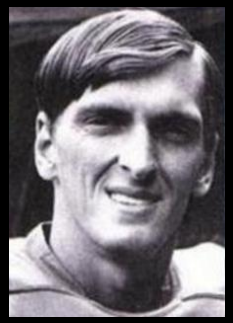
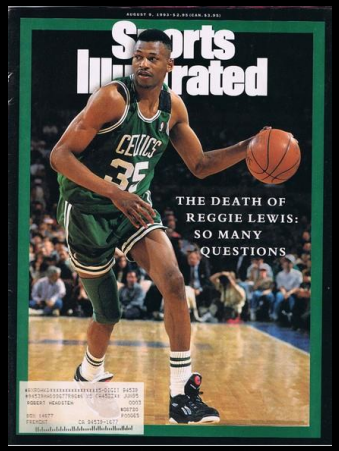
Προαθλητικός καρδιολογικός έλεγχος

Σταύρος Κούνας
Δ/ντής Καρδιολογικού Τμήματος
ΒΙΟΚΛΙΝΙΚΗ ΑΘΗΝΩΝ





1971 Chuck Hughes SD (CAD) NFL

1976 Owen Brown SD (HCM)

1980 1st SD in athletes paper (Maron & Roberts)

1985 Bethesda #16

1987 1st Screening studies (college athletes)

1988 "Pistol" Pete Maravich SD

1990 Hank Gathers SD

1993 Reggie Lewis SD

1995 Commotio Cordis described

1998 Knapp v. Northwestern

2005 Jason Collier Thomas Herrion Jiri Fischer (SD/CA)

2006 Veneto screening data

2009 Causes SD in athletes (n=1866)

2015 AHA/ACC screening recommendations #3

1976 Chris Patton SD (Marfan)

1981 U.S. Sudden Death in Athletes Registry begins

1986 Len Bias SD (cocaine)

1988 ARVC introduced as disease (& cause of SD in athletes)

1994 Bethesda #26

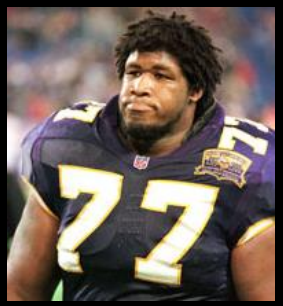
1996 AHA screening recommendations #1

2001 Korey Stringer SD (heat stroke)

2005 Bethesda #36

2007 AHA screening recommendations #2

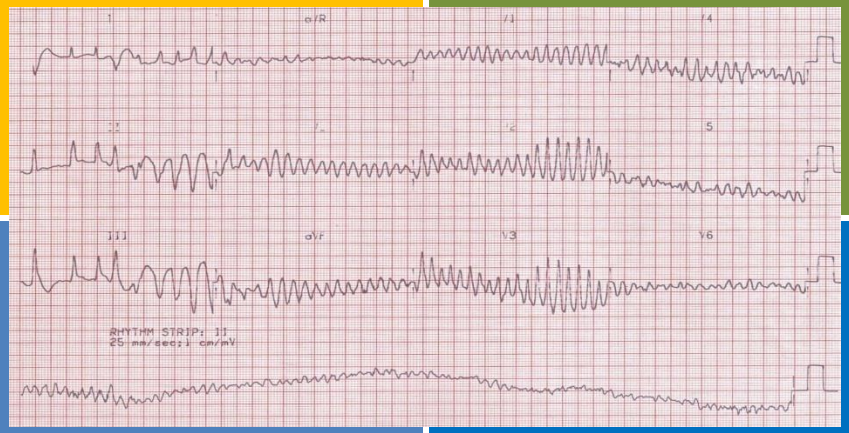
2015 U.S Sudden Death in Athletes Registry (n=3000)



Maron BJ. Am J Cardiol 2015;116:1461-68

Δομικές καρδιοπάθειες

Ισχαιμική καρδιοπάθεια



Αρρυθμιολογικά σύνδρομα

Φάρμακα - άλλα

Pre-participation screening

Young Athletes < 35 years old

Young Athletes - Incidence of SCD

Table 1 Incidence studies in general populations of atheltes

Author	Year	Country	Study design	Case identification	Denominator	Exertional deaths of all?	SCD or SCA+SCD	Years studied	Population	Incidence	Number of years	Age range	Mean age	Number of cardiac deaths
Van Camp[21]	1996	USA	Retrospective cohort	National Center for Catastrophic Injury Research and media database	17 most popular sports, participants in NCAA, NFHS, NAIA, NAJC, added together, conversion factor to account multisport athletes used 'based on discussions with representatives from the national organisations'. 1.9 for high school and 1.2 for college	Exertional	SCD	1983–1993	College athletes and high school	1:300 000	10	17–24	17	100
Maron[26]	1996	USA	Retrospective cohort	US Registry for Sudden Death in Athletes	'Unavoidable selection bias and certainly significantly underestimate'	All	SCD	1985–1995	Athletes	_	10	12–40	17	134
Maron[25]	2003	USA	Retrospective cohort	US Registry for Sudden Death in Athletes	Not possible b/c of selection bias	All	SCD	1985–2000	Athletes	_	25	9–40	17	286
Corrado[22]	2003	Italy	Prospective cohort study	Mandatory death reporting	Registered Italian athletes	All	SCD	1979–1999	Athletes and young people	1:47 600 athlete 1:142 900 young people	20	12 –35	23	55
Maron[8]	2009	USA	Retrospective cohort	US Registry for Sudden Death in Athletes	Estimated 10.7 million athletes <39 participating in sports during 2000–2006 (method not described)	All	SCA+SCD	1980–2006	Athletes	1:163 934	27	8–39	18	690
Holst[6]	2010	Denmark	Retrospective cohort	Review of death certificates—then autopsies if available —15 sports related SCD (SrSCD)	Denmark population statistics	All and sports related	SCD	2000–2006	Athletes and young people	1:82 645 SrSCD 1:26 595 general pop	7	12 –35	26	15 SrSCD 470 SCD
Steinvil[23]	2011	Israel	Retrospective cohort	Retrospective review of two Israeli newspapers by two media researchers	45 000 registered competitive athletes in 2009, extrapolated the growth of the Israeli population age10–40 since 1985 based on that figure and allowed for a presumed doubling of the sporting	All	SCD	1985—19 971 998—2009	Athletes	1st—1:393 702nd—1:37 593	24	12–44	24	24

1 : 50.000 – 80.000

Table 2 Incidence studies in college athletes

Author	Year	Country	Study design	Case identification	Denominator	Exertional deaths of all?	SCD or SCA +SCD	Years studied	Population	Incidence	Number of years	Age range	Mean age	Number of cardiac deaths
Drezner[29]	2005	USA	Retrospective survey	Survey answered by 244/326 (75%) Div I NCAA institutions	Number of athletes at surveyed schools	All	SCD		College athletes	1:67 000	3.3			5
Harmon[3]	2011	USA	Retrospective cohort	Parent Heart Watch database, NCAA Resolutions list, insurance claims—capture–recapture analysis demonstrated 90–100% of deaths were likely identified	Participation data from NCAA	All	SCD	2004–2008	College athletes	1:43 000	5	18–26	20	37
Maron[7]	2014	USA	Retrospective cohort	US Registry for Sudden Death in Athletes and NCAA resolutions list for cardiac cases	Participation data from NCAA	All	SCD	2002–2011	College athletes	1:83 000—confirmed 1:62 000—presumed	10	17–26	20	64

Harmon KG, et al. Heart 2014;100:1227–1234

Sports as a trigger for SCD

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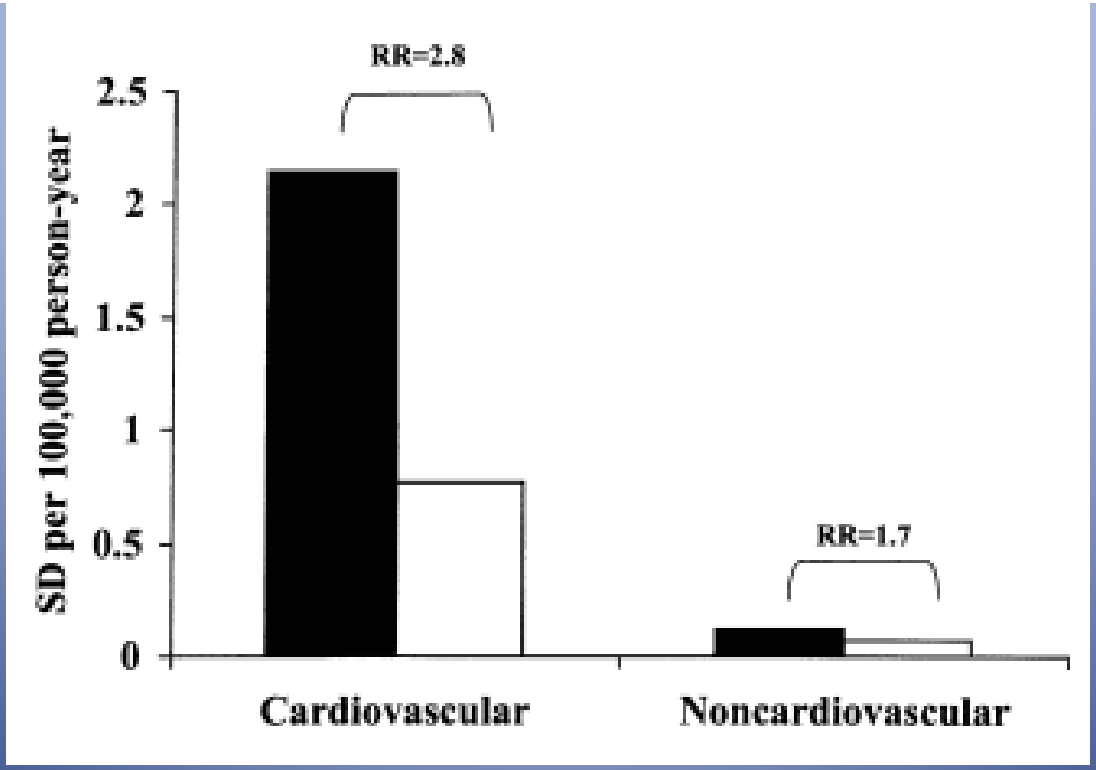
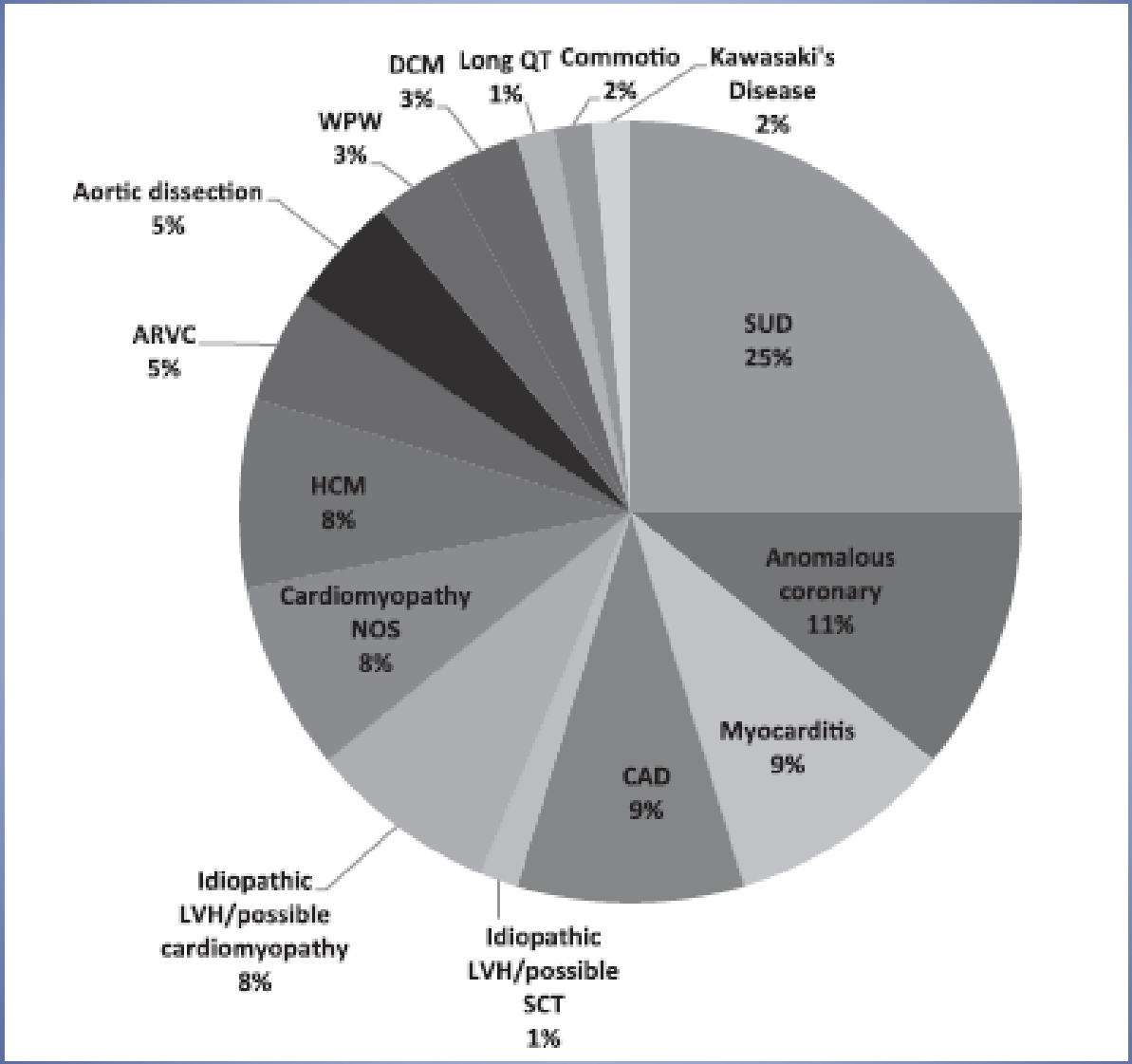
doi:10.1016/j.jacc.2003.03.002

Does Sports Activity Enhance the Risk of Sudden Death in Adolescents and Young Adults?

Domenico Corrado, MD, PHD,* Cristina Basso, MD, PHD,† Giulio Rizzoli, MD,‡
Maurizio Schiavon, MD,§ Gaetano Thiene, MD†
Padua, Italy



Young Athletes - Causes of SCD



Harmon K et al. Circulation. 2015;132:10-19.

Young Athletes - Causes of SCD

Table 3. Incidence of Sudden Cardiac Death in NCAA Athletes

Characteristic	Athlete-Years	SCD	Incidence per Athlete-Year	IRR	95% CI	*P* Value
Overall	4242519	79	1 in 53703	–	–	–
Sex						
Male	2418563	64	1 in 37790	3.22	1.9–5.5	>0.0001*
Female	1823899	15	1 in 121593	1.00	Reference	
Division						
Division 1	1663441	38	1 in 43775	1.98	1.1–3.6	0.0131*
Division 2	930434	22	1 in 42292	2.05	1.1–4.0	0.0231*
Division 3	1648128	19	1 in 86744	1.00	Reference	
Race						
White	3075942	45	1 in 68354	1.00	Reference	
Black	644715	30	1 in 21491	3.18	1.9–5.2	>0.0001*
Hispanic	168763	3	1 in 56254	1.22	0.2–3.8	0.6974
Other	353042	1	1 in 353042	0.19	0.005–1.1	0.0491*

Harmon K et al. Circulation. 2015;132:10-19.

Screening Protocol – AHA 2015

Table 1. The 14-Element AHA Recommendations for Preparticipation Cardiovascular Screening of Competitive Athletes

Medical history*

Personal history

1. Chest pain/discomfort/tightness/pressure related to exertion
2. Unexplained syncope/near-syncope†
3. Excessive and unexplained dyspnea/fatigue or palpitations, associated with exercise
4. Prior recognition of a heart murmur
5. Elevated systemic blood pressure
6. Prior restriction from participation in sports
7. Prior testing for the heart, ordered by a physician

Family history

8. Premature death (sudden and unexpected, or otherwise) before 50 y of age attributable to heart disease in ≥1 relative
9. Disability from heart disease in close relative <50 y 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 conditions in family members

Physical examination

11. Heart murmur‡
12. Femoral pulses to exclude aortic coarctation
13. Physical stigmata of Marfan syndrome
14. Brachial artery blood pressure (sitting position)§

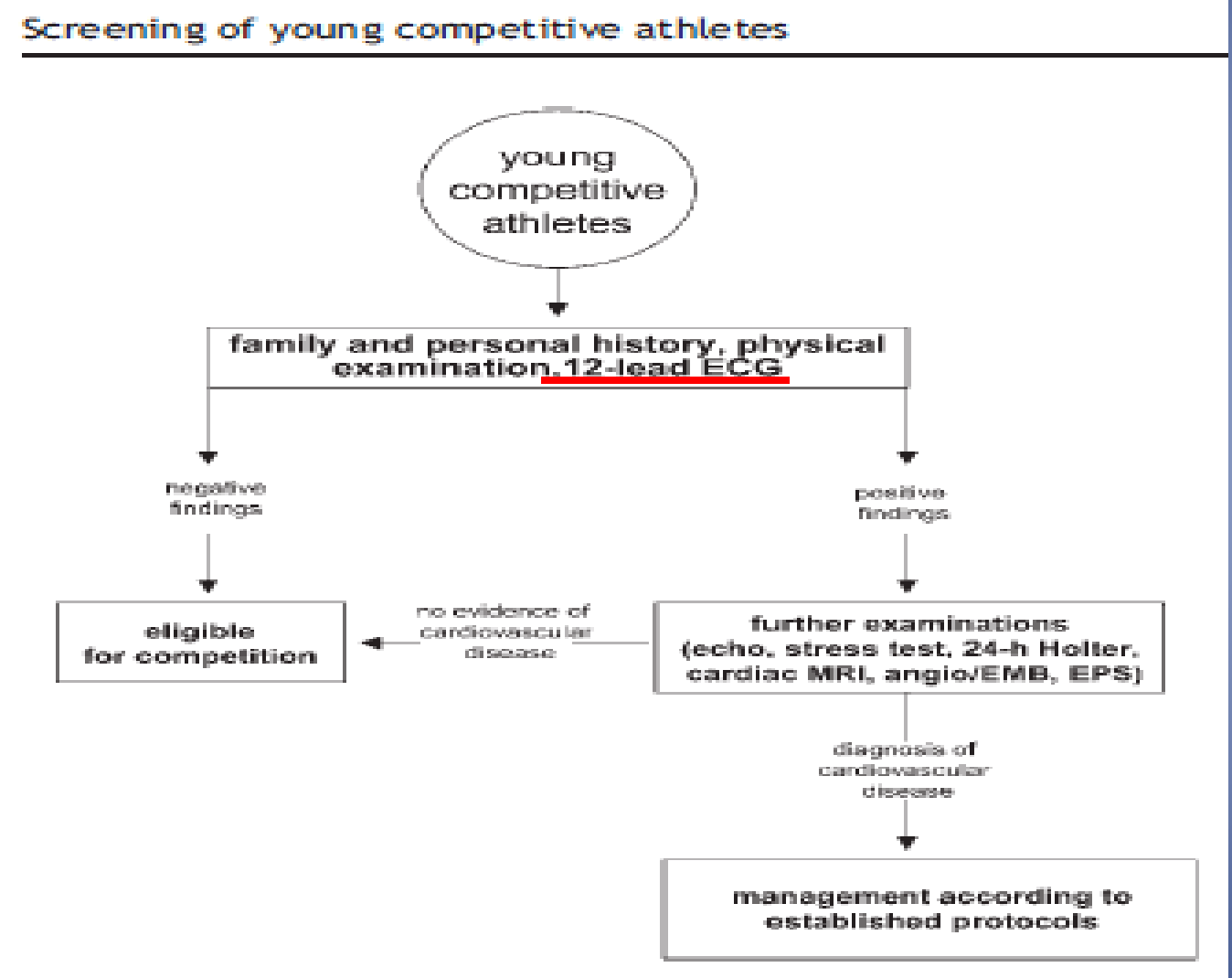
Circulation. 2014;130:1303-1334

Screening Protocol – AHA 2015

- AHA's **14-point** screening guidelines as part of a **comprehensive history taking and physical examination** to detect or raise suspicion of genetic/congenital cardiovascular abnormalities (Class I; Level of Evidence C).
- Screening with **12-lead ECGs** (or echocardiograms) …..may be considered in relatively small cohorts of young healthy people 12 to 25 years of age, not necessarily limited to competitive athletes (Class IIb; Level of Evidence C)
- Mandatory and universal mass screening with 12-lead ECGs in large general populations…. is not recommended for athletes and nonathletes alike (Class III, no evidence of benefit; Level of Evidence C).

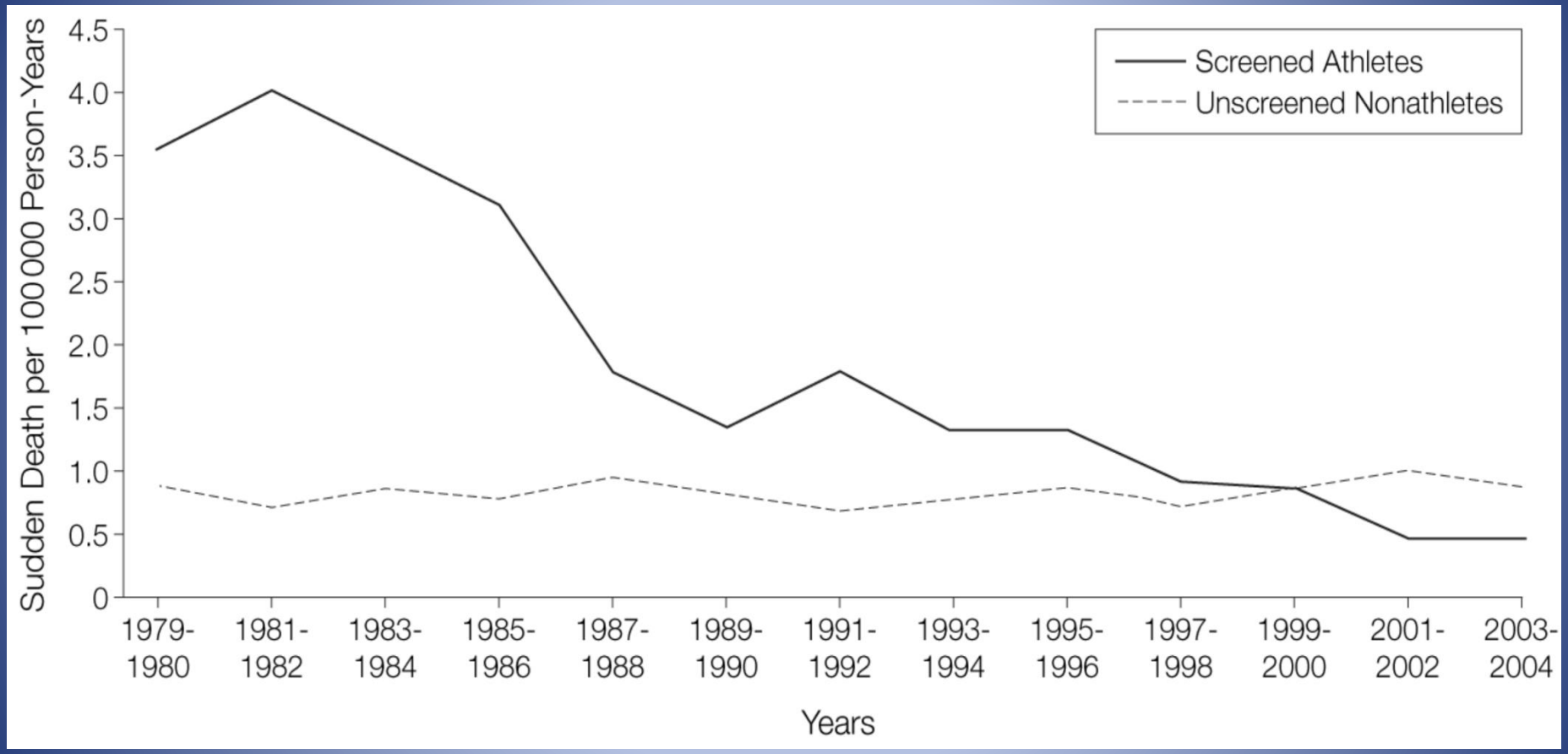
Circulation. 2015;132:e267–e272

Screening Protocol – ESC



European Heart Journal (2005) 26, 516–524

Is ECG effective (mandatory)?



Corrado D et al. JAMA. 2006;296(13):1593-1601

Cost effectiveness

Br J Sports Med. 2016 Jun 23. pii: bjsports-2015-095902. doi: 10.1136/bjsports-2015-095902. [Epub ahead of print]

Results of a nationally implemented de novo cardiac screening programme in elite rugby players in England.

Ghani S[1], Papadakis M[1], Kemp S[2], Zaidi A[1], Sheikh N[1], Gati S[1], Raju H[1], Smith A[3], Palmer C[4], Somauroo J[5], Sharma S[1].

⊕ Author information

Abstract

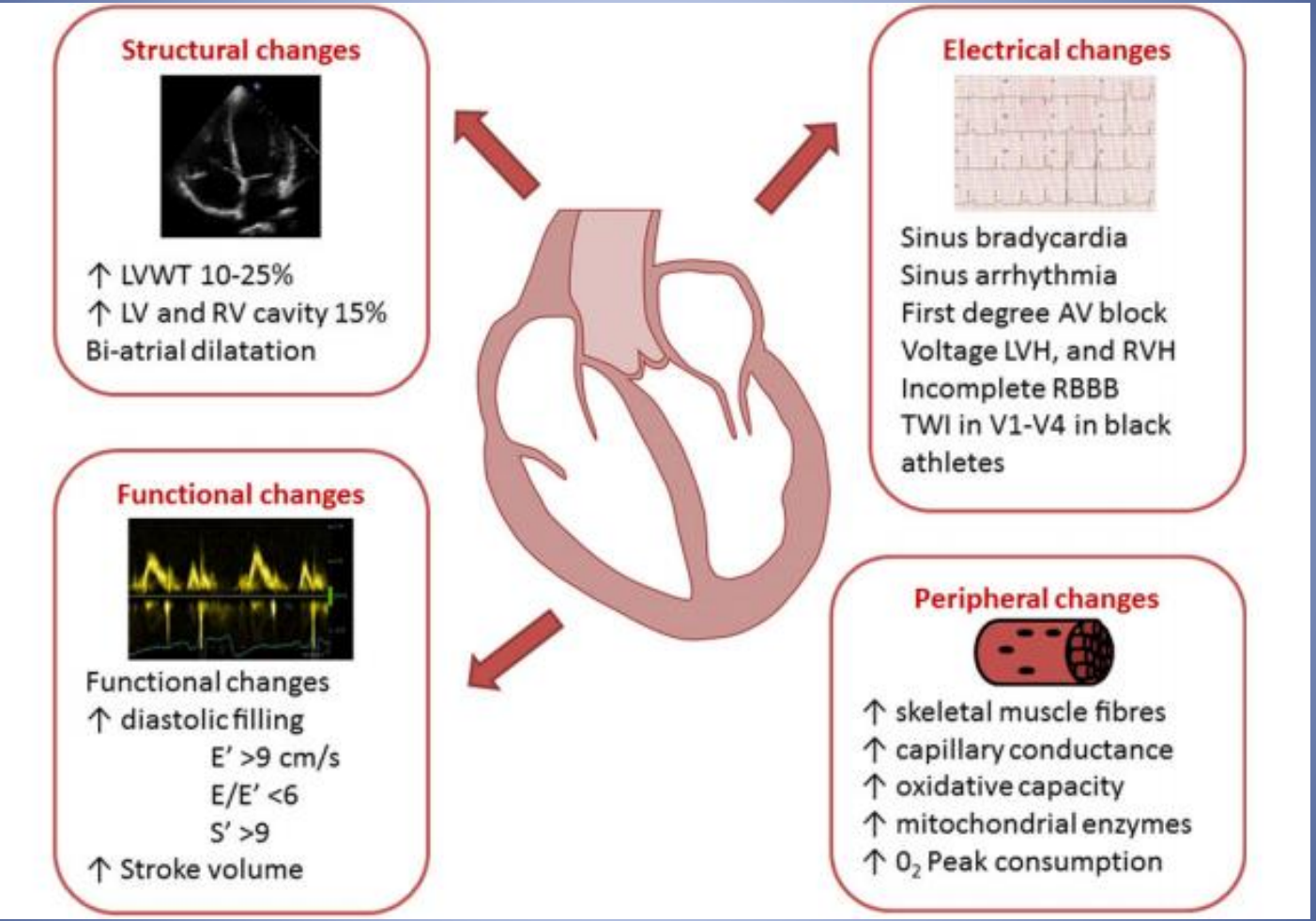
BACKGROUND/AIM: Screening of young competitive athletes remains a contentious issue. In 2010, a nationwide cardiac screening for all elite rugby players was introduced in England. This provided a unique opportunity to prospectively assess the feasibility and cost-effectiveness of a de novo, ECG-based cardiac screening programme.

METHODS: Between 2010 and 2012, 1191 rugby players aged ≥14 years underwent cardiac screening with a health questionnaire, 12-lead ECG and a consultation with a cardiologist. The players with concerning findings on initial evaluation were offered on-site transthoracic echocardiogram (TTE). Athletes were referred for further investigations as deemed necessary. The overall cost of the screening programme was estimated.

RESULTS: After initial evaluation, 9.7% of athletes underwent on-site TTE; 8.2% underwent on-site TTE due to ECG anomalies and 1.4% underwent on-site TTE due to concerns on the questionnaire. After TTE, only 2.9% of the total cohort was referred for further evaluation. Two players were diagnosed with potentially serious conditions; one with Wolff-Parkinson-White, who resumed competition after catheter ablation, and one with hypertrophic cardiomyopathy, who withdrew from competition. During a mean follow-up of 52.8±5.5 months, none of the players who were reassured experienced any adverse cardiac events. The total cost of the screening programme was £59 875, which averaged to a cost of £50 per player or £29 938 per condition identified. Application of refined ECG criteria would reduce the ECG false-positive rate to 4.9%.

CONCLUSIONS: Preparticipation cardiac screening with 12-lead ECG is feasible. Refinement of the ECG criteria, the use of on-site TTE and expert setting can minimise the burden of unnecessary investigations and reduce costs.

Athlete's Heart adaptations



Sharma S et al. European Heart Journal (2015) 36, 1445–1453

Seattle criteria

ECG INTERPRETATION IN ATHLETES

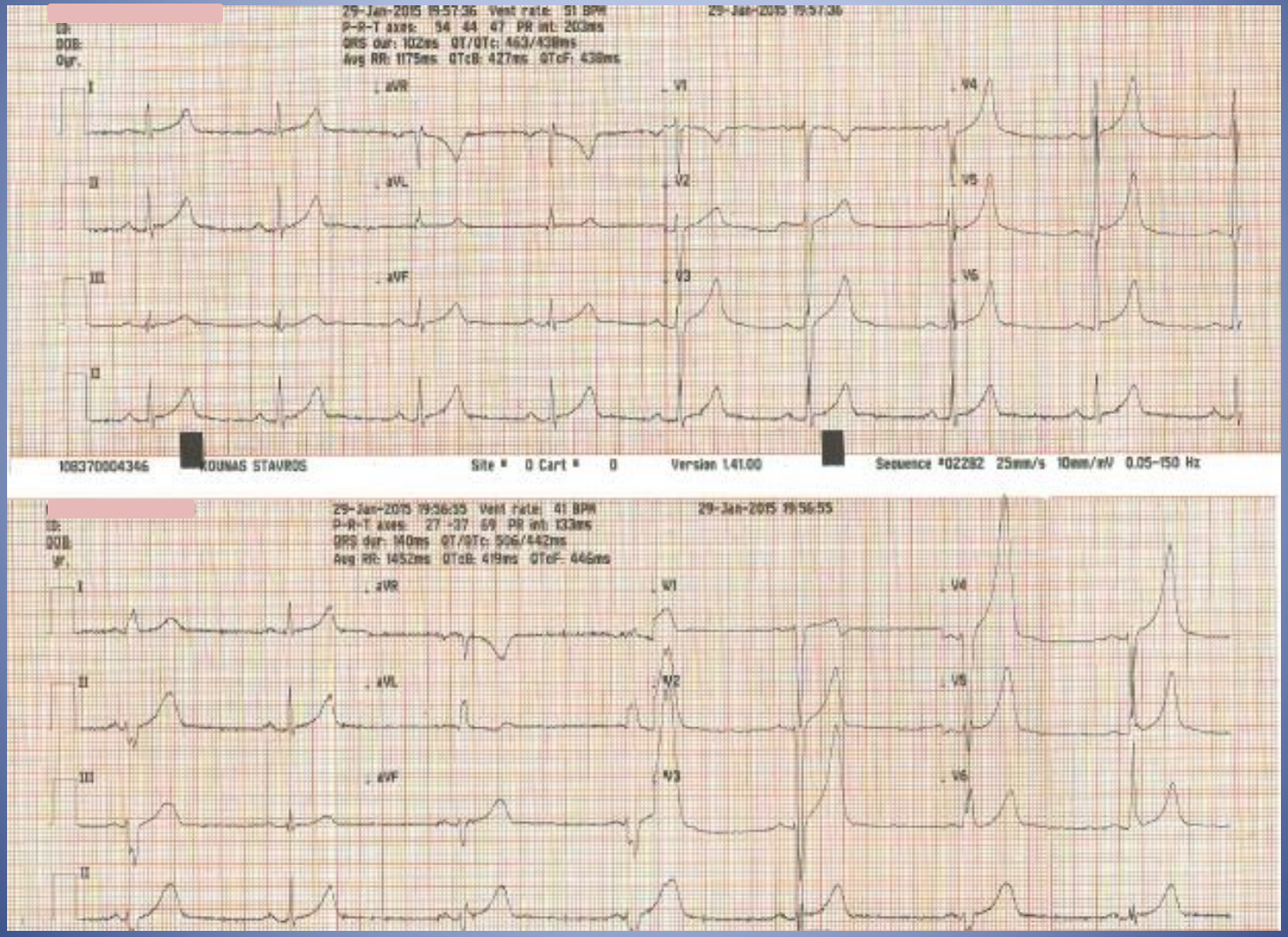
Table 1 Abnormal ECG criteria in Athletes

Any abnormal finding is considered training-unrelated and suggests the possibility of underlying pathological cardiac disease, requiring further diagnostic work-up.

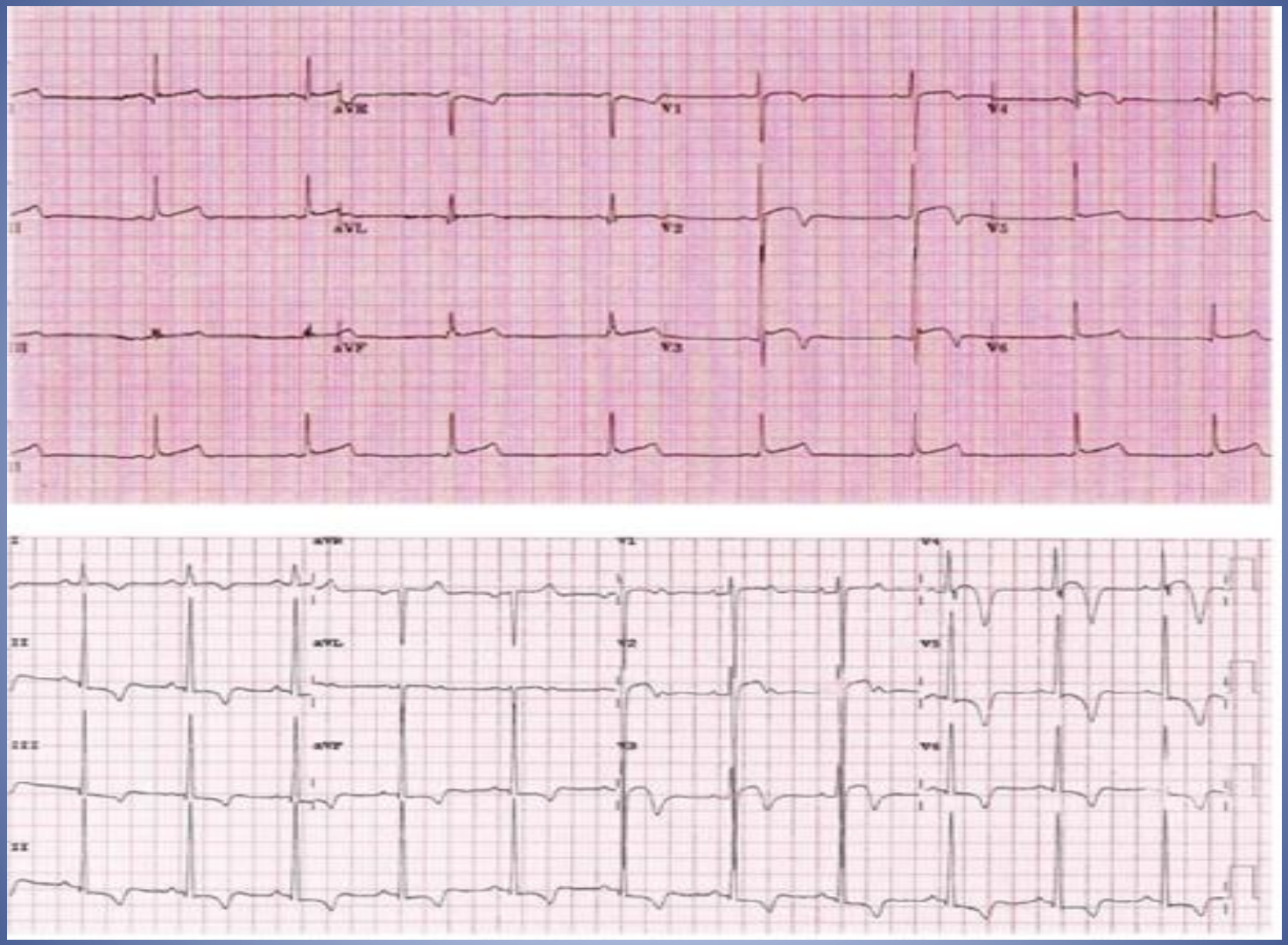
Abnormal ECG finding	Definition
T wave inversion	>1 mm in depth from baseline in two or more adjacent leads not including aVR or V1 ([1]note exception below—figure 1)
ST segment depression	≥1 mm in depth in two or more adjacent leads
Pathological Q waves	>3 mm in depth or >0.04 s in duration in two or more leads
Complete left bundle branch block	QRS >0.12 s, predominantly negative QRS complex in lead V_1 (QS or rS), and upright monophasic R wave in leads I and V_6 (figure 2)
Complete right bundle branch block	QRS >0.12 s, terminal R wave in lead V_1 (rsR′), and wide terminal S wave in leads I and V_6 (figure 3)
Intraventricular conduction delay	Non-specific, QRS >0.12 s
Left atrial enlargement	Prolonged P wave duration of >0.12 s in leads I or II with negative portion of the P wave ≥1 mm in depth and ≥0.04 s in duration in lead V_1
Left axis deviation	−30° to −90°
Right atrial enlargement	High/pointed P wave ≥2.5 mm in leads II and III or V_1
Right ventricular hypertrophy	Right axis deviation ≥120°, tall R wave in V1+persistent precordial S waves ($R\text{-}V_1+S\text{–}V_5$>10.5 mm)
Mobitz type II 2° AV block	Intermittently non-conducted P waves not preceded by PR prolongation and not followed by PR shortening
3° AV block	Complete heart block
Ventricular pre-excitation	PR interval <0.12 s with a delta wave (slurred upstroke in the QRS complex—figure 4)
Long QT interval	QTc ≥0.47 s (99% males) QTc ≥0.48 s (99% females) QTc ≥0.50 s (unequivocal LQTS; figure 5)
Short QT interval	QTc ≤0.34 s
Brugada-like ECG pattern	High take-off and downsloping ST segment elevation in V_1–V_3 (figure 6)
Epsilon wave	Small negative deflection just beyond the QRS in V_1 or V_2 (figure 7)
Profound sinus bradycardia	<30 BPM or sinus pauses ≥3 s
Atrial tachyarrhythmias	Supraventricular tachycardia, atrioventricular nodal reentrant tachycardia, atrial-fibrillation and atrial-flutter
Premature ventricular contractions	≥2 per tracing
Ventricular arrhythmias	Couplets, triplets and non-sustained ventricular tachycardia

Drezner J. Br J Sports Med 2012 46: i6-i8

Case: 25 yo athlete (triathlon)



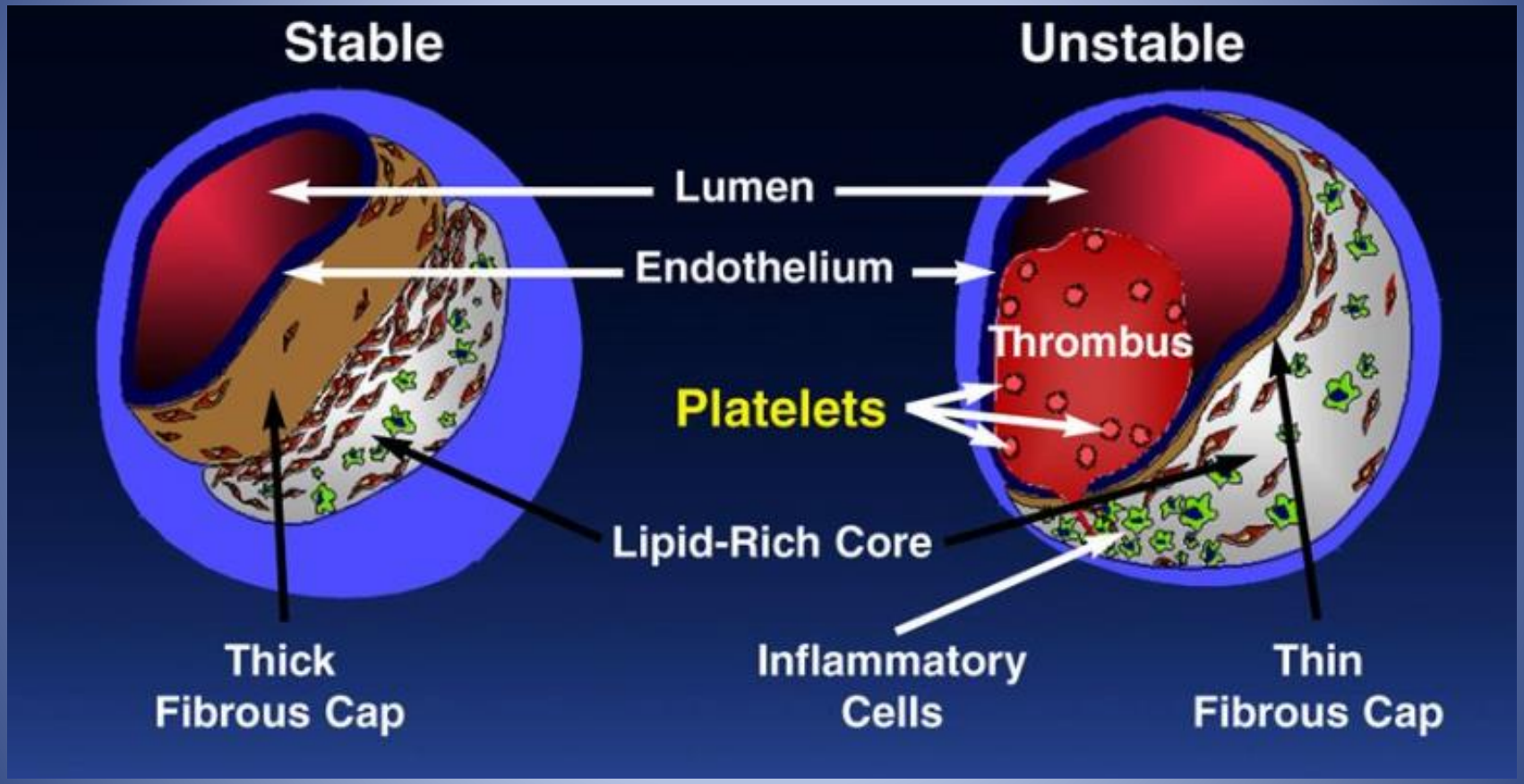
Seattle criteria



Sharma S. European Heart Journal (2015) 36, 1445–1453

Pre-participation screening

Master Athletes - Causes

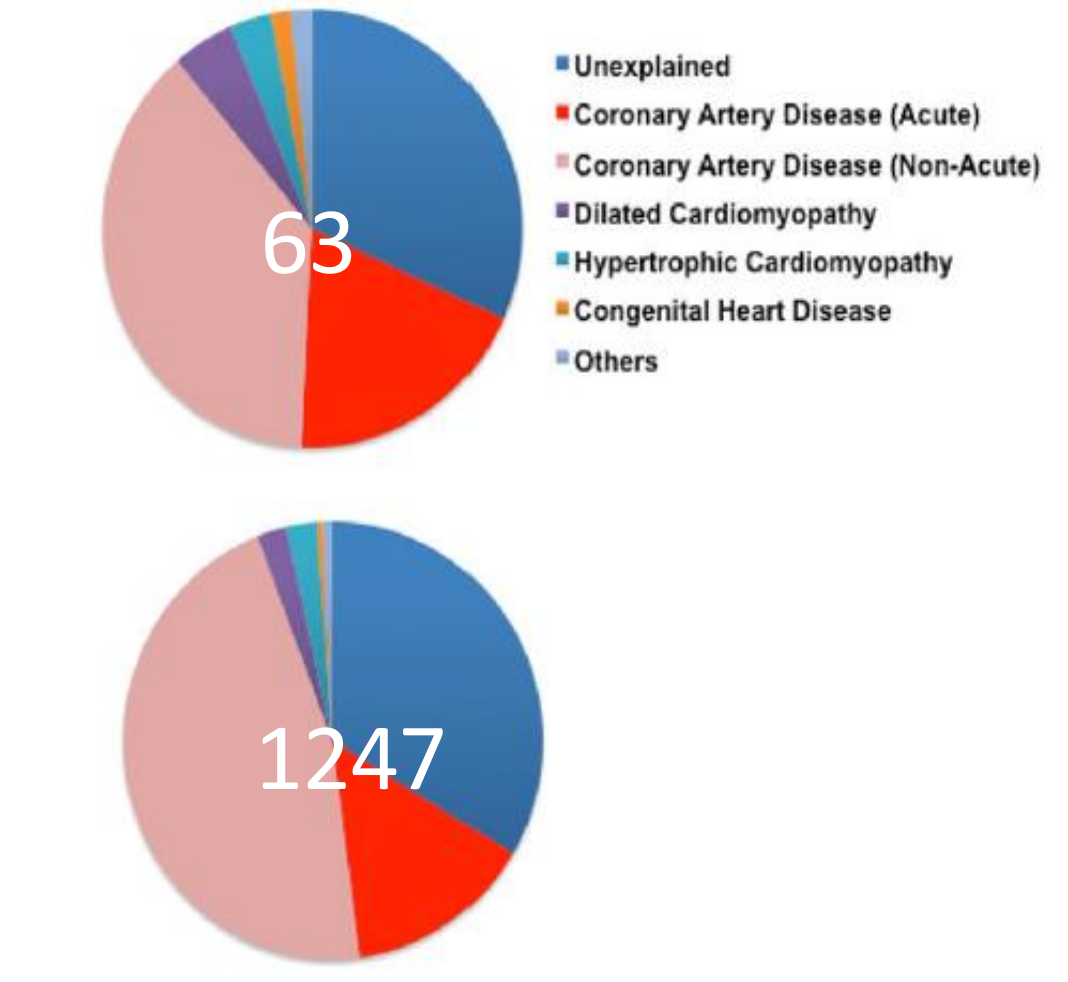


Falk E. Circulation 1995;92:657-671

Master Athletes - Incidence

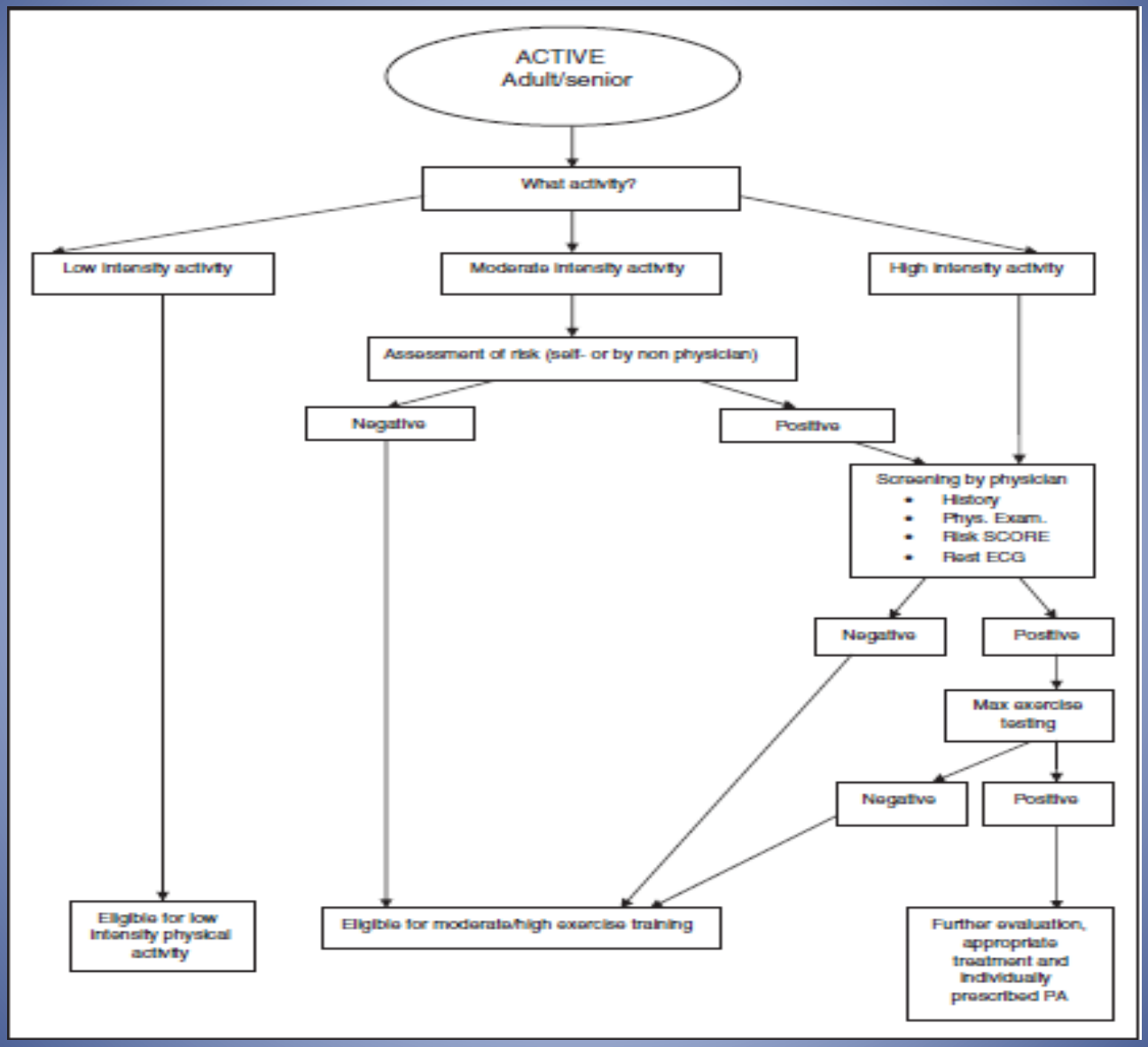
5% of total during sports
(21 per million per year)

- Known pre-existing cardiac disease in 16%
- ≥1 cardiovascular risk factor in 56%
- 36% of cases had typical cardiovascular symptoms during the week preceding SCA



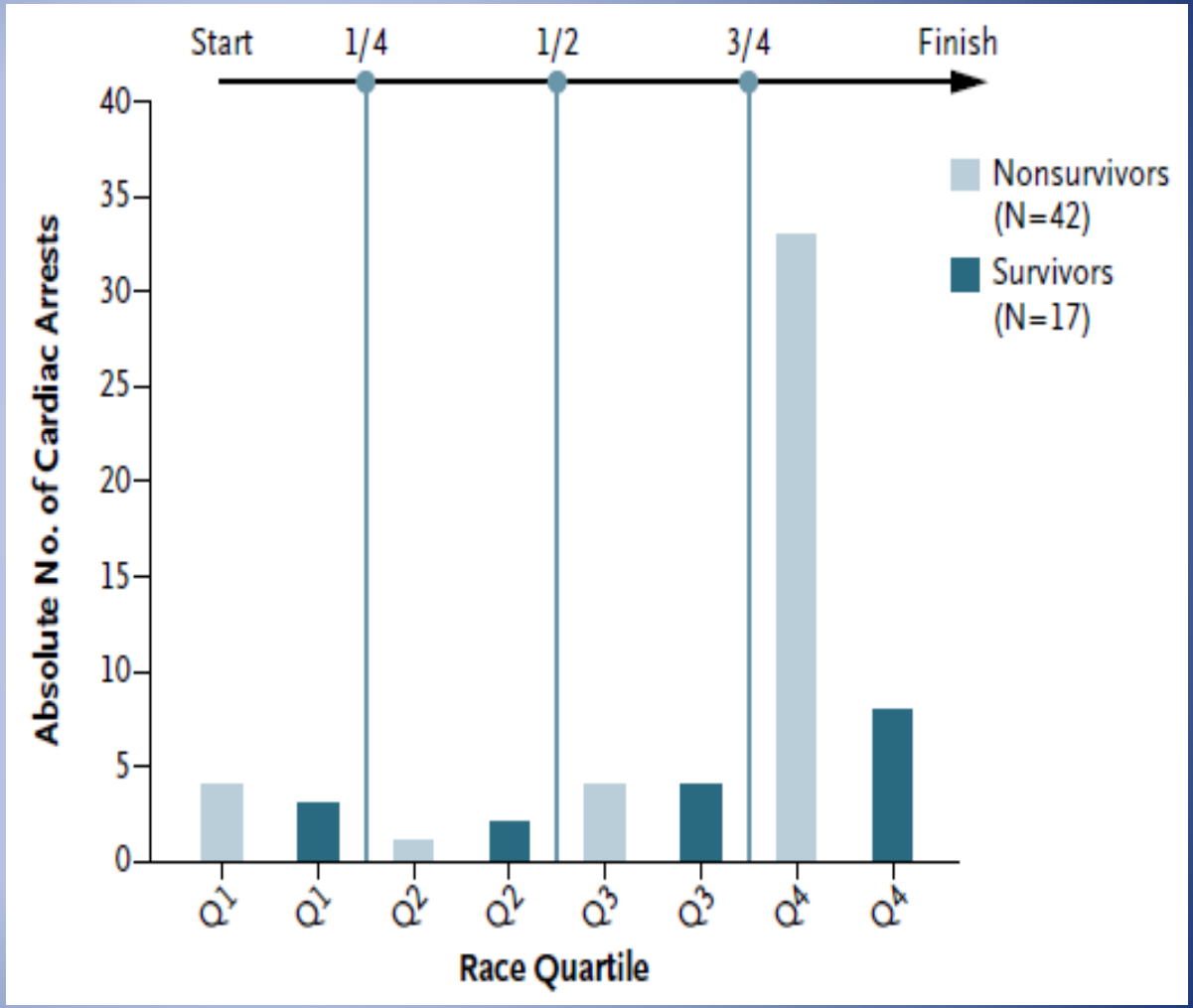
Marijon E et al. Circulation. 2015 April 21; 131(16): 1384–1391

Screening protocol (CAD)



European Journal of Cardiovascular Prevention & Rehabilitation 2011;18(3):446-458

Long distances runners



Kim J et al N Engl J Med 2012;366:130-40

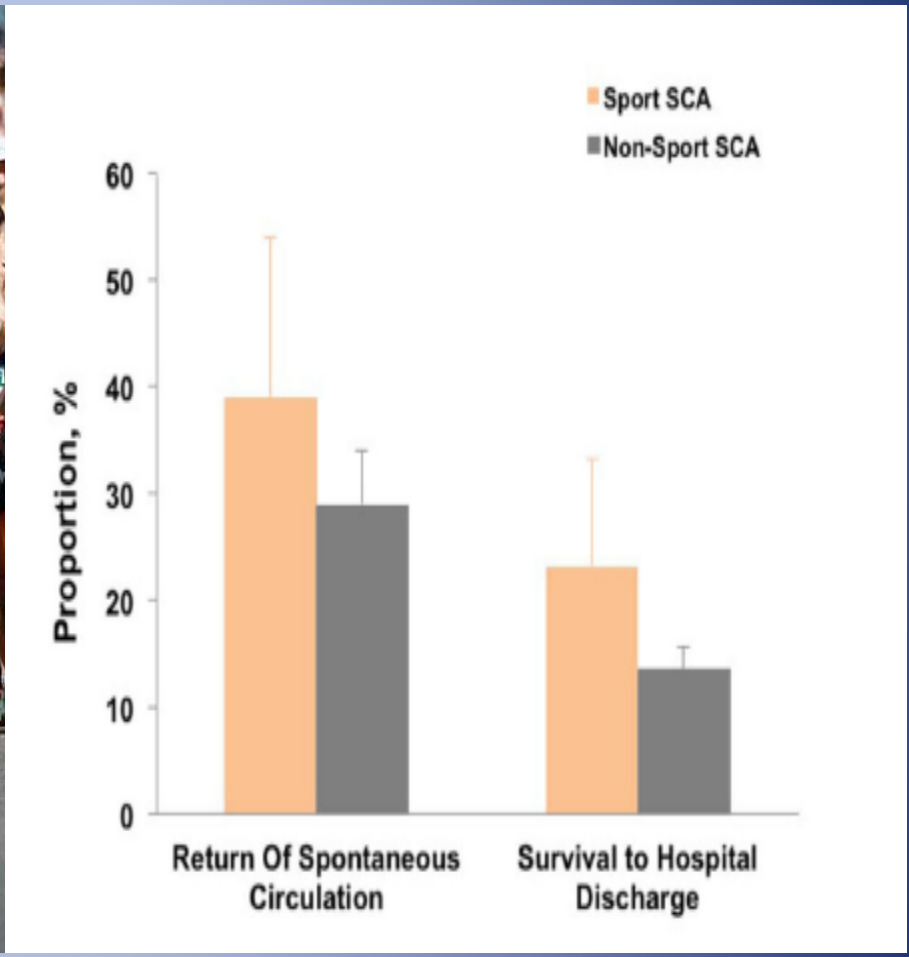
openheart

Prerace aspirin to protect susceptible runners from cardiac arrest during marathons: is opportunity knocking?

Arthur J Siegel[1,2]

- If cardiac arrests during marathons are due to atherosclerotic heart disease in some middle-aged men
- And same-aged asymptomatic marathoners show biomarkers of inflammation during races which predict cardiovascular events in healthy persons
- And aspirin has been conclusively shown to prevent first myocardial infarctions in healthy men
- Pre-race aspirin usage may protect susceptible runners from cardiac arrest.

Better survival rates of exercise SCAs



Marijon E et al. Circulation. 2015 April 21; 131(16): 1384–1391

European Heart Journal (2011) **32**, 2119–2124
doi:10.1093/eurheartj/ehr178

SPECIAL ARTICLE

Consensus document regarding cardiovascular safety at sports arenas

Position stand from the European Association of Cardiovascular Prevention and Rehabilitation (EACPR), section of Sports Cardiology

Table 1 Check list: written medical action plan of sports arenas with >1000 spectators, should include

Name of medical director at arena
Map of the arena with localization of emergency exits and medical care (for opposing teams and spectators)
Arena and event specific planning
Level of care
Personnel
Medical equipment
Communication
Treatment facilities
Transportation resources
Documentation
Collaboration with local emergency medical system and nearest hospital
Continuous quality improvement
External information

Table 2 Recommendations for minimum level of care at sports arenas/events with >1000 spectators/competitors

Arena size	AEDs no	Physicians	Nurses	MTs	Ambulances
<10 000	1–2	1	1	2	0–1
10–50 000	4	2	1–5	2–10	1–2
>50 000	8	2–4	>5	>10	>2

Pre-participation screening

Young Athletes < 35 years old

Master Athletes > 35 years old

Athletes with cardiovascular abnormalities

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http://dx.doi.org/10.1016/j.jacc.2015.09.032

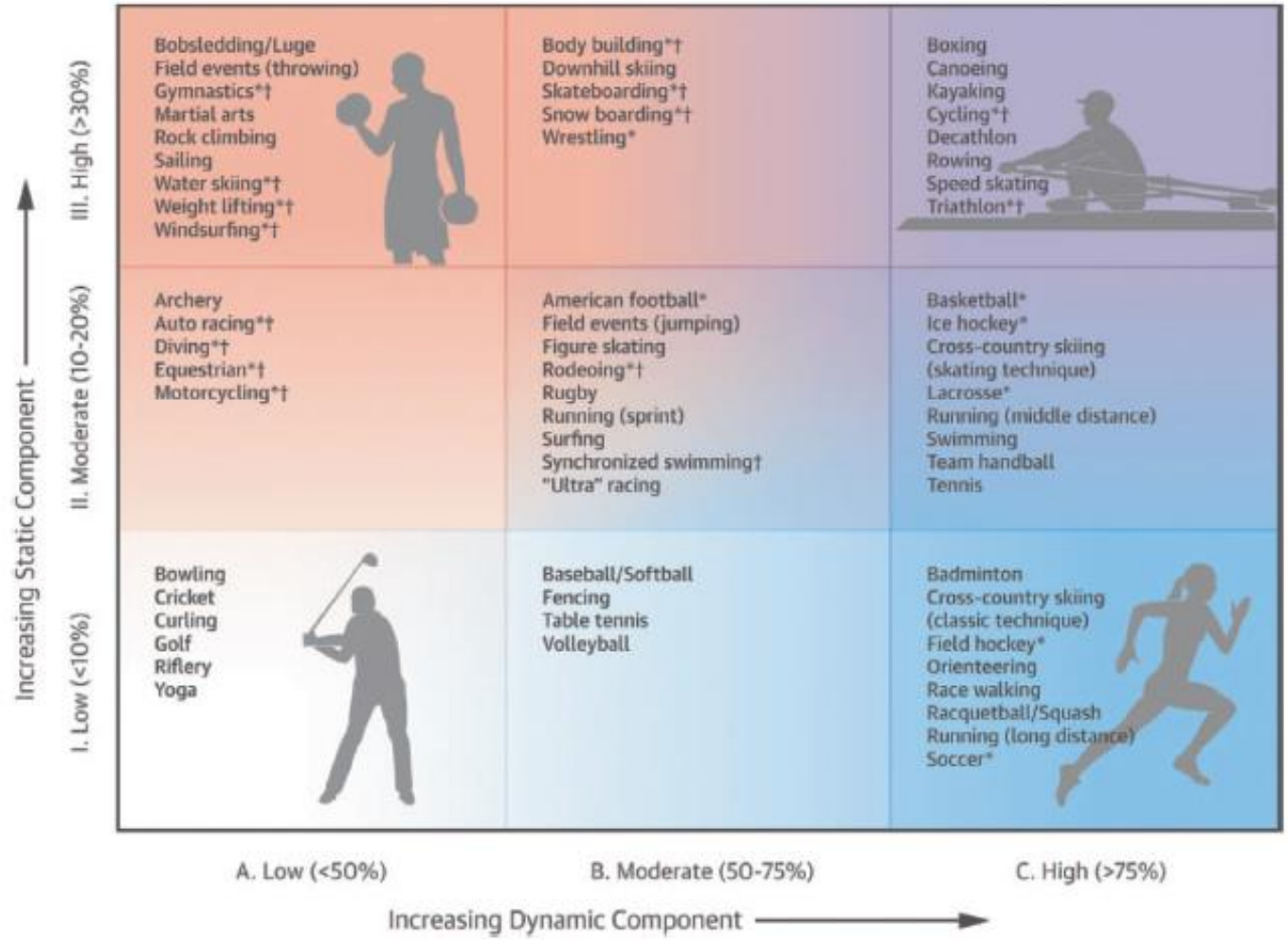
AHA/ACC SCIENTIFIC STATEMENT

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

- Classification
- Pre-participation screening
- Cardiomyopathies (HCM/ARVC)
- Congenital Heart Disease
- Valvular Heart Disease
- Hypertension
- Coronary Artery Disease
- Arrhythmias
- Channelopathies
- Drugs
- Cardiopulmonary Resuscitation
- Commotio Cordis
- Sickle cell trait
- Legal aspects

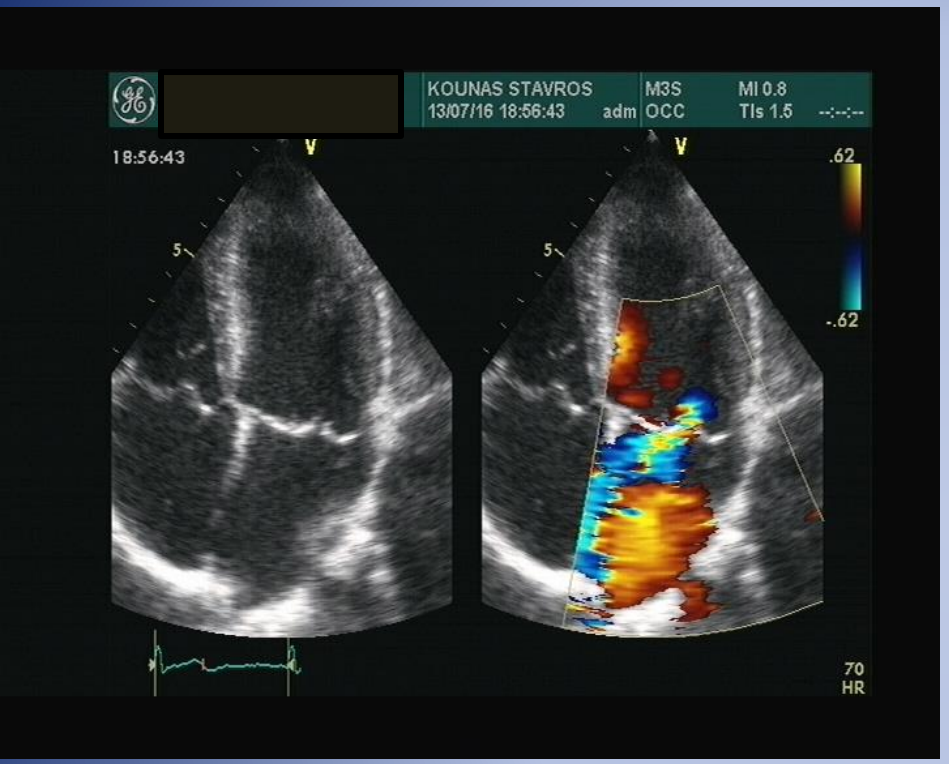
Sports classification

FIGURE Classification of Sports



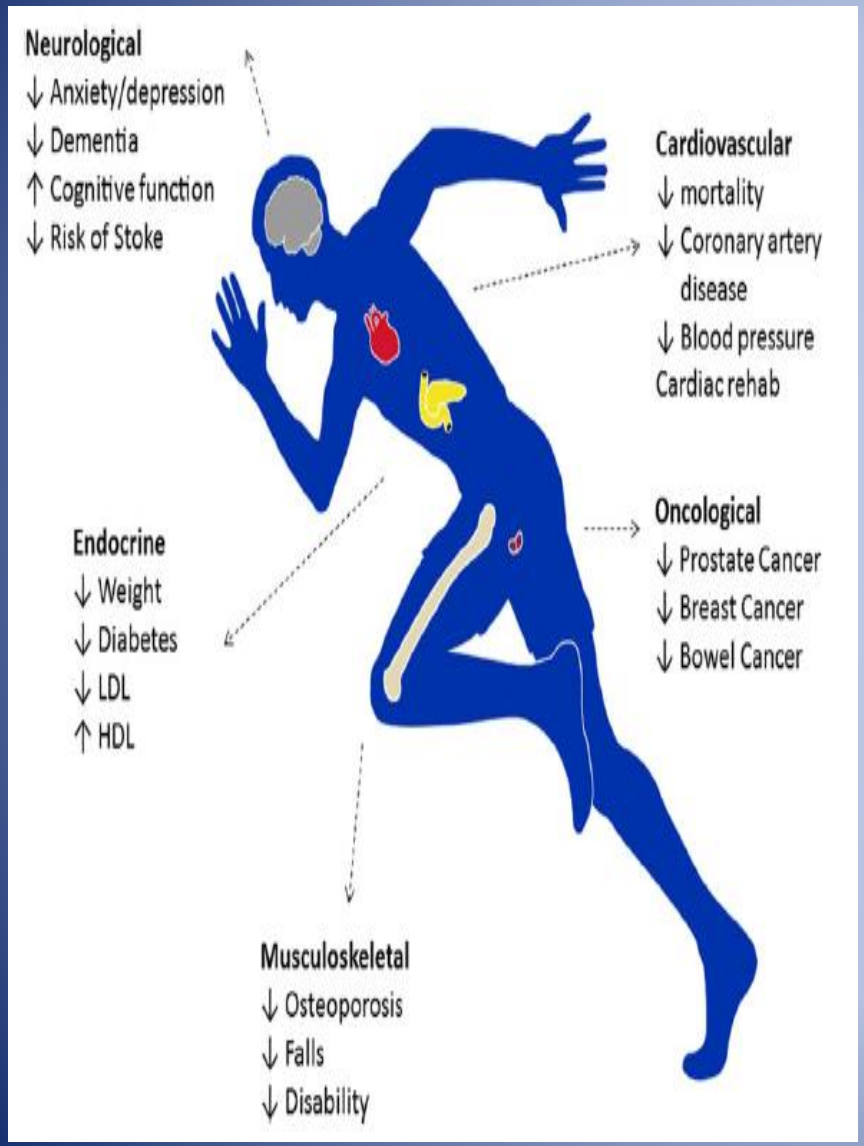
J Am Coll Cardiol. 2015 Dec 1;66(21):2350-2450

Case: 28 yo basketball player



It is reasonable for athletes with moderate MR in sinus rhythm with normal LV systolic function at rest and mild LV enlargement (compatible with that which may result solely from athletic training [LVEDD <60 mm or <35 mm/m2 in men or <40 mm/m2 in women]) to participate in all competitive sports (stage B) (Class IIa; Level of Evidence C).

Take home messages



- Exercise is beneficial!
- Pre-participation screening should attempt to identify athletes with underlying CVD or symptoms
- Screening protocols differ in young and master athletes
- Recommendations for athletes with CVD should be advised before disqualifing

Ευχαριστώ για την προσοχή σας!