Sudden Cardiac Death in Sports: Causes and Current Screening Recommendations

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## Athlete Subgroups

<table>
<thead>
<tr>
<th>Age</th>
<th>Young Athletes ($\leq 35$ yrs)</th>
<th>Older athletes ($&gt;35$ yrs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sports</td>
<td>A variety of sports (ball games)</td>
<td>Jogging and running</td>
</tr>
<tr>
<td>Level</td>
<td>Competitive activity</td>
<td>Leisure sports activity</td>
</tr>
<tr>
<td>Pathology</td>
<td>Large spectrum of cardiac disease (congenital and genetic)</td>
<td>Atherosclerotic coronary artery disease</td>
</tr>
<tr>
<td>Clinical history</td>
<td>Unsuspected heart disease (up to 75%)</td>
<td>Known coronary artery disease (up to 80%)</td>
</tr>
</tbody>
</table>
Relative risk of sport-related SD by cardiovascular and non-cardiovascular diseases in adolescents and young adults

Cardiovascular causes of sudden death associated with sports

Adults (age > 35 years):
- Atherosclerotic coronary artery disease

Young competitive athletes (age ≤35 years):
- Hypertrophic cardiomyopathy
- Arrhythmogenic right ventricular cardiomyopathy
- Congenital anomalies of coronary arteries
- Myocarditis
- Aortic rupture
- Valvular disease
- Preexcitation syndromes and conduction diseases
- Ion channel diseases
- Congenital heart disease, operated or unoperated
Leading causes of sudden cardiovascular death in young competitive athletes

HCM

ARVC/D
Sensitivity of 12-lead ECG in SD victims of HCM

78
SD victims of HCM

53
Prior 12-lead ECG

51/53 (96%)
Positive ECG
(LVH, ST-T changes, q waves)

Sensitivity of 12-lead ECG in sport-related SD victims with ARVC

21 Sport-related SD victims with ARVC

17 Prior 12-lead ECG

15/17 (88%) Positive ECG (negative T-waves beyond V1, QRS widening)

Corrado et al PACE 2002; 25 (abstr) :544
<table>
<thead>
<tr>
<th>Disease</th>
<th>QTc interval</th>
<th>P wave</th>
<th>PR interval</th>
<th>QRS complex</th>
<th>ST interval</th>
<th>T wave</th>
<th>Arrhythmias</th>
</tr>
</thead>
<tbody>
<tr>
<td>HCM</td>
<td>Normal</td>
<td>(left atrial enlargement)</td>
<td>Normal</td>
<td>Increased voltages in mid-left precordial leads; abnormal Q waves in inferior and/or lateral leads; (LAD, LBBB); (delta wave)</td>
<td>Down-sloping (up-sloping)</td>
<td>Inverted in mid-left precordial leads; (giant and negative in the apical variant)</td>
<td>(Atrial fibrillation); (PVB); (VT)</td>
</tr>
<tr>
<td>Arrhythmogenic right ventricular cardiomyopathy/dysplasia</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Prolonged &gt;110 ms in right precordial leads; epsilon wave in right precordial leads; reduced voltages ≤0.5 mV in frontal leads; (RBBB)</td>
<td>(Up-sloping in right precordial leads)</td>
<td>Inverted in right precordial leads</td>
<td>PVB with a LBBB pattern; (VT) with a LBBB pattern</td>
</tr>
<tr>
<td>Dilated cardiomyopathy</td>
<td>Normal</td>
<td>(Left atrial enlargement)</td>
<td>(Prolonged ≥0.21 s)</td>
<td>LBBB</td>
<td>Down-sloping (up-sloping)</td>
<td>Inverted in inferior and/or lateral leads</td>
<td>PVB; (VT)</td>
</tr>
<tr>
<td>Long QT syndrome</td>
<td>Prolonged</td>
<td>(Left atrial enlargement)</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Bifid or biphasic in all leads</td>
<td>(PVB); (torsade de pointes)</td>
</tr>
<tr>
<td>&gt;440 ms in males</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;460 ms in females</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Brugada syndrome</td>
<td>Normal</td>
<td>Prolonged ≥0.21 s</td>
<td>Normal</td>
<td>S1S2S3 pattern; (RBBB/LAD)</td>
<td>Up-sloping coved-type in right precordial leads</td>
<td>Inverted in right precordial leads</td>
<td>(Polymorphic VT); (atrial fibrillation) (sinus bradycardia) (2nd or 3rd degree AV block)</td>
</tr>
<tr>
<td>Lenègre disease</td>
<td>Normal</td>
<td>Normal</td>
<td>Prolonged ≥0.21 s</td>
<td>RBBB; RBBB/LAD; LBBB</td>
<td>Normal</td>
<td>Secondary changes</td>
<td></td>
</tr>
<tr>
<td>Short QT syndrome</td>
<td>Shortened &lt;300 ms</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Secondary changes</td>
<td>Atrial fibrillation (polymorphic VT); Supraventricular tachycardia; (atrial fibrillation)</td>
</tr>
<tr>
<td>Pre-excitation syndrome (WPW)</td>
<td>Normal</td>
<td>Normal</td>
<td>Shortened &lt;0.12 s</td>
<td>Delta wave</td>
<td>Secondary changes</td>
<td>Secondary changes</td>
<td></td>
</tr>
<tr>
<td>Coronary artery diseases</td>
<td>(Prolonged)</td>
<td>Normal</td>
<td>Normal</td>
<td>(Abnormal Q waves)</td>
<td>(Down- or upsloping)</td>
<td>Inverted in ≥2 leads</td>
<td>PVB; (VT)</td>
</tr>
</tbody>
</table>

Less common or uncommon ECG findings are reported in brackets.

QTc: QT interval corrected for heart rate by Bazett's formula. LBBB: left bundle branch block. RBBB: right bundle branch block. LAD: left axis deviation of -30° or more. PVB: either single or coupled premature ventricular beats. VT: either non-sustained or sustained ventricular tachycardia.

Corrado et al. Eur Heart J 2005

*aCoronary artery diseases: either premature coronary atherosclerosis or congenital coronary anomalies.*

*bAbnormal Q waves (see Table 3).*
ESC Report

Cardiovascular pre-participation screening of young competitive athletes for prevention of sudden death: proposal for a common European protocol

Consensus Statement of the Study Group of Sport Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology

Domenico Corrado¹*, Antonio Pelliccia², Hans Halvor Bjørnstad³, Luc Vanhees⁴, Alessandro Biffi², Mats Borjesson⁵, Nicole Panhuyzen-Goedkoop⁶, Asterios Deligiannis⁷, Erik Solberg⁸, Dorian Dugmore⁹, Klaus P. Mellwig¹⁰, Deodato Assanelli¹¹, Pietro Delise¹², Frank van-Buuren¹⁰, Aris Anastasakis¹³, Hein Heidbuchel⁴, Ellen Hoffmann¹⁴, Robert Fagard⁴, Silvia G. Priori¹⁵, Cristina Basso¹⁹, Eloisa Arbustini¹⁶, Carina Blomstrom-Lundqvist¹⁷, William J. McKenna¹⁸, and Gaetano Thiene¹⁹
Young Competitive Athletes (12-35 years)

Family and personal history, physical examination, 12-lead ECG

- negative findings
  - Eligibility for competition
  - no evidence of cardiovascular disease
- positive findings
  - Further examinations
    - Noninvasive: echocardiography, stress test, 24-h Holter, cardiac MR
    - Invasive: angiography, endomyocardial biopsy, electrophysiologic study
  - diagnosis of cardiovascular disease
  - Management according to established protocols
SCREENING FOR HYPERTROPHIC CARDIOMYOPATHY IN YOUNG ATHLETES

DOMENICO CORRADO, M.D., CRISTINA BASSO, M.D., MAURIZIO SCHIAVON, M.D., AND GAETANO THIENE, M.D.

ABSTRACT

Background For more than 20 years in Italy, young athletes have been screened before participating in competitive sports. We assessed whether this strategy results in the prevention of sudden death from hypertrophic cardiomyopathy, a common cardiovascular cause of death in young athletes.

Methods We prospectively studied sudden deaths among athletes and nonathletes (35 years of age or less) in the Veneto region of Italy from 1979 to 1996. The causes of sudden death in both populations were compared, and the pathological findings in the athletes were related to their clinical histories and electrocardiograms. Cardiovascular reasons for disqualification from participation in sports were investigated and follow-up was performed in a consecutive series of 33,735 young athletes who underwent preparticipation screening in Padua, Italy, during the same period.

Results Of 269 sudden deaths in young people, 49 occurred in competitive athletes (44 male and 5 female athletes; mean [±SD] age, 23±7 years). The most common causes of sudden death in athletes were arrhythmogenic right ventricular cardiomyopathy (22.4 percent), coronary atherosclerosis (18.4 percent), and anomalous origin of a coronary artery (12.2 percent). Hypertrophic cardiomyopathy caused only 1 sudden death among the athletes (2.0 percent) but caused 16 sudden deaths in the nonathletes (7.3 percent). Hypertrophic cardiomyopathy was detected in 22 athletes (0.07 percent) at preparticipation screening and accounted for 3.5 percent of the cardiovascular reasons for disqualification. None of the disqualified athletes with hypertrophic cardiomyopathy died during a mean follow-up period of 8.2±5 years.

Conclusions The results show that hypertrophic cardiomyopathy was an uncommon cause of death in these young competitive athletes and suggest that the identification and disqualification of affected athletes at screening before participation in competitive sports may have prevented sudden death. (N Engl J Med 1998;339:364-9.)
Preparticipation Athletic Screening
(Padua: 1979-1996)

- Athletes screened: 33,735
- Athletes disqualified: 1,058 (3%)
- Cardiovascular causes of disqualification: 621 (59%)
- Hypertrophic Cardiomyopathy: 22 (0.07% of 33,735)
<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
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</thead>
<tbody>
<tr>
<td>N.</td>
<td>22</td>
</tr>
<tr>
<td>Age</td>
<td>20±4 yrs</td>
</tr>
<tr>
<td>Sex (% male)</td>
<td>90</td>
</tr>
<tr>
<td>Reason for echo (80%)</td>
<td>ECG changes</td>
</tr>
<tr>
<td>LV wall Thickness</td>
<td>19±3 mm</td>
</tr>
<tr>
<td>LV cavity</td>
<td>43±2 mm</td>
</tr>
<tr>
<td>LVH after detraining</td>
<td>unchanged</td>
</tr>
</tbody>
</table>

Prevalence of HCM in young white people

ECG: **0.07%** (22 of 33,735)
Corrado et al NEJM, 1998

ECHO: **0.10%** (2 of 2,030)
Maron et al Circulation, 1995
Prevalence of Cardiomyopathy in Italian Asymptomatic Children With Electrocardiographic T-Wave Inversion at Preparticipation Screening

Federico Migliore, MD; Alessandro Zorzi, MD; Pierantonio Michieli, MD, PhD; Martina Perazzolo Marra, MD, PhD; Mariachiara Siciliano, MD; Ilaria Rigato, MD, PhD; Barbara Bauce, MD, PhD; Cristina Basso, MD, PhD; Daniela Toazza, MD; Maurizio Schiavon, MD; Sabino Iliceto, MD; Gaetano Thiene, MD; Domenico Corrado, MD, PhD

Background—T-wave inversion on a 12-lead ECG is usually dismissed in young people as normal persistence of the juvenile pattern of repolarization. However, T-wave inversion is a common ECG abnormality of cardiomyopathies such as hypertrophic cardiomyopathy and arrhythmogenic right ventricular cardiomyopathy, which are leading causes of sudden cardiac death in athletes. We prospectively assessed the prevalence, age relation, and underlying cardiomyopathy of T-wave inversion in children undergoing preparticipation screening.

Methods and Results—The study population included 2765 consecutive Italian children (1914 male participants; mean age, 13.9±2.2 years; range 8–18 years) undergoing preparticipation screening including an ECG. Of 229 children (8%) who underwent further evaluation because of positive findings at initial preparticipation screening, 33 (1.2%) were diagnosed with cardiovascular disease. T-wave inversion was recorded in 158 children (5.7%) and was localized in the right precordial leads in 131 (4.7%). The prevalence of right precordial T-wave inversion decreased significantly with increasing age (8.4% in children <14 years of age versus 1.7% in those ≥14 years; P<0.001), pubertal development (9.5% of children with incomplete versus 1.6% with complete development; P<0.001), and body mass index below the 10th percentile (P<0.001). Incomplete pubertal development was the only independent predictor for right precordial T-wave inversion (odds ratio, 3.6; 95% confidence interval, 1.9–6.8; P<0.001). Of 158 children with T-wave inversion, 4 (2.5%) had a diagnosis of cardiomyopathy, including arrhythmogenic right ventricular cardiomyopathy (n=3) and hypertrophic cardiomyopathy (n=1).

Conclusions—The prevalence of T-wave inversion decreases significantly after puberty. Echocardiographic investigation of children with postpubertal persistence of T-wave inversion at preparticipation screening is warranted because it may lead to presymptomatic diagnosis of a cardiomyopathy that could lead to sudden cardiac death during sports. (Circulation. 2012;125:529-538.)

Key Words: arrhythmogenic right ventricular dysplasia ■ athletes ■ cardiomyopathy, hypertrophic ■ child ■ electrocardiography
Children
N=2765

T-wave inversion in ≥2 leads
N=158 (5.7%)

No T-wave inversion
N=2607 (94.3%)

T-wave inversion in inferior leads (LII/aVF)
N=24 (0.9%)
- Echocardiography
  - No cardiomyopathies

T-wave inversion in right precordial leads (V1-V3)
N=131 (4.7%)
- Echocardiography
  - “definitive” ARVC (N=1) *
  - “borderline” ARVC (N=2) †

T-wave inversion in lateral leads (V4-V6 and/or LI-aVL)
N=3 (0.1%)
- Echocardiography
  - HCM (N=1) ‡
ECG and echocardiographic findings in a 14-year-old male
ECG and echocardiographic findings in a 15-year-old male
Trends in Sudden Cardiovascular Death in Young Competitive Athletes After Implementation of a Preparticipation Screening Program

Domenico Corrado, MD, PhD
Cristina Basso, MD, PhD
Andrea Pavei, MD
Pierantonio Michieli, MD, PhD
Maurizio Schiavon, MD
Gaetano Thiene, MD

Context A nationwide systematic preparticipation athletic screening was introduced in Italy in 1982. The impact of such a program on prevention of sudden cardiovascular death in the athlete remains to be determined.

Objective To analyze trends in incidence rates and cardiovascular causes of sudden death in young competitive athletes in relation to preparticipation screening.

Annual Incidence Rates of Sudden Cardiovascular Death in Screened Competitive Athletes and Unscreened Nonathletes Aged 12 to 35 Years in the Veneto Region of Italy (1979-2004)

*Corrado et al JAMA 2006;296:1593-1601*
Mortality trend for sudden death from Cardiomyopathies

RR=0.10

Corrado et al. JAMA 2006;296:1593-1601
Cardiovascular conditions causing disqualification from competitive sports in 879 athletes over 2 consecutive screening periods (1982-1992 and 1993-2004) at the Center for Sports Medicine in Padua, Italy

<table>
<thead>
<tr>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td>Rhythm and conduction abnormalities</td>
<td>345 (39)</td>
<td>166 (36)</td>
<td>179 (42.2)</td>
<td>0.13</td>
</tr>
<tr>
<td>- ventricular arrhythmias</td>
<td>173 (19.6)</td>
<td>81 (18)</td>
<td>92 (21.6)</td>
<td>0.20</td>
</tr>
<tr>
<td>- supraventricular arrhythmias</td>
<td>73  (8.3)</td>
<td>39 (8.6)</td>
<td>34  (8.0)</td>
<td>0.56</td>
</tr>
<tr>
<td>- WPW Syndrome</td>
<td>55   (6.3)</td>
<td>29 (6.3)</td>
<td>26  (6.1)</td>
<td>0.88</td>
</tr>
<tr>
<td>- LBBB or RBBB &amp; LAD</td>
<td>26   (3.0)</td>
<td>8  (1.7)</td>
<td>18  (4.2)</td>
<td>0.10</td>
</tr>
<tr>
<td>- second Degree AV Block</td>
<td>13   (1.5)</td>
<td>7 (1.5)</td>
<td>6  (1.4)</td>
<td>0.89</td>
</tr>
<tr>
<td>- long QT Syndrome</td>
<td>5     (0.6)</td>
<td>2 (0.4)</td>
<td>3  (0.7)</td>
<td>0.93</td>
</tr>
<tr>
<td>Systemic hypertension</td>
<td>205  (23)</td>
<td>118 (25.9)</td>
<td>87  (20.5)</td>
<td>0.96</td>
</tr>
<tr>
<td>Valvular disease (including MVP):</td>
<td>184  (21)</td>
<td>106 (23.3)</td>
<td>78  (18.4)</td>
<td>0.09</td>
</tr>
<tr>
<td>Cardiomyopathies</td>
<td>60   (6.8)</td>
<td>20 (4.4)</td>
<td>40  (9.4)</td>
<td>0.005</td>
</tr>
<tr>
<td>- hypertrophic</td>
<td>30   (3.4)</td>
<td>14 (3.0)</td>
<td>16  (3.8)</td>
<td>0.50</td>
</tr>
<tr>
<td>- arrhythmogenic right ventricular</td>
<td>16   (1.8)</td>
<td>2 (0.4)</td>
<td>14  (3.3)</td>
<td>0.004</td>
</tr>
<tr>
<td>- dilated</td>
<td>14   (1.6)</td>
<td>4 (0.9)</td>
<td>10  (2.4)</td>
<td>0.21</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>11   (1.3)</td>
<td>2 (0.4)</td>
<td>9  (2.1)</td>
<td>0.05</td>
</tr>
<tr>
<td>Other</td>
<td>74   (8.4)</td>
<td>43 (9.5)</td>
<td>31  (7.3)</td>
<td>0.42</td>
</tr>
</tbody>
</table>
Case report

- 23 years-old male competitive athlete (soccer)
- Preparticipation cardiovascular screening:
  - Asymptomatic
  - Unremarkable family history
  - Normal physical examination
- Normal laboratory exams
- ECG
12-lead ECG
Contrast-enhanced Cardiac MR

BL: 23 years-old man
24-hour Holter monitoring
(including training)
6 months later...
Screening of young athletes for Cardiovascular diseases
(Center for Sports Medicine, Padua 1979-2004)

Athletes screened
42,386

Positive findings
3,914 (9%)

Heart diseases
879 (2%)

Potentially lethal heart diseases
91 (0.2%)

False positive ≈ 7%

Corrado et al JAMA 2006; 296: 1593-1601
Recommendations for interpretation of 12-lead electrocardiogram in the athlete

Domenico Corrado¹*, Antonio Pelliccia², Hein Heidbuchel³, Sanjay Sharma⁴, Mark Link⁵, Cristina Basso⁶, Alessandro Bifﬁ², Gianfranco Buja¹, Pietro Delise⁷, Ihor Gussac⁸, Aris Anastasakis⁹, Mats Borjesson¹⁰, Hans Halvor Bjørnstad¹¹, François Carrè¹², Asterios Deligiannis¹³, Dorian Dugmore¹⁴, Robert Fagard³, Jan Hoogsteen¹⁵, Klaus P. Mellwig¹⁶, Nicole Panhuyzen-Goedkoop¹⁷, Erik Solberg¹⁸, Luc Vanhees³, Jonathan Drezner¹⁹, N.A. Mark Estes, III⁵, Sabino Iliceto¹, Barry J. Maron²⁰, Roberto Peidro²¹, Peter J. Schwartz²², Ricardo Stein²³, Gaetano Thiene⁶, Paolo Zeppilli²⁴, and William J. McKenna²⁵ On behalf of the Sections of Sports Cardiology of the European Association of Cardiovascular Prevention and Rehabilitation; and the Working Group of Myocardial and Pericardial Disease of the European Society of Cardiology
Appropriate interpretation of the athlete's electrocardiogram saves lives as well as money

ECG abnormalities in the athlete

(Group 1)
Common (up to 80%)
- Sinus bradycardia;
- First degree AV block;
- Notched QRS in V1 or incomplete RBBB;
- Early repolarization;
- Isolated QRS voltage criteria for left ventricular hypertrophy

(Group 2)
Uncommon (<5%)
- T-wave inversion;
- ST-segment depression;
- Pathological Q waves;
- Left atrial enlargement;
- Left axis deviation/left Anterior hemiblock;
- Right axis deviation/left posterior hemiblock;
- Right ventricular hypertrophy;
- Complete LBBB or RBBB;
- Long or short QT interval;
- Brugada-like early repolarization;
- Ventricular arrhythmias

National health system

- In Italy screening is made feasible because of its limited costs in the setting of a mass-program.
- National health system developed in terms of health care and prevention services
- Infrastructure
- Sports physicians
# How much is an athlete’s life?

<table>
<thead>
<tr>
<th>Description</th>
<th>Cost</th>
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</thead>
<tbody>
<tr>
<td>Athletes screened</td>
<td>1,000,000</td>
</tr>
<tr>
<td>Estimate cost to initially screen all athletes (30 €)</td>
<td>€ 30,000,000</td>
</tr>
<tr>
<td>Estimate cost to evaluate ~100,000 athletes with positive findings (60 €)</td>
<td>€ 6,000,000</td>
</tr>
<tr>
<td>Total cost to of screening</td>
<td>€ 36,000,000</td>
</tr>
<tr>
<td>N° of SDs in unscreened athletes (mortality 4/100000 athlete-years)</td>
<td>40</td>
</tr>
<tr>
<td>N° of SDs in screened athletes (mortality 0.4/100000 athlete-years)</td>
<td>4</td>
</tr>
<tr>
<td>Lives saved</td>
<td>36</td>
</tr>
<tr>
<td>Cost for a life saved</td>
<td>€ 1,000,000</td>
</tr>
<tr>
<td>Cost for one year of life saved (YLS): 10 additional years of life</td>
<td>€ 100,000/YLS</td>
</tr>
<tr>
<td>Cost for one year of life saved: 20 additional years of life</td>
<td>€ 50,000/YLS</td>
</tr>
<tr>
<td>Cost for one year of life saved: 30 additional years of life</td>
<td>€ 33,000/YLS</td>
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# Athlete Subgroups

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Exercise-related sudden death in middle aged/senior leisure time athletes

- Habitual sport activity in middle aged and older population offers protection over the long-term from the overall risk of myocardial infarction and sudden death.

- Physical exercise may acutely trigger sudden cardiac arrest mostly in persons who did not exercise regularly.

*Siscovick DS. NEJM 1984;311:874*
## Incidence of SCD during Marathon

<table>
<thead>
<tr>
<th>Author</th>
<th>Events</th>
<th>Study population</th>
<th>Study period</th>
<th>SCD rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>BJ Maron (JACC, 1998)</td>
<td>Marine Corps (WA) Twin Cities (MN) Marathons</td>
<td>215,413 Marathon runners</td>
<td>30 years (1976-1994)</td>
<td>0.5/100 000</td>
</tr>
<tr>
<td>DA Redelmeier (BMJ 2007)</td>
<td>26 (USA) Marathons</td>
<td>3,292,268 Marathon runners</td>
<td>30 years (1975-2004)</td>
<td>0.8/100 000</td>
</tr>
<tr>
<td>Kim JH (NEJM 2012)</td>
<td>USA Marathon &amp; half-Marathon</td>
<td>10,900,000 Marathon runners</td>
<td>10 years (2000-2010)</td>
<td>0.5/100 000</td>
</tr>
</tbody>
</table>
Profile of middle-aged victims of SCD during sports

- adult man
- Asymptomatic
- No prior documentation of heart disease
- Cardiac arrest due to FV
- Post-mortem: atherosclerotic plaque lesions obstructing ≥one epicardial coronary vessel(s) in 71% to 87%
Sudden death of a 47-year old marathon runner

Obstructive atherosclerotic coronary artery disease of both left (anterior descending branch) and right coronary arteries (A,B)

C) Histology of the myocardium shows replacement type fibrosis due to previous myocardial infarction.
Pre-participation basal 12-lead ECG has been proven to be life-saving in young competitive athletes (<35 years) in whom SCD is mostly caused by ECG detectable genetic cardiomyopathies.
Pre-participation basal 12-lead ECG appears to be a non-accurate test for screening coronary artery disease in older athletes engaged in leisure-time sports such as Marathon running.
Exercise test screening

Because of its established prognostic value, widespread availability and low cost, exercise testing is widely deemed the best available test for screening asymptomatic adults prior to an exercise programme.

Exercise testing has a low predictive value for CAD in the general asymptomatic population, but in a patient population with risk factor for CAD it may identify patients with markedly increased risk for coronary events.
Cardiovascular evaluation of middle-aged/senior individuals engaged in leisure-time sport activities: position stand from the sections of exercise physiology and sports cardiology of the European Association of Cardiovascular Prevention and Rehabilitation

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SEDENTARY

Low intensity activity

Self-assessment of risk

NO on every question

Screening by physician: Hx, Phys.exam., RISK SCORE, ECG

YES on every question

High intensity activity

Maximal exercise testing

Low risk

Positive

Further evaluation, Appropriate treatment and individually prescribed physical activity

Eligible

Negative
ACTIVE
Adult/senior

Low intensity activity  Moderate intensity activity  High intensity activity

Self-assessment of risk

NO on every question  YES on every question

Screening by physician:
Hx, Phys.exam., RISK SCORE, ECG

Low risk  High risk

Maximal exercise testing

Eligible

Negative  Positive

Further evaluation,
Appropriate treatment and individually prescribed physical activity

Consensus document of EACPR
Conclusions (1)

The available evidence indicates that ECG screening has to be considered an efficient health strategy for prevention of SCD of young competitive athletes.

At risk-cardiovascular diseases are accurately identified by ECG screening at a pre-symptomatic phase.

Early identification and management of asymptomatic athletes favourably modify the outcome of the underlying diseases leading to substantial prevention of in-the-field mortality.
Conclusions (2)

Identification of middle-aged/senior athletes with coronary artery disease at risk of SCD is difficult and the utility of preparticipation screening by means of resting and exercise ECG testing remains unproven.

Cardiovascular medical evaluation is recommended by most Associations of Cardiology and Sports Medicine as a prudent measure before entering leisure-time sport activity in the light of our current understanding of the cardiovascular risks and benefits of exercise in this age group.