The Heart of a Champion

Daniel J. O'Rourke, MD, MS, MEd

October 1999

February 2024

Outline

Exercise

- Types of Exercise
- Physical Activity & Health
- Physiologic Effects

Athlete's Heart

- Cardiovascular Effects
- Clinical Findings
- Gender Differences

Hypertrophic Cardiomyopathy

- Genetics
- Pathophysiology
- Clinical Findings
- Screening & Diagnostic Evaluation
- Treatment

Sudden Cardiac Death in the Athlete

- Epidemiology
- Etiologies
- Screening
- Prevention

Types of Exercise – Improved Health

Endurance (Aerobic)

- Brisk walking or jogging
- Dancing
- Swimming
- Biking
- Playing tennis or basketball

Strength (Anaerobic/Resistance)

- Lifting weights
- Carrying groceries
- Arm curls
- Push-ups
- Using a resistance band

Balance

- Tai Chi
- Standing on one foot
- Heel-to-Toe walk
- Standing from a seated position

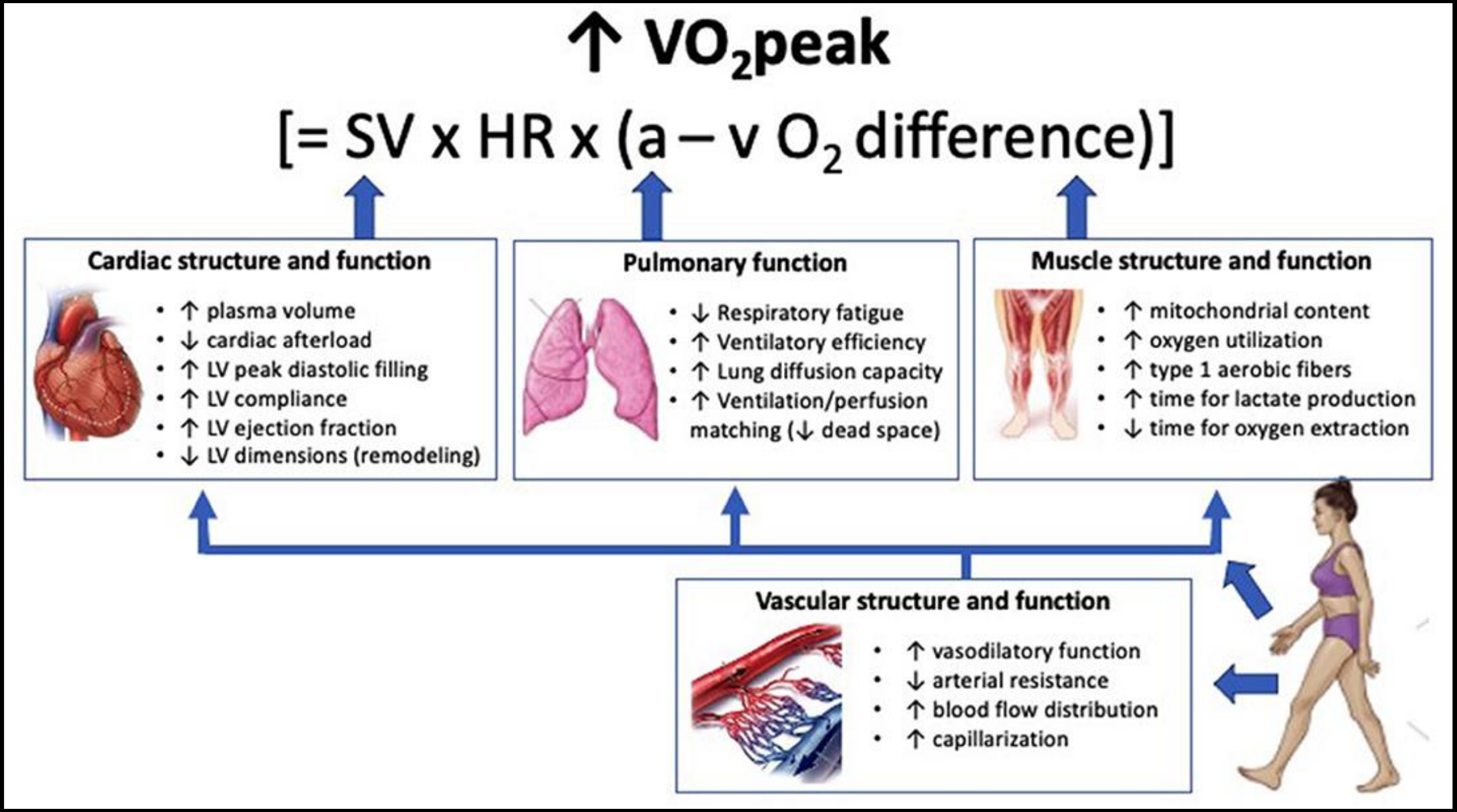
Flexibility (Stretching)

- Torso
- Back
- Quads
- Gluteals

https://www.nia.nih.gov/health/exercise-and-physical-activity/four-types-exercise-can-improve-your-health-and-physical#endurance

Exercise Physiology

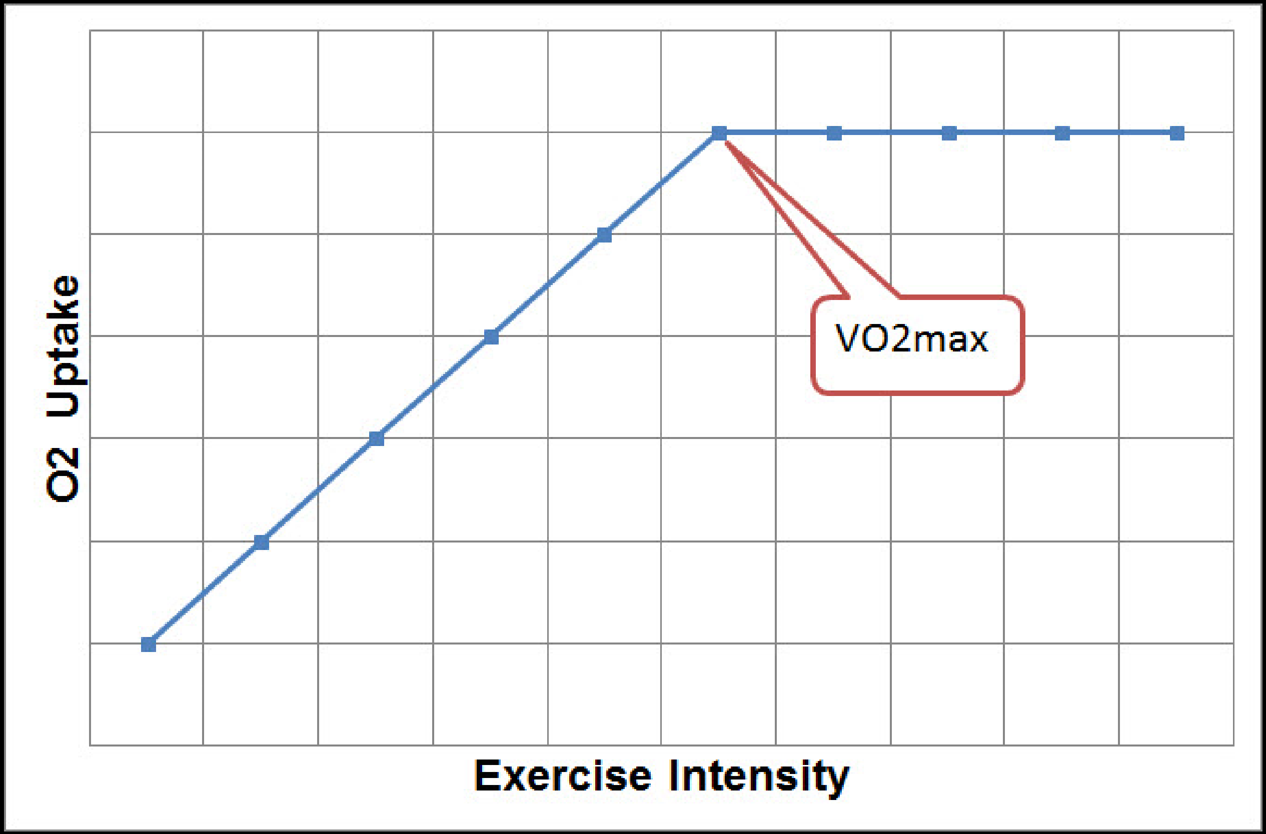
- During aerobic exercise, the cardiac output (stroke volume X heart rate) increases
- Initial increase in output is due to both ↑ stroke volume and ↑ heart rate.
- Above 50% VO_2 max, cardiac output rises solely from continued ↑ in heart rate
- Maximal attainable heart rate decreases with age
- With training, peripheral skeletal muscles increases capillary density that facilitates high levels of oxygen extraction



Most widely recognized measure of cardiopulmonary fitness

A function of the capacity of the heart, lungs and blood to transport oxygen to the working muscles and ability of the muscles to use oxidative phosphorylation to create ATP aerobically

Front. Cardiovasc. Med., 03 September 2021. Volume 8 - 2021 | https://doi.org/10.3389/fcvm.2021.734278


VO2max
O2 Uptake
Exercise Intensity

INDICES OF EXERCISE INTENSITY FOR ENDURANCE SPORTS FROM MAXIMAL EXERCISE TESTING AND TRAINING ZONES 2020 ESC GUIDELINES

Intensity	VO2 max (%)	HR max (%)	HRR (%)	Rate of Perceived Exertion (RPE) Scale	Training zone
Low/light exercise	< 40	<55	<40	2-3	Aerobic
Moderate	40-69	55-74	40-69	4-6	Aerobic
High	70-85	75-90	70-85	7-8	Aerobic + Lactate
Very high	>85	>90	>85	9-10	Aerobic + Lactate + Anaerobic

Health Benefits of Physical Activity for Adults

IMMEDIATE

A single bout of moderate-to vigorous physical activity provides immediate benefits for your health.

LONG-TERM

Regular physical activity provides important health benefits for chronic disease prevention.

Sleep
Improves sleep quality

Less Anxiety
Reduces feelings of anxiety

Blood Pressure
Reduces blood pressure

Brain Health
Reduces risks of developing dementia (including Alzheimer's disease) and reduces risk of depression

Heart Health
Lowers risk of heart disease, stroke, and type 2 diabetes

Cancer Prevention
Lowers risk of eight cancers: bladder, breast, colon, endometrium, esophagus, kidney, lung, and stomach

Healthy Weight
Reduces risk of weight gain

Bone Strength
Improves bone health

Balance and Coordination
Reduces risks of falls

Emerging research suggests physical activity may also help boost immune function.

Nieman, "The Compelling Link," 201–217.
Jones, "Exercise, Immunity, and Illness," 317–344.

CDC

ACTIVE PEOPLE, HEALTHY NATION™ — CREATING AN ACTIVE AMERICA, TOGETHER

Source: *Physical Activity Guidelines for Americans*, 2nd edition

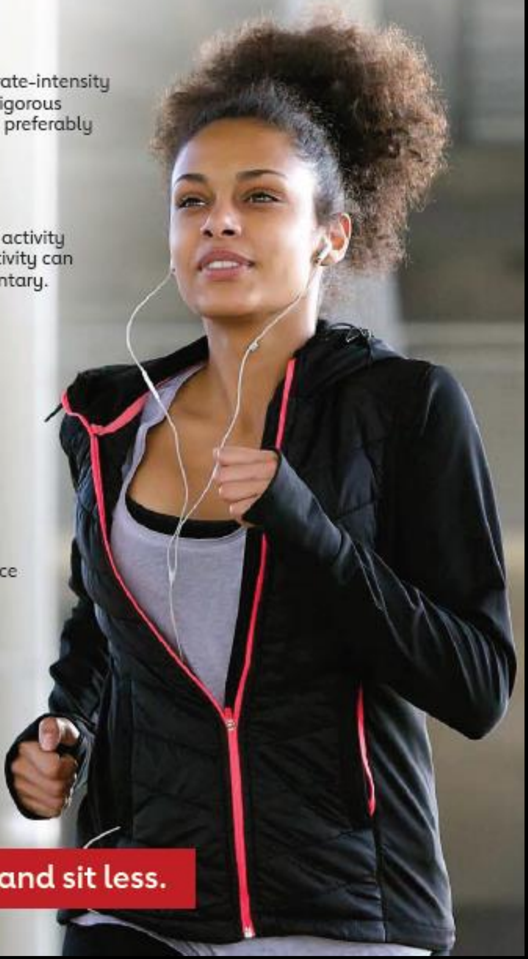
To learn more, visit: https://www.cdc.gov/physicalactivity/basics/adults/health-benefits-of-physical-activity-for-adults.html

August 2020

American Heart Association.
Healthy for Good™

How much **physical activity** do you need?

Here are the American Heart Association recommendations for adults.



Fit in 150+

Get at least 150 minutes per week of moderate-intensity aerobic activity or 75 minutes per week of vigorous aerobic activity (or a combination of both), preferably spread throughout the week.

Move More, Sit Less

Get up and move throughout the day. Any activity is better than none. Even light-intensity activity can offset the serious health risks of being sedentary.

Add Intensity

Moderate to vigorous aerobic exercise is best. Your heart will beat faster, and you'll breathe harder than normal. As you get used to being more active, increase your time and/or intensity to get more benefits.

Add Muscle

Include moderate- to high-intensity muscle-strengthening activity (like resistance or weight training) at least twice a week.

Feel Better

Physical activity is one of the best ways to keep your body and brain healthy. It relieves stress, improves mood, gives you energy, helps with sleep and can lower your risk of chronic disease, including dementia and depression.

Move more, with more intensity, and sit less.

Find out how at heart.org/movemore.

https://www.heart.org/en/healthy-living/fitness/fitness-basics/aha-recs-for-physical-activity-infographic

Athlete's Heart

- **Regular exercise promotes structural, functional, and electrical remodeling of the heart → referred to as the *"athlete's heart."***

- **Athlete's heart refers to normal changes from regular exercise, i.e., an adaptive increase in cardiac chamber size and wall thickness that is promoted by the volumes and pressure loads of exercise resulting in improved function and efficiency.**

- **Significant exercise-related remodeling is rarely associated with adverse clinical effects. Although afib is more common in endurance athletes (related to remodeling or genetics?).**

(J Am Coll Cardiol 2022;80:1346–1362)

Athlete's Heart: Past to Present

- 1890's – Swedish physician Henschen used auscultation and percussion to demonstrate increased cardiac dimensions in Nordic skiers.

- Early 1900's – Paul Dudley White studied radial pulse rate and patterns in Boston Marathon competitors. Noted high prevalence of sinus bradycardia.

- 1960's to 1970's – CxR & ECGs
- 1970's to 2000's – Echocardiography
- 2000 – Present – Echo + MRI

(Circulation. 2011;123:2723-2735.)

Athlete's Heart: Adaptive vs. Maladaptive

- 1902 – Postulated that cardiac enlargement in athletes is a form of overuse pathology.
- 100+ year debate with no clear evidence to substantiate its validity.
- Long-term studies are needed.
- Modern view – athlete's heart implicates adaptive physiology, not preclinical disease.
- There remains evolving ambiguity and controversies.

Hemodynamic Effects of Exercise

Resistance (Anaerobic)

Normal or slightly ↑ CO

↑ PVR

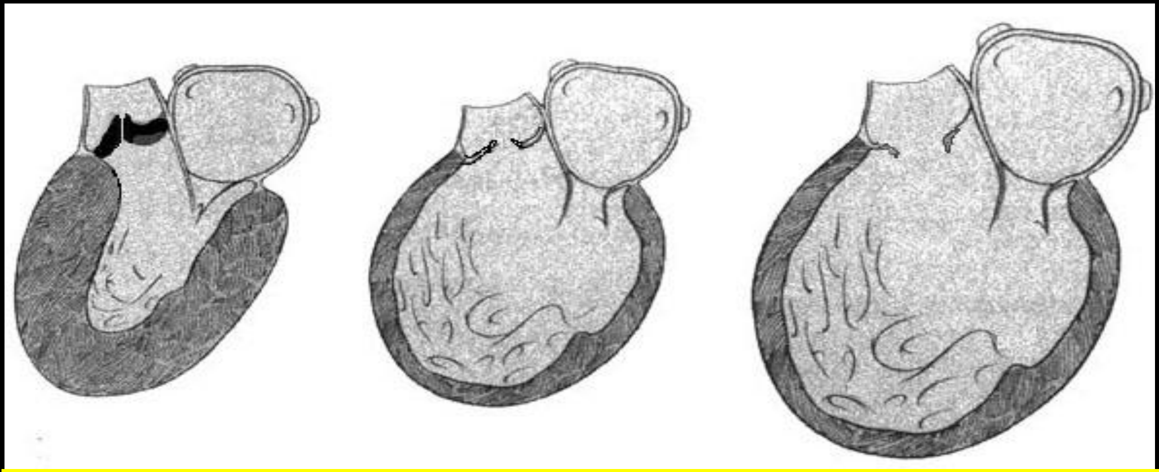
↑ LV wall thickness

Endurance (Aerobic)

Sustained ↑ CO → ↑ SV

Normal or ↓ PVR

↑ cavity size

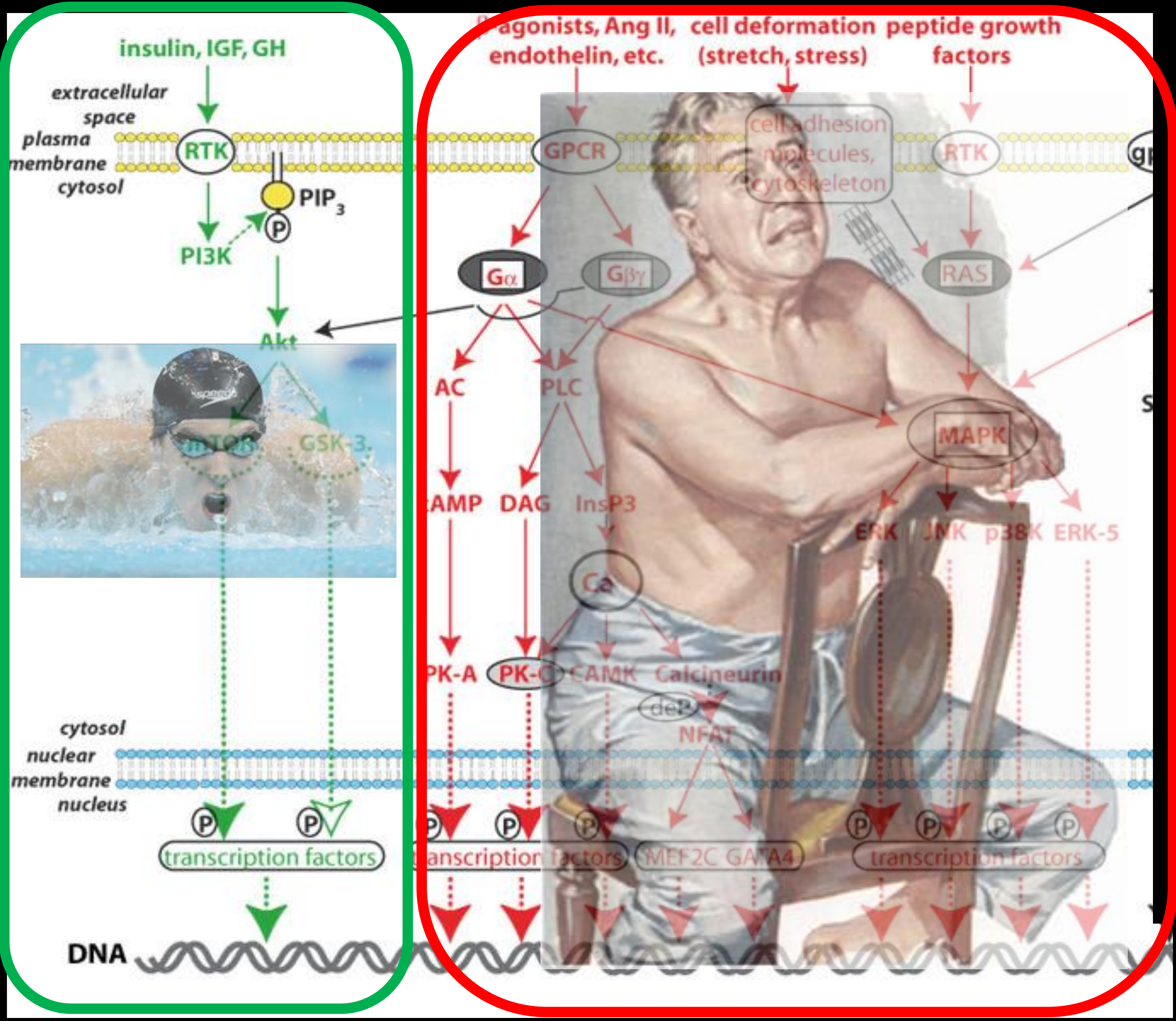


CONCENTRIC NORMAL ECCENTRIC

“Hypertrophy” “Dilatation”

Hypertrophic Signaling

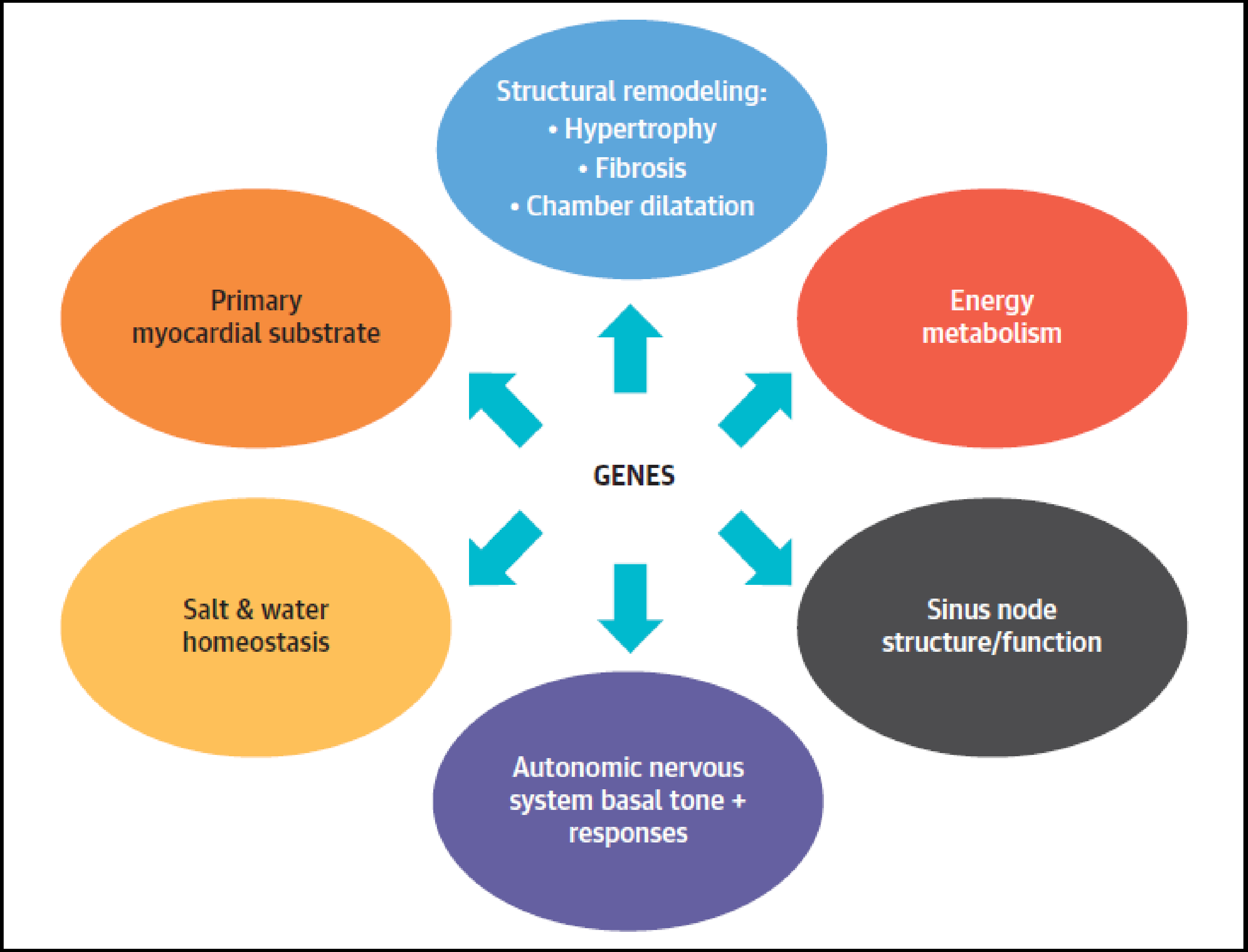
Hypertrophy (Proteomics)



ADAPTIVE

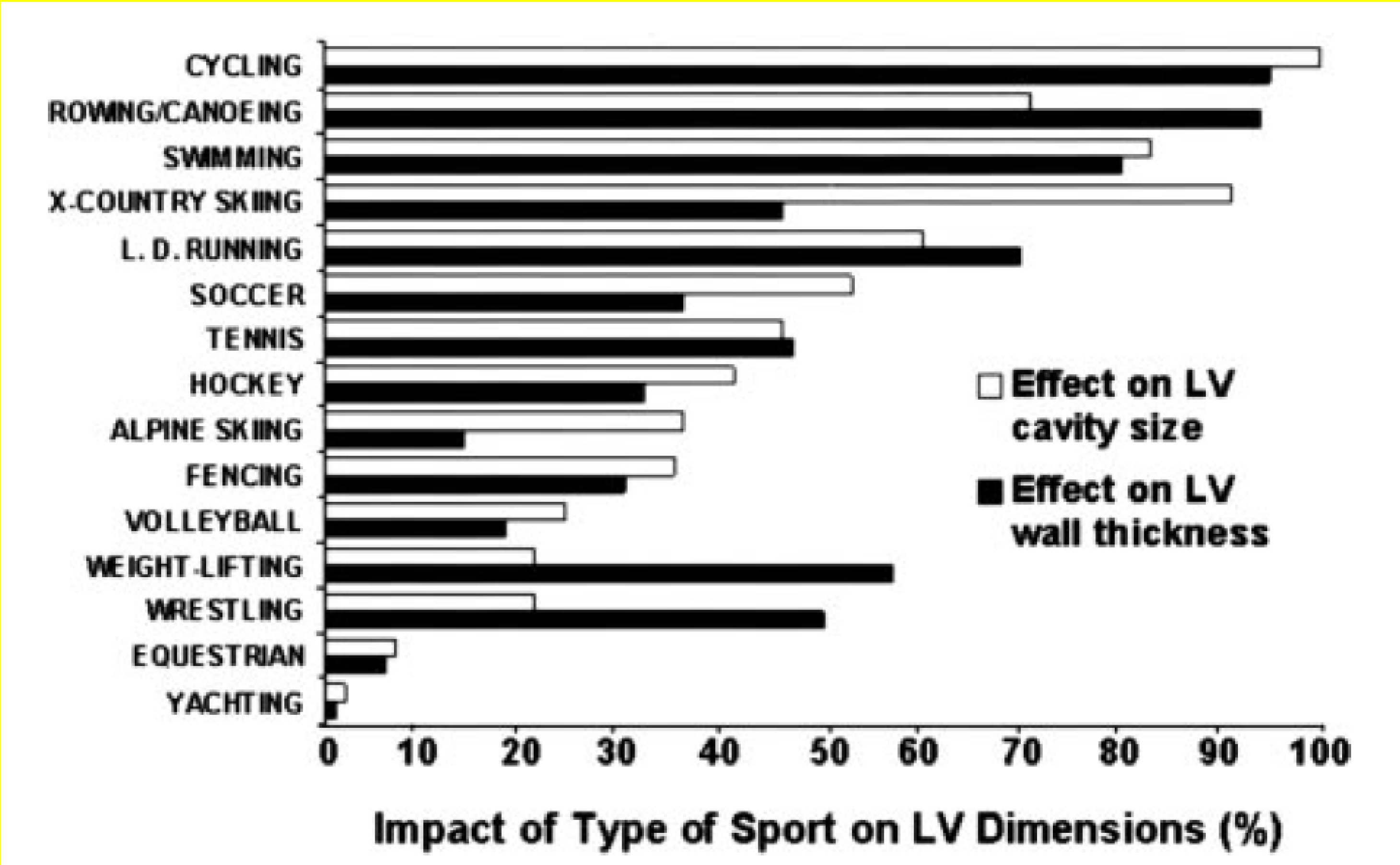
MALADAPTIVE

Are Athletes Born or Created?

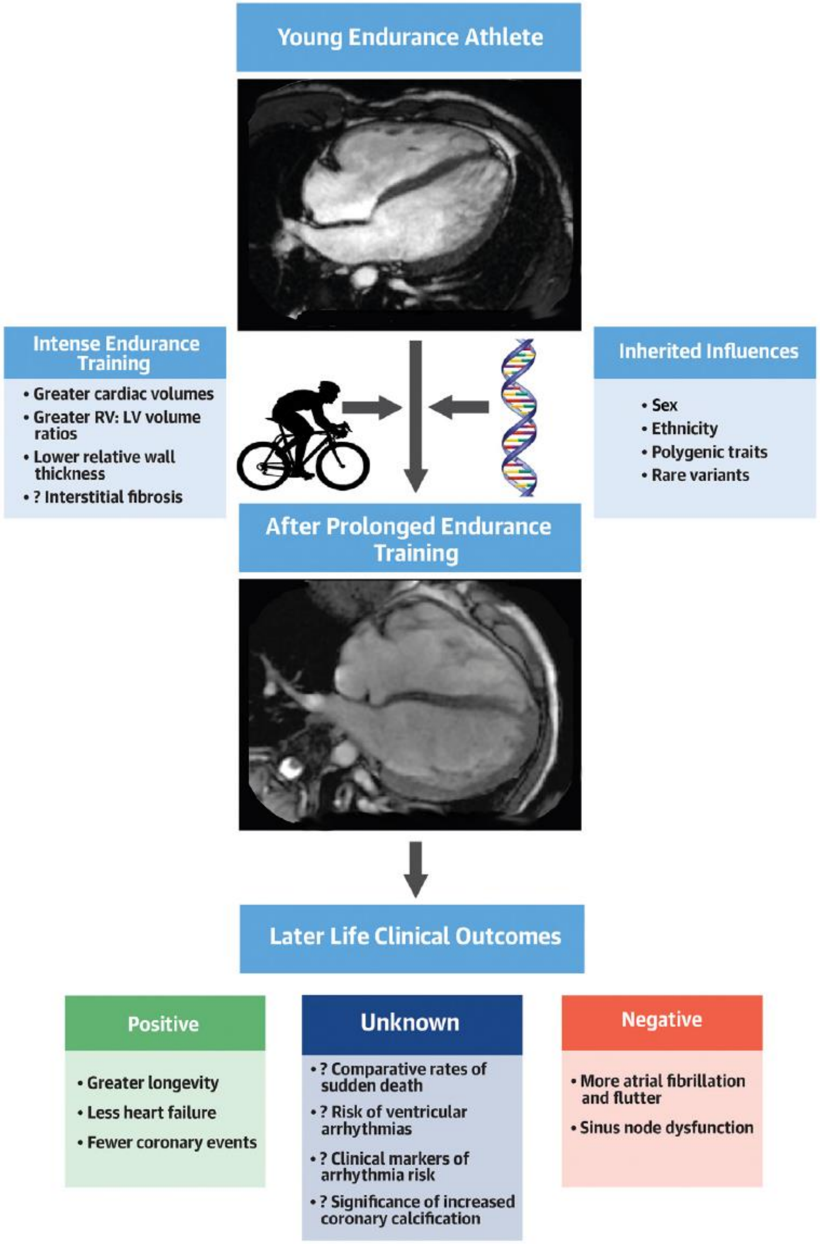


J Am Coll Cardiol 2022;80:1346–1362.

	Low Dynamic	**Moderate Dynamic**	**High Dynamic**
Low Static	Bowling Golf	Baseball Tennis (doubles) Volleyball	X-Country skiing Raquetball Running (long) Tennis (singles) Soccer
Moderate Static	Archery Auto racing Equestrian	Football (Amer) Rugby Figure Skating	Basketball Ice hockey Football (Aust.) Swimming Running (mid)
High Static	Gymnastics Karate/Judo Rock climbing Weight lifting	Downhill skiing Wrestling	Boxing Canoeing Cycling Rowing



Maron B. and Pelliccia A. Circulation. 2006;114:1633-1644.



La Gerche A, et al. J Am Coll Cardiol. 2022;80(14):1346-1362.

Female Athlete's Heart

- **Women are under-represented in sports cardiology research.**

- **2020 study**

  720 Olympic athletes (360 men, 360 women)

  To define the electrocardiographic and morphological features of female athlete's heart, with special attention to differences related to sex and sport

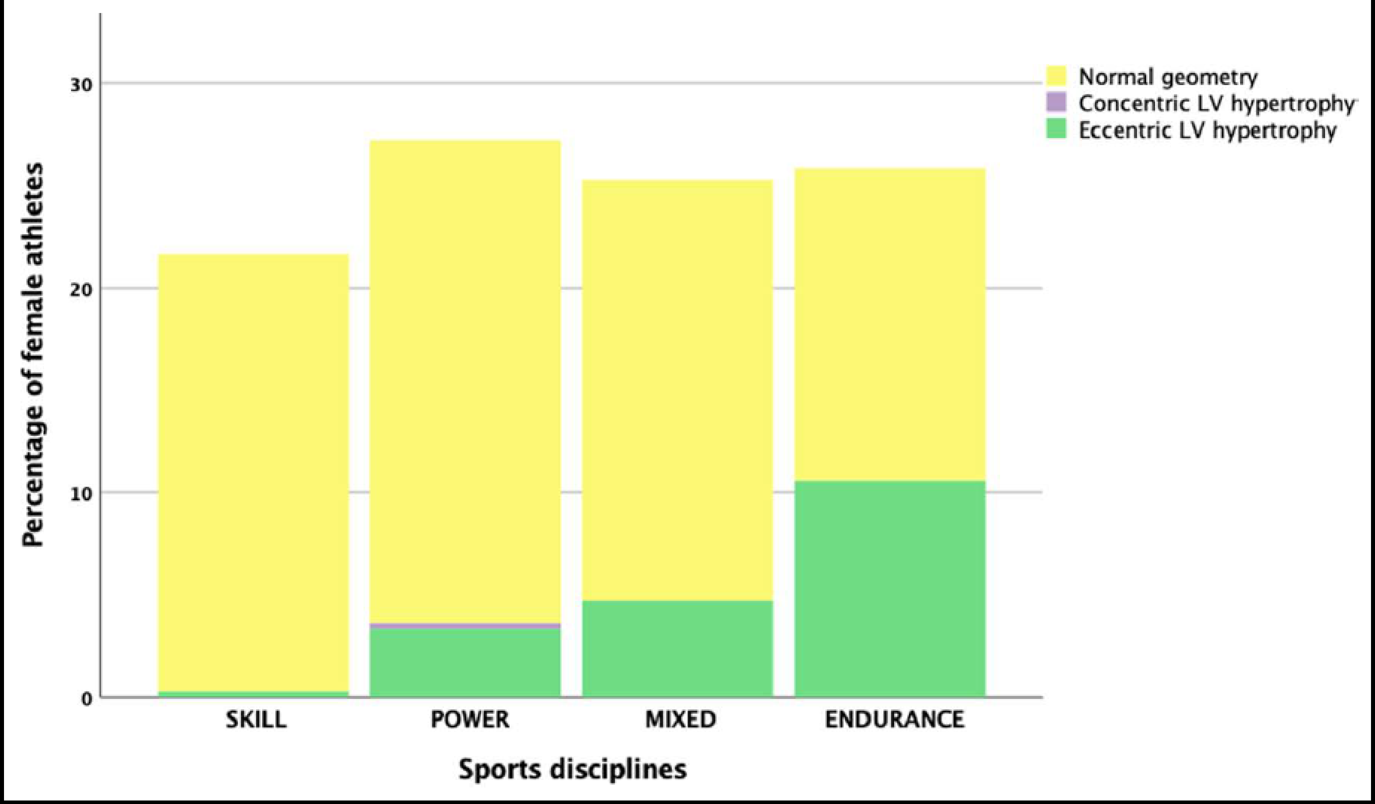
  Mean age: 23±5 years

  Clinical evaluation, resting ECG, exercise stress test and echocardiography

Circ Cardiovasc Imaging. 2020;13:e011587. DOI: 10.1161/CIRCIMAGING.120.011587

Findings – Female Athletes

- **ECG** Higher proportion of anterior T wave inversion
- **ETT** Lower response in absolute systolic and diastolic BP
- **Echo**



Circ Cardiovasc Imaging. 2020;13:e011587. DOI: 10.1161/CIRCIMAGING.120.011587

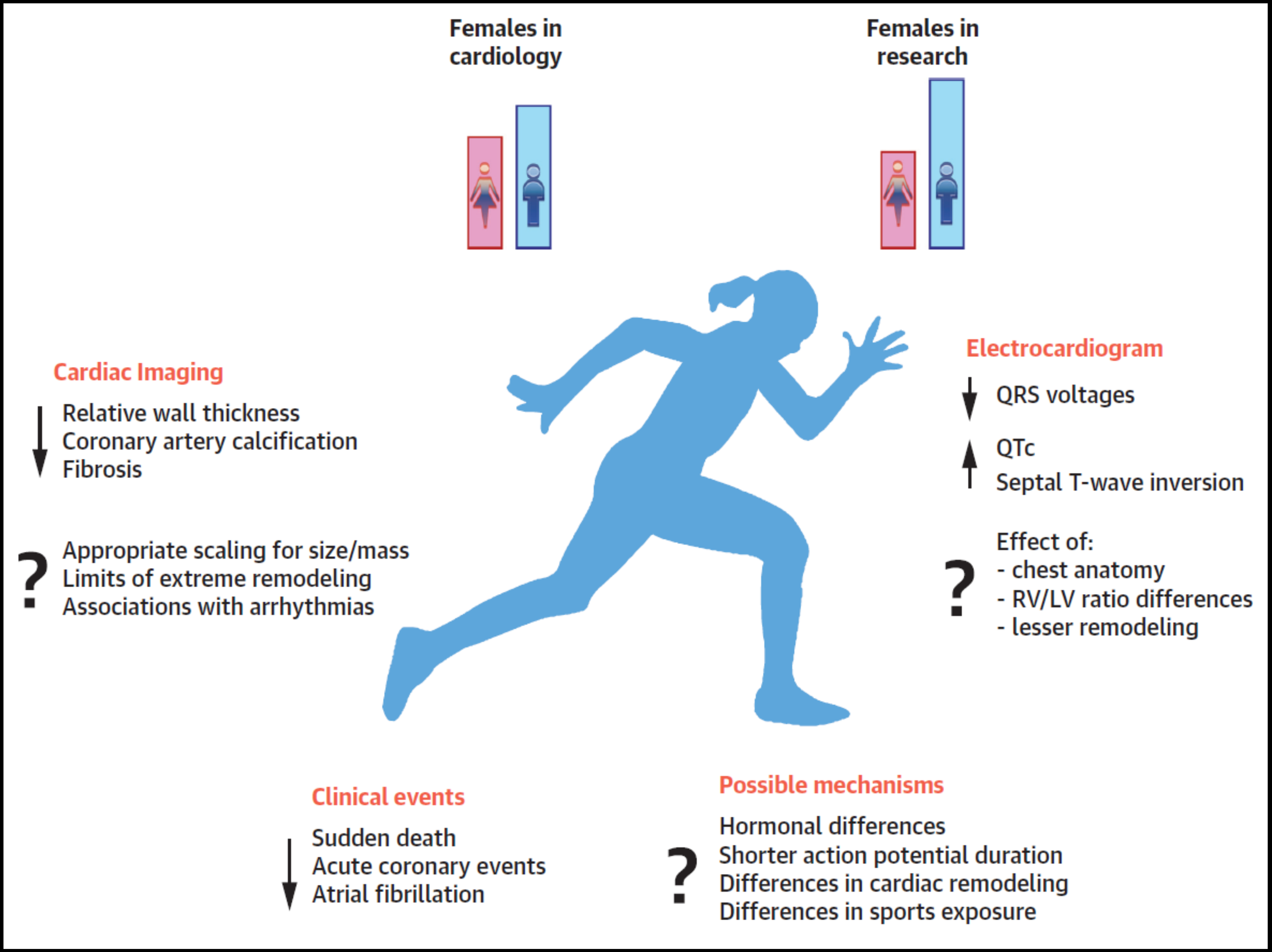
Conclusions

Electrical and structural cardiac remodeling significantly differs between male and female athletes, with females:

- **presenting a higher proportion of anterior T wave inversion**
- **lower blood pressure response to exercise**
- **less criteria of biventricular hypertrophy**
- **a different pattern of LV and RV remodeling.**
- **Female athletes usually maintain a normal LV geometry with larger relative increase of LV and RV cavity dimensions compared with male athletes when normalized to body size.**

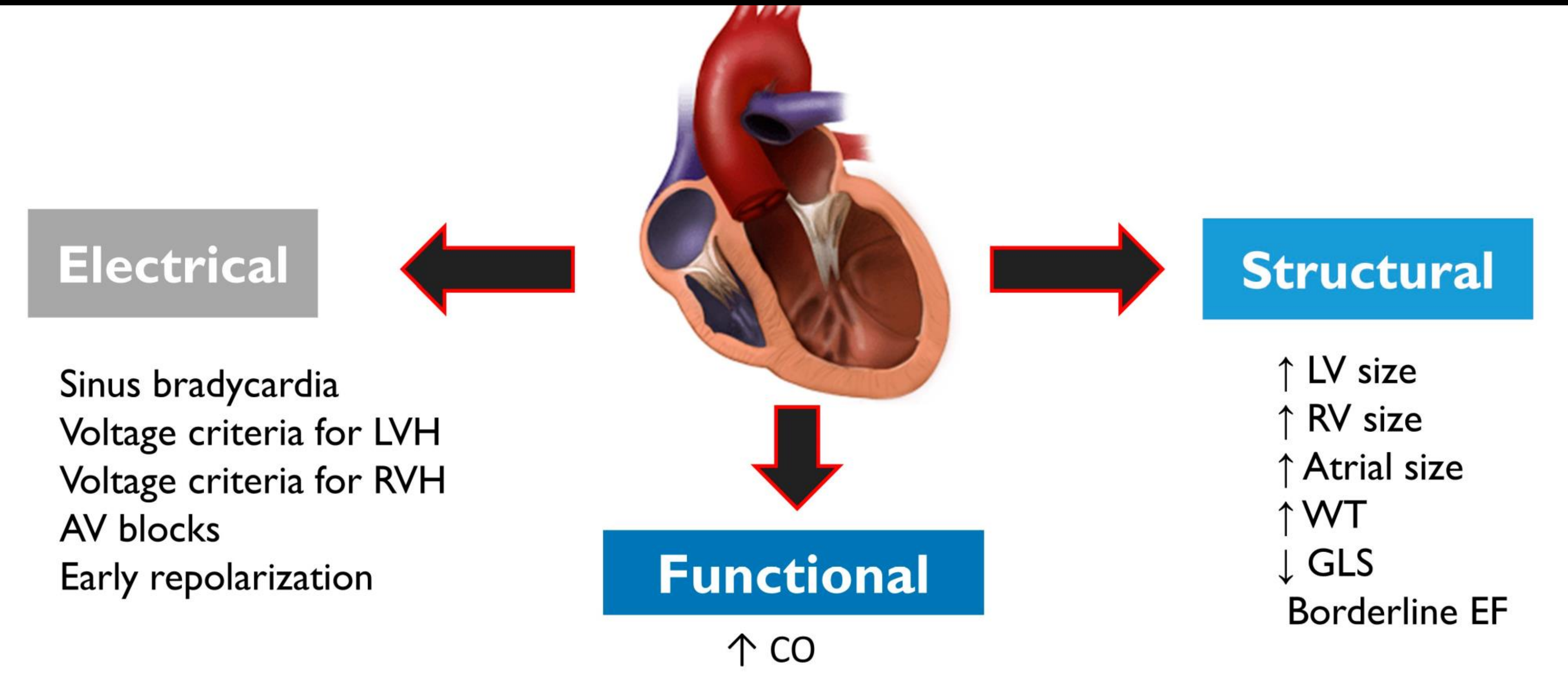
Overall, these findings support the concept that sex has a profound effect on cardiac remodeling, and theorems derived from men cannot be directly applied to female athletes.

Female Athlete's Heart

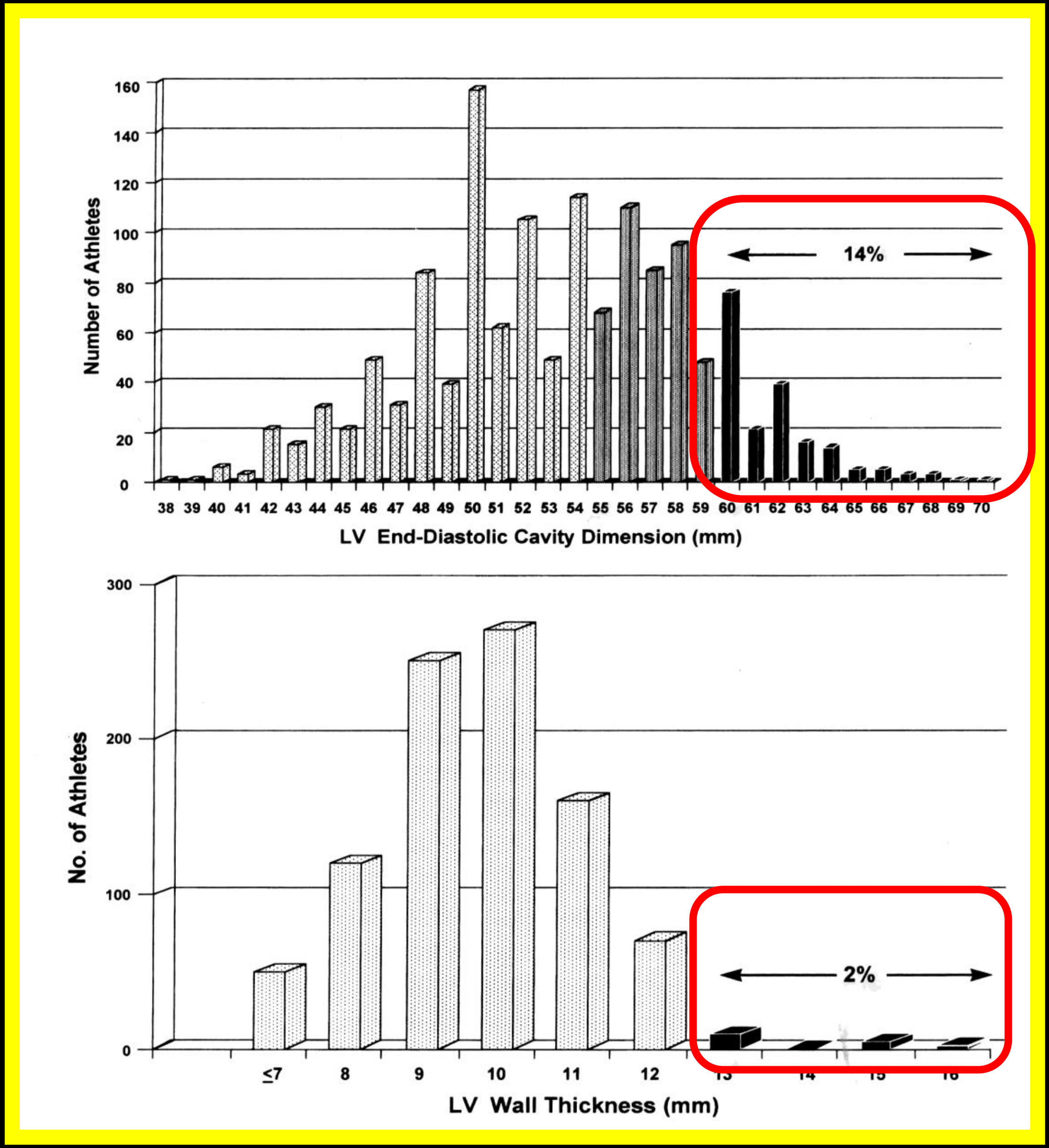


J Am Coll Cardiol 2022;80:1346–1362.

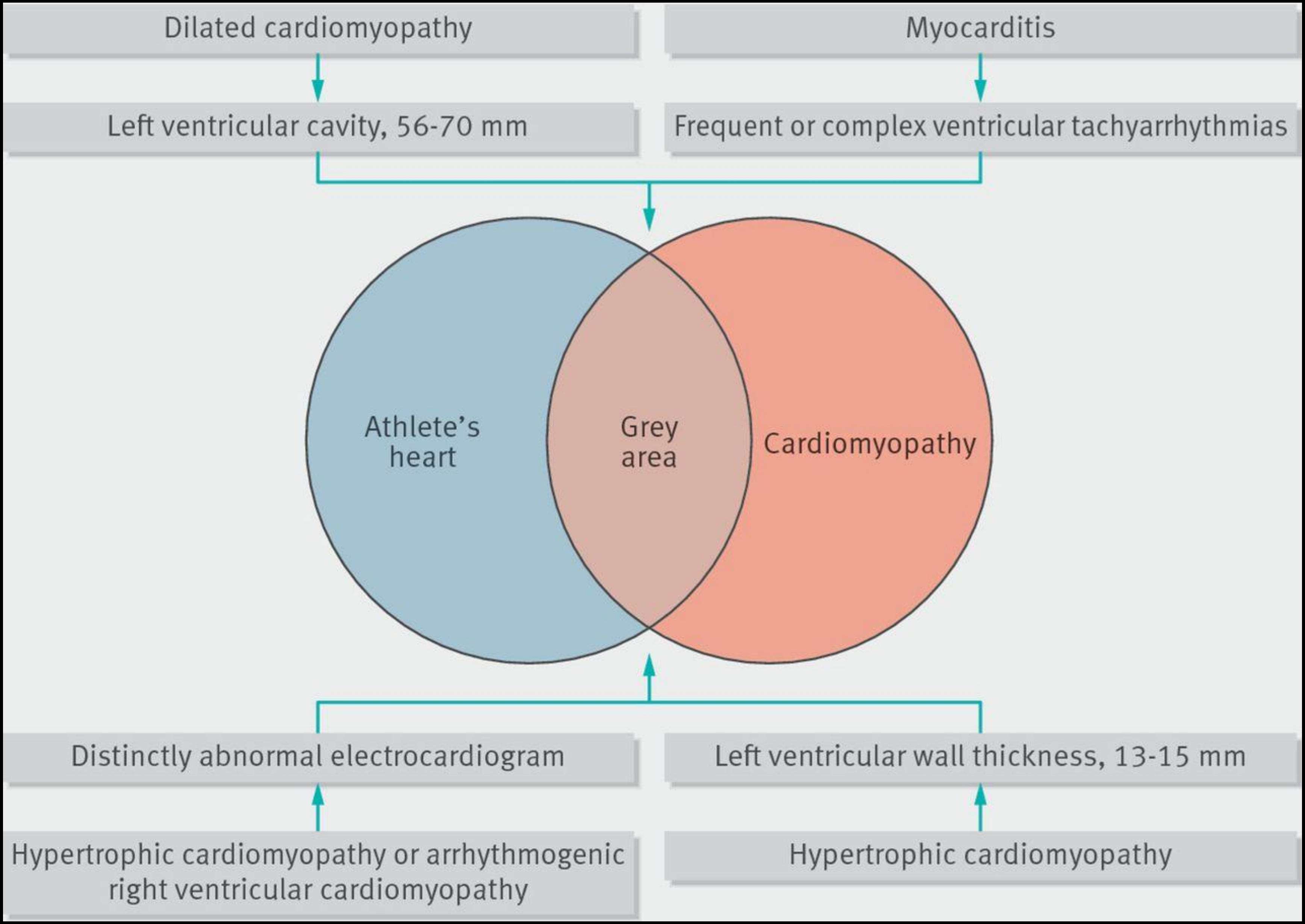
Common Findings in Athletes



J. Cardiovasc. Dev. Dis. 2023, 10, 68. https://doi.org/10.3390/jcdd10020068

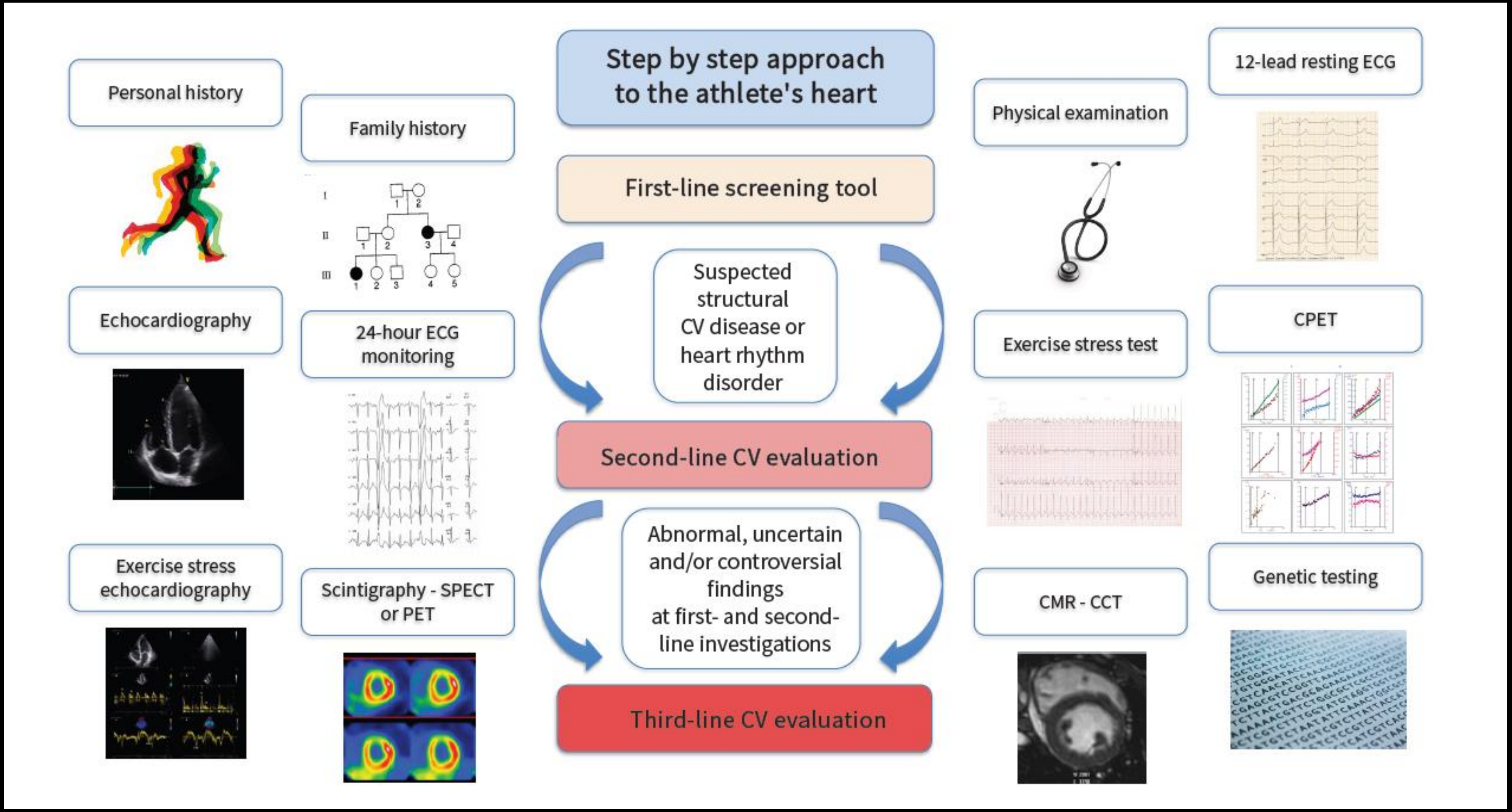


Pellicicia A et al. NEJM 1991;324:295-301.



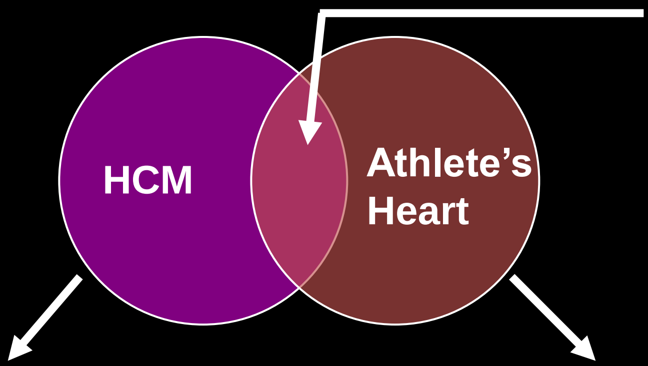
Semsarian C, Sweeting J, Ackerman MJ. Sudden cardiac death in athletes. *British J of Sports Med.* 2015;49:1017-1023.

Shrinking the Grey Zone



Rev. Cardiovasc. Med. 2023; 24(5): 151

“Grey Zone” of LV Wall Thickness (13-15 mm)



HCM	Feature	Athlete’s Heart
+	Unusual patterns of LVH	-
+	LV Cavity < 45 mm	-
-	LV Cavity > 55 mm	+
+	Marked LA Enlargement	-
+	Bizarre ECG Patterns	-
+	Abnormal LV Filling	-
+	Female Gender	-
-	↓Thickness w/ Decondition	+
+	Family History of HCM	-
-	Max VO_2 > 45 ml/kg/min > 100% predicted	+

Maron BJ. *J Am Coll Cardiol.* 2005.

Hypertrophic Cardiomyopathy

Inheritance Pattern

+/- +/+

+/-

Autosomal Dominant

Sex Distribution

50% 50%

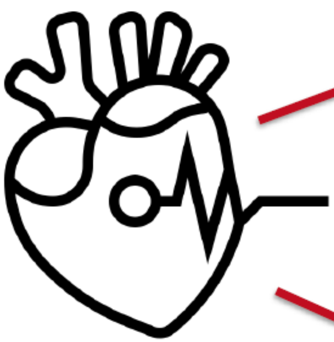
Women diagnosed less commonly

Disease Prevalence

Estimated 1:200 – 1:500

Triggers for Evaluation

Symptoms
Cardiac Event
Heart Murmur
Abnormal EKG
Cardiac Imaging
Family Studies



HCM

LV Outflow Tract Obstruction (LVOTO)

⅔ have LVOTO

⅓ do not have LVOTO

Other non-HCM Causes of LV Hypertrophy

Metabolic & Multi-organ Syndromes

- RASopathies
- Mitochondrial myopathies
- Glycogen / Lysosomal storage diseases
- Amyloidosis
- Sarcoidosis
- Hemochromatosis
- Danon disease

Secondary Causes

- Athlete's heart
- Hypertension
- Valvular & subvalvular stenosis

Adapted from: 2020 ACC/AHA Guideline for the Diagnosis and Treatment of Patients with Hypertrophic Cardiomyopathy

Hypertrophic Cardiomyopathy

~30-60% of HCM patients have an identifiable pathogenic or likely-pathogenic genetic variant

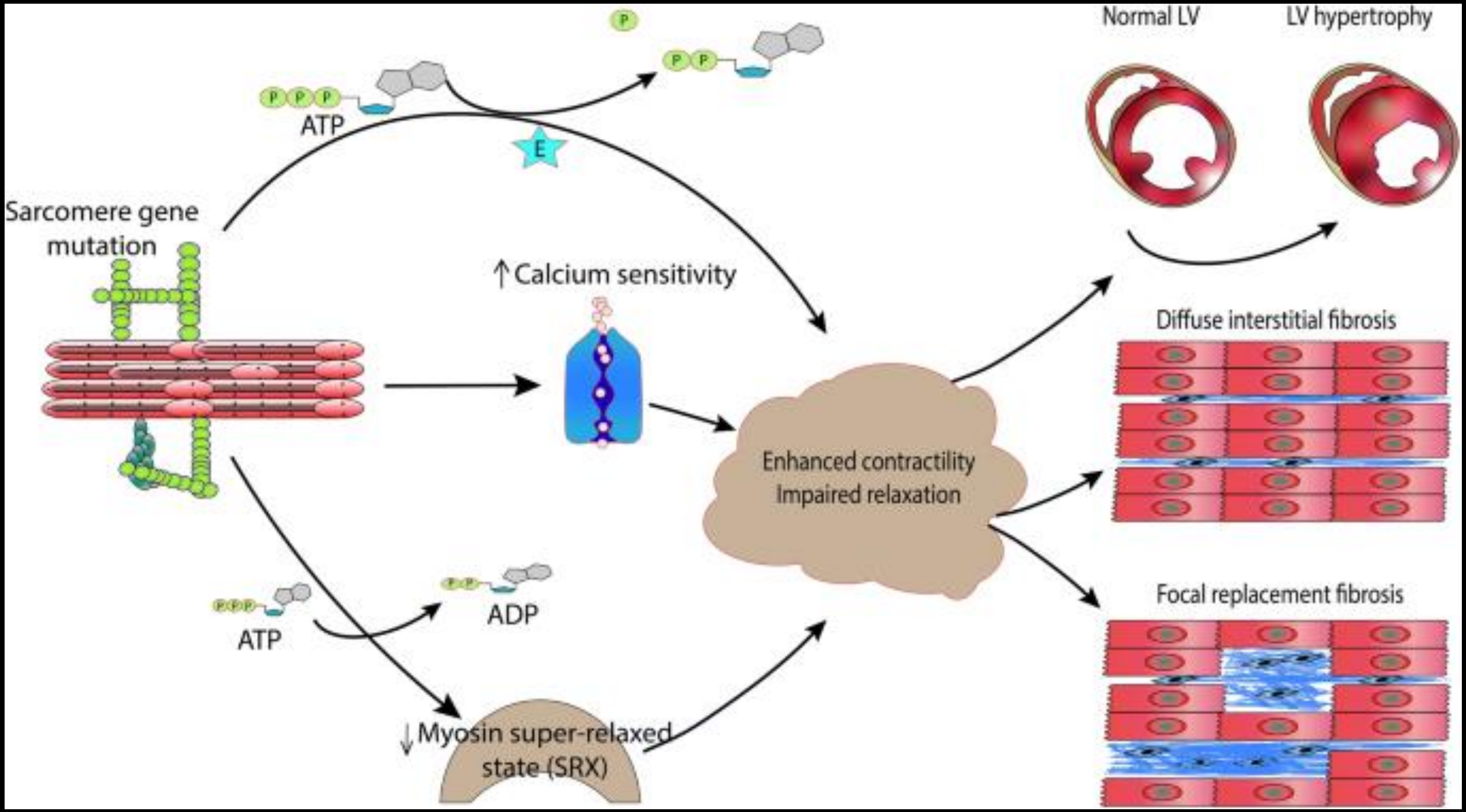
Many others have no genetic evidence of disease
and / or
no other affected family members

Sarcomere Genes Implicated in HCM
MYH7
MYBPC3
TNNI3
TNNT2
TPM1
MYL2
MYL3
ACTC1

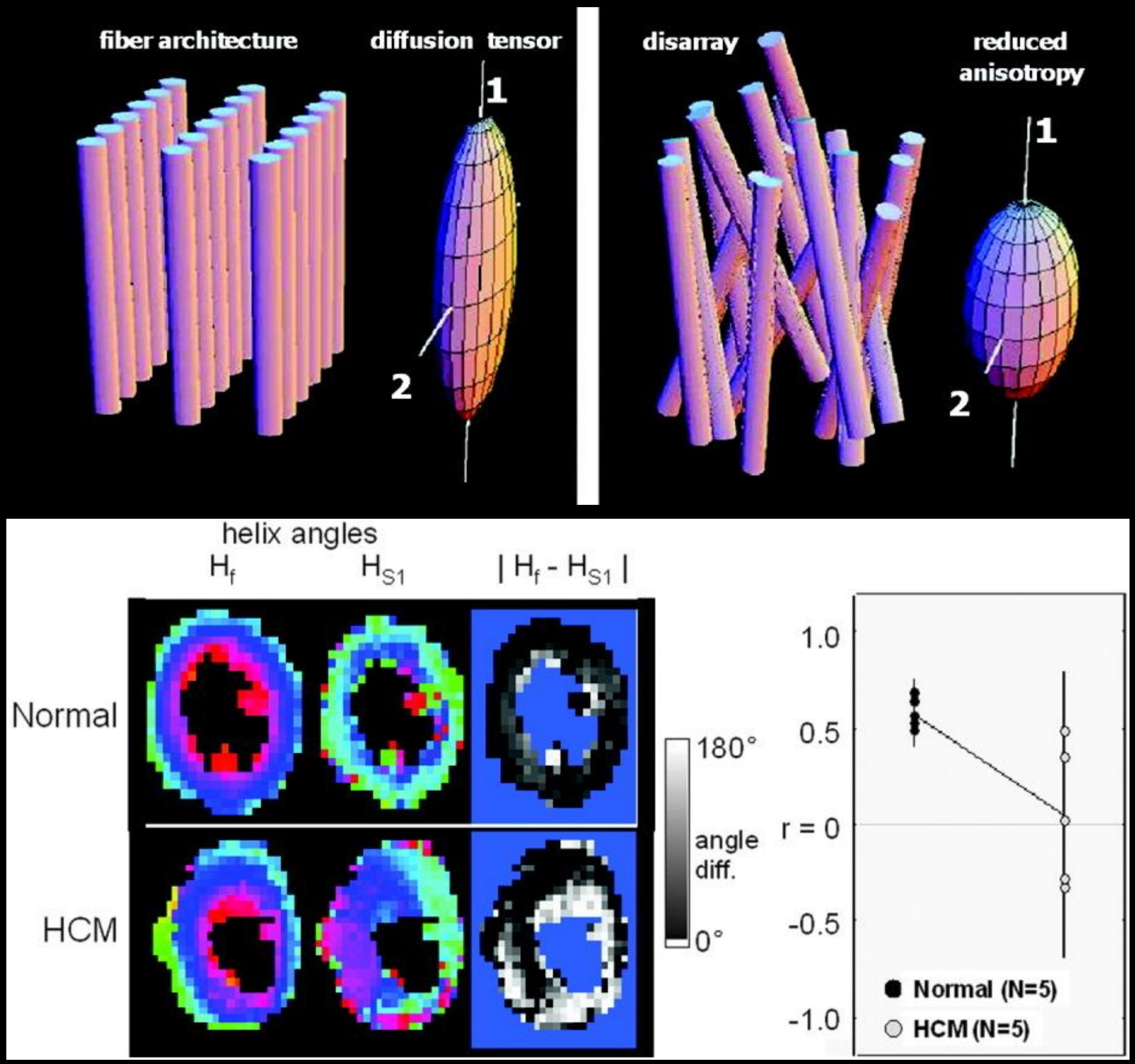
Two most common genes that harbor pathogenic variants in HCM (70%) (*MYH7*, *MYBPC3*)

Adapted from: 2020 ACC/AHA Guideline for the Diagnosis and Treatment of Patients with Hypertrophic Cardiomyopathy

Hypertrophic Cardiomyopathy

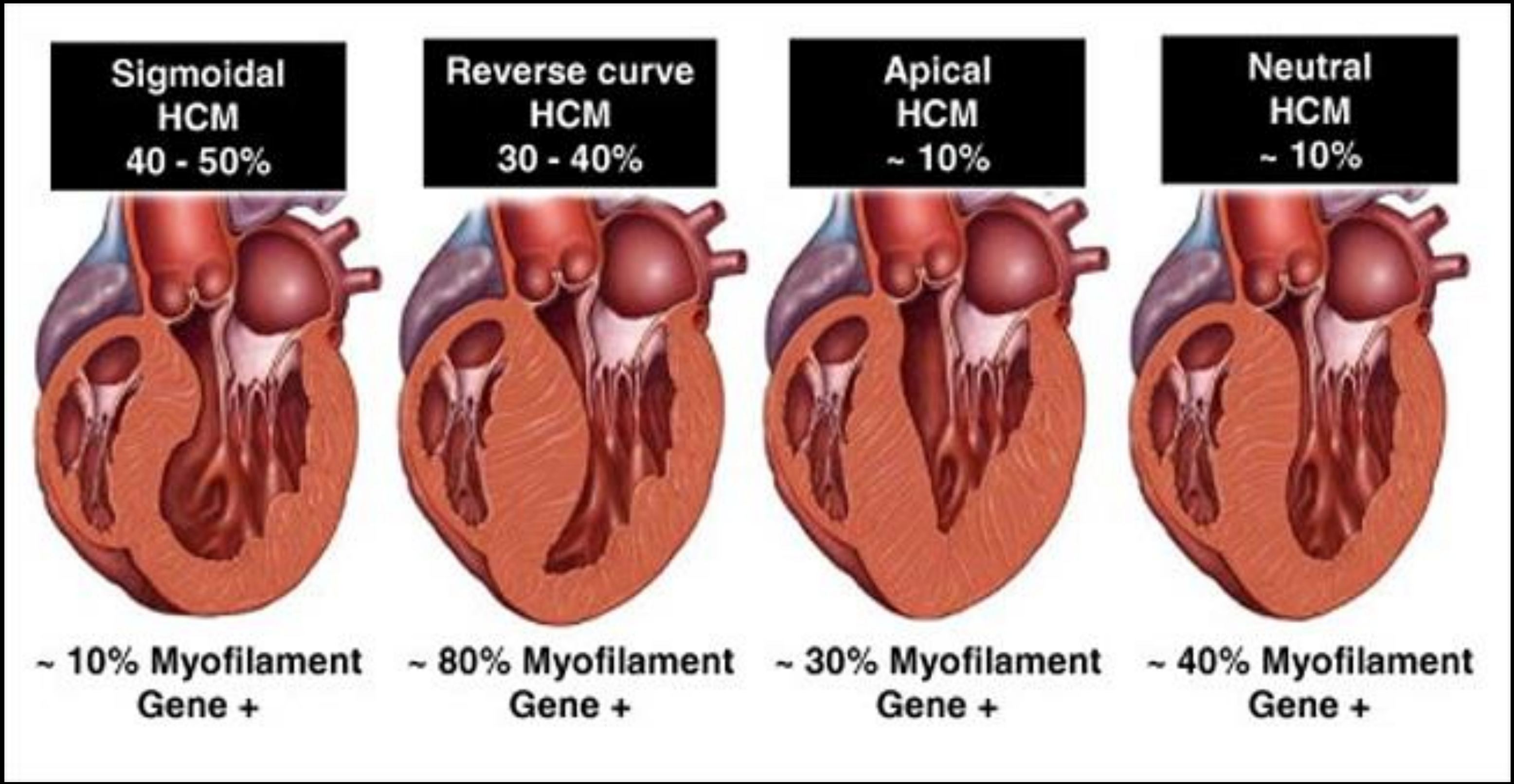


***Heart Fail Rev* 26, 1023–1036 (2021). https://doi.org/10.1007/s10741-020-09931-1**



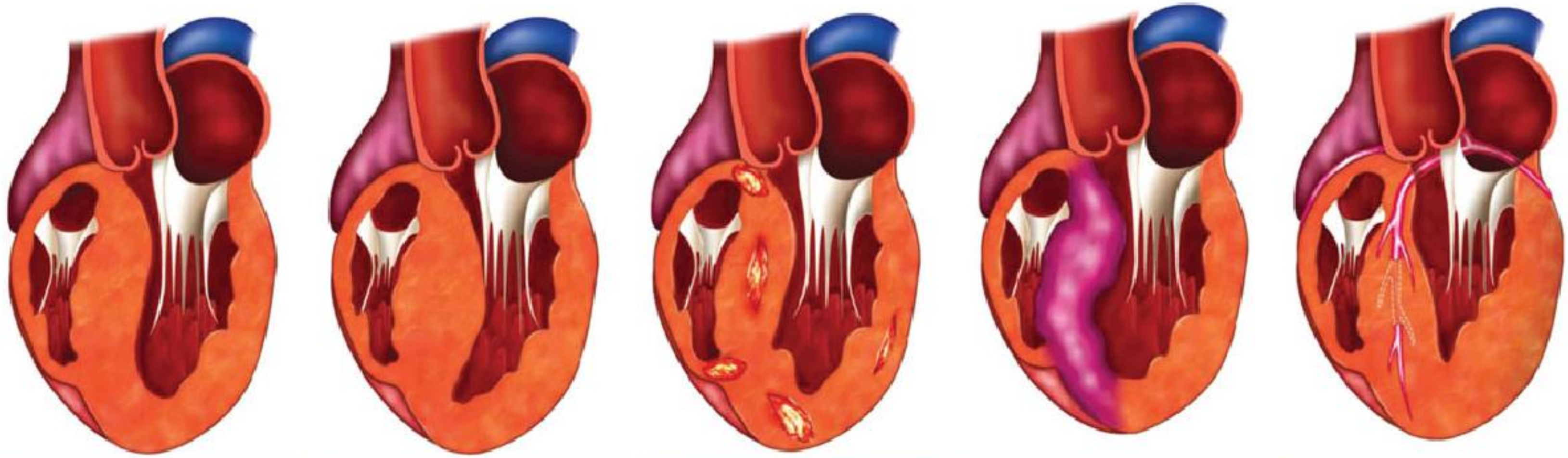
Magnetic Resonance Imaging, Volume: 23, Issue: 1, Pages: 1-8, First published: 05 December 2005, DOI: (10.1002/jmri.20473)

Morphologic Variants



J Am Coll Cardiol **2009;54:201-211.**

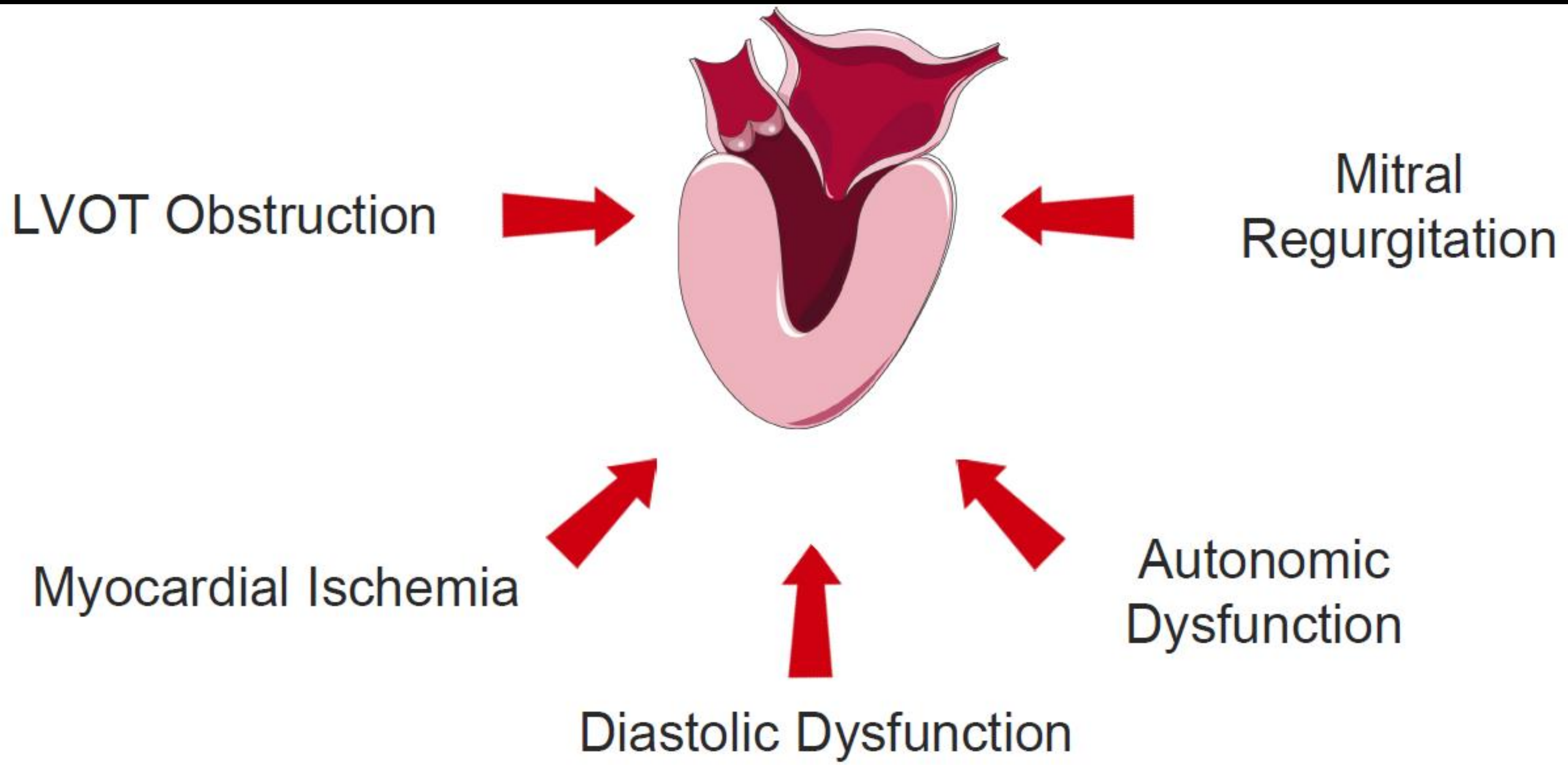
Pathologic & Imaging Variants



	LVH (Echo, CMR, CCT)	LVOTO (Echo, CMR, CCT)	Fibrosis (CMR, CCT)	Microvascular ischemia (CNI, CMR, Echo)	Myocardial bridging (CCT)
SCD	++	+(?)	+(?)	++	+(??)
HF	++	++	++	++	–
AF/STROKE	++	++	+	+	–
ANGINA	+	++	–	++	+

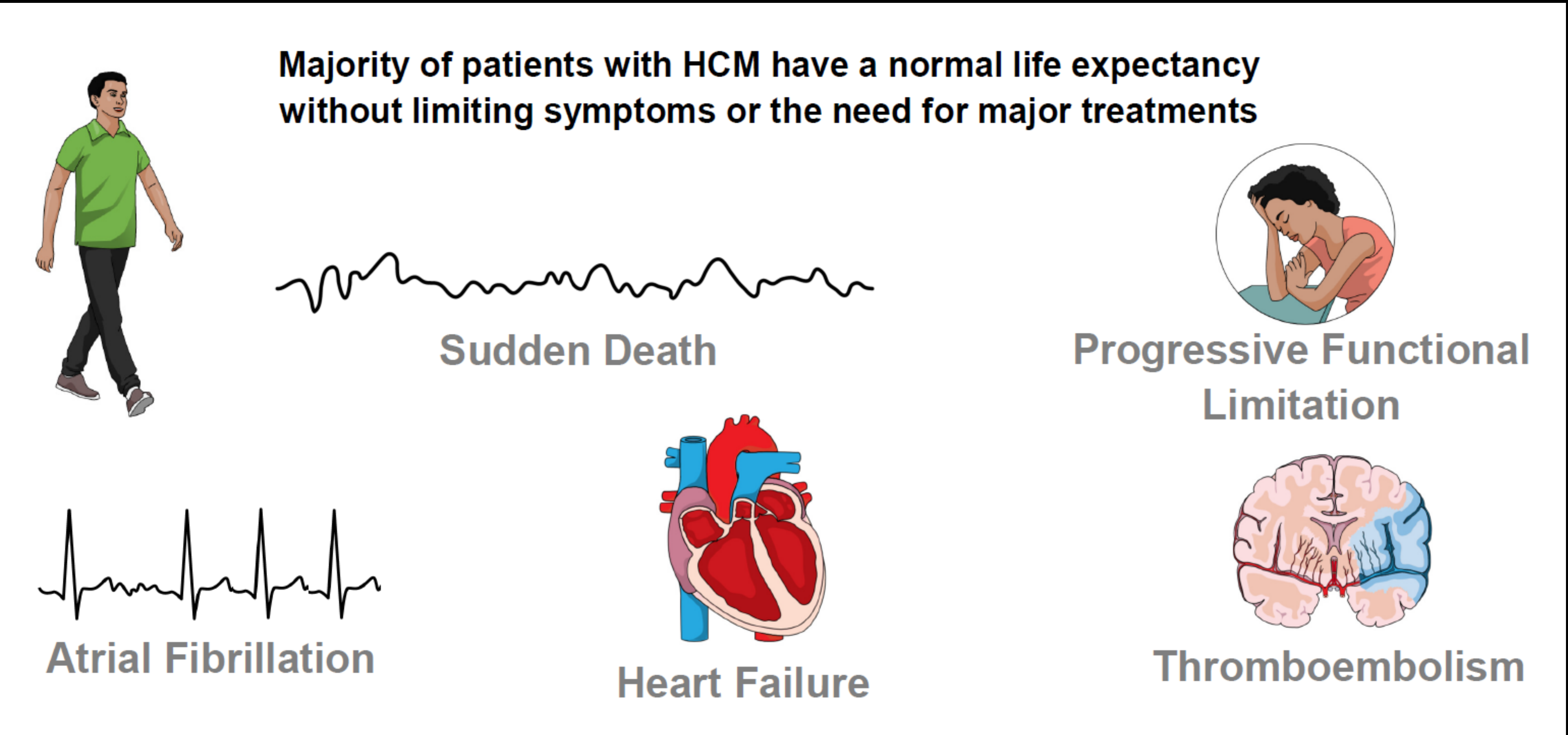
European heart journal cardiovascular Imaging **16 3 (2015): 280.**

Pathophysiology



Adapted from: 2020 ACC/AHA Guideline for the Diagnosis and Treatment of Patients with Hypertrophic Cardiomyopathy

Potential Adverse Outcomes



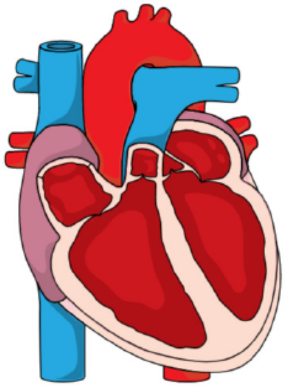
Adapted from: 2020 ACC/AHA Guideline for the Diagnosis and Treatment of Patients with Hypertrophic Cardiomyopathy

Mortality & Heart Failure

American Heart Association.

Hypertrophic Cardiomyopathy Mortality Rates Now < 1% per Year

Sudden Death



Heart Failure

Improvements in Risk Stratification

ICD Implantation

Now the greatest unmet treatment need in adults

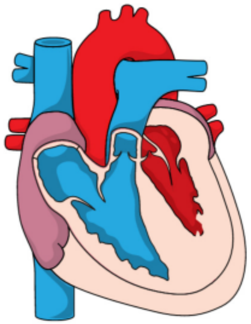
Adapted from: 2020 ACC/AHA Guideline for the Diagnosis and Treatment of Patients with Hypertrophic Cardiomyopathy

Diagnosis

HCM

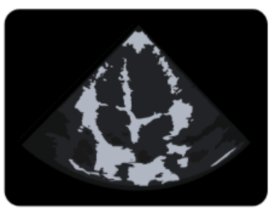
American Heart Association.

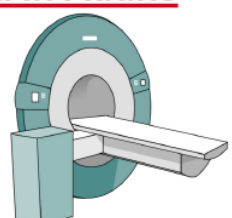
Defining Hypertrophic Cardiomyopathy in 2020



- Morphologic expression confined solely to the heart
- Characterized by left ventricular (LV) hypertrophy
  Basal anterior septum in continuity with the anterior free wall = most common
- No other cardiac, systemic or metabolic disease capable of producing the magnitude of hypertrophy present
- Disease-causing sarcomere (or sarcomere-related) variant identified or genetic etiology unresolved

Diagnostic Criteria in Adults





2D echocardiography or cardiac MRI

Maximal end-diastolic LV wall thickness ≥ 15 mm

Maximal end-diastolic LV wall thickness 13-14 mm in family member of HCM pt. or in conjunction with positive genetic test

Other Nondiagnostic Morphologic Abnormalities Associated with HCM

Systolic anterior motion (SAM) of the mitral valve

Hyperdynamic LV function

Hypertrophied & apically displaced papillary muscles

Myocardial crypts

Anomalous papillary muscle insertion in anterior MV leaflet

Elongated mitral valve leaflets

Myocardial bridging

Right ventricular hypertrophy

Adapted from: 2020 ACC/AHA Guideline for the Diagnosis and Treatment of Patients with Hypertrophic Cardiomyopathy

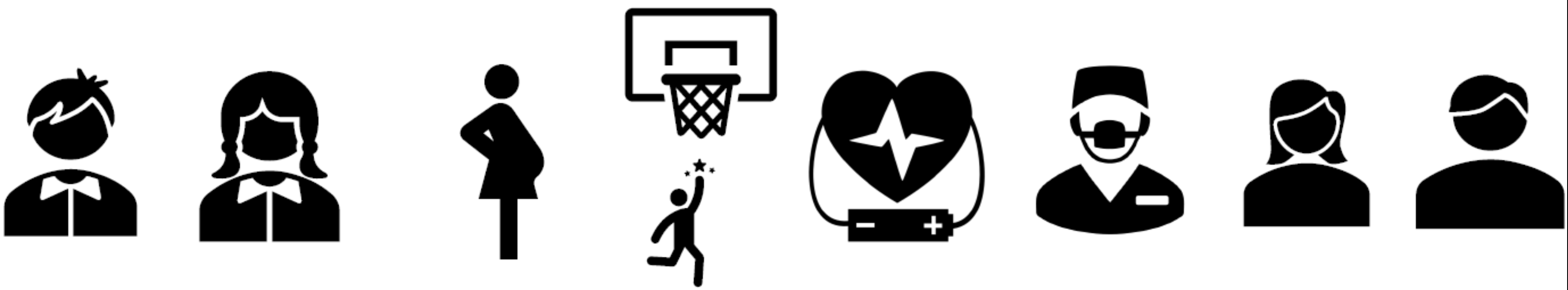
Recommendations for Shared Decision-Making in HCM

Discussions should involve:

-- Disclosure of risk and benefits

-- Anticipated outcomes of all options

--Goals, concerns and preferences of the patient (and family if the patient is a minor)



Shared decision discussions should be applied to:

--Genetic testing

--Sudden death risk assessment and ICD implantation

--Participation in high-intensity exercise and competitive sports

--Medical and invasive therapies for LVOT obstruction

Adapted from: 2020 ACC/AHA Guideline for the Diagnosis and Treatment of Patients with Hypertrophic Cardiomyopathy

Teams Based Approach to Hypertrophic Cardiomyopathy Care

Cardiologists Working Outside of HCM Centers:

- Initial and Surveillance Testing
- Initial Treatment Recommendations
- Rapid Assessment for Change in Disease Course

HCM Centers:

- Confirmation of Diagnosis
- Genetic Counseling and Testing
- Advanced Treatment Decisions and Procedures

Comprehensive HCM Centers:

- Complex Invasive Septal Reduction Therapies
- Catheter Ablation for Ventricular and Complex Atrial Tachyarrhythmias
- Advanced Heart Failure Therapies

Adapted from: 2020 ACC/AHA Guideline for the Diagnosis and Treatment of Patients with Hypertrophic Cardiomyopathy

Screening & Follow-up

Age of First-Degree Relative	Initiation of Screening	Repeat ECG, Echo
Pediatric		
Children and adolescents from genotype-positive families, and families with early onset disease	At the time HCM is diagnosed in another family member	Every 1-2 y
All other pediatric	At any time after HCM is diagnosed in a family member but no later than puberty	Every 2-3 y
Adults	At the time HCM is diagnosed in another family member	Every 3-5 y

Adapted from: 2020 ACC/AHA Guideline for the Diagnosis and Treatment of Patients with Hypertrophic Cardiomyopathy

Risk Assessment of Sudden Cardiac Death (SCD) in HCM

At initial evaluation and every 1-2 years (Class I)

Assess the following (Class I):

- ✓ Personal history of cardiac arrest, sustained ventricular arrhythmia, OR unexplained syncope suspected to be arrhythmic
- ✓ Family history of premature SCD in a close relative
- ✓ Maximal LV wall thickness, EF<=50%, apical aneurysm
- ✓ NSVT episodes on continuous ambulatory electrocardiographic monitoring; In select adult patients without major SCD risk factors, ICD may be considered in NSVT present on ambulatory monitoring (Class IIb).

IF none of the above:

CMR to help decision regarding ICD (Class I)

Reasonable to obtain echocardiographic LA diameter and LVOT gradient (Class IIa)

Management

Lifestyle modifications

- Optimal hydration
- Avoidance of caffeine and alcohol
- Appropriate exercise and diet to maintain fitness

Asymptomatic patients do not require medications

- Beta-blockers
- Calcium channel blockers (e.g., Verapamil)
- Disopyramide
- Diuretics – cautiously as needed

American Heart Association.

Pharmacologic Management Based on Type of HCM

Obstructive

Discontinue
Vasodilators
Digoxin
High dose diuretics

Symptoms r/t LVOTO

non-vasodilating β blockers

If not effective:

CCBs *

If persistent severe symptoms:

Add Disopyramide OR **Septal reduction therapy in eligible patients**

Persistent dyspnea with volume overload

Cautious use of low-dose oral diuretics

Verapamil is potentially harmful in severe dyspnea at rest, very high gradients and in children < 6 weeks

Acute Hypotension

IV Fluids

If no response:

Phenylephrine ± β blockers

Nonobstructive/Preserved EF
(symptoms of exertional angina or dyspnea)

ARBs/ACEi in symptomatic patients is not well established.

Therefore treatment includes:

β blockers or CCBs*

If dyspnea continues:

Diuresis

In persistent NYHA class III/VI + apical HCM

Treat with GDMT for HFpEF

Apical myectomyⱡ

Nonobstructive/Preserved EF
(asymptomatic)

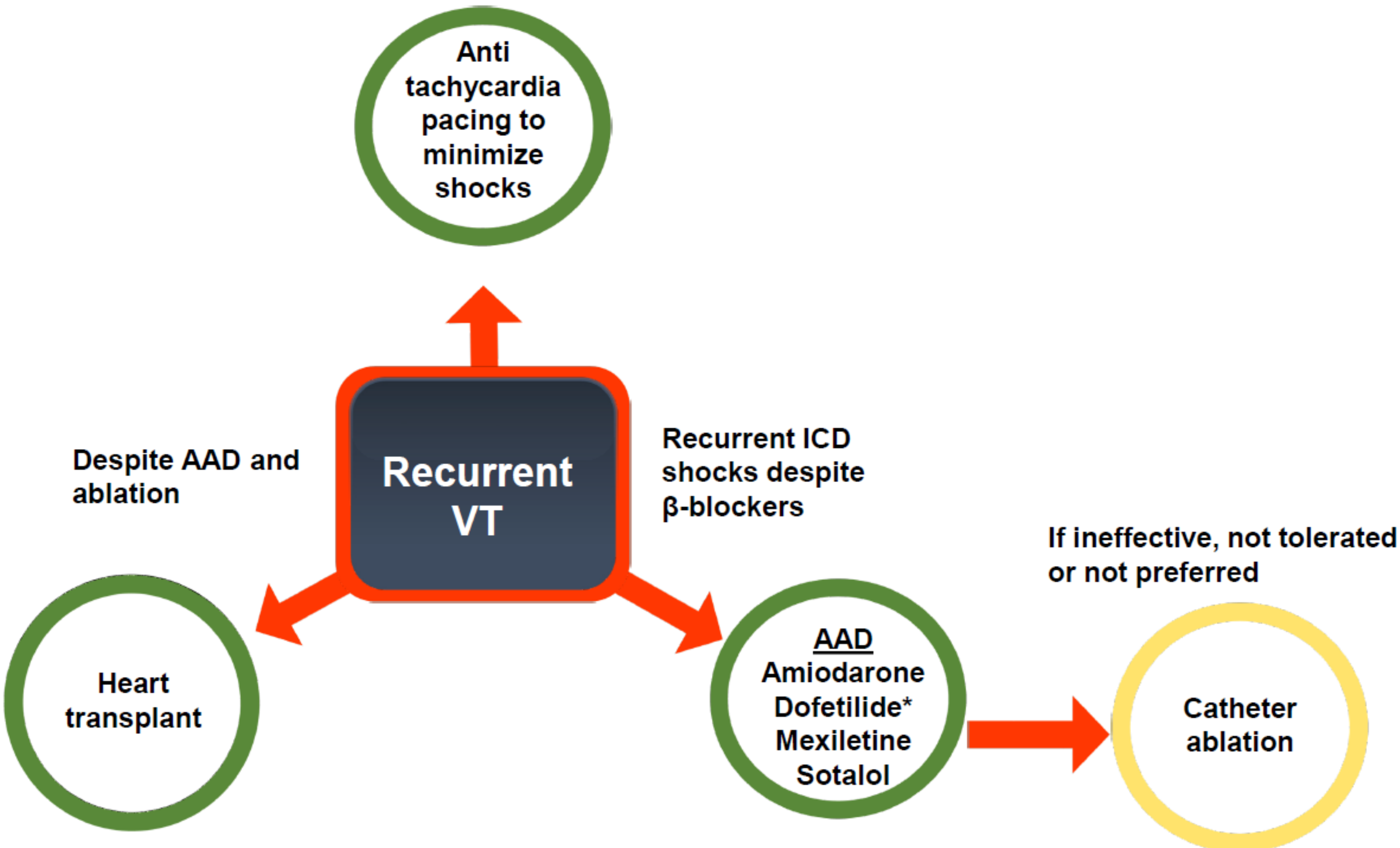
The following is not well established: β blockers or CCBs if asymptomatic

*non-dihydropyridine calcium channel blockers (CCBs)

ⱡLV end-diastolic volume <50 mL/m2 and LV stroke volume <30 mL/m2

Adapted from: 2020 ACC/AHA Guideline for the Diagnosis and Treatment of Patients with Hypertrophic Cardiomyopathy

Management of HCM and Ventricular Arrhythmias



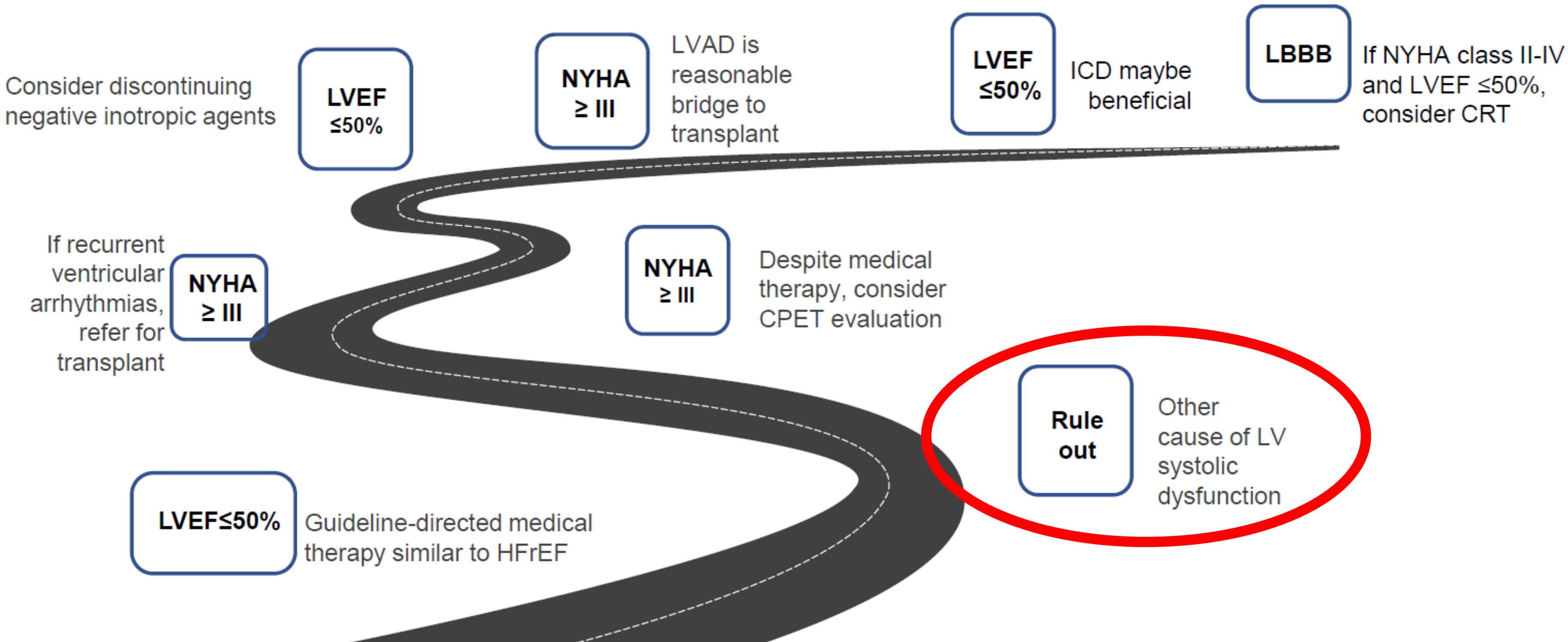
* Not in children

Adapted from: 2020 ACC/AHA Guideline for the Diagnosis and Treatment of Patients with Hypertrophic Cardiomyopathy

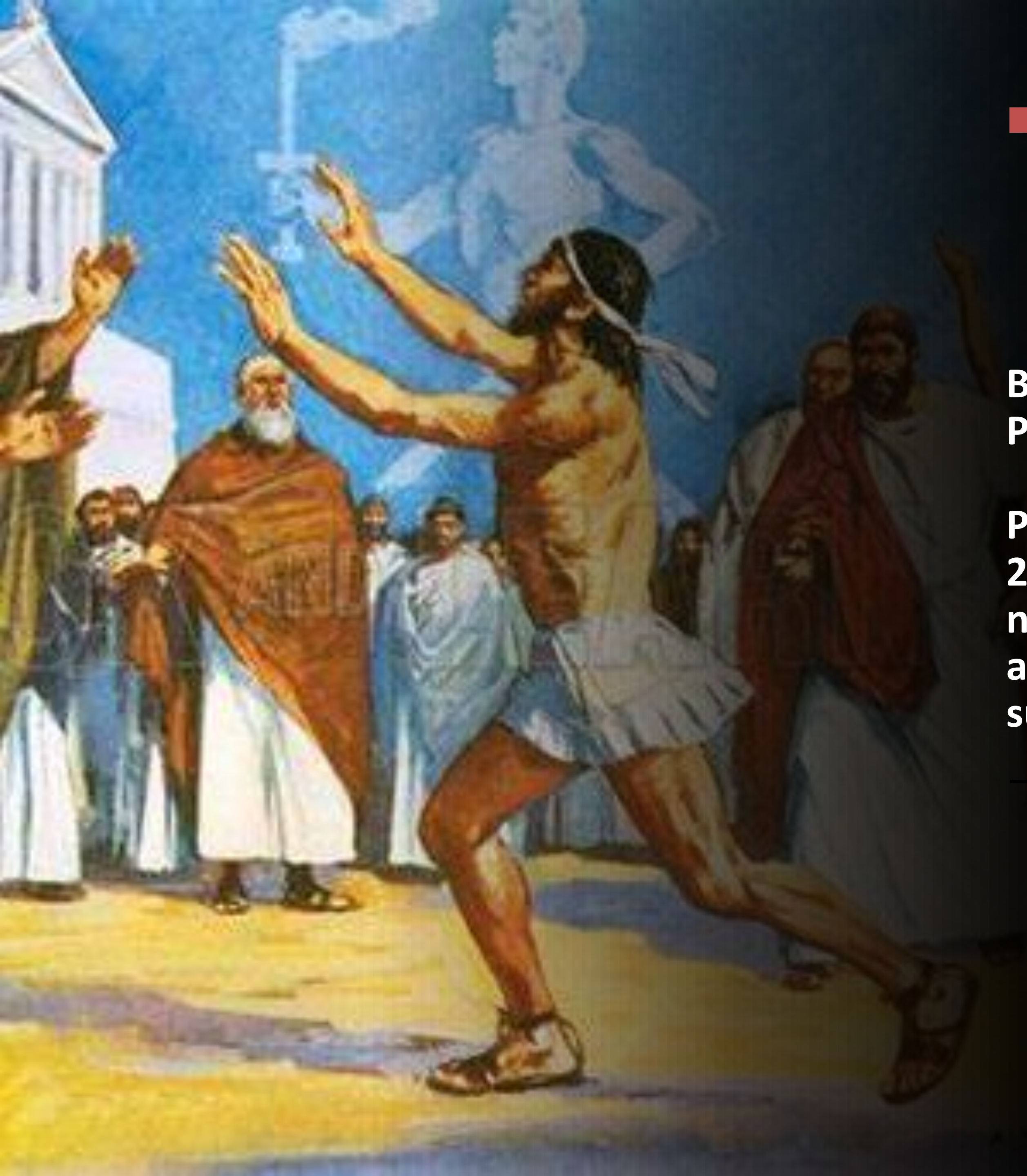
HCM

American Heart Association.

Hypertrophic Cardiomyopathy with Advanced Heart Failure



Adapted from: 2020 ACC/AHA Guideline for the Diagnosis and Treatment of Patients with Hypertrophic Cardiomyopathy



Battle of Marathon (490 BC)
Persian invasion of Greece

Pheidippides, after running 25 miles from the battlefield near Marathon to Athens to announce the Greek victory, suddenly collapsed and died.

Sudden Cardiac Death in Athletes

Sudden Cardiac Death

Athlete	Diagnosis	Incidence	Common Mutation
Reggie Lewis	HCM	1 in 500	Cardiac myosin binding protein C, β-myosin heavy chain, Troponin I, T, α-tropomyosin
Marc Vivien Foe	HCM	1 in 500	
Miklos Feher	HCM	1 in 500	
Zena Ray Upshaw	HCM	1 in 500	
Nick Knapp	HCM	1 in 500	
Hank Gathers	Idiopathic		N/A
Antonio Puerta	ARVD	1 in 2500 - 5000	Plakophilin-2, Desmoglein-2, Desmoplakin, Desmocollin-2
Wes Leonard	DCM	0.57 in 100,000 (≤18 years)	Titin, β-myosin heavy chain, α-myosin, myopalladin, troponin T
Flo Hyman	Marfans Syndrome	1 in 5000	Fibrillin-1, TGF-β receptor 1, 2
Sergei Grinkov	Myocardial infarction	12.9 per 1000 (30 - 34 years)	PLA-2 variant
Alexander Dale Oen	Myocardial infarction	12.9 per 1000 (30- 34 years)	
Darryl Kile	Myocardial infarction	12.9 per 1000 (30-34 years)	
Pete Maravich	Absent LMCA	Rare	N/A
Jim Fixx	Myocardial infarction	600 in 100,000	N/A

HCM = hypertrophic obstructive cardiomyopathy; ARVD = arrhythmogenic right ventricular dysplasia; DCM = dilated cardiomyopathy; CMN = cystic medial necrosis; MS = marfan syndrome; MI = myocardial infarction; PLA = platelet antigen gene; LMCA = left main coronary artery

Epidemiology

- 60 million kids ages 6-18 play sports per year
- 7,600,000 High School Athletes
- 550,000 College Athletes
- 187,000 Division I College Athletes
- 35,000 Professional Athletes (88% men)

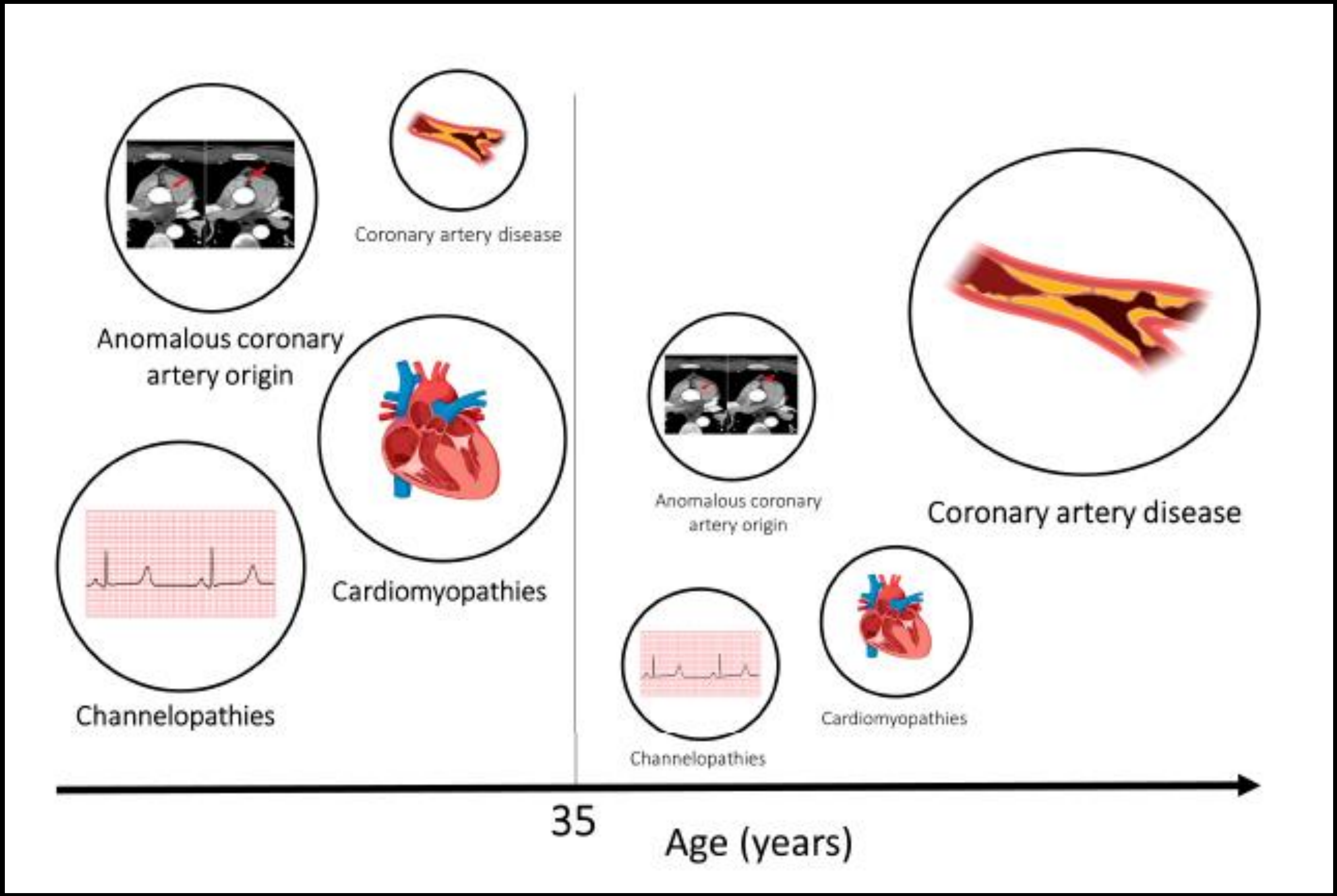
Risk of SCD

- Variable estimates due to variable definitions of an athlete
- Factors altering the risk Gender, Race, Type of Sport
- Wide range 1 in 3,000 to 1 in 1,000,000
- High School Athletes 1 in 200,000

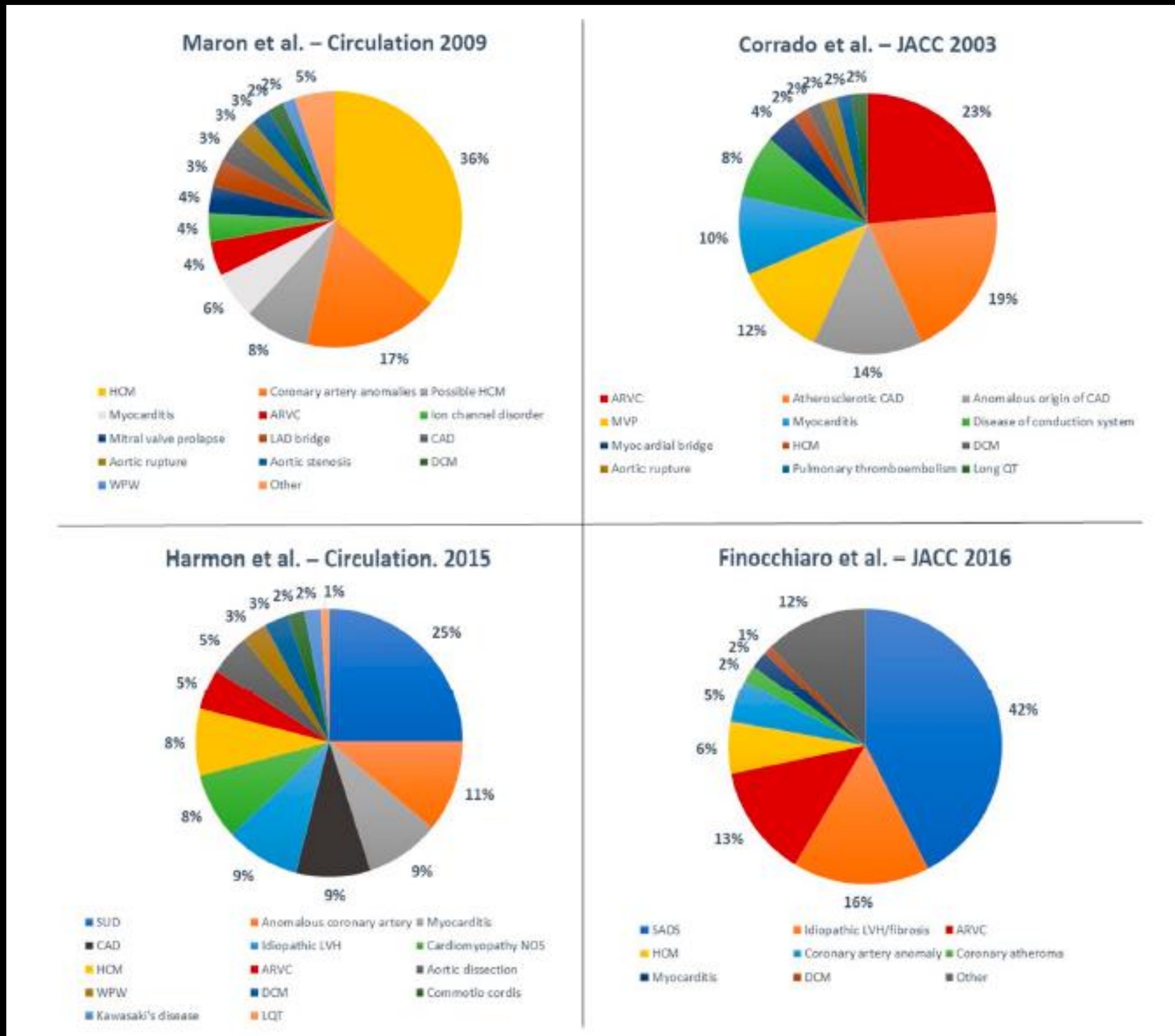
Characteristic	Increased Risk Group	Decreased Risk Group
Overall	1 in 53,703 athlete-years[14]	
Gender	Males: 1 in 37,790	Females: 1 in 121,593
Race	Black: 1 in 21,491	White: 1 in 68,354 Hispanic: 1 in 56,254
Sports	Men's Basketball: 1 in 8,978 Men's Soccer: 1 in 23,689 Men's Football: 1 in 35,951	N/A

Methodist Debakey Cardiovasc J. 2016 Apr-Jun; 12(2): 76–80.

Etiology Stratified by Age

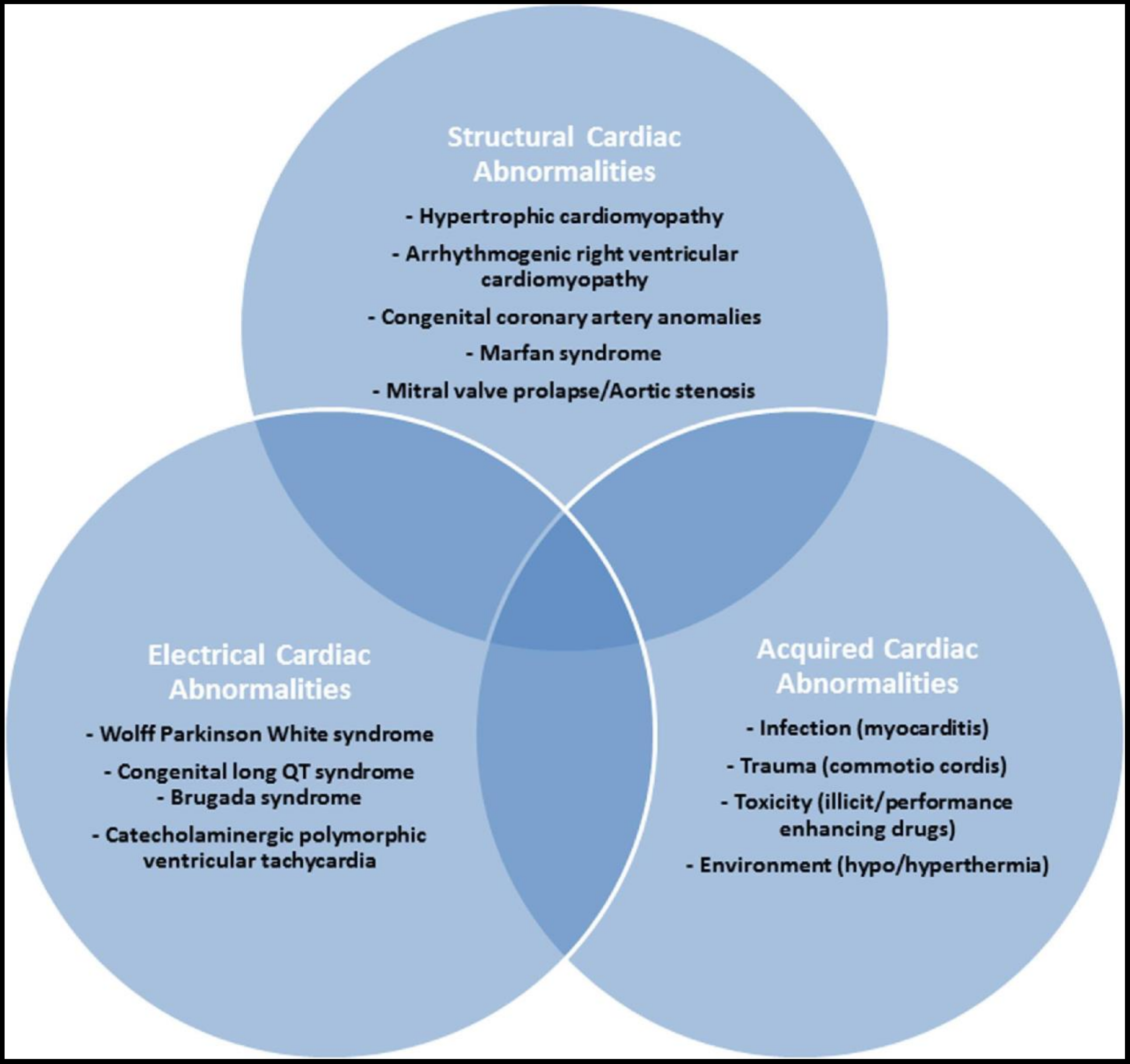


J. Cardiovasc. Dev. Dis. 2023, 10, 68.

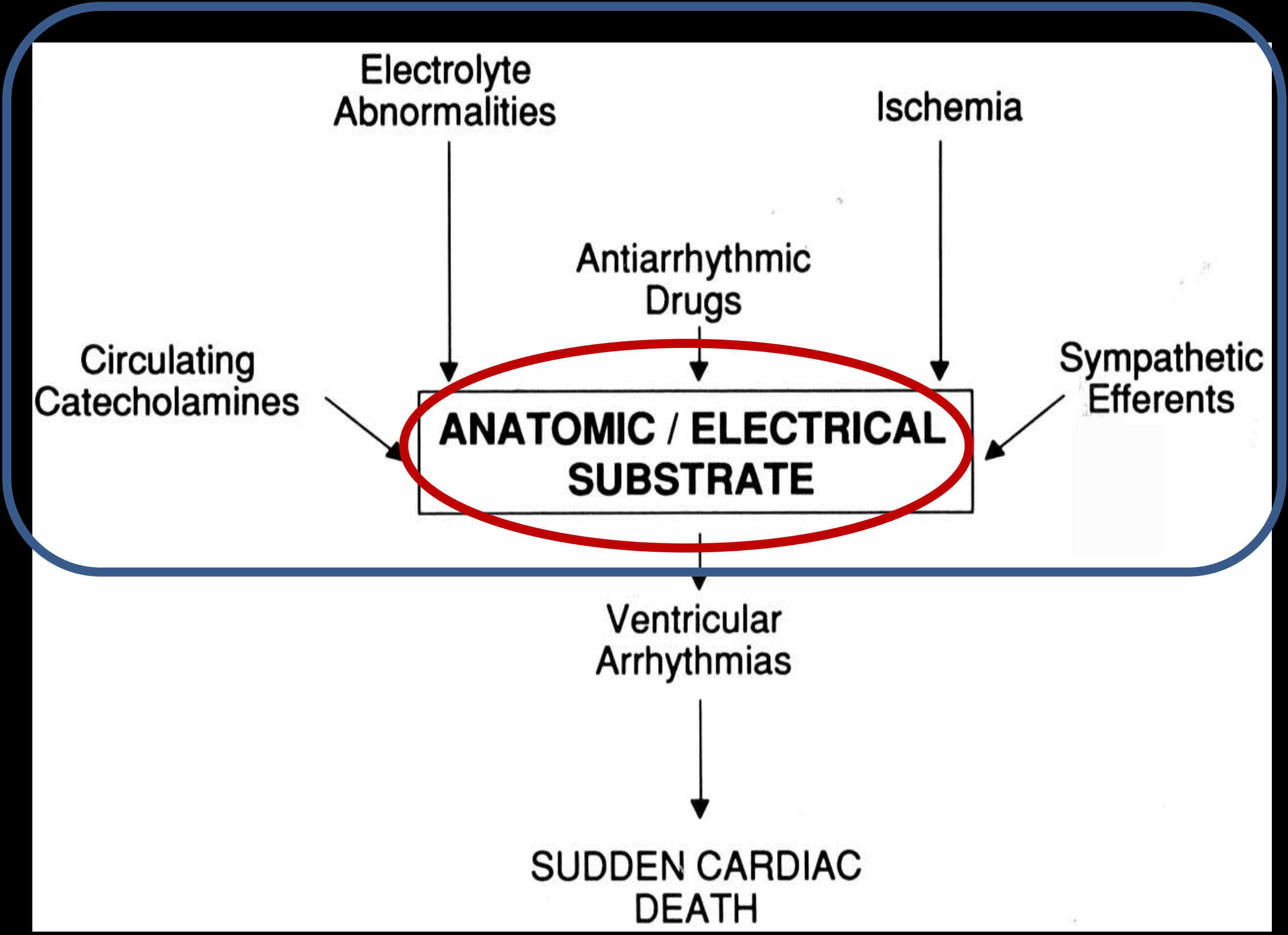


J. Cardiovasc. Dev. Dis. 2023, 10, 68.

Pathophysiology

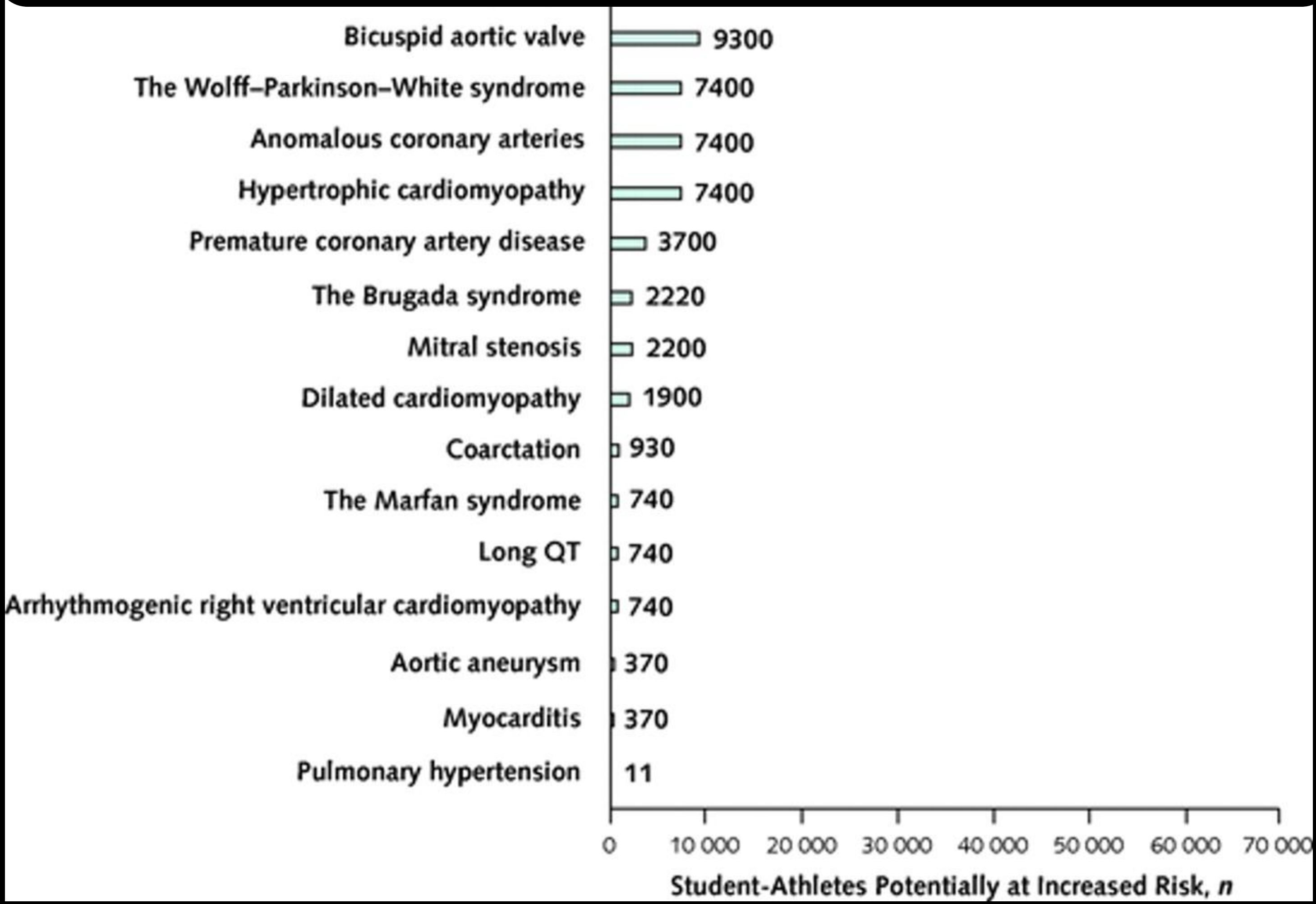


JACC. Volume 61. Issue 10. 12 March 2013, Pages 1027-1040

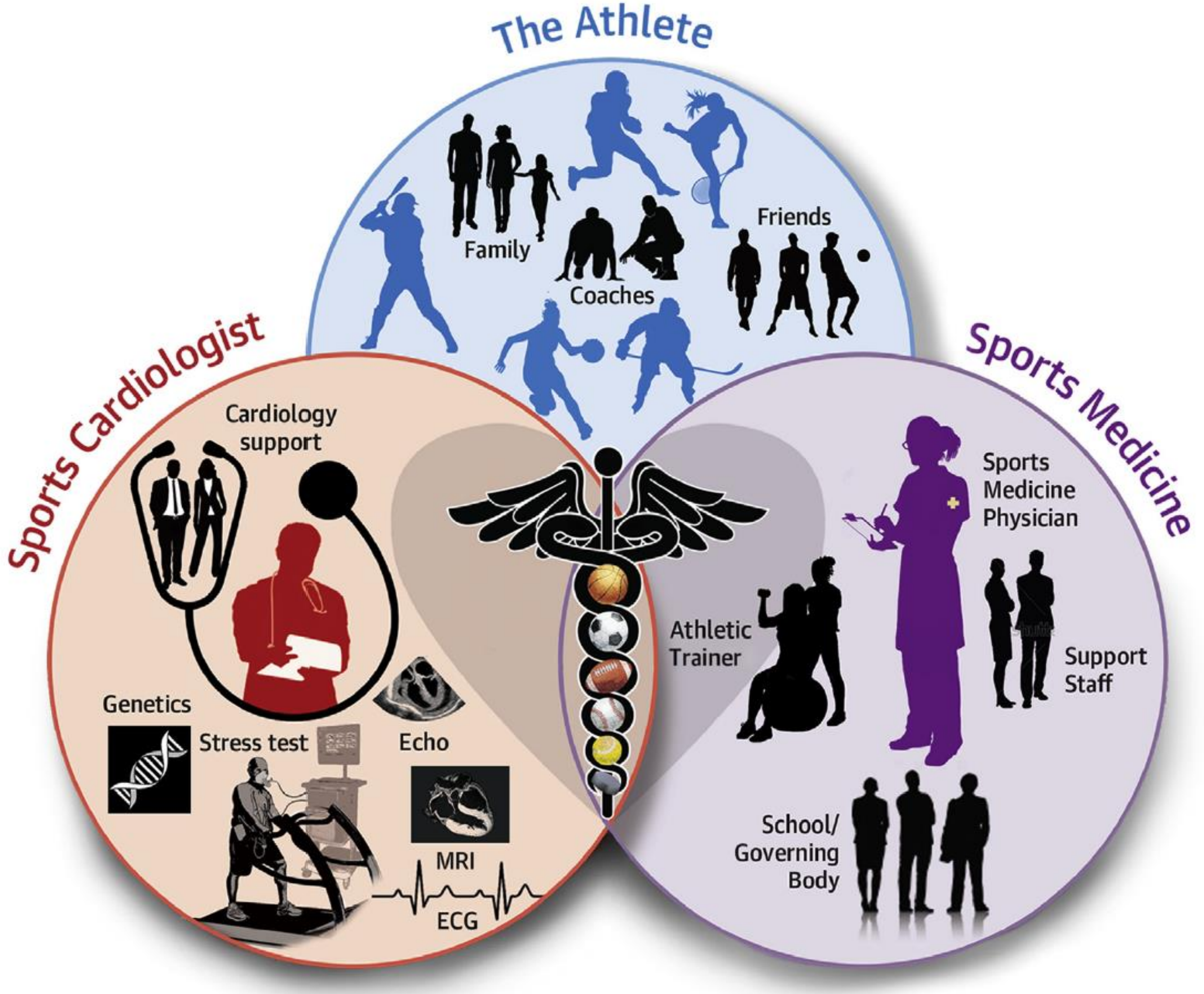


Gilman JK, Naccarelli GV. Curr Prob Cardiol Nov 1992.

Prevalence of Cardiac Abnormalities in Student-Athletes



Team-Based Approach to the Cardiovascular Care of Athletes



Emery, M.S. et al. J Am Coll Cardiol HF. 2018;6(1):30-40.

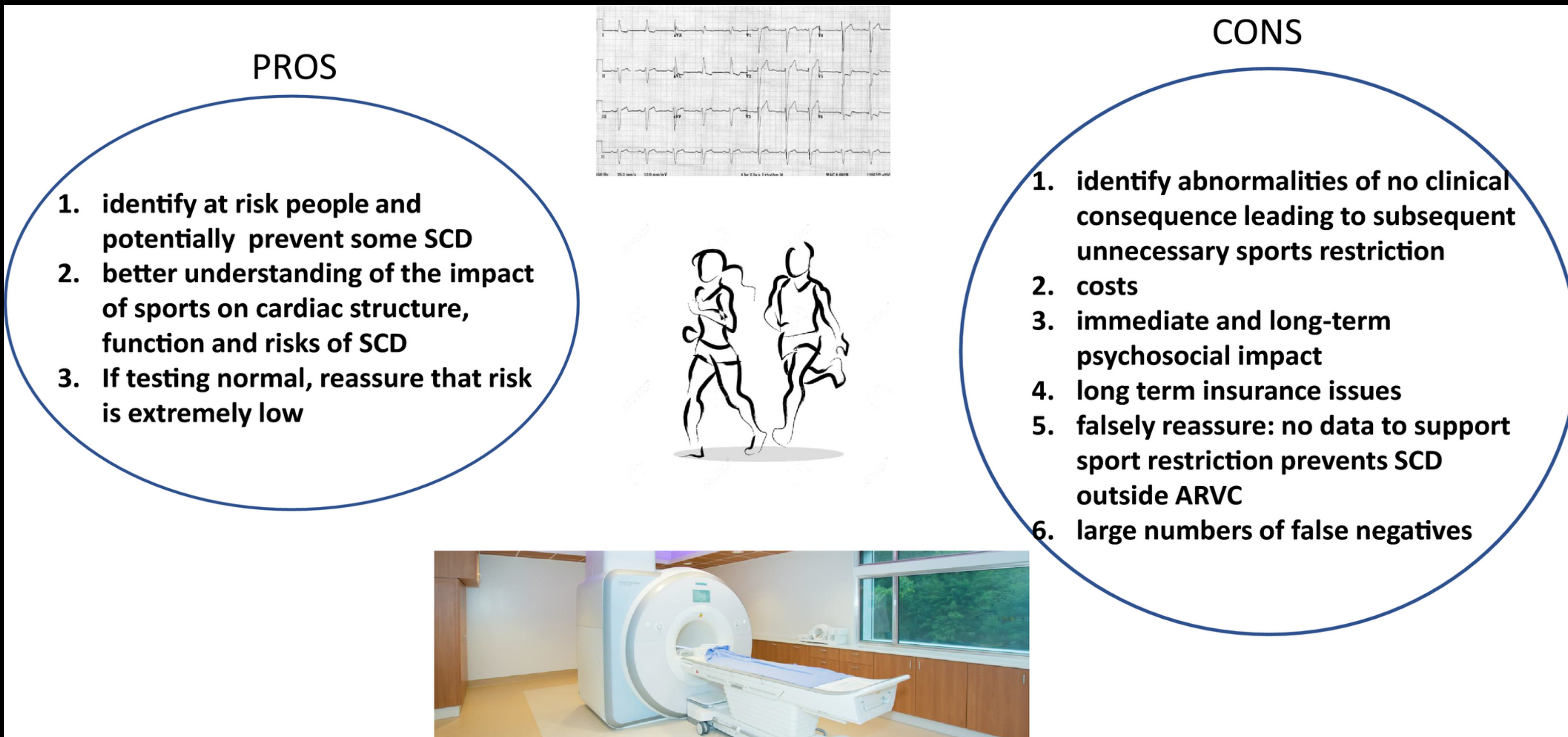
AHA Recommendations	Pre-Participation Physical Evaluation
Medical history*	
Personal History	Heart Health Questions About You
1. Chest pain/discomfort/tightness/pressure related to exertion	6. Have you ever had discomfort, pain, tightness, or pressure in your chest during exercise?
2. Unexplained syncope/near syncope†	5. Have you ever passed out or nearly passed out DURING or AFTER exercise?
3. Excessive and unexplained dyspnea/fatigue or palpitations, associated with exercise	12. Do you get more tired or short of breath more quickly than your friends during exercise? 10. Do you get lightheaded or feel more short of breath than expected during exercise? 7. Does your heart ever race or skip beats (irregular beats) during exercise?
4. Prior recognition of a heart murmur 5. Elevated systemic blood pressure	8. Has a doctor ever told you that you have any heart problems? If so, check all that apply: ☐ High blood pressure ☐ A heart murmur ☐ High cholesterol ☐ A heart infection ☐ Kawasaki disease Other: __________
6. Prior restriction from sports	1. Has a doctor ever denied or restricted your participation in sports for any reason?
7. Prior testing for heart disease, ordered by a physician	9. Has a doctor ever ordered a test for your heart? (For example, ECG/EKG, echocardiogram)
	11. **Have you ever had an unexplained seizure?**
Family History	Heart Health Questions About Your Family
8. Premature death (sudden and unexpected or otherwise) before 50 yrs of age attributable to heart disease ≥1 relative	13. Has any family member or relative died of heart problems or had an unexpected death before age 50 (including drowning, unexplained car accident, or sudden infant death syndrome)?
9. **Disability from heart disease in a close relative <50 yrs of age**	
10. Hypertrophic or dilated cardiomyopathy, long QT syndrome or other ion channelopathies, Marfan syndrome, or clinically significant arrhythmias; specific knowledge of genetic cardiac condition in family member	14. Does anyone in your family have hypertrophic cardiomyopathy, Marfan syndrome, arrhythmogenic right ventricular cardiomyopathy, long QT syndrome, short QT syndrome, Brugada syndrome, or catecholaminergic polymorphic ventricular tachycardia?
	15. **Does anyone in your family have a heart problem, pacemaker, or implanted defibrillator?**
	16. **Has anyone in your family had unexplained fainting, unexplained seizures, or near drowning?**
Physical examination	Physical examination
11. Heart murmur‡	a. Heart ■ Murmurs (auscultation standing, supine, with or without Valsalva) ■ Location of point of maximal impulse
12. Femoral pulses to exclude coarctation	b. Pulses ■ Simultaneous femoral and radial pulses
13. Physical stigmata of Marfan syndrome	c. Appearance ■ Marfan stigmata (kyphoscoliosis, high-arched palate, pectus excavatum, arachnodactyly, arm span >height, hyperlaxity, myopia, MVP, aortic insufficiency)
14. Brachial artery blood pressure (sitting position)§	d. Blood pressure

J Am Coll Cardiol HF2018;6:30–40.

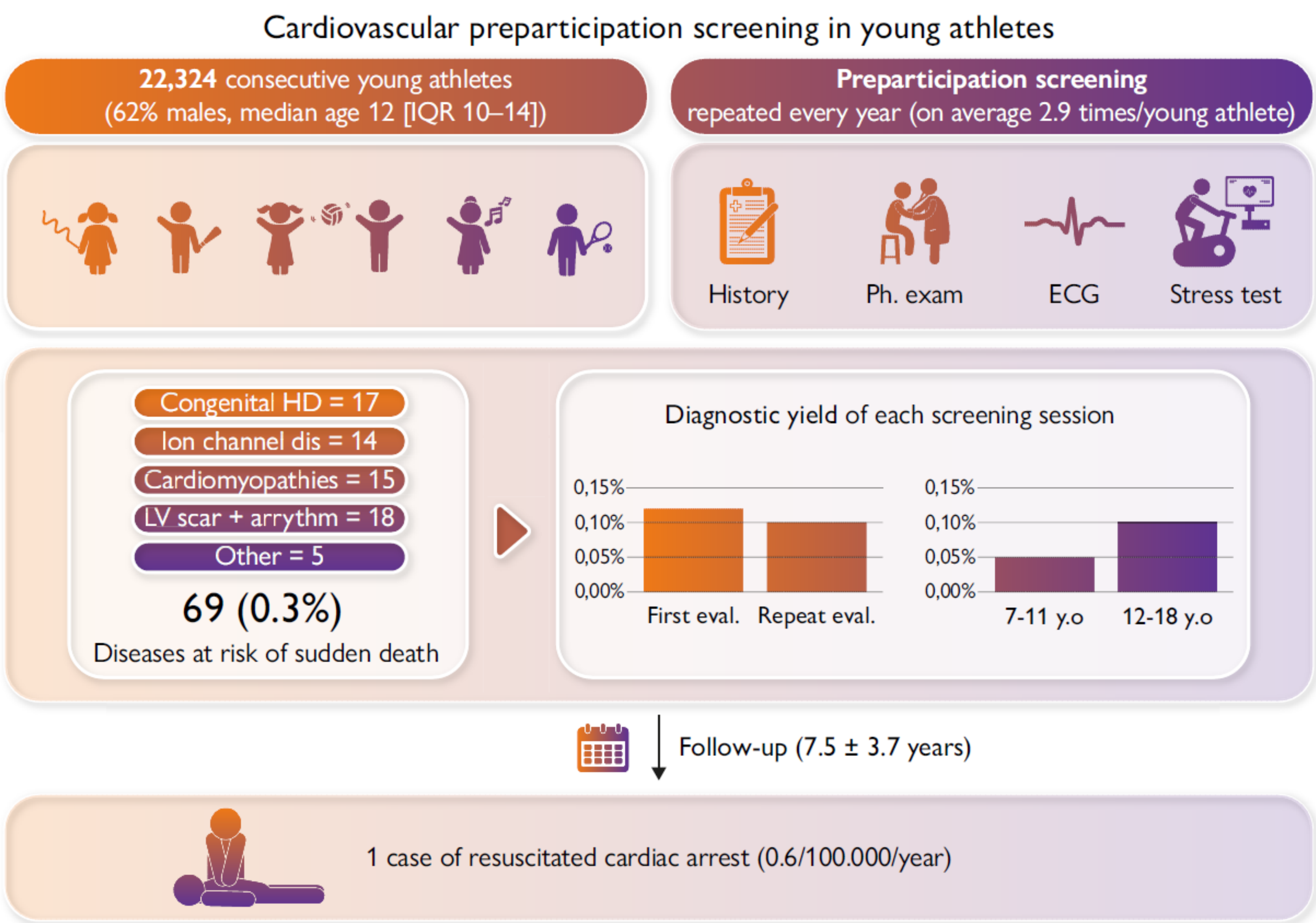
Prevention

- Sudden cardiac death in athletes may be prevented through the implementation of policies aimed at identifying cardiac conditions that may pose a risk in asymptomatic individuals (screening) and policies that increase the likelihood of successful resuscitation of cardiac arrests.

Screening



Paul Dorian. Journal of the American Heart Association. Policies to Prevent Sudden Cardiac Death in Young Athletes: Challenging, But More Testing Is Not the Answer, Volume: 9, Issue: 8, DOI: (10.1161/JAHA.120.016332)



European Heart Journal (2023) 44, 1084–1092
https://doi.org/10.1093/eurheartj/ehad017

No Consensus

United States

- History
- Physical

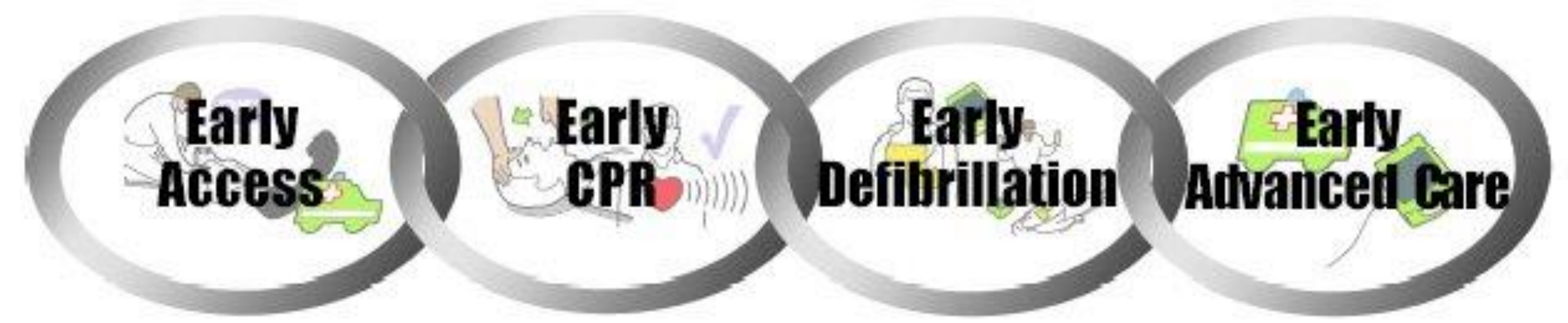
Europe

- History
- Physical
- 12-lead ECG

Italy

- + Echo

Emergency Action Plan



Summary

Exercise

- Basics overview of exercise physiology
- Types of exercise & cardiac remodeling
- Beneficial effects of physical activity & your health

Athlete's Heart

- Regular exercise promotes structural, functional, and electrical remodeling of the heart
- Gender differences & the need for increased research involving women

Hypertrophic Cardiomyopathy

- Genetics
- Morphologic and pathophysiologic phenotypes
- Clinical Findings
- Diagnostic evaluation & management
- Screening

Sudden Cardiac Death in the Athlete

- Epidemiology
- Etiologies
- Pathophysiology
- Screening & Prevention