Pre-competition screening in competitive athletes and leisure sportsmen
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time course:

1. The paradox of sports – is sport healthy?

2. The prevention of sports-associated sudden cardiac death (SCD)

3. Latest news, outlook and discussion
suggested literature:

Pelliccia et al., Recommendations for competitive sports in athletes with cardiovascular disease; EHJ May 2005
Maron et al., 36th Bethesda Conference: Eligibility Recommendations for Competitive Athletes With Cardiovascular Abnormalities. JACC Vol. 45, No. 8, 2005
Corrado et al.: Recommendations for Interpretation of 12-lead Electrocardiogram in the Athlete, EHJ 2010;31(2):243-59
Marcus FI, McKenna WJ; Diagnosis of arrhythmogenic right ventricular cardiomyopathy/dysplasia: proposed modification of the Task Force Criteria. Eur Heart J. 2010 Apr;31(7):806-14.
the less you are trained - the more you benefit!
myocardial infarctions overall
myocardial infarctions survived
myocardial infarctions lethal
per 100'000 „patient years“

- Physically active people are protected, but…

- sport is also a trigger for SCD!
  (Mittleman et al. 1993: SCD increased within 26 hours)
Sports-associated sudden cardiac death (SCD)

physiology, causes and prevention
• >90% of sports related sudden deaths are due to cardiovascular events

• incidence of SCD: 0.5 to 2.3:100,000 per year in competitive athletes (under the age of 35)

• in more than 80-90% there are underlying cardiac conditions that could have been detected

• e.g. athletes with hypertrophic cardiomyopathy show specific hallmarks in 12-lead resting-ECG in >90%
"Trigger"

cardiac substrate/underlying disorder

sport/physical exercise

structural disorder conduction disorder collagenoses, etc.
Causes of sudden cardiac death:

- > 35 years: mostly coronary heart disease (>80%)
Causes of sudden cardiac death:

- **> 35 years:** mostly coronary heart disease (>80%)

- **< 35 years:**
  - hereditary cardiomyopathies
  - coronary anomalies
  - arrhythmias, conduction anomalies
  - myocarditis
  - commotio cordis
  - Marfan syndrome/ruptured aortic aneurysma
  - etc.
Causes of SD in 387 Young Athletes (Maron BJ. NEJM 2003; 349: 1064–1075)
The exception proves the roule…

…Commotio Cordis
Commotio cordis:

Caused by a blunt, non-penetrating blow over the pre-cordial region, triggering ventricular fibrillation.

*Children and adolescents are specifically prone.*
“Trigger”

Cardiac substrate/underlying disorder

Sport/physical exercise

Structural disorder
Conduction disorder
Collagenoses, etc.
«The will to win is important, but the will to prepare is vital.»

Joe Paterno (Football Coach)
Current concepts in cardiac pre-competition screening/medical assessment (PCMA)
3 initial questions:

THE ATHLETE WITH A „NORMAL HEART“

COMPETITIVE OR NON-COMPETITIVE?

AGE > 35 OR < 35?

THE ATHLETE WITH AN „ABNORMAL HEART“
What are the current guidelines in cardiac screening?

THE ATHLETE WITH A „NORMAL HEART“

<table>
<thead>
<tr>
<th>&lt;35</th>
<th>history</th>
<th>clinical exam</th>
<th>12-lead-resting-ECG</th>
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</table>

| >35 | history | additional stress-test…? | ECG |

| „competitive“ | ? |

| „non-competitive“ | additional stress-test…? |
**competitive sports:**
(Definition Circulation 2004;109:2807-2816.),
AHA Scientific Statement

A competitive athlete is one who participates in an organized team or individual sport that requires systematic training and regular competition against others and that places a high premium on athletic excellence and achievement.
What are the current guidelines in cardiac screening?

THE ATHLETE WITH AN „ABNORMAL HEART“

<table>
<thead>
<tr>
<th>„competitive“</th>
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<tr>
<td>„ESC-/Bethesta-Guidelines“</td>
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### Table of Competitive Sports Participation Recommendations

<table>
<thead>
<tr>
<th>Increasing Static Component</th>
<th>Increasing Dynamic Component</th>
</tr>
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<tbody>
<tr>
<td><strong>Low</strong> (&lt;40% Max O₂)</td>
<td><strong>A. Low</strong> (&lt;40% Max O₂)</td>
</tr>
<tr>
<td><strong>Moderate</strong> (40-70% Max O₂)</td>
<td><strong>B. Moderate</strong> (40-70% Max O₂)</td>
</tr>
<tr>
<td><strong>High</strong> (&gt;70% Max O₂)</td>
<td><strong>C. High</strong> (&gt;70% Max O₂)</td>
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#### Low (≤40% Max O₂)
- **AVS** (Early)
- History, PE, ECG, ET, Echo
- Severe stenosis
  - Mild-to-moderate regurgitation,
  - Normal LV size and function,
  - Normal exercise testing,
  - No significant rhythmia
- Mild-to-moderate regurgitation,
  - Proof of progressive LV dilatation

#### Moderate (40-70% Max O₂)
- **AVS** (Early)
- No competitive sports

#### High (>70% Max O₂)
- **MVS** (Late)
- All sports

#### Follow-up
- **MVS** (Late)
- Yearly

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**Recommendations for competitive sports participation in athletes with cardiovascular disease**, EHJ (2005) 26, 1422-1445
What are the current guidelines in cardiac screening?

### The Athlete with a "Normal Heart"

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Cardiac screening – “European Style”

starting at the age of 12-14, repeated every 1-2 years

Cardiac screening – "European Style"

Core Components:

- Medical History
- Physical examination
- 12-lead resting ECG

In case of positive findings: Further tests
Cardiac screening – “European Style”

Modern „Pre-Competition Medical Screening“

- time frame: about 15 minutes
- costs: about 30-50 Euro

> 80-90% of exercise-related SCD could be prevented!!
Cardiac screening – “European Style”

Core Components:
Medical History

Physical examination
12-lead resting ECG

In case of positive findings
Further tests
Medical History

• The International Olympic Committee (IOC) implemented a specified questionnaire ("Lausanne recommendations").

• integrates personal and family history (most common causes of SCD in sports are inherited)

• simple, underestimated but very effective tool!

• ask accurately, systematically and specifically (e.g. involve parents in children and adolescents)
Cardiac screening – “European Style”

Core Components:

- Medical History
- 12-lead resting ECG

Physical examination

In case of positive findings

Further tests
Physical examination

Pitfalls and Hallmarks:

- blood pressure (bilateral) and heart rate after 5 minutes rest

- meticulous cardiac auscultation
differentiation of „physiologic“ and „suspicious“ murmurs and sounds (>60% of athletes have a „murmur“!)
Physical examination

Ghent Criteria in Marfan Syndrome
Cardiac screening – “European Style”

Core Components:
- Medical History
- Physical examination
- 12-lead resting ECG

In case of positive findings

Further tests
12-lead resting ECG

Pitfalls and Hallmarks:

Correct performance:
- lead positions
- after 5-minutes rest
- modulations of measurement conditions (e.g. amplitude and sweep)

Correct analysis

nota bene: high rate of „false-positive“ findings!
Interpretation of 12-lead resting ECG

Table 3 Criteria for a positive 12-lead ECG

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Details</th>
</tr>
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<tr>
<td>P wave</td>
<td>left atrial enlargement: negative portion of the P wave in lead V1 ≥ 0.1 mV in depth and ≥0.04 s in duration; right atrial enlargement: peaked P wave in leads II and III or V1 ≥ 0.25 mV in amplitude.</td>
</tr>
<tr>
<td>QRS complex</td>
<td>frontal plane axis deviation: right ≥ +120° or left -30° to -90°;</td>
</tr>
</tbody>
</table>

Update:
Corrado et al.: Recommendations for Interpretation of 12-lead Electrocardiogram in the Athlete, EHJ 2010;31(2):243-59

ST-segment, T-waves, and QT interval
- ST-segment depression or T-wave flattening or inversion in two or more leads;
- Prolongation of heart rate corrected QT interval >0.44 s in males and > 0.46 s in females.

Rhythm and conduction abnormalities
- Premature ventricular beats or more severe ventricular arrhythmias;
- Supraventricular tachycardias, atrial flutter, or atrial fibrillation;
- Short PR interval (<0.12 s) with or without ’delta’ wave;
- Sinus bradycardia with resting heart rate ≤40 beats/min\(^a\);
- First (PR ≥ 0.21 s\(^b\)), second or third degree atrioventricular block.

\(^a\)Increasing less than 100 beats/min during limited exercise test.
\(^b\)Not shortening with hyperventilation or limited exercise test.

Modified from Corrado et al.\(^3\)
Interpretation of 12-lead resting ECG

COMMON TRAINING RELATED
- Sinus bradycardia
- 1° AV block
- incomplete RBBB
- early repolarisation
- isolated high voltage

COMMON TRAINING UN-RELATED
- T-wave inversion
- ST-segment segment depression
- pathological Q-waves
- left atrial enlargement
- left or right axis deviation
- RV hypertrophy
- ventricular pre-excitation
- complete LBBB/RBBB
- long or short QT interval
- Brugada like pattern
Causes of Sudden Death in 387 Young Athletes (Maron BJ. Sudden death in young athletes. New England Journal of Medicine 2003; 349: 1064–1075)
Causes of Sudden Death in Athletes and Non-Athletes 35 years of age or less in the Veneto region of Italy 1979 to 1996 (Corrado, et al., New England Journal of Medicine, Volume 339:364-369):

- More black athletes in the USA
  - (genetic reasons)

Main reason: ECG

(HCM detectable in > 90%!)
Cardiac screening – “European Style”

Core Components:

- Medical History
- Physical examination
- 12-lead resting ECG

In case of positive findings → Further tests
Echocardiography

- principal diagnostic tool for many cardiac pathologies (e.g. hypertrophic cardiopathy, HCM)

- Some (important) pathologies are not visible (e.g. conduction anomalies)
- Some are difficult to see (e.g. ARVC)
- Structural changes however are difficult to detect (e.g. ARVC, sometimes congenital coronary anomalies)

Conclusion:

Echocardiography is not recommended as a primary tool for screening of young athletes in current guidelines. (cost-effectiveness, borderline findings)

- In trained athletes echocardiography carries the potential for false-positive results, especially concerning borderline values for left ventricular wall thickness.
Imaging in cardiac screening

**CT scan:**
- coronary anomalies
- (premature) coronary heart disease

**MRI:**
- myocarditis
- new diagnostic criterias for ARVC
CT scan: 

Source: university Hospital Zurich
<table>
<thead>
<tr>
<th>Original task force criteria</th>
<th>Revised task force criteria</th>
</tr>
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<tbody>
<tr>
<td>I. Global or regional dysfunction and structural</td>
<td></td>
</tr>
<tr>
<td>alterations*</td>
<td></td>
</tr>
<tr>
<td>Major</td>
<td></td>
</tr>
<tr>
<td>• Severe dilatation and reduction of RV</td>
<td>• Regional RV akinesia, dyskinesia, or aneurysm</td>
</tr>
<tr>
<td>ejection fraction with no (or only mild) LV</td>
<td>• and 1 of the following (end diastole):</td>
</tr>
<tr>
<td>impairment</td>
<td>—  PLAX RVOT ≥ 32 mm (corrected for body size [PLAX/BSA] ≥ 19 mm/m²)</td>
</tr>
<tr>
<td>• Localized RV aneurysms (akinetic or dyskinetic</td>
<td>—  PSAX RVOT ≥ 36 mm (corrected for body size [PSAX/BSA] ≥ 21 mm/m²)</td>
</tr>
<tr>
<td>areas with diastolic bulging)</td>
<td>—  or fractional area change ≤ 33%</td>
</tr>
<tr>
<td>• Severe segmental dilatation of the RV</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>By MRI:</td>
<td>• Regional RV akinesia or dyskinesia or dyssynchronous RV contraction</td>
</tr>
<tr>
<td>• Regional RV akinesia or dyskinesia or</td>
<td>• and 1 of the following:</td>
</tr>
<tr>
<td>dyssynchronous RV contraction</td>
<td>—  Ratio of RV end-diastolic volume to BSA ≥ 110 mL/m² (male) or ≥ 100 mL/m² (female)</td>
</tr>
<tr>
<td>• and 1 of the following:</td>
<td>—  or RV ejection fraction ≤ 40%</td>
</tr>
<tr>
<td>By RV angiography:</td>
<td></td>
</tr>
<tr>
<td>• Regional RV akinesia, dyskinesia, or aneurysm</td>
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By 2D echo:

By MRI:

Electrophysiologic testing

Flecainid test:
- umasking type II and III Brugada

Electrophysiologic testing:
- risk assessment and therapy
Laboratory exams

Genetic testing: e.g. HCM, ARVC, long- and short-QT, M. Lenègre/M.Lev

Blood tests: in specific clinical constellations
Sports-associated cardiac death is very rare and therefore not relevant.

The ECG generates too many “false-positive” findings and consequently causes unnecessary costs, uncertainty and even disqualification.

The evidence mainly bases on Italian data.

...what are the pleas of the critics?

- the individuum counts
- older/leisure athletes!
  (greyzone…)
- Improving the guidelines e.g. black, female athletes
- specifically skilled physicians
- „core labs“/working groups
- the others are challenged!
The role of stress tests in cardiac screening

**THE ATHLETE WITH A „NORMAL HEART“**

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<td>&gt;35</td>
<td>additional stress-test…?</td>
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The role of stress tests in cardiac screening

18% sensitivity and 92% specificity of a pos. test to predict 1 exercise-related cardiovascular event in asymptomatic men with dyslipidemia (Siskovick et al.)

Striktly dependant of the pre-test propability

adequate risk-assessment!!
Screening of sedentary “older” individuals

Screening of regularly active “older” individuals

Latest news, outlook and discussion
Current topics in cardiac screening:

„closing the gaps“…

Cardiac Screening in

- athletes of different ethnias
- „elderly“ athletes (> 35 years)
- competitive female athletes
Presenation of the FIFA pre-competition medical screenings/PCMA prior to the FIFA World Championship in South Africa 2010
FIFA/F-MARC PCMA in Gabon/Western Africa
Cardiac screening of all referees and assistant referees
Qualified for the FIFA Football World Championship 2010
Support of the teams in their cardiac screening prior to the Women’s U-20 and U-17 Football World Championship 2010.
Looking for new risk factors
Der Puls von Ottmar Hitzfeld vor, während und nach einem Spiel mit Bayern München

Pulsschläge/Minute
120
110
100
90
80
70
1 Minute vor Spielbeginn
Spielbeginn
(Halbzeitpause
Bayern
Bayer
Beginn 2. Halbzeit
3:0
Chance FC Bayern
Spielende
Pressekonferenz
16:30; 17:00 Uhr
17:00; 17:30 Uhr
17:30; 18:00 Uhr
18:00; 18:30 Uhr
18:30; 19:00 Uhr
19:00; 19:30 Uhr
19:30; 20:00 Uhr
20:00; 20:30 Uhr
20:30; 21:00 Uhr
21:00; 21:30 Uhr
21:30; 22:00 Uhr
22:00; 22:30 Uhr
22:30; 23:00 Uhr
Myocardial infarctions during football World Championship 2006 in Germany
Life expectancy and heart rate in animals

Levine et al (1997)

<table>
<thead>
<tr>
<th>Animal</th>
<th>Life Expectancy (Years)</th>
<th>Heart Rate (bpm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mouse</td>
<td>3</td>
<td>1000</td>
</tr>
<tr>
<td>Hamster</td>
<td>4</td>
<td>500</td>
</tr>
<tr>
<td>Rat</td>
<td>8</td>
<td>300</td>
</tr>
<tr>
<td>Marmot</td>
<td>10</td>
<td>200</td>
</tr>
<tr>
<td>Dog</td>
<td>15</td>
<td>100</td>
</tr>
<tr>
<td>Monkey</td>
<td>20</td>
<td>50</td>
</tr>
<tr>
<td>Cat</td>
<td>25</td>
<td>20</td>
</tr>
<tr>
<td>Giraffe</td>
<td>30</td>
<td>10</td>
</tr>
<tr>
<td>Ass</td>
<td>40</td>
<td>5</td>
</tr>
<tr>
<td>Elephant</td>
<td>80</td>
<td>2</td>
</tr>
<tr>
<td>Horse</td>
<td>100</td>
<td>1</td>
</tr>
<tr>
<td>Lion</td>
<td>150</td>
<td>0.5</td>
</tr>
<tr>
<td>Whale</td>
<td>200</td>
<td></td>
</tr>
<tr>
<td>Man</td>
<td>100</td>
<td></td>
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Levine et al (1997)
Thank you for your attention!
1. Is there a rhythm other than sinus rhythm?
2. If sinus rhythm, is the P wave normal?

- left atrial enlargement: negative portion of the P wave in lead V1 ≥ 0.1mV in depth and ≥ 0.04s in duration
- right atrial enlargement: peaked P wave in leads II and III or V1 ≥ 0.25mV in amplitude

Interpretation of 12-lead resting ECG
Interpretation of 12-lead resting ECG

QRS complex?
Interpretation of 12-lead resting ECG

Repolarisation, ST-Segment, T-wave, QT-interval
Interpretation of 12-lead resting ECG

- premature ventricular beats or more severe ventricular arrhythmias
- supraventricular tachycardias, atrial flutter, or atrial fibrillation
- short PR interval (<0.12s) with or without “delta” wave
- sinus bradycardia with resting heart rate ≤40 beats/min (only if increasing less than 100/min in stress test)
- first degree (PR ≥0.21s) AV block (only if not shortening through hyperventilation or exercise test)
- second or third degree AV block