We investigated the long-term consequences of popular ultra-endurance sports in non-elite athletes.

**Methods:** Male runners of the 2011 Grand Prix of Bern, a 10 mile race were included. Athletes with an office Blood Pressure (BP) > 140/90 mmHg were excluded. Subjects were stratified according to their former participations in long-distance competitions: leisure-time runners (active controls), marathon runners, and ultra-endurance athletes (78 and 150 km races, long-distance triathlons). Ventricular morphology and function was assessed by echocardiography, including Doppler Tissue Imaging (DTI) and 2D speckle tracking. 24-hour ambulatory Holter and BP monitoring was performed. Results were adjusted for lifetime training hours.

**Results:** Two-hundred-and-eighty athletes applied, 107 were randomly selected and 97 were included in the final analysis. Mean age was 42±8 years. RV end-diastolic and end-systolic areas, fractional area change, Tricuspid Annular Plane Systolic Excursion (TAPSE), DTI of the RV lateral wall, and RV strain and strain rate did not differ between the groups. Ventricular ecotox was low and equally distributed between the groups (Table 1). In a stepwise multiple regression analysis, including age, lifetime training hours, type of endurance sports, and mean systolic BP, lifetime training hours were positively associated with RV size and TAPSE. Type and volume of sports activity showed no correlation with other RV functional parameters or etiology.

**Conclusion:** In elite athletes, long-term ultra-endurance sport practice was not associated with RV dysfunction or increased ventricular ectopy. Our data suggests that this phenomenon may be restricted to the unique subgroup of professional power-endurance athletes.

**1787 I BEDSIDE**

**Frequency of significant ECG abnormalities in 1000 sport active children**

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In consensus documents the begin and pathological ECG abnormalities in athletes have been defined. This was done rather for adults than young teenagers. In consensus documents the begin and pathological ECG abnormalities in athletes have been defined. This was done rather for adults than young teenagers. In consensus documents the begin and pathological ECG abnormalities in athletes have been defined. This was done rather for adults than young teenagers. In consensus documents the begin and pathological ECG abnormalities in athletes have been defined. This was done rather for adults than young teenagers. In consensus documents the begin and pathological ECG abnormalities in athletes have been defined. This was done rather for adults than young teenagers.

**Methods:** We analyzed standard 12-leads ECGs of 1000 sport active children (243 girls and 757 boys, mean age 12±2.7 years). ECGs were recorded during TeleInterMed screening. Data were collected in 3 sport medicine clinics and transmitted for analysis to central station. Electrocardiograms analyzed by 2 doctors were reevaluated by experienced supervisor. We analyzed the presence of reported in consensus documents significant ECG abnormalities.

**Results:** Mean RR interval was 834±35 ms, QTc 433±25 ms. Important abnormalities were present in 14% ECGs. QTc ≥ 440 ms was present in 1.3% ECGs. Other findings included: left atrium abnormalities in 3.5%, left axis deviation in 1.4%, pathological Q waves in 2.2%, RBBB 1.2%, left anterior hemiblock 1.1%, preexcitation 0.4%, ST elevation ≥ 2 mm in any leads* 4.2 vs 23.2 mmHg, vs 25 (p=0.01). Ventricular premature beats 2 (5) 4 (14) 4 (12) 0.542

**Conclusion:** Significant ECG abnormalities are present in more than 10% sport active children.

**1788 I BEDSIDE**

**The new Seattle Criteria reduce the prevalence of abnormal ECG findings in professional football players**

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**Purpose:** To compare the new ‘Seattle Criteria’ for ECG interpretation in athletes, with former recommendations from ESC.

**Methods:** In 2008, 574 professional football players (mean age 25, range 18-38 years) in Norway underwent preparticipation ECG-screening (CARDIOLINE, ReallClick version 3.5.4, Milan, Italy). ECGs were read manually by one investigator, measuring the global QRS complex with calipers. Analyses were performed according to the definitions of Corrado et al. 2010, as “updated” by Uboeri et al. 2011, and the new “Seattle criteria” by Dreizner et al. 2013, and the results were compared.

**Results:** Main differences are presented in Table 1. Altogether 111 (19%) players had abnormal ECG-findings according to the “Seattle Criteria”, compared to 292 (51%) applying the former ESC recommendations (p<0.001).

**Table 1: Main differences in normal and abnormal ECG findings between ESC versus “Seattle Criteria” in interpreting athletes’ ECG**

<table>
<thead>
<tr>
<th>Uncommon and abnormal findings; ESC recommendations, <strong>ESC</strong></th>
<th>Difference, <strong>Seattle criteria</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>ST elevation (≥ 2 mm in any lead)* 50 (172) 23 (72)</td>
<td>ST elevation (≥ 2 mm in any lead)* 33 (112) 21 (68)</td>
</tr>
<tr>
<td>TWI in any lead except V1,V2 ili II, aVR or TWI (≥ 2 mm in &gt;2 contiguous leads)* 70 (NA) 40 (28)</td>
<td>TWI in any lead except V1,V2 ili II, aVR or TWI (≥ 2 mm in &gt;2 contiguous leads)* 32 (NA) 20 (28)</td>
</tr>
<tr>
<td>LVH voltage criteria and another pathologic finding 33 (108) 3 (23)</td>
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<tr>
<td>RVH voltage criteria (&lt; 30 years old, ≥ right atrial abnormalities, TWI≤ 1 mm in V2 or QRS axis ≥-115, V1, ≥ QRS axis &lt; 120°) 33 (108) 33 (108)</td>
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</tr>
<tr>
<td>Non-specific intraventricular conduction delay (QRS duration &gt;120 ms, vs ≤ 140 ms) 18 (61) 13 (94)</td>
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<tr>
<td>Short QTc (&lt;340 ms, vs &gt;320 ms) 12 (21) 11 (21)</td>
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<tr>
<td>Q-waves ≥ 3 mm and/or &gt;40 ms (in any leads except III, aVR and V1, vs ≥ 2 leads except III and aVF) 27 (83) 18 (57)</td>
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</tr>
<tr>
<td>ST depression (≥ 0.5 mm in any lateral wall, &gt;0.5 mm in any other lead, vs ≥ 0.5 mm in two or more leads) 9 (27) 2 (7)</td>
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</table>

**Conclusion:** The significant reduction of 32% abnormal ECG-findings applying the new “Seattle Criteria” versus the ESC’s recommendations will probably reduce the need and costs of extended examinations after preparticipation ECG-screening of athletes.

**1813 I BEDSIDE**

**Familial aggregation of aortic stenosis**

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**Purpose:** Aortic stenosis is the most common valve disease in the elderly and can be considered multifactorial and genetic components have been suggested. However, knowledge on familial occurrence on a population based level is missing. We aimed to describe the familial aggregation of aortic stenosis in the adult population.

**Methods:** From nationwide Danish registers we included a cohort of persons born in or after 1910, with identifiable relatives. From 1977 to 2012 we identified all cases diagnosed with aortic stenosis in the cohort and relatives aged 35 years or more, who did not have a registered diagnosis of cardiovascular disease before age 35 years. We also assessed for familial occurrence of aortic stenosis presenting with frequently seen cardiac co-morbidities such as ischemic heart disease and endocarditis in cohort members. We estimated incidence rate ratios (IRR) adjusted for sex, age and calendar period for aortic stenosis by family history of aortic stenosis in first degree relatives; this was done by log-linear Poisson regression analysis.

**Results:** We followed a cohort of 4,764,969 persons free of CVD before age 35 years, for 98,871,697 persons-years and identified 59,362 persons with aortic stenosis. The median age at diagnosis of aortic stenosis in persons (n=258) with a family history of aortic stenosis in a first degree relative was 58 years, and in those (n=59,104) without such a family history the median age was 74 years (p<0.001). Given a first degree relative with aortic stenosis the IRR was 9.5% (95% confidence interval (CI) for aortic stenosis was 1.61 (95% CI 1.42-1.82). In persons aged 35-55 years the IRR was 1.36 (95% CI 1.12-1.65) for aortic stenosis in those aged 56 to 75 years the IRR was 2.13 (95% CI 1.75-2.59), and in those aged 76 years or older the IRR was 1.89 (95% CI 1.43-2.50). The risk by family history in those with certain cardiac co-morbidities was stronger.

**Conclusion:** To our knowledge this is the first nationwide study to report and quantify the risk of aortic stenosis by family history of aortic stenosis. This risk...