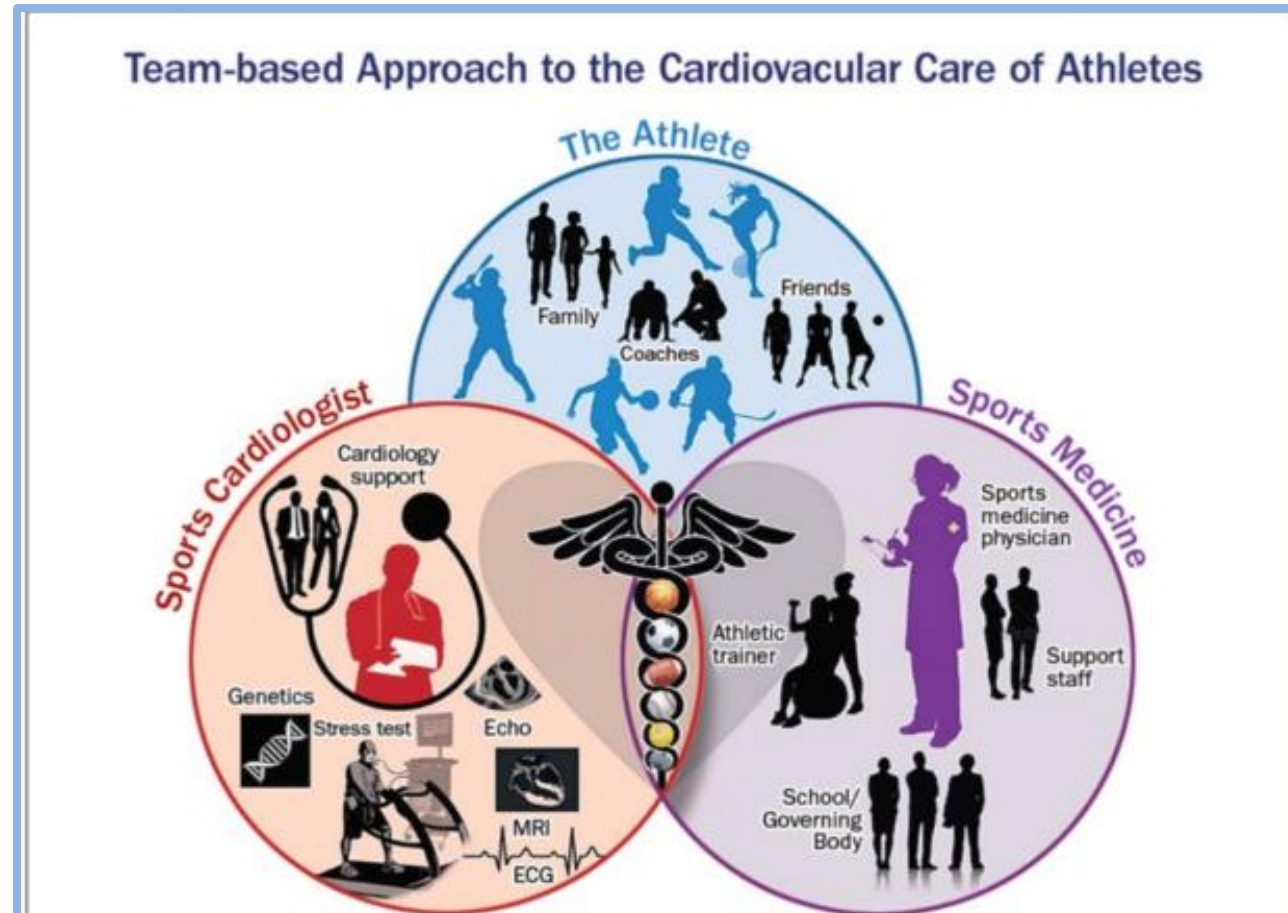
Athlete with SCA Risk

Would YOU allow this athlete with screening-based, incidentally detected HCM (asymptomatic) to continue with his/her competitive sport?

- 1. YES**
- 2. NO**
- 3. MAYBE**



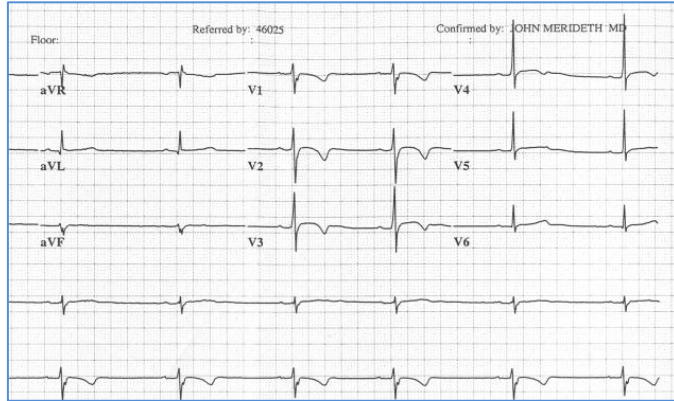
Cardiac Care of the Athlete



**Multidisciplinary Athlete-Centered Care (“Athlete Care Team”)
in Evaluating and Managing Athletes at Risk of SCD**

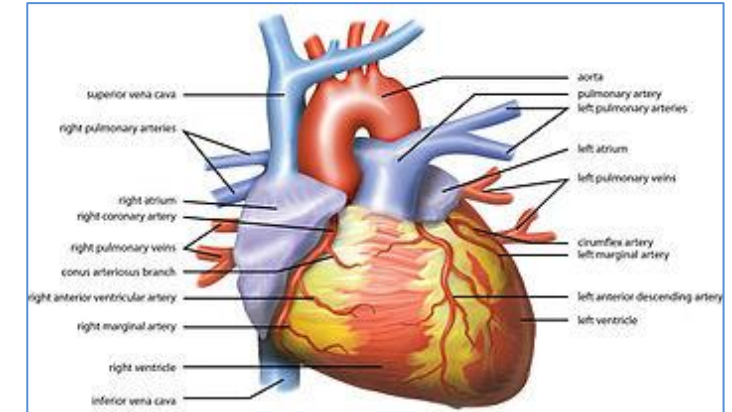


Sudden Cardiac Death in *Young Athletes*



Structural Abnormalities

Hypertrophic cardiomyopathy
RV cardiomyopathy
Artery anomalies
Marfan syndrome
Valve disease



Electrical Abnormalities

Wolff Parkinson White syndrome
Long QT syndrome
Brugada syndrome
CPVT

Acquired Abnormalities

Infection (myocarditis)
Trauma (commotio cordis)
Drugs
Environment (heat/cold)



Traditional etiologies of SCD in Athletes (<40 years)

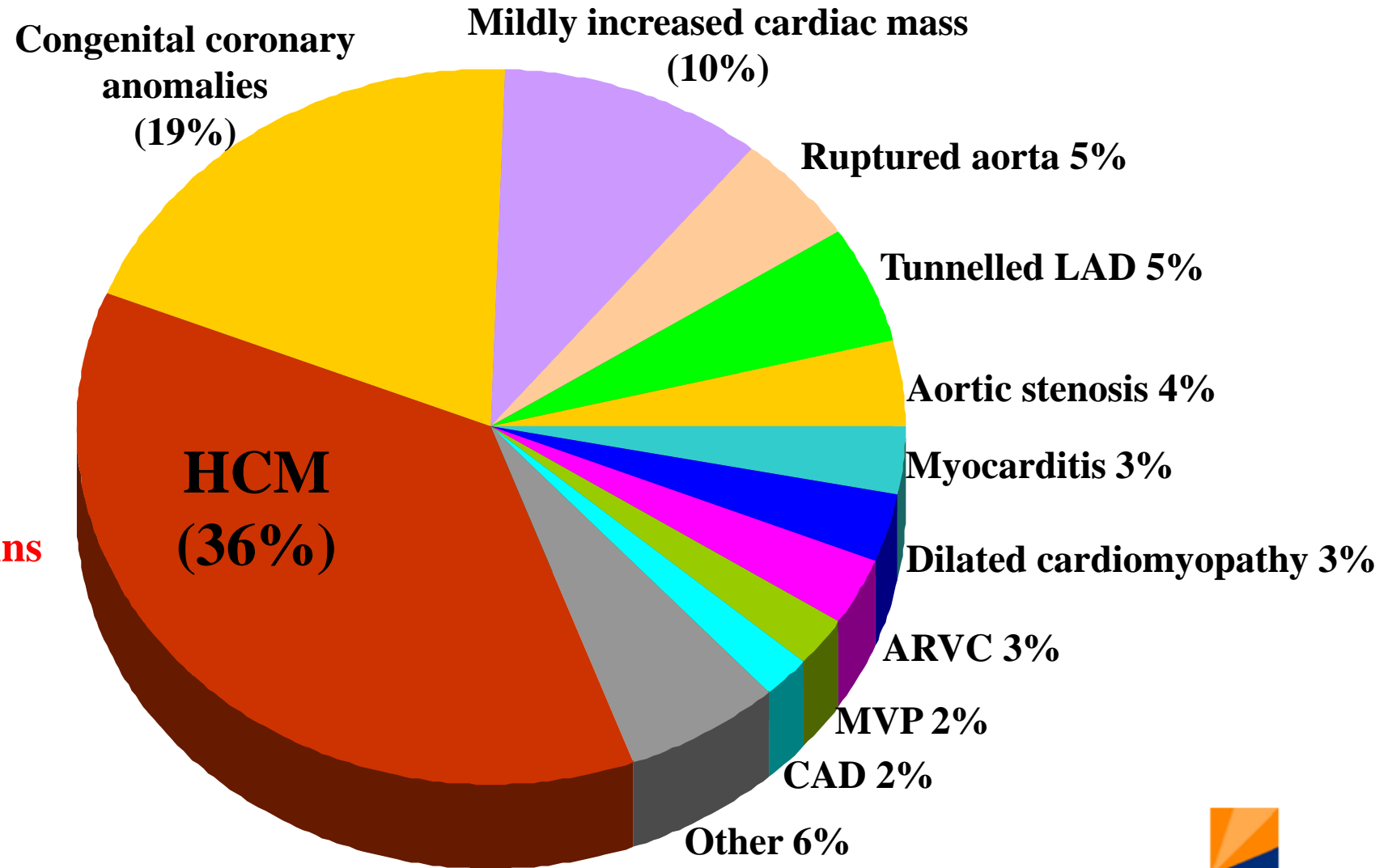
1980 - 2005

Multiple updates:
2007, 2009, 2016

N = 2406

Confirmed CV deaths = 840+

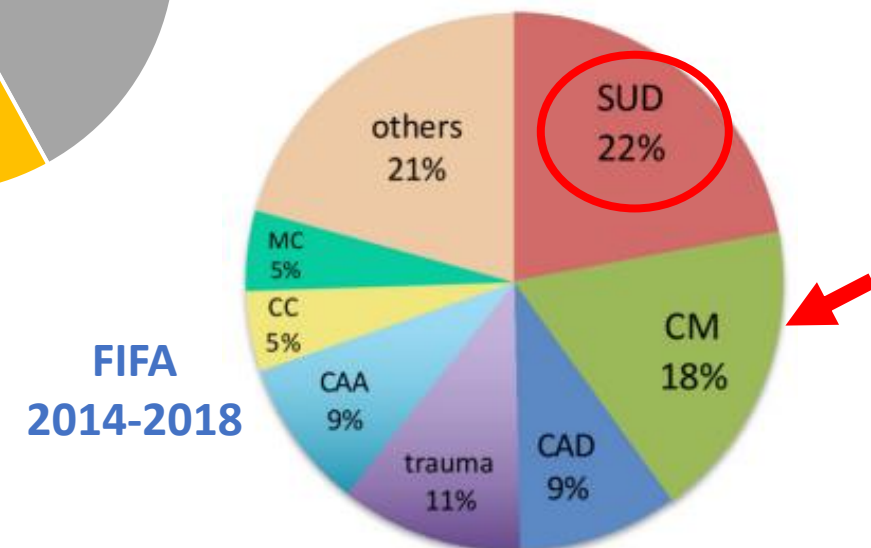
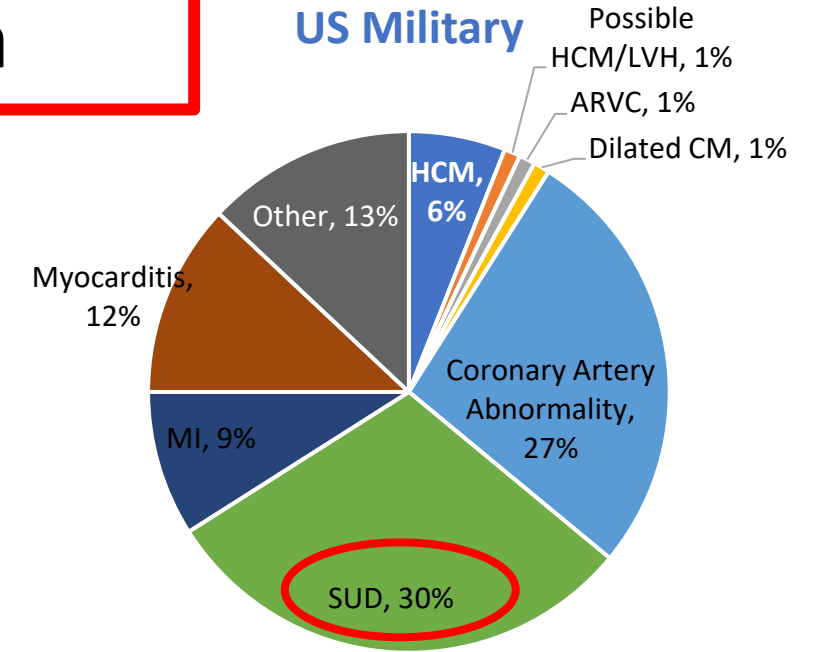
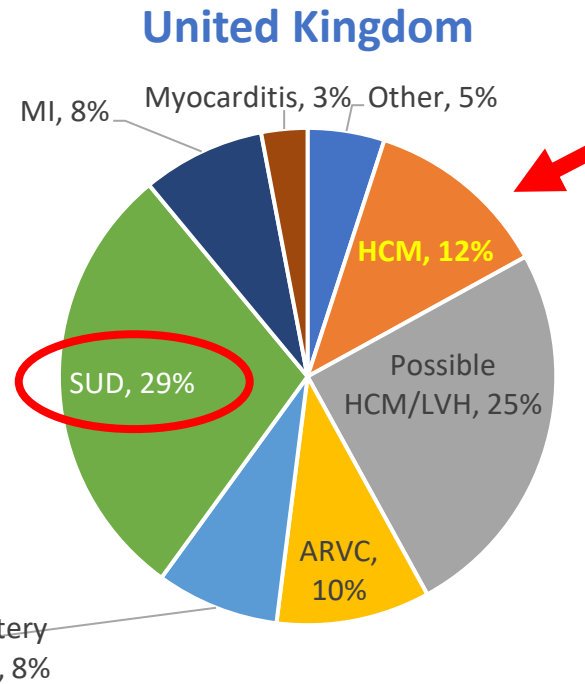
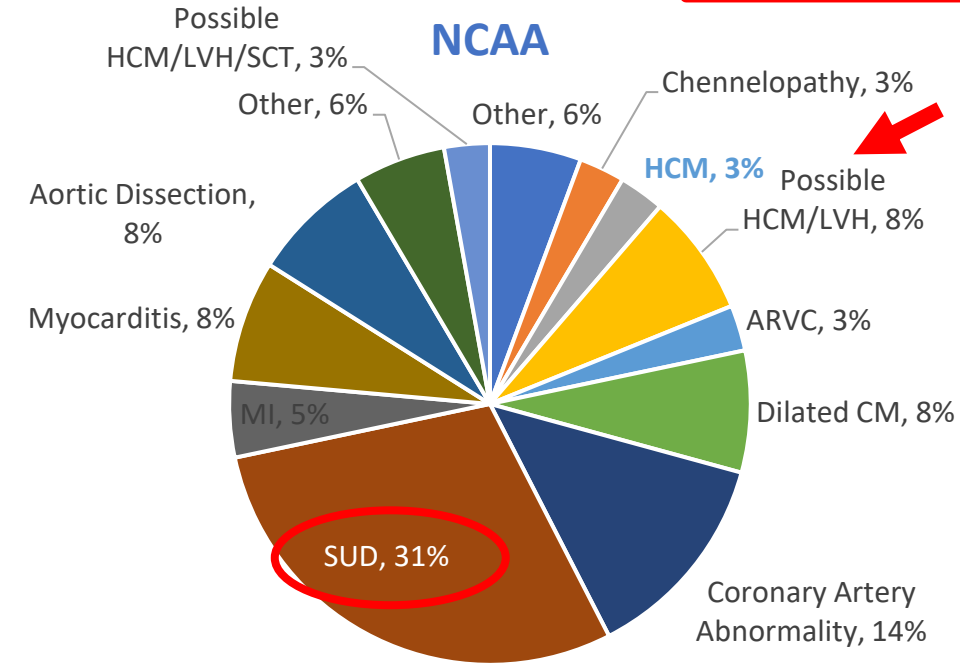
**The data and message remains
largely unchanged**



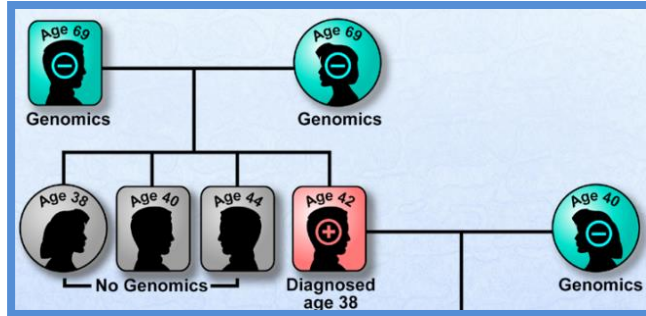
Maron BJ et al. *Circulation*. 1996;94:850-56.

Contemporary Estimates of SCD

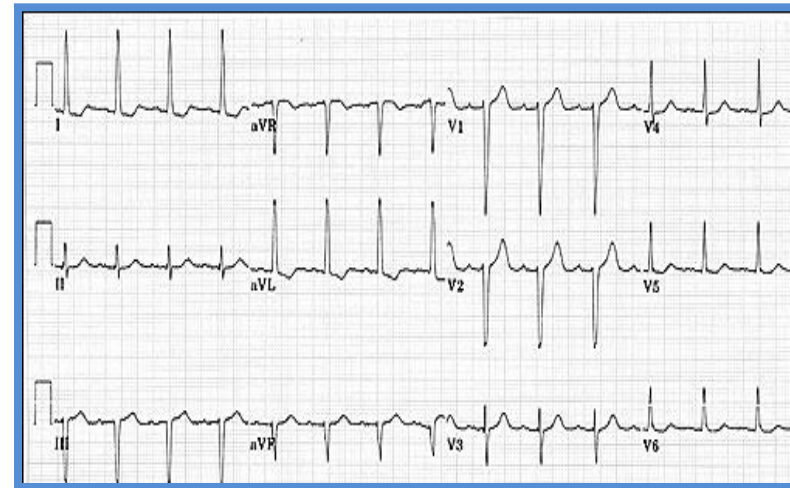
- > challenge prior data



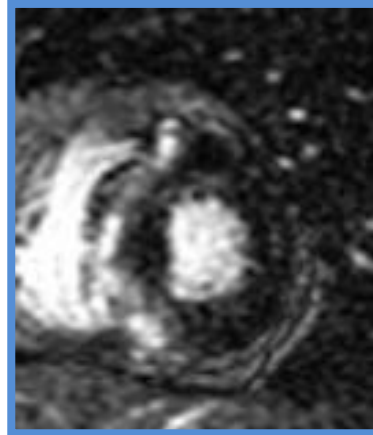
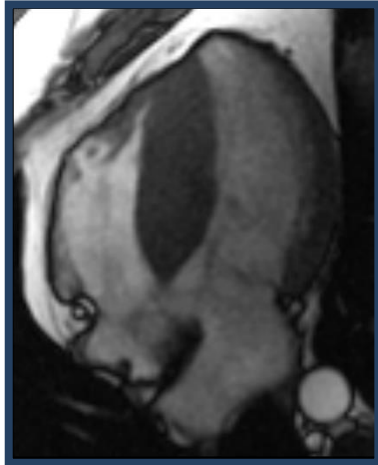
Athlete Evaluation



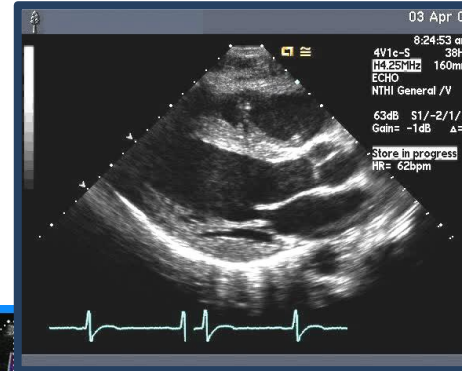
History



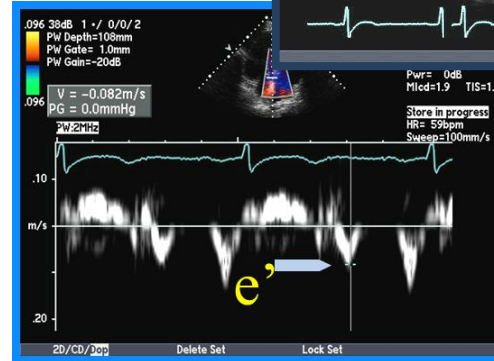
Athlete Evaluation



MRI



Echo

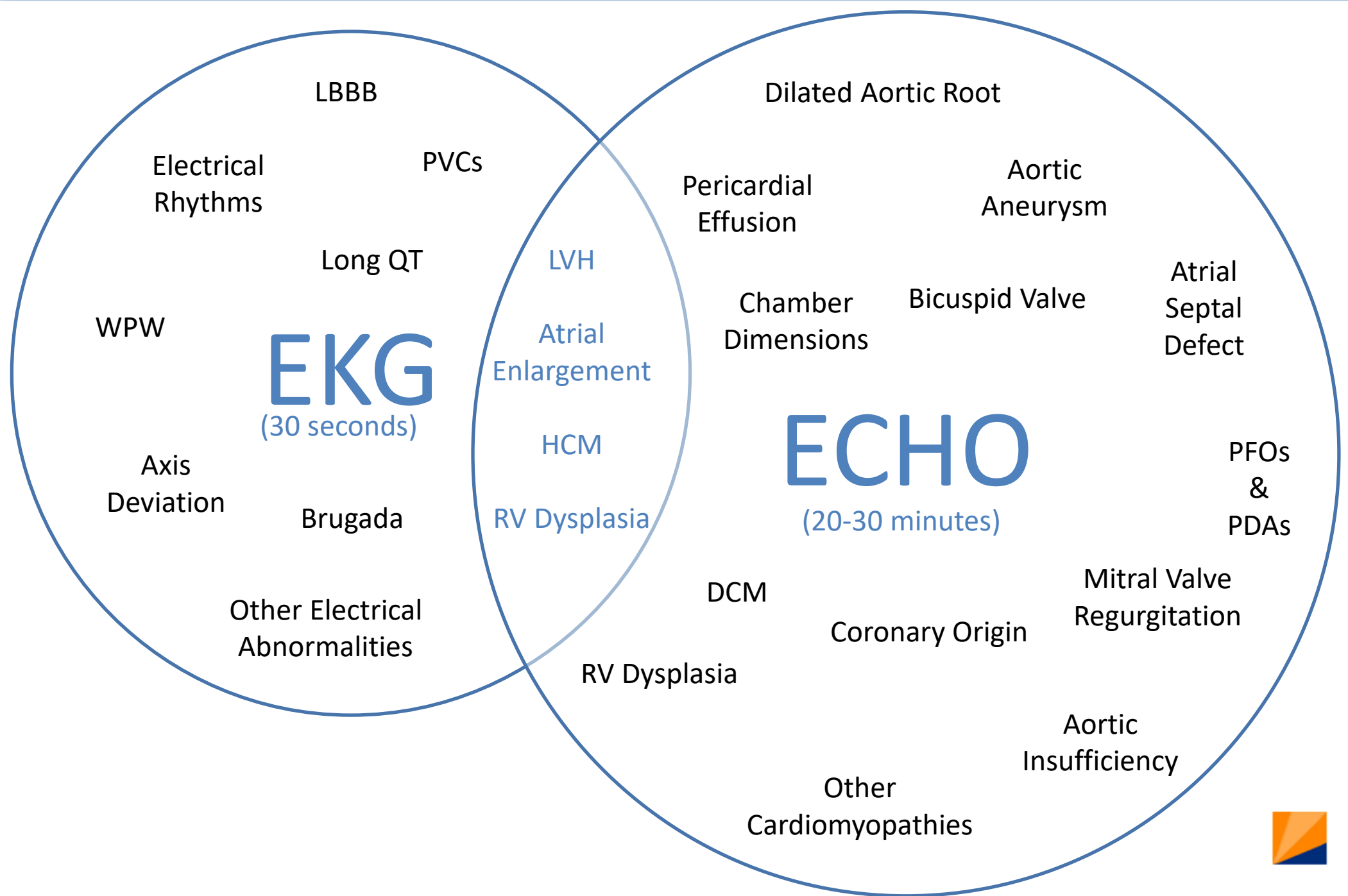


Comparison of Screening Strategies for Elite Athletes

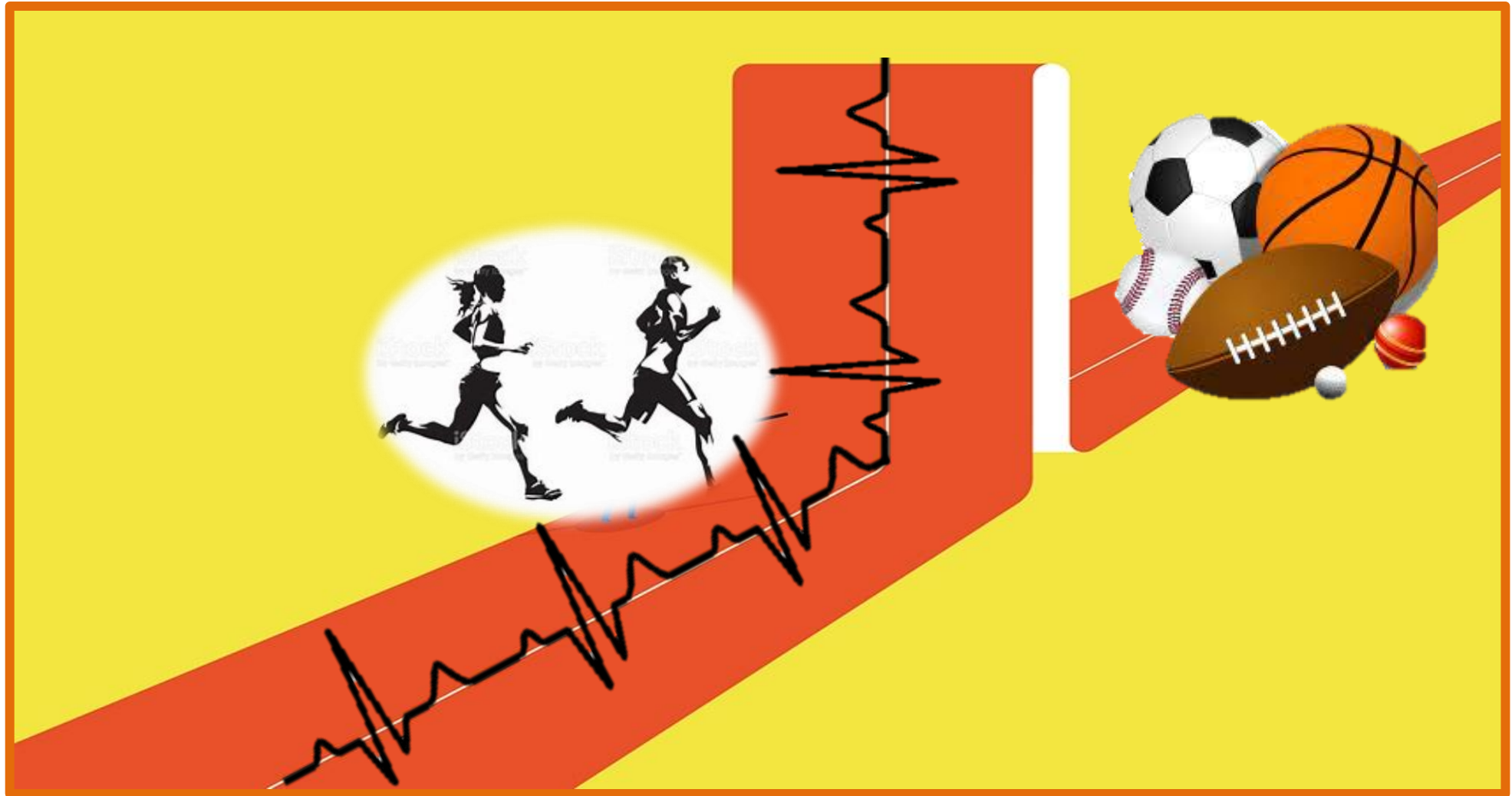
	IOC/ USOC	FIFA	MLB	MLS	NBA/ WNBA	NFL	NHL	Premier League
Combine			X		X	X	X	
H&P	X [‡]	X [‡]	X	X	X	X	X [‡]	X
ECG	X	X	X	X [^]	X	X	X	X [^]
Echo		X		X	X	X		X [^]
Stress test ECG	X [*]	X [*]						
Stress Echo					X			
Additional Testing As needed	X	X	X	X	X	X	X	X

[‡] Unique H&P; others use AHA
[^] Every 2 years
^{*} Stress ECG if >35 years old

Screening findings: EKG vs ECHO



Barriers to Participation



Screening

- Goal: Early identification of cardiac conditions at risk for sudden cardiac arrest & death (SCA/D)
- Optimal strategy unclear
- AMSSM: “assist the individual physician in assessing the most appropriate screening strategy unique to their athlete population, community needs and resources”
- “mitigating risk through individualized, patient-centered and disease-specific medical management.”

POSITION STATEMENT

AMSSM Position Statement on Cardiovascular
Preparticipation Screening in Athletes: Current Evidence,
Knowledge Gaps, Recommendations, and
Future Directions

Jonathan A. Drezner, MD, Francis G. O'Connor, MD, MPH,† Kimberly G. Harmon, MD,*
Karl B. Fields, MD,‡ Chad A. Asplund, MD,§ Irfan M. Asif, MD,¶ David E. Price, MD,||
Robert J. Dimeff, MD,**†‡§ David T. Bernhardt, MD,§§¶ and William O. Roberts, MD, MS|||*



Purpose of Cardiovascular Screening

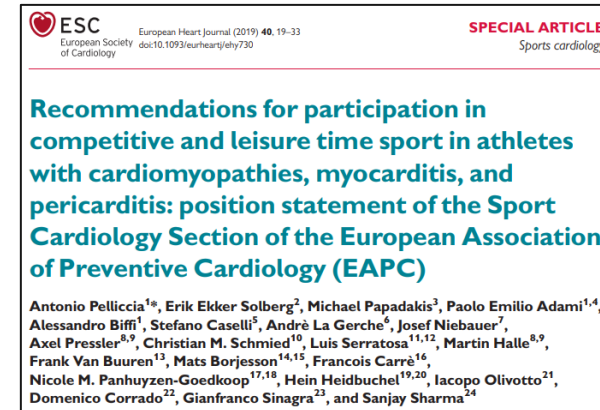
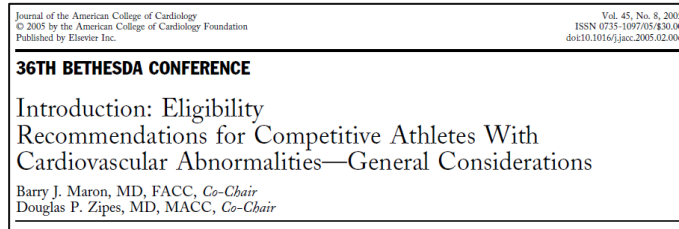
- Historically, athletes with Cardiovascular disease had been advised against competitive sport participation
 - 2011 ACCF/AHA HCM Guidelines
 - 2015 AHA/ACC Eligibility and Disqualification Recommendations for Competitive Athletes With Cardiovascular Abnormalities

“Athletes with a probable or unequivocal clinical expression and diagnosis of CV disease (ie, HCM) should not participate in most competitive sports, with the exception of those of low intensity (class IA sports). This recommendation is independent of age, sex, magnitude of LV hypertrophy, particular sarcomere mutation, presence or absence of LV outflow obstruction (at rest or with physiological exercise), absence of prior cardiac symptoms, presence or absence of late gadolinium enhancement (fibrosis) on CMR, and whether major interventions such as surgical myectomy or alcohol ablation have been performed previously.” (2015 AHA/ACC Guidelines)

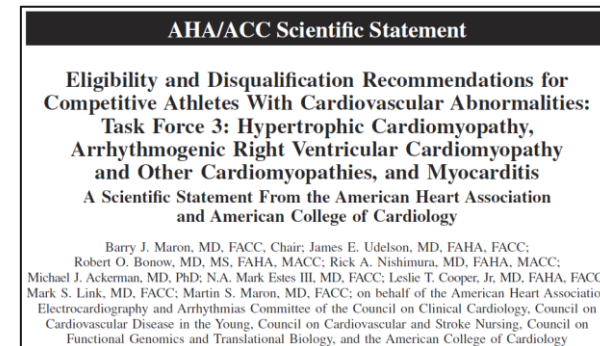
Recommendations for Athletes



2005



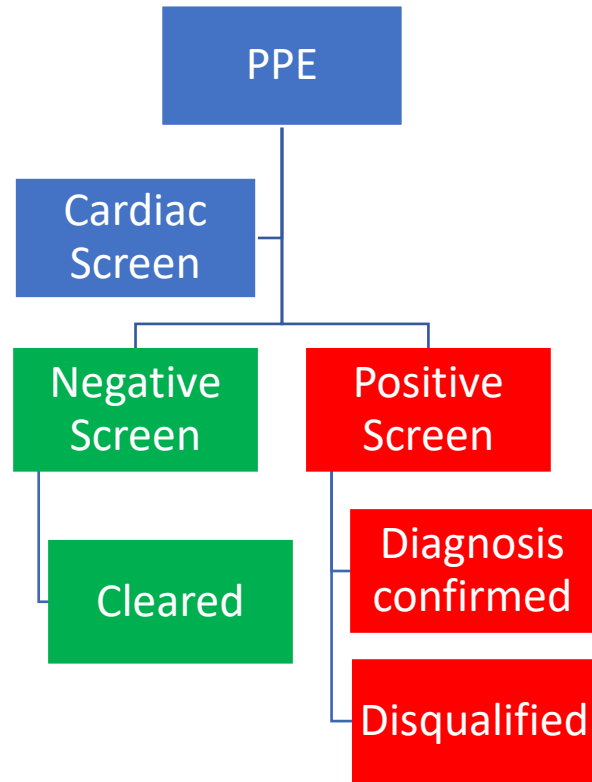
2019



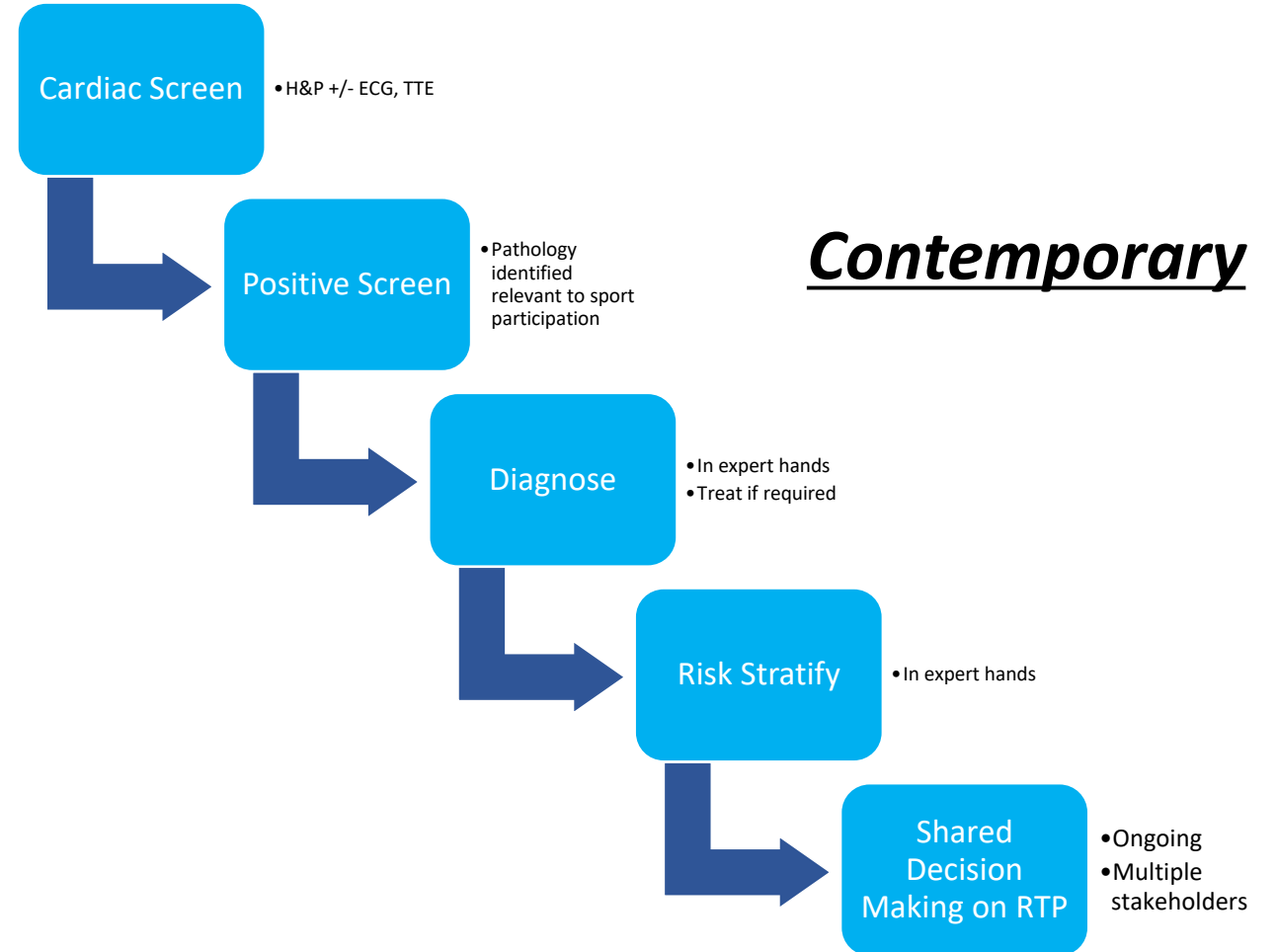
2015

Guidelines now indicate we need more than yes or no.
-> Advocate risk assessment

Paradigm Shift in Sports Cardiology



Traditional



Some young athletes with heart disease cleared to play under new recommendations

By AMERICAN HEART ASSOCIATION NEWS



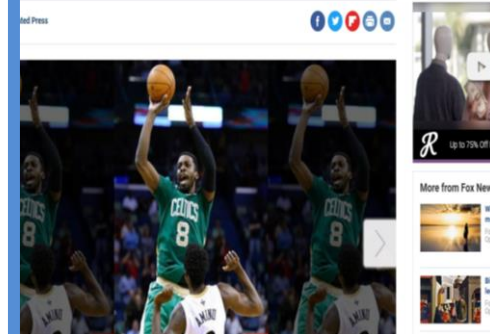
Michigan DT Maurice Hurst, potential first-round pick, cleared to play after heart condition diagnosis



Frank Schwab
Shutdown Corner March 22, 2018

Follow

For 4 NBA players with serious heart ailments, a fraternity has helped get them through it



Baylor's Jared Butler medically cleared to play in NBA: Sources

By Shams Charania and The Athletic Staff
July 17, 2021 Updated 7:21 PM EDT

8 Comments



Cleared by cardiologists, Sierra Leone's Alhaji Kamara joins D.C. United

By Steven Goff
May 10, 2018

D.C. United has acquired Alhaji Kamara, a forward from Sierra Leone whose career was jeopardized by a heart condition this winter.

In the past week, the MLS cardiology consultant and a heart specialist at MedStar Georgetown University Hospital examined Kamara and determined that he could resume playing soccer, United General Manager Dave Kasper said Tuesday.

With medical clearance, United finalized a deal with Kamara's previous employer, IFK Norrköping of Sweden. D.C. did not pay a transfer fee but will compensate the Swedish team if Kamara, 22, meets performance incentives or is sold in the future.



Alhaji Kamara (Norrköping)



Assassin's Creed Odyssey - PlayStation 4

NBA NHL

Clint Dempsey cleared to play again after heart problems

- Seattle Sounders striker missed end of last season with medical condition
- Dempsey says he hopes to make return to US national team soon

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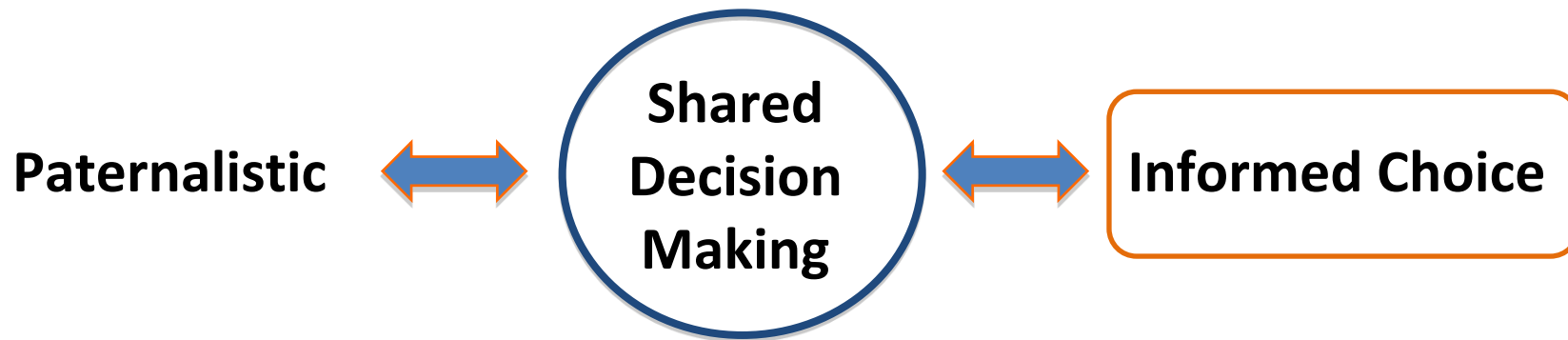
Christian Eriksen joins Brentford in remarkable return to football after cardiac arrest



Shared Decision Making

SDM is an approach where clinicians and patients make decisions together using the best available evidence.

(Elwyn et al. BMJ 2010)



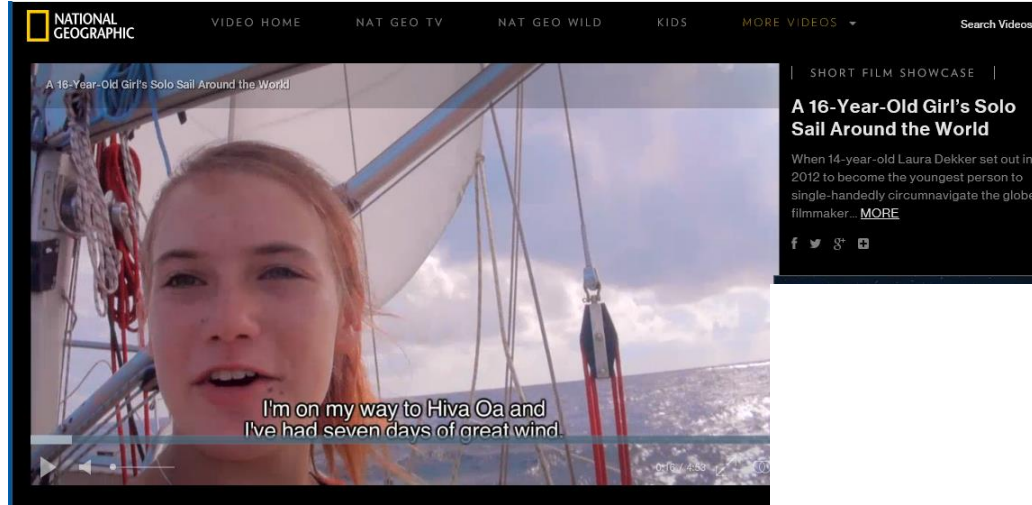
Patient well informed (**Knowledge**)

Knows what's important to them
(**Patient values elicited**)

Decision consistent with values



Spectrum of comfort with risk



Would you let your 16 year old daughter (or son) sail around the world by herself?

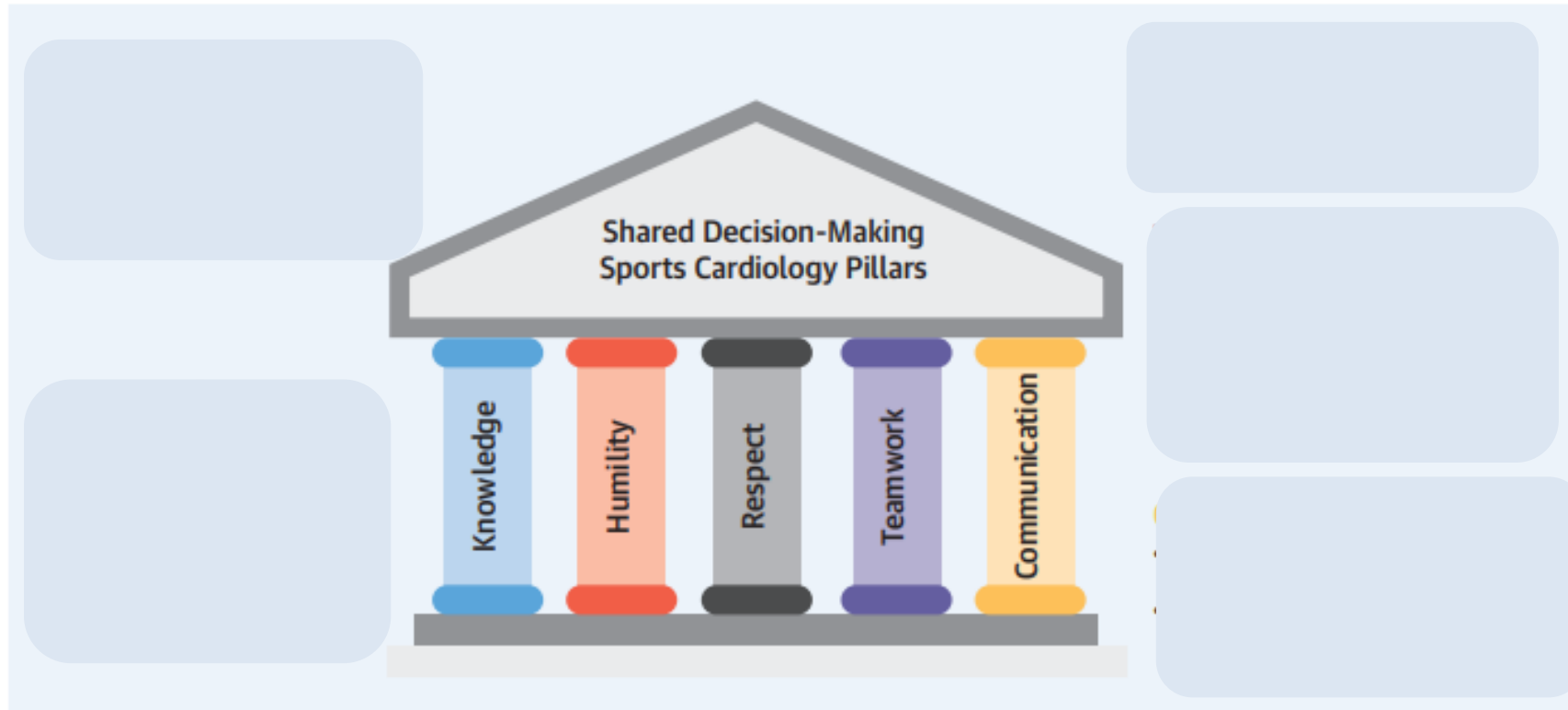
1) Yes

2) No



SDM in Sports Cardiology

FIGURE 3 SDM Sports Cardiology Pillars



An illustration of the 5 pillars that make up a prescribed method of shared decision-making (SDM) that includes the required points to guide the patient-physician discussion.



CLINICAL SCIENCES

clinical commentary

The medical care of competitive athletes: the role of the physician and individual assumption of risk

BENJAMIN D. LEVINE and JAMES STRAY-GUNDERSEN

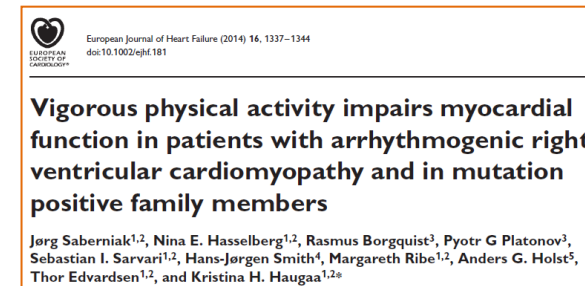
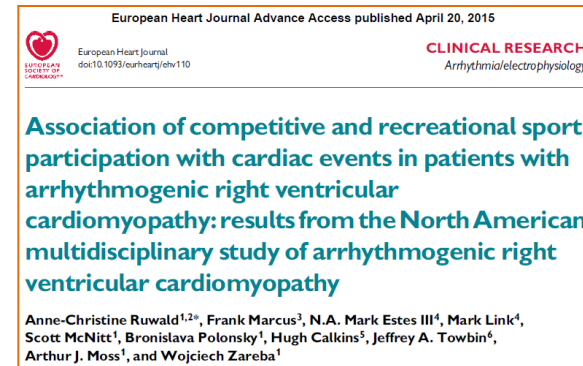
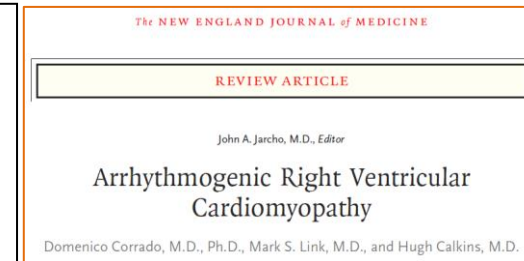
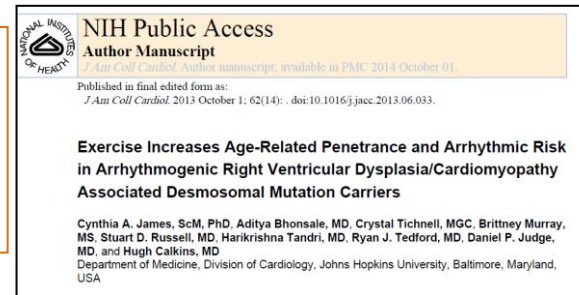
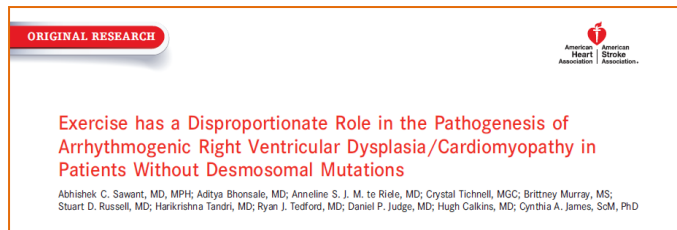
*Institute for Exercise and Environmental Medicine,
Presbyterian Hospital of Dallas,
The Baylor/UT Southwestern Sports Science Laboratory, and
The University of Texas Southwestern Medical Center at Dallas*

LEVINE, B. D. and J. STRAY-GUNDERSEN. The medical care of competitive athletes: the role of the physician and individual assumption of risk. *Med. Sci. Sports Exerc.*, Vol. 26, No. 10, pp. 1190–1192, 1994.



Shared Decision-Making ≠ Low Risk for All

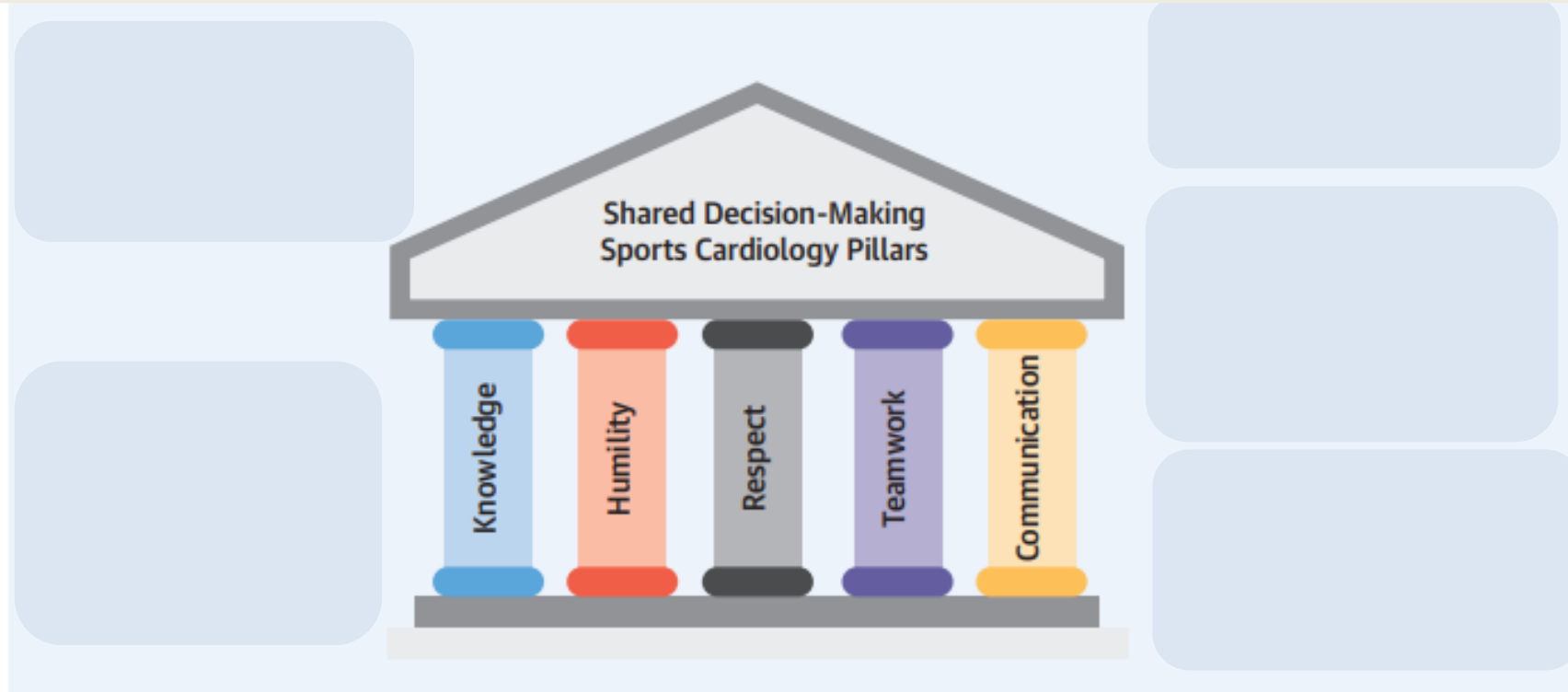
Exercise and Arrhythmogenic RV Cardiomyopathy



Exercise increases the risk of ventricular arrhythmias and worsens RV function



**4. Document: Discussed risk and potential for harm even with an AED.
Plan for surveillance → ?change in risk.**



Who makes the final decision?



Who makes the final decision?



**All parties in agreement:
Expert Provider, Patient, Family and
the University/Institution**



KNAPP v. NORTHWESTERN UNIVERSITY

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United States Court of Appeals, Seventh Circuit.

Nicholas KNAPP, Plaintiff-Appellee, v. NORTHWESTERN UNIVERSITY, an Illinois not-for-profit corporation, and Rick Taylor, Defendants-Appellants.

No. 96-3450.

Decided: November 22, 1996

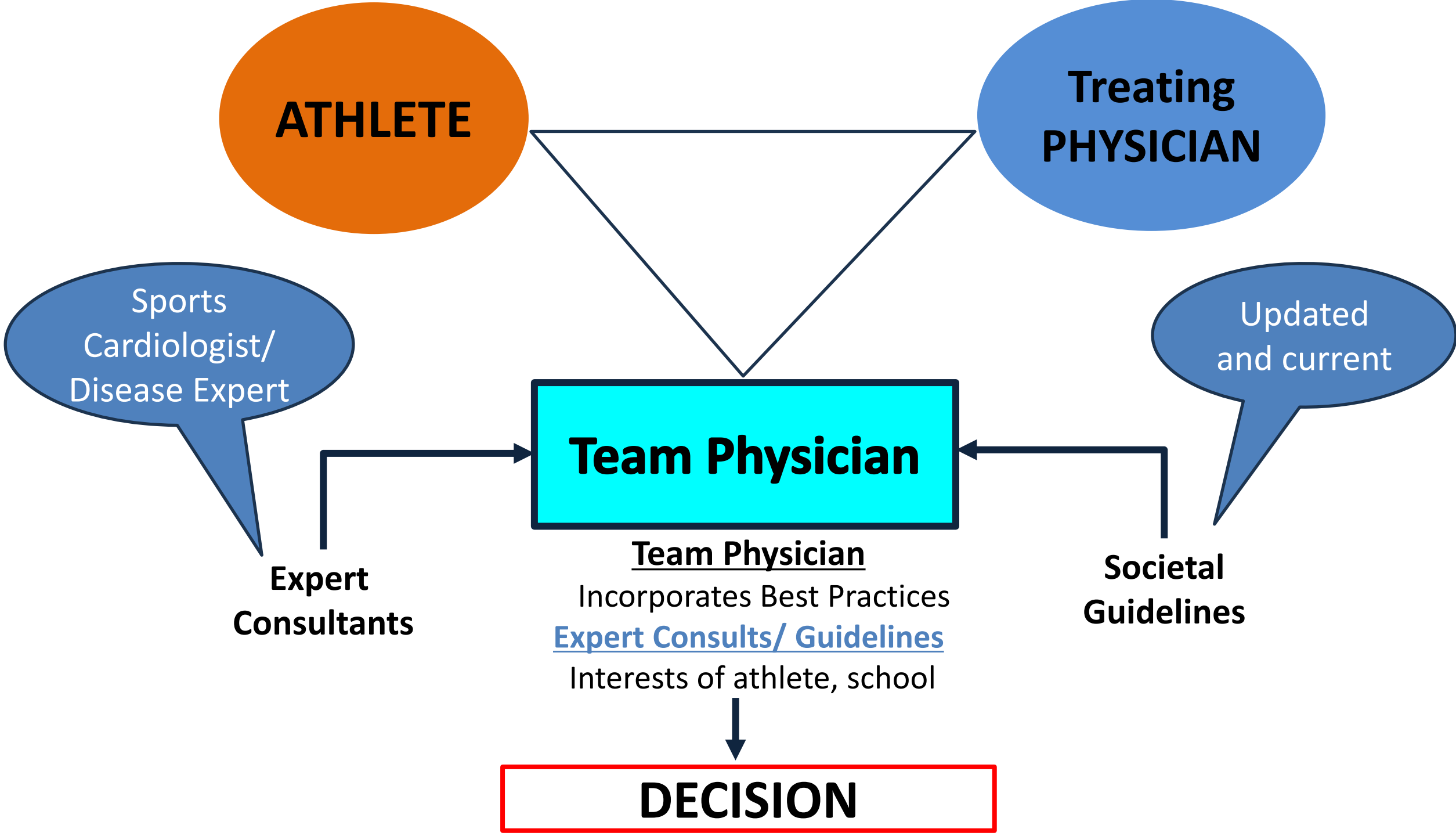
In closing, we wish to make clear that we are not saying Northwestern's decision necessarily is the right decision. We say only that it is not an illegal one under the Rehabilitation Act. On the same facts, another team physician at another university, reviewing the same medical history, physical evaluation, and medical recommendations, might reasonably decide that Knapp met the physical qualifications for playing on an intercollegiate basketball team. Simply put, all universities need not evaluate risk the same way. What we say in this case is that if substantial evidence supports the decision-maker here Northwestern-that decision must be respected.

Shared Decision Making

cases involving risk of future injury, a school's perception of the threat of such injury cannot be based on unfounded fears or stereotypes; it must be based on objective evidence. Chiari, 920 F.2d at 317. But here, where Northwestern acted rationally and reasonably rather than paternalistically, no Rehabilitation Act violation has occurred. The Rehabilitation Act "does not require a school to engage in reflexive actions to actual or perceived handicaps with actions that are not justified by the facts." Arline, 480 U.S. at 284-85, 107 S.Ct. at 1129.

Based on evidence,
expert review.
NOT – because I said so.





Evolving Science re: Cardiovascular Conditions:

- What are the risks of exercise in specific conditions?
- Consider the denominator
- Address the knowledge gaps

Circulation
JOURNAL OF THE AMERICAN HEART ASSOCIATION

The Recognition and Significance of Pathological T-Wave Inversions in Athletes
Frédéric Schnell, Nathan Riding, Rory O'Hanlon, Pierre Axel Lentz, Erwan Donal, Gaelle Kervio, David Matelot, Guillaume Leurent, Stéphane Doutreleau, Laurent Chevalier, Sylvain Guerard, Mathew G. Wilson and François Carré

Circulation, published online November 10, 2014;
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:

<http://circ.ahajournals.org/content/early/2014/11/10/CIRCULATIONAHA.114.011038>



Circulation

Volume 138, Issue 13, 25 September 2016; Pages e272-e391
<https://doi.org/10.1161/CIR.0000000000000549>



AHA/ACC/HRS GUIDELINE

2017 AHA/ACC/HRS Guideline for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death

A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society

Sana M. Al-Khatib, MD, MHS, FACC, FAHA, FHRS, William G. Stevenson, MD, FACC, FAHA, FHRS, Michael J. Ackerman, MD, PhD, William J. Blom, MD, PhD, David J. Callans, MD, FACC, FHRS, Anne B. Curtis, MD, FACC, FAHA, FHRS, Michael J. Ackerman, MD, PhD, William J. Blom, MD, PhD, David J. Callans, MD, FACC, FHRS, Anne B. Curtis, MD, FACC, FAHA, FHRS, Gregg C. Fonarow, MD, FACC, FAHA, FHFSA, Anne Christopher B. Granger, MD, FACC, FAHA, Stephen C. Har A. Hlatky, MD, FACC, FAHA, José A. Joglar, MD, FACC, FAHA, Daniel D. Matlock, MD, MPH, Robert J. Myerburg, MD, FACC, FAHA, FHRS

ORIGINAL RESEARCH

Cardiac Events During in Children and Adolescents

Kristina D. Chambers, BA, Virginia Beasly D, Vassilios Bezerides

Return-to-Play for Elite Athletes With Genetic Heart Diseases Predisposing to Sudden Cardiac Death

Katherine A. Martinez,^{1,2,3} J. Martijn Bos, MD, PhD,^{1,2,3,4,5} Aaron L. Baggish, MD,^{4,6} Dermot M. Phelan, MD, PhD,⁷ Kathryn E. Tobert, BS,² Darrel B. Newman,¹ Erica Scherer,¹ Bradley J. Petek, MD,^{1,4,6} Michael J. Ackerman, MD, PhD,^{1,2,3,4} Matthew W. Martinez, MD⁸

ABSTRACT

BACKGROUND People diagnosed with genetic heart diseases (GHDs) associated with sudden cardiac death (SCD) have historically been restricted from competitive sports. Recent data documenting return-to-play (RTP) experiences following shared decision making (SDM) suggest that cardiac event rates for athletes with a GHD are lower than previously described, thereby suggesting an opportunity to reconsider this paradigm.

OBJECTIVES The purpose of this study was to evaluate clinical outcomes among National Collegiate Athletic Association Division I university and professional athletes diagnosed with a GHD.

METHODS A multicenter retrospective analysis was performed to examine demographics, clinical characteristics, RTP outcomes, and cardiac events among elite athletes with a GHD.

RESULTS A total of 76 elite (66%, Division I, 34% professional) athletes (age 19.9 ± 5 years, 28% women) diagnosed with a GHD (hypertrophic cardiomyopathy [53%], long QT syndrome, long QT syndrome [26%]) comprise this cohort. Most athletes were asymptomatic (48 of 76, 63%) before diagnosis and had their GHD detected during routine pre-participation cardiovascular screening. Most athletes (55 of 76, 72%) were initially disqualified from their sport but subsequently opted for unrestricted RTP after comprehensive clinical evaluation and SDM. To date, (mean follow-up 7 ± 6 years), only 1 exercise-related (1.3%) and 2 nonexercise-related GHD-associated adverse cardiac events occurred. There have been no fatalities during follow-up.

CONCLUSIONS This is the first study describing the experience of athletes with a known SCD-predisposing GHD who are competing at the elite level. After careful evaluation, risk stratification, and tailoring of their GHD therapy, RTP following SDM appears associated with low, nonfatal event rates at elite levels of sport. (J Am Coll Cardiol 2023;82:661-670) © 2023 by the American College of Cardiology Foundation.

2005 Bethesda Conference criteria for long QT syndrome (LQTS) were used to identify athletes with LQTS. Long QT syndrome (LQTS) is a condition with highly variable expression and is the leading cause of sudden cardiac death in young athletes worldwide.¹ The condition is characterized by a prolonged QT interval on a 12-lead electrocardiogram (ECG) and is associated with an extraordinarily high level of risk for sudden cardiac death. Traditional methods for differentiating LQTS from other causes of prolonged QT interval (athlete's heart) have relied on parameters derived from the ECG, including QT interval, QTc interval, and QT dispersion. However, such algorithms may not be diagnostic in all cases.

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Cardiomyopathies

Clinical Profile of Athletes With Hypertrophic Cardiomyopathy

Nabeel Sheikh, MRCP; Michael Papadakis, MD; Frédéric Schnell, PhD; Vasileios Panoulas, MD, PhD; Anil Malhotra, MRCP; Mathew Wilson, PhD; François Carré, PhD; Sanjay Sharma, MD

Background—The phenotype of individuals with hypertrophic cardiomyopathy (HCM) who exercise regularly is unknown. This study characterized the clinical profile of young athletes with HCM.

Methods and Results—The electrical, structural, and functional cardiac parameters from 106 young (14–35 years) athletes with HCM were compared with 101 sedentary HCM patients. A subset of athletes with HCM exhibiting morphologically mild (13–16 mm), concentric disease was compared with 55 healthy athletes with mild physiological left ventricular hypertrophy (LVH). Most athletes with HCM (96%) exhibited T-wave inversion and had milder LVH (15.8±3.4 mm versus 19.7±6.5 mm, $P<0.001$), larger left ventricular cavity dimensions (47.8±6.0 mm versus 44.3±7.7 mm, $P<0.001$), and superior indices of diastolic function (average E/E' 7.9±2.4 versus 10.7±3.9, $P<0.001$) compared with sedentary HCM patients. In athletes with HCM, LVH was frequently (36%) confined to the apex and only 15 individuals (14%) exhibited mild concentric LVH mimicking physiological LVH. In these 15 athletes, conventional structural and functional cardiac parameters showed modest sensitivity and specificity for differentiating HCM from physiological LVH: 13% had a left ventricular cavity >54 mm, 87% had a left atrium ≤40, and 100% had an E/E' <12.

Conclusions—Athletes with HCM exhibit less LVH, larger left ventricular cavities, and normal indices of diastolic function compared with sedentary patients. Only a minority of athletes with HCM constitute the conventional gray zone of mild, concentric LVH. In this minority, conventional echocardiographic parameters alone are insufficient to differentiate HCM from physiological LVH and should be complemented by additional structural and functional assessments to minimize the risk of false reassurance. (Circulation. 2016;133:1000–1008)

Key Words: echocardiography

THE NEW ENGLAND JOURNAL OF MEDICINE

ORIGINAL ARTICLE

Outcomes in Athletes with Marked ECG Repolarization Abnormalities

Antonio Pelliccia, M.D., Fernando M. Di Paolo, M.D., Filippo M. Quattrini, M.D., Cristina Basso, M.D., Franco Culasso, Ph.D., Gloria Popoli, M.D., Rosanna De Luca, M.D., Antonio Spataro, M.D., Alessandro Biffi, M.D., Gaetano Thiene, M.D., and Barry J. Maron, M.D.

ABSTRACT

BACKGROUND Young, trained athletes may have abnormal 12-lead electrocardiograms (ECGs) without evidence of structural cardiac disease. Whether such ECG patterns represent the initial expression of underlying cardiac disease with potential long-term adverse consequences remains unresolved. We assessed long-term clinical outcomes in athletes with ECGs characterized by marked repolarization abnormalities.

METHODS From a database of 12,550 trained athletes, we identified 81 with diffusely distributed and deeply inverted T waves (≥2 mm at least three leads) who had no apparent cardiac disease and who had undergone serial clinical, ECG, and echocardiographic studies for a mean (±SD) of 9.7 years (range, 1 to 27). Comparisons were made with 229 matched control athletes with normal ECGs from the same database.

RESULTS Of the 81 athletes with abnormal ECGs, 5 (6%) ultimately proved to have cardiomyopathies, including one who died suddenly at the age of 24 years from clinically undetected arrhythmogenic right ventricular cardiomyopathy. Of the 80 surviving athletes, clinical and phenotypic features of hypertrophic cardiomyopathy developed in 3 after 12±5 years (at the ages of 27, 32, and 50 years), including 1 who had an aborted cardiac arrest. The fifth athlete demonstrated dilated cardiomyopathy after 9 years of follow-up. In contrast, none of the 229 athletes with normal ECGs had a cardiac event or received a diagnosis of cardiomyopathy 9±3 years after initial evaluation ($P=0.001$), classified by 2 electrophysiologists.

and benefits of sports participation in athletes with HCM and details the challenges and limitations of shared decision-making when all parties may not agree.

observational ICU sports safety registry quantified risks associated with sports participation for athletes receiving ICDs on the basis of standard criteria. Initial results (2013³) demonstrated no death, failure to defibrillate, or injury resulting from arrhythmia or shock during sports. On the basis of these data, the 2015 eligibility and disqualification recommendations for competitive athletes with cardiovascular disease⁴ now state that

participating in competitive sports is contraindicated for athletes with a known SCD-predisposing GHD who are competing at the elite level. After careful evaluation, risk stratification, and tailoring of their GHD therapy, RTP following SDM appears associated with low, nonfatal event rates at elite levels of sport. (J Am Coll Cardiol 2023;82:661-670) © 2023 by the American College of Cardiology Foundation.

questionnaires and medical records. Median age was 38 years (19–53 years). Median follow-up period was 67 months (11–249 months). Idiopathic ventricular fibrillation (VF) was the most frequent indication (20%) for implantation. There were 22 patients (31%) who experienced 12 shock episodes, of which 112 were appropriate; 15% of shocks occurred during physical

LQTS

Return to play? Athletes with congenital long QT syndrome

Jonathan N Johnson,¹ Michael J Ackerman^{1,2,3}

BJSM 2013

- Low rate of cardiac events and no deaths in over 650 athlete-years of follow-up

Sports Participation in Genotype Positive Children With Long QT Syndrome

Peter F. Aziz, MD,* Tammy Sweeten, MS,† Ramon L. Vogel, MD,† William J. Bonney, MD,†
Jacqueline Henderson, RN,† Akash R. Patel, MD,† Maully J. Shah, MBBS†

JACC 2015

- No cardiac events and no deaths in **treatment-compliant** children with LQTS in 755 patient-years of follow-up

Shared Decision-Making in Cardiovascular Disease



EXERCISE IN GENETIC CARDIOVASCULAR DISEASE (LIVE-HCM)

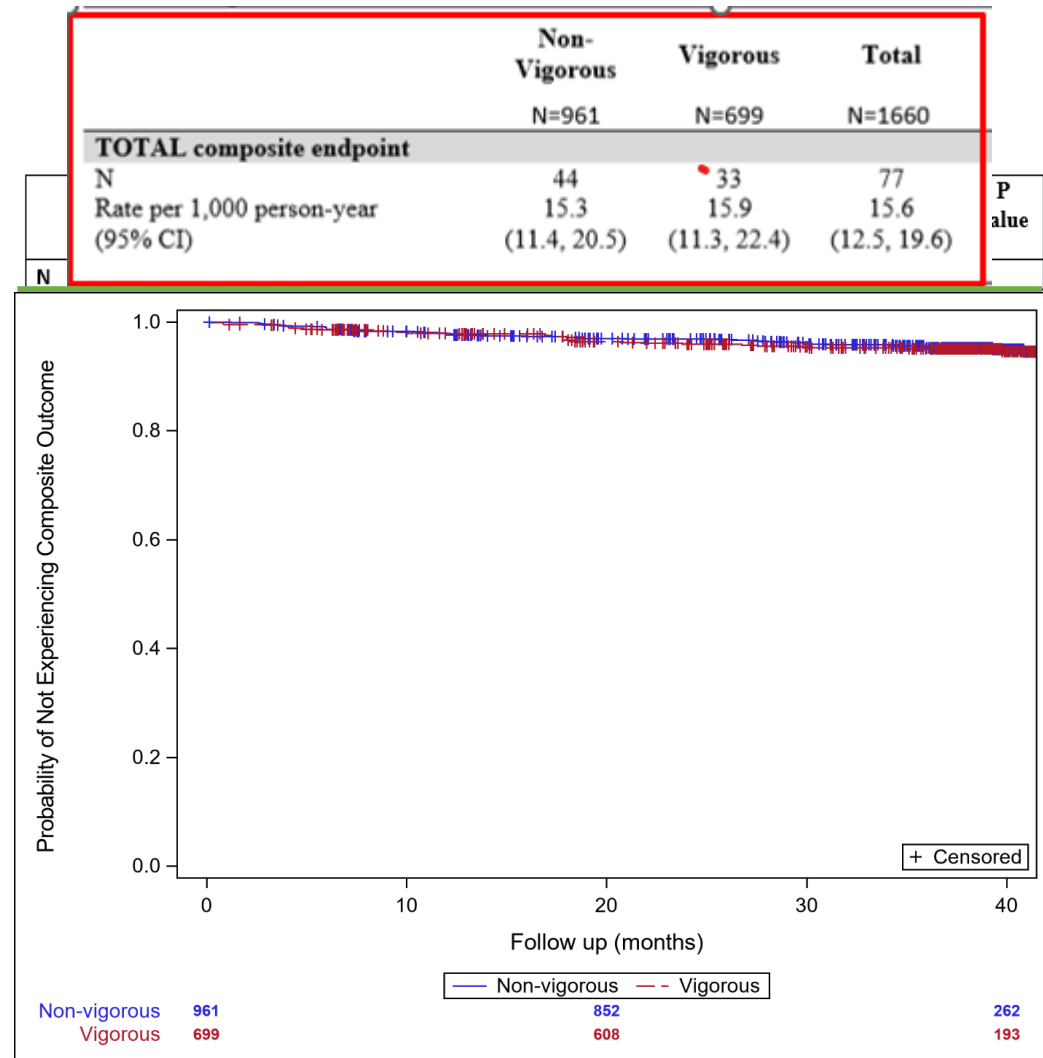
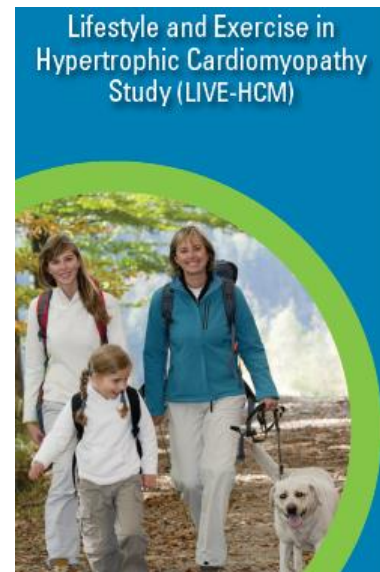
Aim 1: Incidence **arrhythmic events** over 3 years
Comparison moderate or vigorous exercisers vs sedentary

Aim 2: **Quality of life**
Comparison moderate or vigorous exercisers vs sedentary

Age 8-60 years, with OR without ICD
Any level exercise

3 years of follow up

NIH R01 HL125918-01

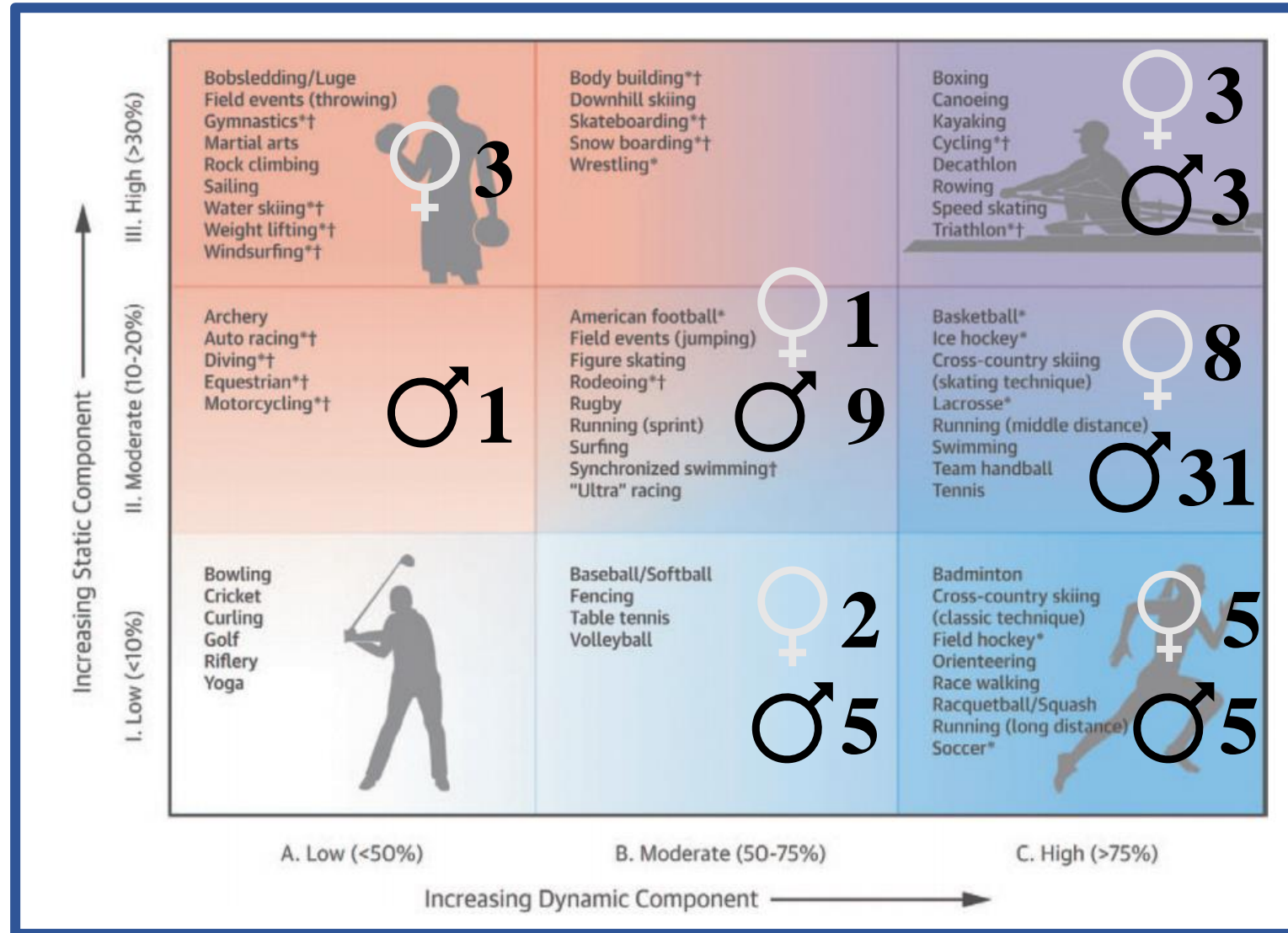


RTP for Elite Level Athletes With Sudden Cardiac Death Predisposing Heart Conditions

Demographics	Total Cohort
Total Number of Athletes	76
Mean Age at Return-to-Play (years)	19.9 ± 5.0
Female (%)	21 (28)
Race (%)	
White	38 (50)
Black	28 (37)
Not Disclosed/Unknown	4 (5)
Other	2 (3)
Asian	1 (1)
Hispanic	1 (1)
Indian	1 (1)
Pacific Islander	1 (1)
Diagnosis (%)	
HCM	40 (53)
LQTS	20 (26)
DCM	5 (7)
Other	5 (7)
ARVC	4 (5)
IVF	2 (3)
Symptomatic Prior to Diagnosis (%)	28 (37)
Syncope	10 (13)
Other	6 (8)
SCA	6 (8)
Irregular Heart Rate / Palpitations	4 (5)
Sustained VT	2 (3)

- 49 (64%) Division I and 27 (36%) professional athletes.
- 55 athletes (72%) were initially disqualified but opted to RTP after comprehensive clinical evaluation and SDM.
- 73 out of 76 athletes (96%) chose to RTP.
- 1 patients (1.3%) had ≥ 1 breakthrough cardiac event (BCE) with exercise, 2 (2.6%) without exercise.
- NO deaths.

Return-to-Play for Elite Athletes With Genetic Heart Diseases Predisposing to Sudden Cardiac Death



Return-to-Play for Elite Athletes With Genetic Heart Diseases Predisposing to Sudden Cardiac Death

Demographics	Total Cohort
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Conclusion:

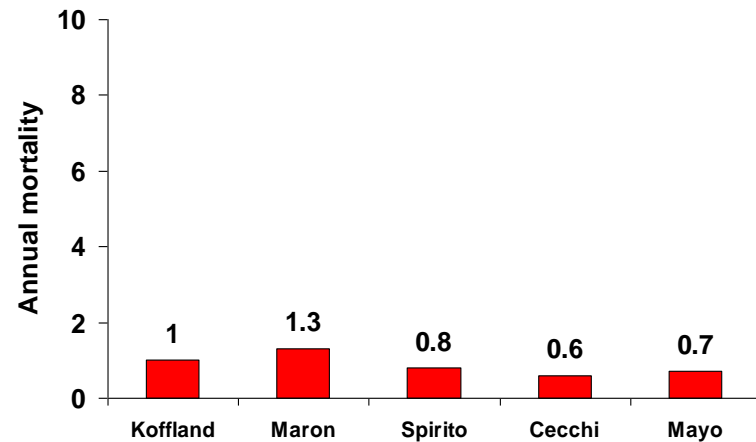
After careful evaluation by an expert, risk stratification, and use to SDM, an exercise plan can be put into place for Division I and professional athletes to RTP.

breakthrough cardiac event (BCE) with exercise, 2 (2.6%) without exercise.

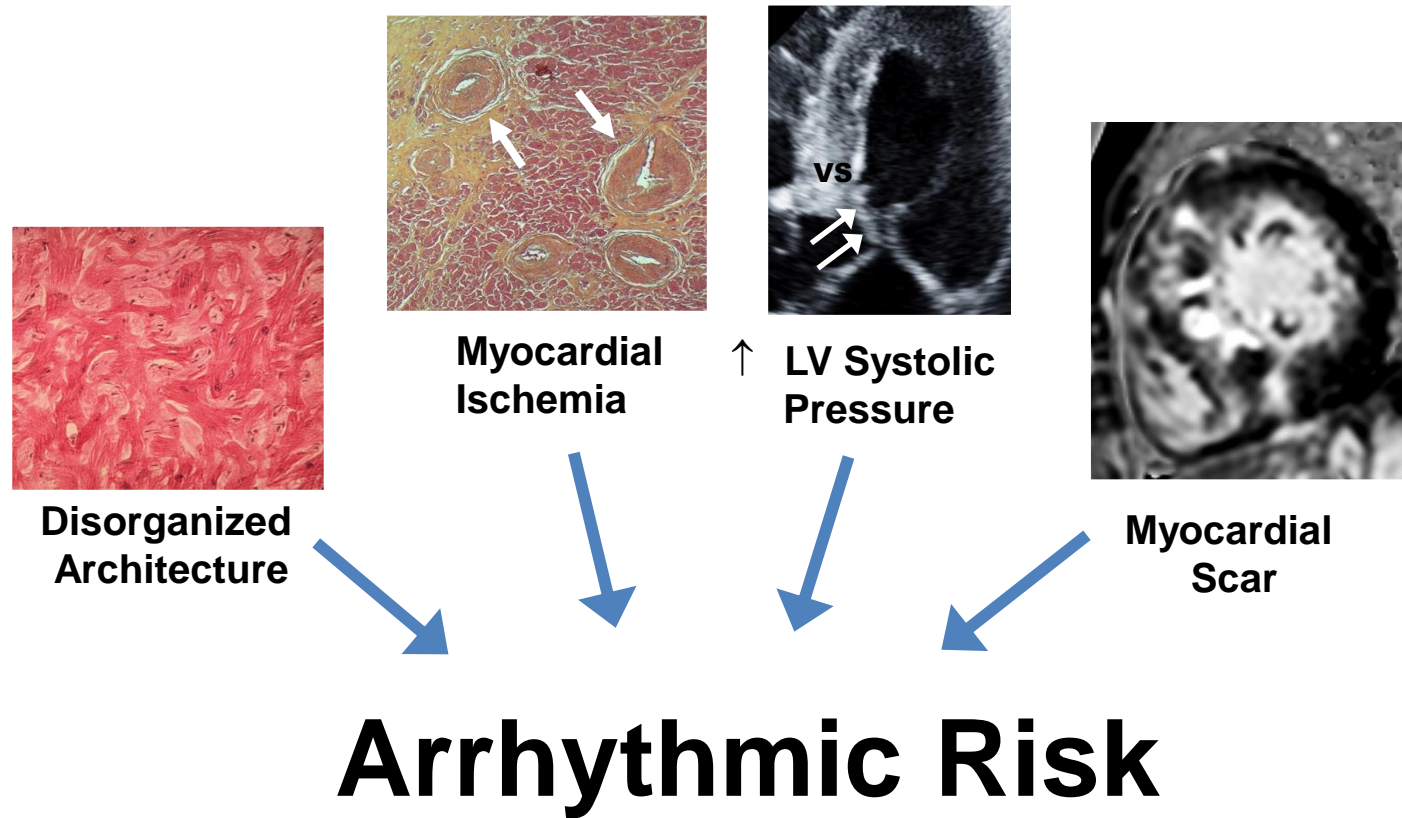
- NO deaths.

Clinical Dilemma

Low Annual Mortality Overall



HCM



Clinical Risk Factors for HCM Sudden Cardiac Arrest



Family history of sudden death from HCM

Massive LVH ($>30\text{mm}$)

Unexplained syncope

HCM w/ LV systolic dysfunction ($\text{EF} < 50\%$)

LV apical aneurysm

Extensive LGE on CMR

NSVT on ambulatory monitor

Enhanced American College of Cardiology/American Heart Association Strategy for Prevention of Sudden Cardiac Death in High-Risk Patients With Hypertrophic Cardiomyopathy

Martin S. Maron, MD; Ethan J. Rowin, MD; Benjamin S. Wessler, MD; Paula J. Mooney, RN; Amber Fatima, MD; Parth Patel, MD; Benjamin C. Koethe, MPH; Mikhail Romashko, MD; Mark S. Link, MD; Barry J. Maron, MD

2019

- 2094 patients with HCM. Observational data. Single center
- Mean follow-up 4.7 years
- 12 of 1567 (0.8%) had SCA
- 527 received primary prevention ICDs
- Very low annual mortality. 99% survival without ICD
- 16% experienced device therapy–terminated VT/VF;
- 20 patients w/ ICD interventions -> 85% had LGE

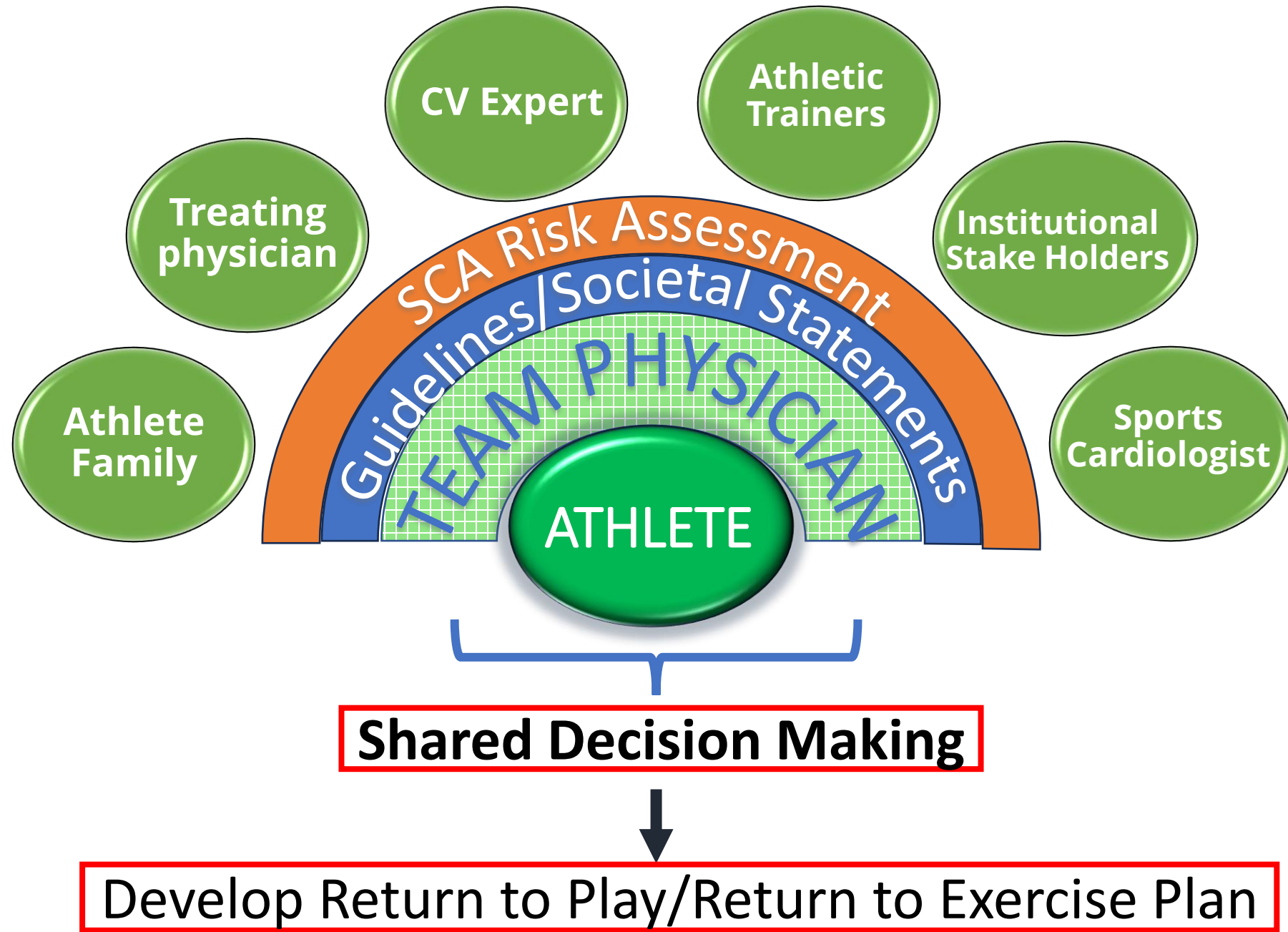
Risk stratification and prevention model averted nearly all SCD

Enhanced ACC/AHA clinical risk factor strategy for predicting SCD events was 95% sensitive

2020 AHA/ACC Guideline for the Diagnosis and Treatment of Patients With Hypertrophic Cardiomyopathy

A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines

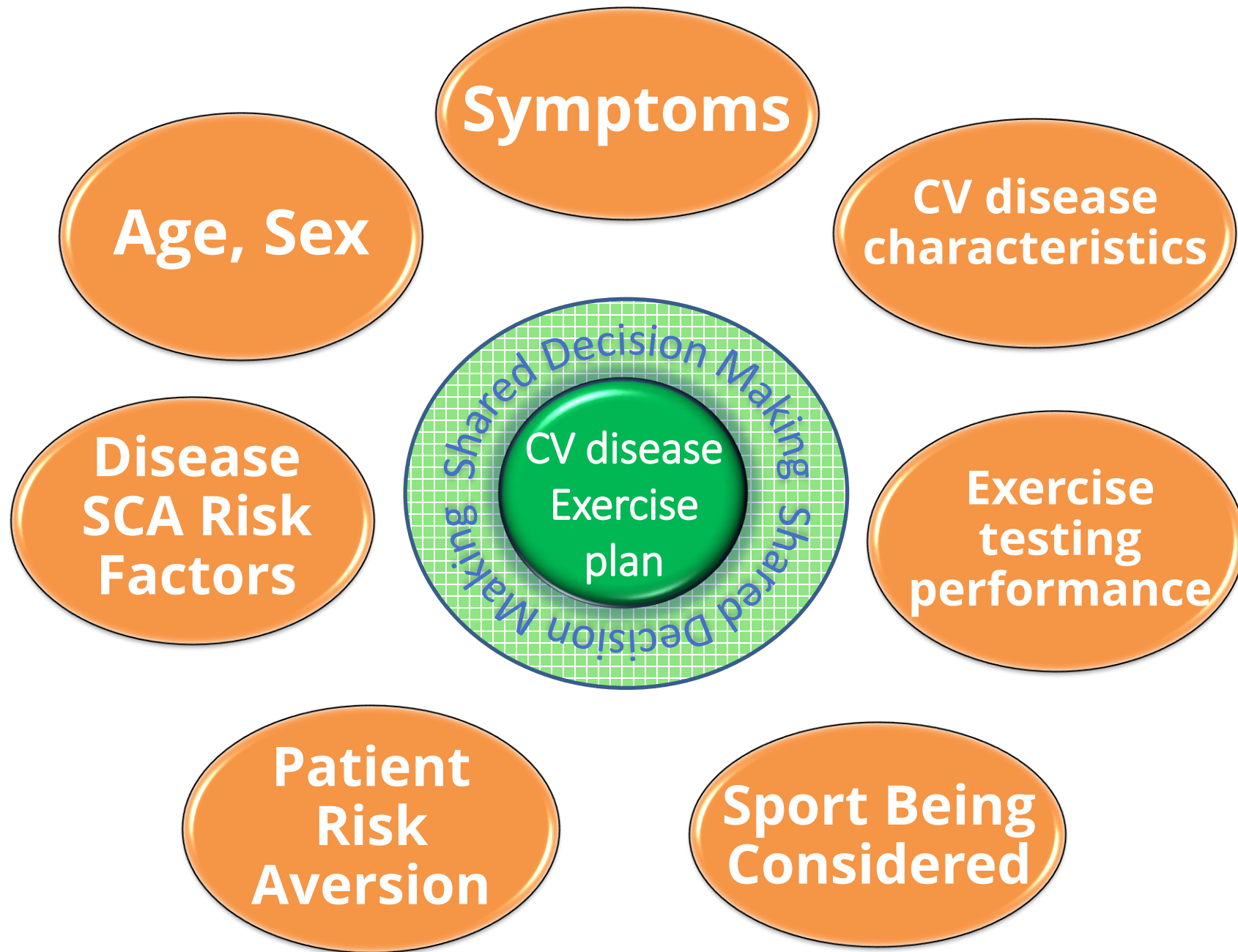
COR	LOE	RECOMMENDATIONS
1	B-NR	1. For most patients with HCM, mild- to moderate-intensity recreational* exercise is beneficial to improve cardiorespiratory fitness, physical functioning, and quality of life, and for their overall health in keeping with physical activity guidelines for the general population (1-3).
1	C-EO	2. For athletes with HCM, a comprehensive evaluation and shared discussion of potential risks of sports participation by an expert provider is recommended (4).
2a	C-EO	3. For most patients with HCM, participation in low-intensity competitive sports is reasonable (5,6).
2b	C-LD	5. For patients with HCM, participation in high-intensity recreational activities or moderate- to high-intensity competitive sports activities may be considered after a comprehensive evaluation and shared discussion, repeated annually with an expert provider who conveys that the risk of sudden death and ICD shocks may be increased, and with the understanding that eligibility decisions for competitive sports participation often involve third parties (c.g., team physicians, consultants, and other institutional leadership) acting on behalf of the schools or teams (4,7-11).



Playing with HCM

- Patient/player autonomy in health care decisions is important
- Absolute risk is difficult to quantify for all
- Risk avoidance/acceptance is individual decision
- Potential harms of disqualification
 - Loss of self-identity
 - Loss of scholarship/education
 - Loss of income
 - Depression/mental health concerns



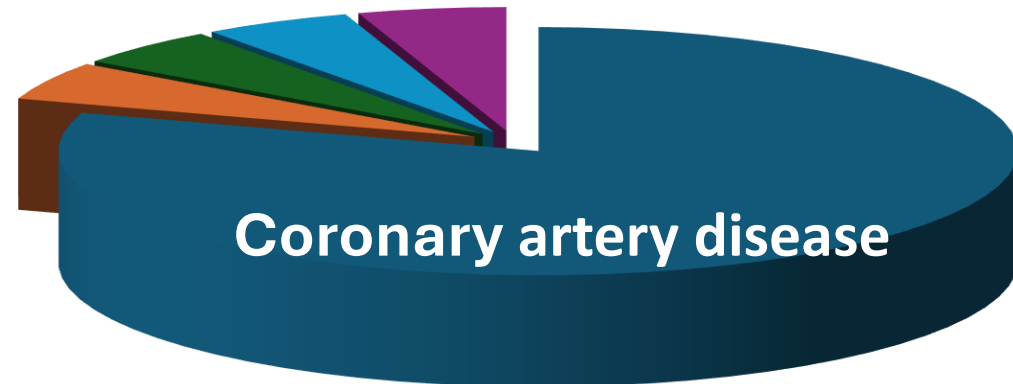
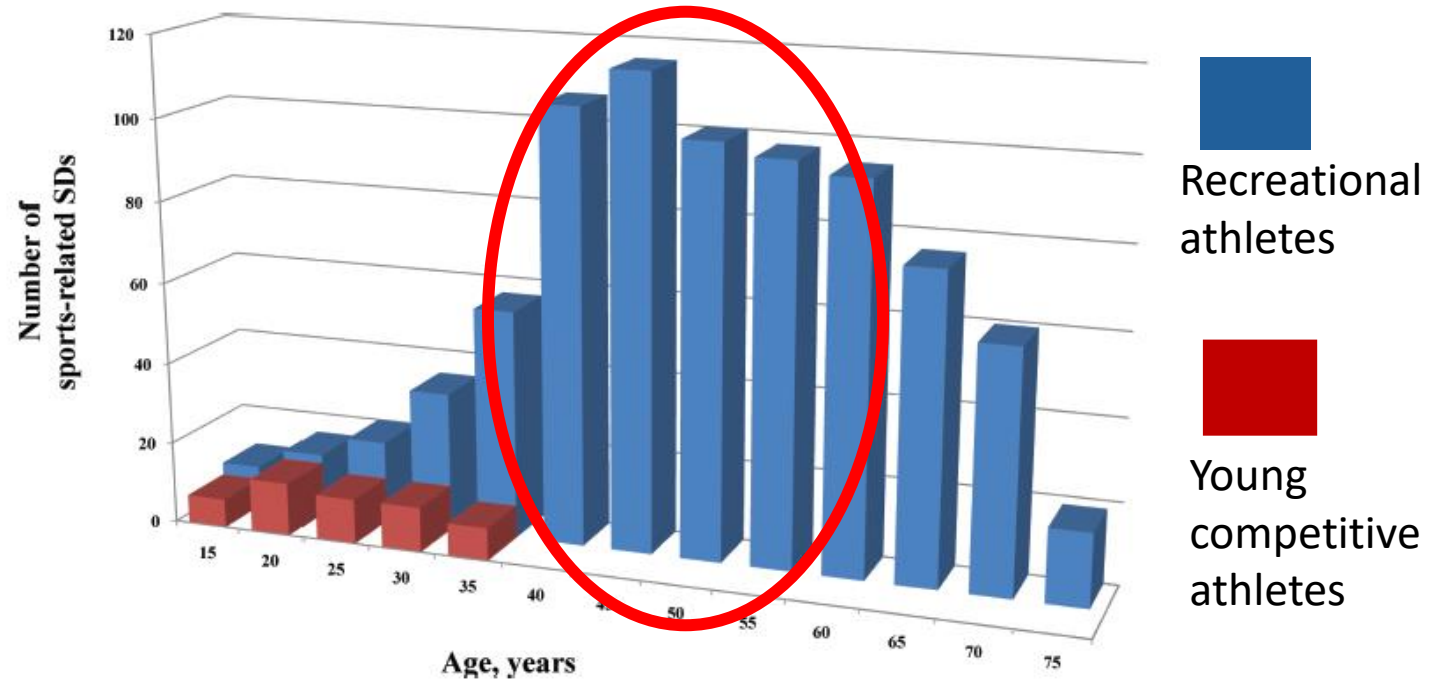




The Exercise Paradox



Can trigger myocardial infarction and promote fatal arrhythmias in predisposed individual



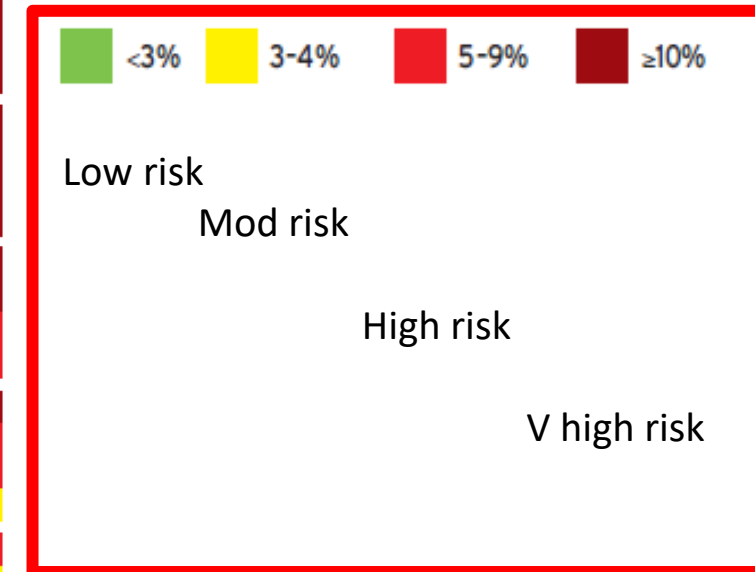
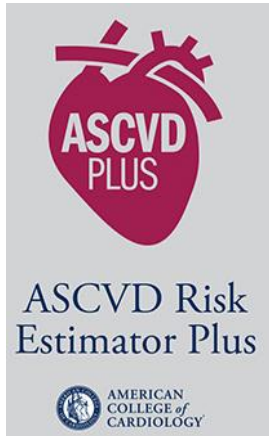
Causes of exercise related SCD in the community

Identifying Individuals at High Risk of Coronary Artery Disease

Symptoms

Habitual activity level

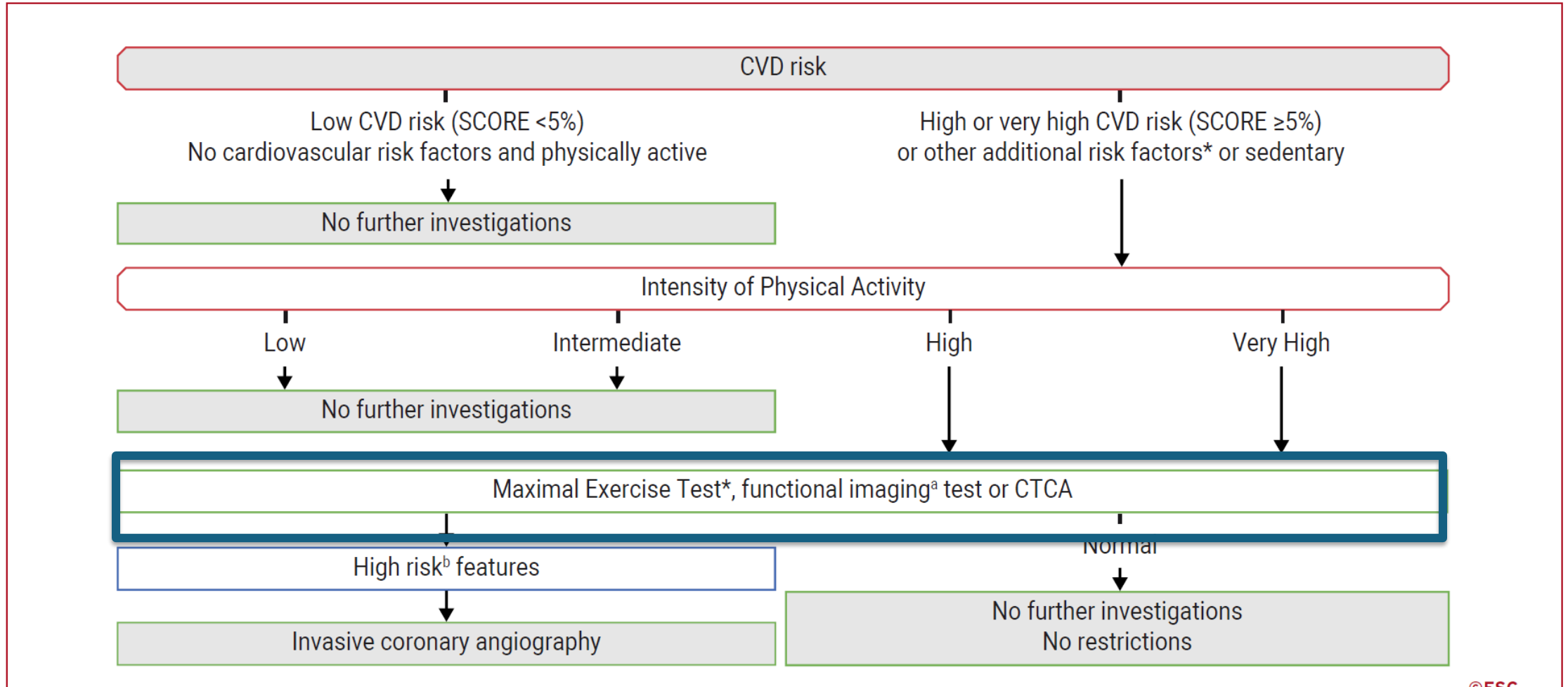
10-year risk



Caveats:

Not validated in Masters athlete
CRF not included in these equations
May overestimate risk in athletes

Proposed algorithm for pre-participation cardiovascular assessment in individuals aged >35 years



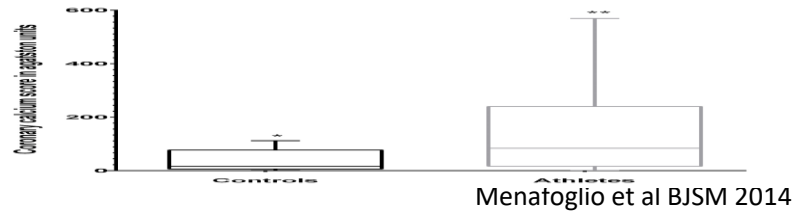
Exercise Stress Testing

Good prognostic value in individuals with angina

Low sensitivity and specificity in asymptomatic individuals

Symptomatic Sens 68% Spec 77%

Asymptomatic Sens 46% Spec 16%



76 athletes had an exercise test

3 already had a diagnosis of cardiac disease.

4 (5.5%) had a positive test.

Subsequent investigation in all 4 failed to show significant coronary artery disease

False positive rate 100%

The Diagnostic Accuracy of Exercise Electrocardiography in Recreational and Competitive Athletes

Van de Sande. Scan J Med Sci Sport 2015

1298 athletes (45 ± 13 years); 88% male

Exercised for 6.6 ± 3.7 hr/week

Low atherosclerotic risk profile

53 (4.1%) positive exercise tests

Data regarding further investigation in 38 athletes (72%):
MPS in 35 and CTCA in 3

False positive rate 95%

Coronary Artery Calcium (CAC)/ Coronary Computed Tomography Angiography (CCTA)



- CAC is a surrogate for atherosclerotic volume and a robust predictor of CV morbidity and mortality over and above traditional risk factors.

- CAC score is used for risk stratification and intensifying therapy for recognised risk factors for atherosclerosis.

- CCTA determines the magnitude of atherosclerotic coronary artery disease distribution and quantitative and qualitative plaque analysis.

- Increasing utilization of CCTA in middle-aged endurance athletes



Athletes with possible or known CV disease

Assess Symptoms



Assess Risk



Manage Risk



- Blood pressure
- Lipid panel & Novel risk markers
- HgbA1c
- Smoking status
- Family history of early CAD (M <55 yo; F <65 yo)
- Supplement use (e.g., testosterone)

CAC Score:

- If they already have it...
- If not, discuss the pros/cons

Fitness, Coronary Calcification and Cardiac Events

8425 men without cardiovascular disease

Cardiorespiratory fitness assessment

+

Coronary artery calcium (CAC) score



Follow up 8.4 years

Deaths, Myocardial infarction and stroke in individuals with 4 categories of CAC

CAC score 0 CAC score 1-99 CAC score 100-399 CAC score ≥ 400

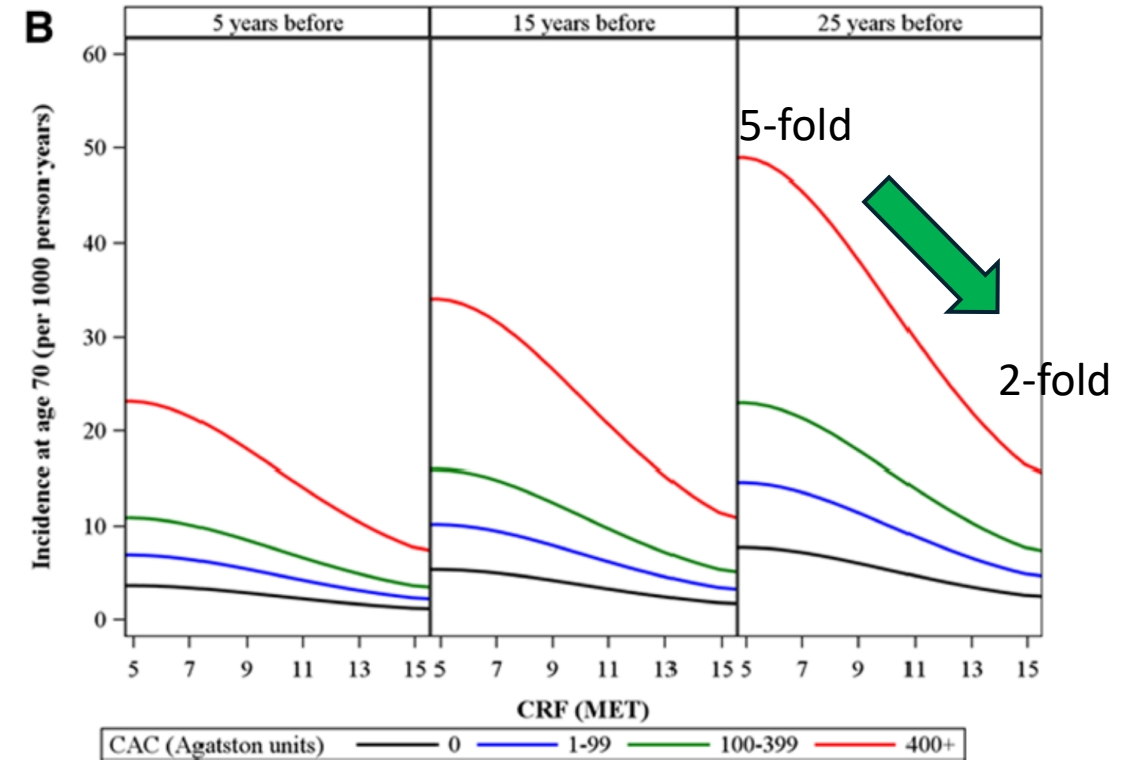
0.9

2.0

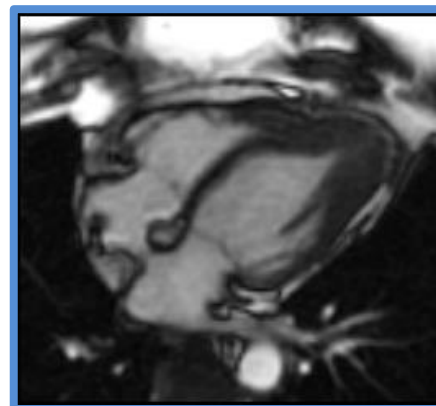
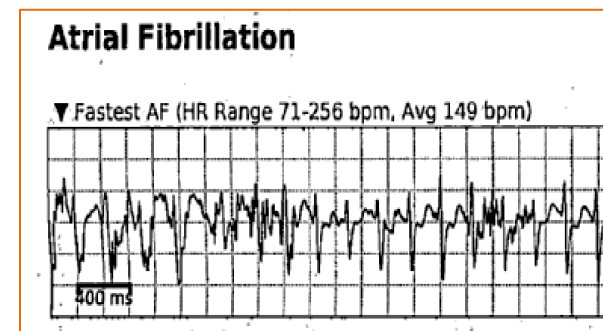
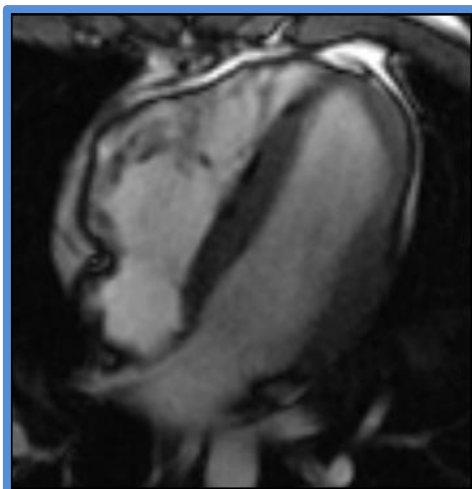
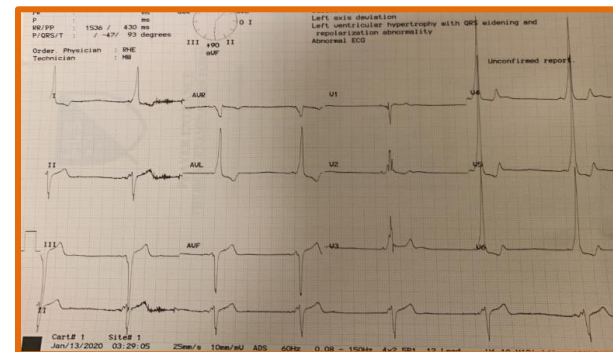
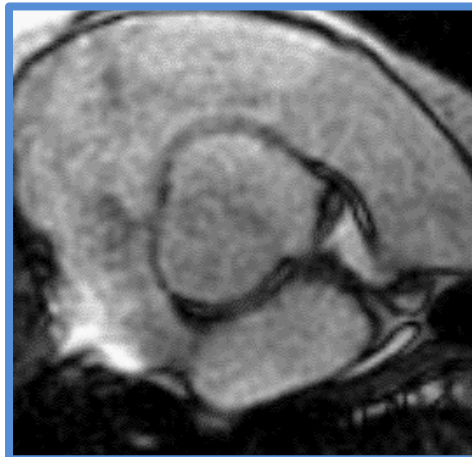
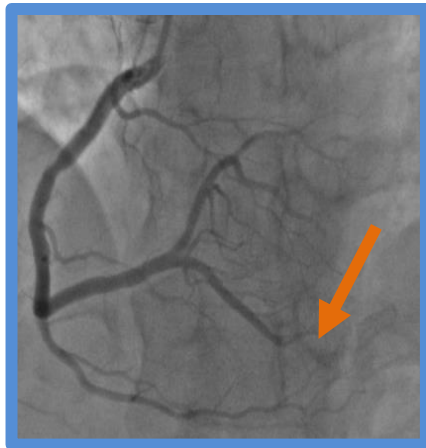
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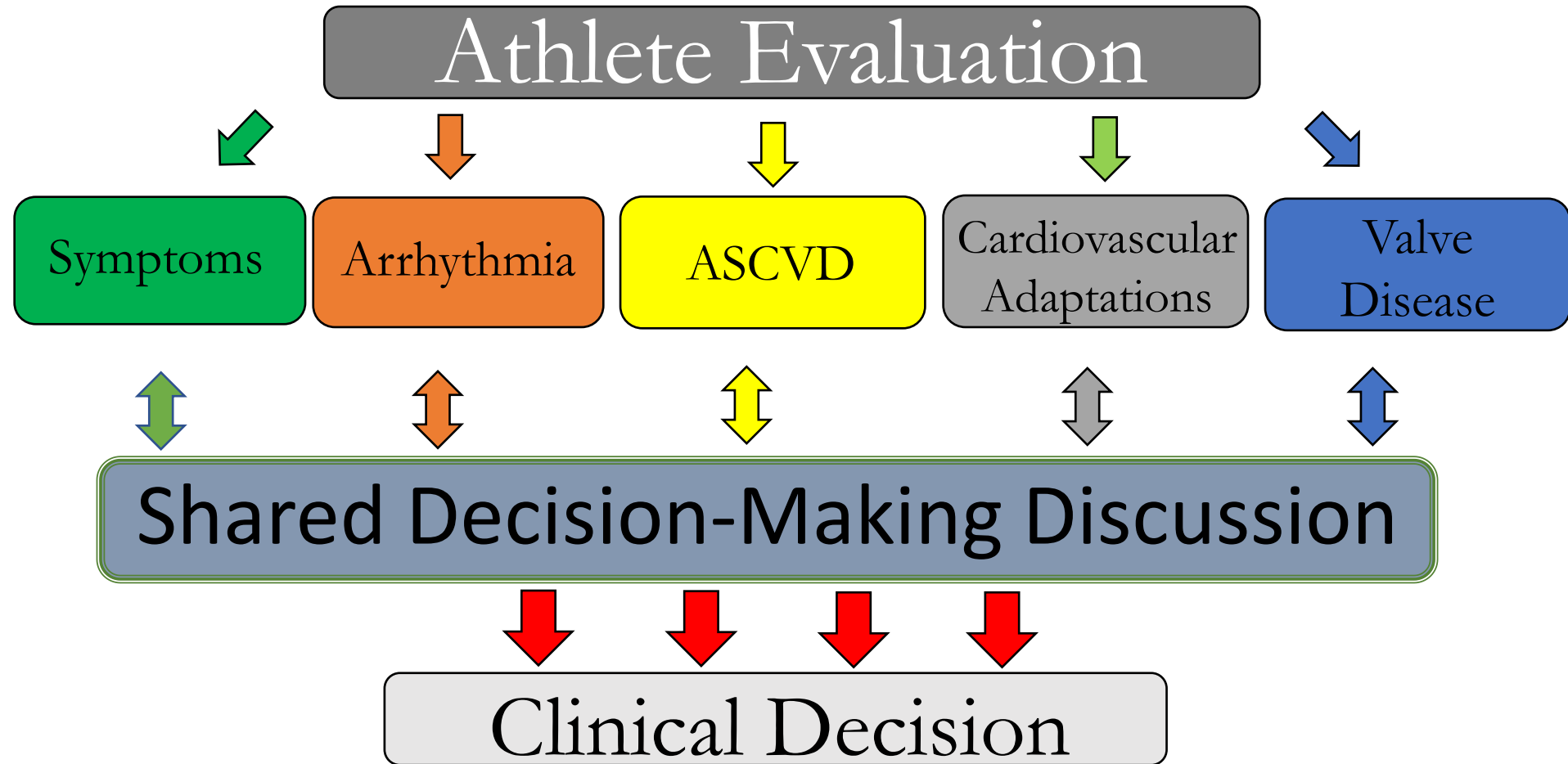
5.9

Hard event rates per 1000 person years



The risk of hard CV events decreased 14% for each MET increase across all CAC scores adjusted for risk factors





Case Presentation

17 y.o elite Caucasian Female
American Soccer midfielder

Asymptomatic.

No prior syncope, CV limitations.

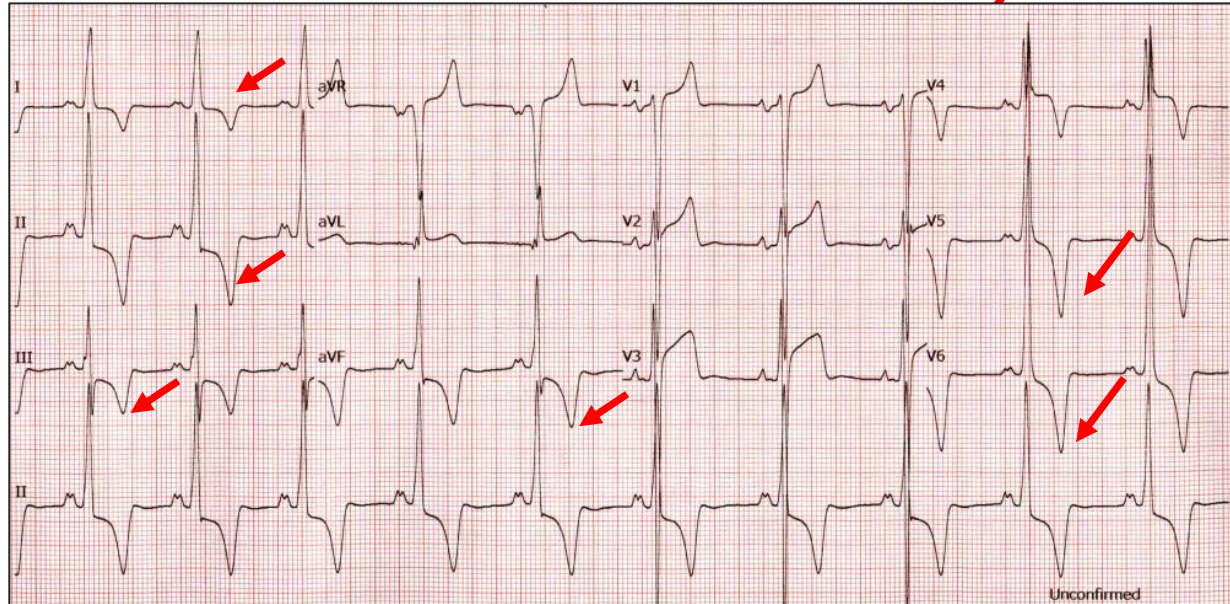
No family hx of cardiomyopathy or sudden cardiac arrest.

Preparticipation screening evaluation including ECG.

ECG was abnormal.



Electrocardiogram



Sinus bradycardia
Normal QTc
TWI II, III, aVF, V3-V6

No prior ECG

Referred for ECHO



ECHO

- Normal biventricular size and function.
- LVEF = 65%.
- Normal wall motion. No valve disease: SAM or MVP. Abnormal diastolic function
- 17mm septal thickness.
- **Exercise Echo** – 16minutes + sprints; no arrhythmias or obstruction, Normal HR and BP response
- **48hr Ambulatory monitor**: Rare PVCs



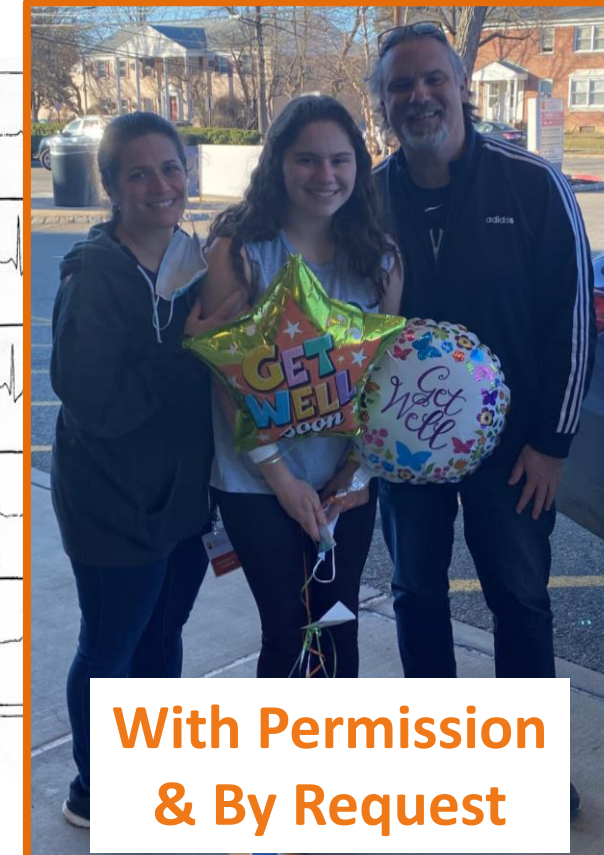
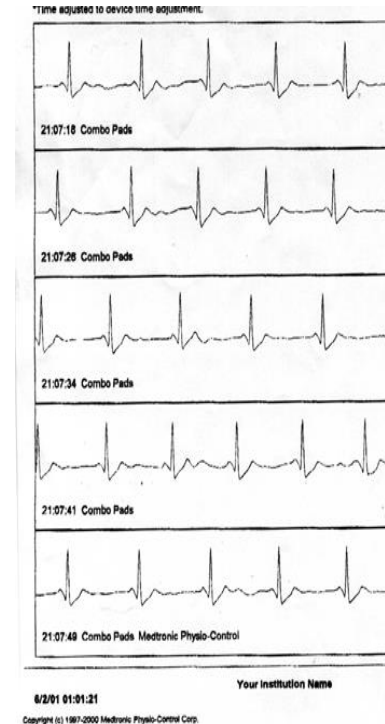
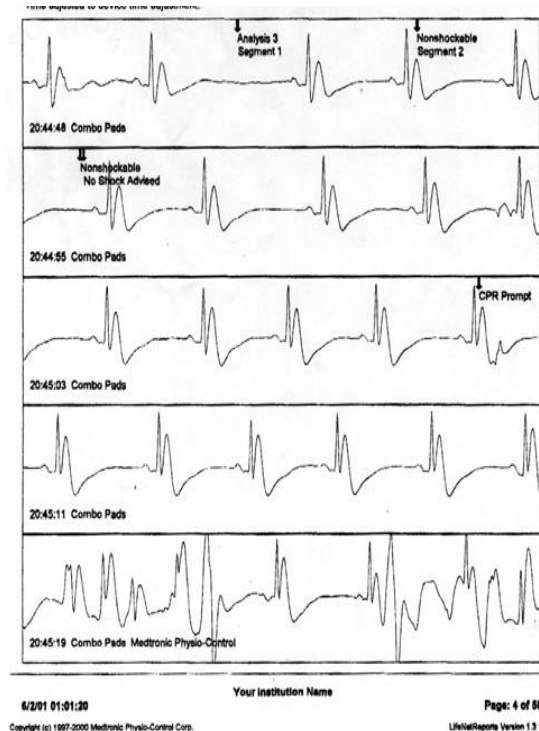
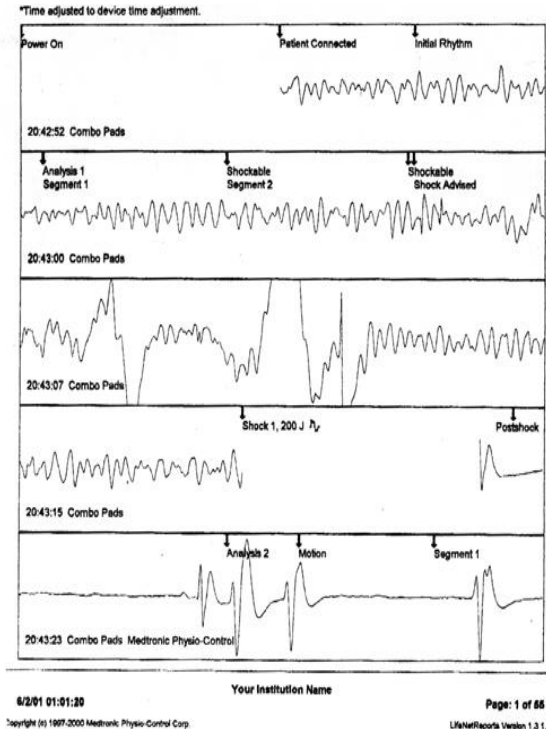
Case

20 y.o Caucasian Female Soccer
No high risk features.

- We discussed her risk and options
- We discussed her current low-risk features
- Importance of ongoing annual surveillance
- After several discussions:
 - She decided not to play

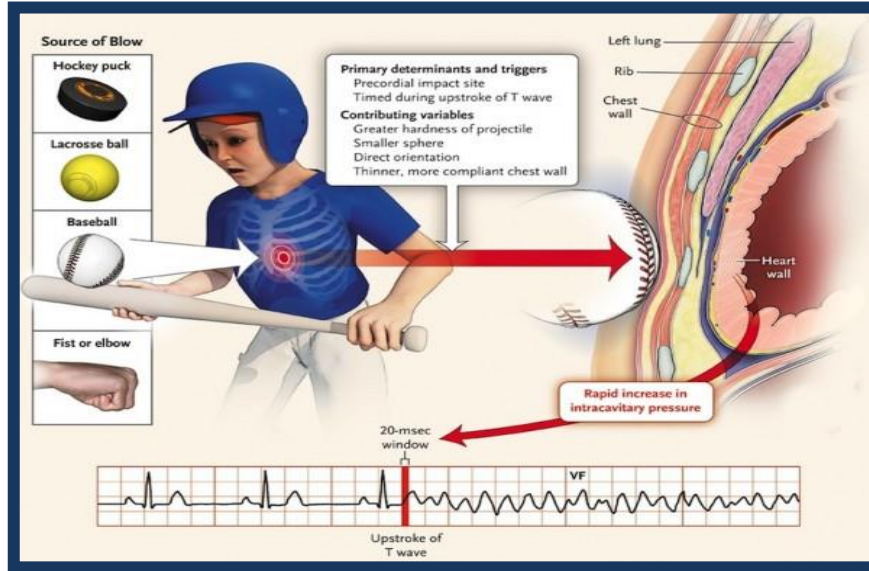


We identify patients at LOW risk for sudden death



Patients at LOW risk for sudden death can still die suddenly

NO perfect evaluation process



Initial Arrhythmic Event



Low Risk
No Risk



Sudden Cardiac Arrest Management

Pre-participation assessments are never perfect

EAP and AED are still important

Signs of cardiac arrest:

Sudden non-contact collapse, passed out athlete





ORCCA

OUTCOMES REGISTRY FOR
CARDIAC CONDITIONS IN ATHLETES



DO YOU KNOW A COLLEGE, SEMI-PROFESSIONAL
OR PROFESSIONAL ATHLETE DIAGNOSED WITH A
CARDIAC CONDITION?



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Thank you

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