Prevalence of abnormal ECGs in male soccer players decreases with the Seattle criteria, but is still high

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Electrocardiogram (ECG) and echocardiography are mandatory in preparticipation cardiac screening in soccer players. Abnormal ECG findings usually require follow-up investigations. The main aim of this study was to compare the prevalence of abnormal ECG findings in male professional soccer players according to European Society of Cardiology’s (ESC) recommendations and the Seattle criteria, and to assess the need for echocardiography. ECGs from 587 of 595 (99%) players were recorded with ClickECG, and measurements were derived with visually adjusted on-screen calipers on the computer-based averaged PQRST complex. Echocardiographic recordings were performed with Vivid 7/i and categorized according to reference values for athlete’s heart. After the initial screening, 32 (5.5%) players were recommended for follow-up. The prevalence of abnormal ECGs was 29.3% vs 11.2% according to the ESC’s recommendations and the Seattle criteria, respectively. None of the players with abnormal ECGs only according to the ESC’s recommendations had abnormal echocardiograms. Echocardiography alone detected one player with abnormalities (athlete’s heart). The Seattle criteria reduced the number of athletes with abnormal ECGs considerably compared with the ESC recommendations. Based on echocardiographic evaluations, this increased the specificity of the Seattle criteria, without increasing the number of false-negative ECGs. The need for mandatory echocardiography in soccer players seems limited.

Electrocardiogram (ECG) has a low sensitivity and specificity to detect potentially lethal cardiac disorders in athletes, speaking against its inclusion in preparticipation cardiac screening (Bahr, 2010). Application of the recently published Seattle criteria for interpreting ECG in athletes will lead to a reduction in abnormal ECG findings by categorizing more commonly found alterations as normal (Drezner et al., 2013). The prevalence of abnormal ECG findings relates to age, gender, ethnicity, level of activity, and sporting discipline (Magalski et al., 2008; Le et al., 2010; Borjesson & Dellborg, 2011; Papadakis et al., 2011; Wilson et al., 2012; Kervio et al., 2013). Prior participation in screening will also influence the prevalence. However, many studies compile all results despite a heterogeneous population (Pelliccia et al., 2000; Corrado et al., 2006). In order to identify ECG findings that may discriminate normal from pathological remodeling, we need standards comprising all phases of the ECG recording, processing, and interpretation. The ECG devices’ technical specifications, lead placements, and methods for measurement can all result in significant differences in the reported amplitudes, intervals, and diagnostic statements (Kligfield et al., 2007; Berge et al., 2014). However, such information is rarely presented (Kligfield et al., 2007).

During the last decade, ECG devices with built-in algorithms for measuring intervals from earliest wave onset to latest offset in all leads have become more utilized, and must be accounted for in comparative studies (Kligfield et al., 2007).

Echocardiography is mandatory in preparticipation cardiac screening in soccer players (UEFA Medical Committee, 2006), but not recommended for athletes in general (Maron et al., 2007; Corrado et al., 2010). The prevalence of hypertrophic cardiomyopathy seems to be less than expected in athletes (Basavarajaiah et al., 2008b; Brosnan et al., 2013). The need for mandatory echocardiography in soccer players might be questioned.

The main aim of this study was to compare the prevalence of abnormal ECG findings between the European Society of Cardiology’s (ESC) recommendations as specified by Uberoi et al. (2011), with the new Seattle criteria for interpreting ECG, in professional soccer players in Norway (Drezner et al., 2013). The secondary aim was to study if the prevalence of abnormal findings
on echocardiography made this examination useful in athletes.

**Methods**

**Participants**

Five hundred and ninety-five male professional soccer players in Norway underwent mandatory preparticipation cardiac screening during a preseason training camp in 2008 (UEFA Medical Committee, 2006). Players aged 18–40 years responded to a questionnaire regarding their height, weight, ethnicity [Caucasian (white), Afro-American (black), or other], whether they had experienced symptoms during or in close proximity to exercise (Table 1), and about cardiovascular disease in first-degree relatives < 55 years (Table 1). All participants gave written informed consent, and the study was approved by the regional ethical committee.

**Physical examination**

Sitting blood pressure (BP) was registered after at least 1-h rest as the mean of two consecutive BP measurements from a validated automatic BP monitor (Dinamap ProCare DPC300N, GE, Milwaukee, Wisconsin, USA), and categorized as high BP when systolic BP ≥ 140 mmHg or diastolic BP ≥ 90 mmHg.

**Electrocardiography**

With the player in a supine position, the precordial ECG electrodes were placed according to recommendations (Kligfield et al., 2007), and the four limb lead electrodes were placed on the arms and legs just distal to the shoulders and hips. ClickECG with Real Click software version 3.2.10 (Cardiette Cardioline, Milan, Italy) collected the simultaneous 10-s digital recordings with a front-end sampling rate of 2000 per second and compression ratio 4:1. Paper speed was 25 mm/s with 10 mV gain. The frequency response was 0.05–150 Hz, the baseline filter was always on, network filter was set at 50 Hz, and muscular filter at 40 Hz. The software recognized waveforms with amplitudes of at least 25 μV and durations of at least 6 ms. The average PQRST complex in each lead was calculated by the trimmed mean, discarding the first and last quartile of the data. The superimposed global complex (SGC) was composed of 12 representative beats from 12 leads (Fig. 1), and the duration of the QRS interval was measured from the earliest wave onset to the latest offset in the SGC. According to the Seattle criteria, intraventricular conduction was categorized as abnormal if the computer-derived QRS interval duration was ≥ 140 ms after visual assessment of first onset and latest offset in all leads in the SGC.

The visual analysis was performed on 100–400% magnified signals on a 24-inch screen with 1680 × 1050 MP resolution, using on-screen calipers (Real Click, software version 3.5.4). The P-wave, PR interval, and QRS durations were measured to the nearest 2 ms from the averaged PQRST complex in lead II. If the PR interval was < 120 ms in lead II, all leads were measured, and the PR interval was categorized as short if < 120 ms in all leads. The R- and S-wave amplitudes were measured to the nearest 1 μV as the mean of the highest amplitudes in the QRS complexes, and the maximum P-wave amplitude was measured in lead II. The offset of the QT interval was adjusted visually using the intersection between a tangent drawn from the descending part of the T wave to the horizontal line drawn from the PR interval in lead II. QTc was defined according to Bazett’s formula and categorized as long if ≥ 470 ms in any lead. The QT duration was visually assessed in all leads if ≤ 330 ms or QTc < 340 ms (ESC criteria).

<table>
<thead>
<tr>
<th>Symptoms related to exercise</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest pain</td>
<td>22 (3.7)</td>
</tr>
<tr>
<td>Palpitations</td>
<td>25 (4.3)</td>
</tr>
<tr>
<td>Syncope/near syncope</td>
<td>17 (2.9)</td>
</tr>
<tr>
<td>Breathlessness</td>
<td>35 (6.0)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Family history in relatives &lt; 55 years</th>
</tr>
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<tbody>
<tr>
<td>Sudden cardiac death</td>
</tr>
<tr>
<td>Cardiovascular disease</td>
</tr>
<tr>
<td>Hypertension</td>
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<tr>
<td>Diabetes</td>
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</tbody>
</table>

Values are presented as n (%).

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**Fig. 1.** Superimposed global PQRST complex computed from eight leads’ raw data in electrocardiogram recording of a 27-year-old professional white male Norwegian soccer player.
or QTc ≤ 320 ms (Seattle criteria), and only regarded as short if these criteria were fulfilled in all leads. The following amplitudes were visually assessed on-screen in the precordial leads and limb leads, and measured with on-screen calipers in the separate leads if borderline: ST segment depression was categorized as > 0.5–1.0 mm or > 1.0 mm in any lateral leads (I, aVL, V5, or V6) and > 1.0 mm in any other lead; ST segment elevation was categorized as > 1.0–2.0 mm or > 2.0 mm; pathological T-wave inversion as > 1.0 or > 2.0 mm (or negative part of biphasic T wave > 1 mm); and pathological Q-wave amplitudes as ≥ 3.0 mm and/or 40 ms in duration. All amplitudes were related to the PR line.

Heart rate in beats per minute (bpm) and QRS axis in degrees were derived from the computer and visually confirmed.

For detailed differences between the specified ESC recommendations and the Seattle criteria, see Supporting Information Tables S1–S3.

Echocardiography

All echocardiographic recordings were performed with a 2.5-MHz transducer (Vivid 7 and Vivid i; GE Vingmed Ultrasound AS, Horten, Norway) as described previously (Berge et al., 2013b).

Interpretations and follow-up

At the time of screening, on site, 11 experienced cardiologists, one resident, and one cardiology fellow (both trained in echocardiography) decided if the players were eligible for professional soccer based on history, BP, ECG, and echocardiographic evaluation. At a second assessment in 2008, most of the ECGs were reanalyzed by four experienced cardiologists, and abnormal ECG findings were related to echocardiographic findings by three cardiologists. After consensus, they recommended additional follow-up based on best clinical judgment at that time. Subsequently, for the present study, all ECGs were retrospectively reassessed and measured by one investigator (H. M. B.), who also developed separate syntaxes adjusted to the different criteria, and possible rhythm or conduction disturbances were discussed with a cardiac electrophysiologist (K. G.). The ECGs were categorized as normal, including common and training-related ECG changes, or as abnormal.

Statistical analyses

Because most of the ECG measurements had non-Gaussian distribution, data are presented as medians and interquartile ranges. The Mann-Whitney U-test was used to test for significant differences between two groups. Correlation between QRS duration from the SGC and visual analysis from the average PQRST complex in lead II was tested using Pearson’s bivariate correlation analysis. Differences between two subgroups were analyzed using t-tests for continuous variables, and χ² or Fisher’s exact tests for categorical variables. P < 0.05 was considered statistically significant, and all tests were two tailed. All statistical analyses were conducted using SPSS (PASW Statistics 18; IBM Corporation 2010, Armonk, New York, USA).

Results

ECG of good quality (all 12 leads measureable) was available from 587 (99%) of 595 players. Their median age was 25.0 years (21.0–28.0), height 183.0 cm (179–187), and weight 79.0 kg (74.0–84.0). The skin color was white in 497 (85%), black in 48 (8%), mixed for 13 (2%), and other for 29 (5%). History of symptoms during sports activity and cardiovascular disease in first-degree relatives are presented in Table 1, and there were no significant differences in these between players with abnormal vs normal ECGs. Median office BP was 121/69 mmHg (115.0–128.5/64.0–74.5) and 39 (7%) players had high BPs.

ECG findings according to the ESC and Seattle criteria

Abnormal findings were present in 172 (29.3%) players’ ECG according to the specified ESC recommendations (Uberoi et al., 2011), including all the 66 (11.2%) players with abnormal ECGs according to the Seattle criteria (Fig. 2) (Supporting Information Figs. S1–S2) (Drezner et al., 2013). The main differences were

Fig. 2. Flow chart showing abnormal electrocardiogram (ECG) findings according to the specified European Society of Cardiology’s (ESC) recommendations and Seattle criteria.
accounted for by the reduction in T-wave inversion (−7.1%), right atrial enlargement (−5.6%), right ventricular hypertrophy (−4.5%), and intraventricular conduction abnormalities (−3.9%) (Fig. 2). Common and training-related findings were found in 383 (65.2%) and 512 (87.2%) players according to the specified ESC recommendations and Seattle criteria, respectively. The increased prevalence was mainly driven by more incomplete right bundle branch block (≥7.5%) and first-degree AV block (≥4.8%) (Supporting Information Fig. S3).

The median QRS axis was 78.5° (64.0–88.8). The median QRS duration of SGC and average PQRST complex in lead II was 110 ms (100–120) and 104 ms (98–108), respectively, and they correlated moderately, r = 0.43 (P < 0.0001). For visually analyzed intervals and amplitudes, see Supporting Information Table S4. After visual assessment, 4 out of 7 ECGs with computer-derived QRS duration of ≥140 ms in the SCG were categorized as abnormal non-specific intraventricular conduction delay. One of 12 ECGs with short PR interval in lead II had short PR interval in all leads.

Recommended follow-up
After the initial assessment 593 (99.7%) players were found eligible for competitive soccer while two were temporarily excluded due to hypertension grade II (≥160/95 mmHg) and left ventricle hypertrophy (LVH) on echocardiography. One of them had abnormal ECG findings according to both criteria, while the other had a normal ECG. Two players were already under medical attention when the screening was performed [Wolff–Parkinson–White syndrome (n = 1) and idioventricular escape rhythm (n = 1)]. After the second assessment, another 28 players received letters with recommendations for follow-up due to abnormal ECG findings (n = 13), high BP (n = 4), valve insufficiencies (n = 9), concentric hypertrophy (n = 2), eccentric hypertrophy (n = 1), atrium septum aneurism (n = 1), or combinations of these. Only one individual with symmetric concentric hypertrophy (assessed as athlete’s heart) was detected on the basis of echocardiography alone and had a normal history and ECG, although his heart rate was borderline: 31 bpm. Six of the 28 players were recommended for follow-up due to causes retrospectively considered normal and training related [Mobitz type I second-degree AV block (n = 2), ectopic atrial rhythm (n = 2), junctional escape rhythm (n = 1), and moderate eccentric hypertrophy (n = 1)]. None of the players were recommended for follow-up based on family history or symptoms alone.

All players with abnormal ECG findings according to the ESC recommendations only had normal echocardiograms. Nine players with normal ECGs and valve insufficiencies grade I–II (n = 8), atrium septum aneurism (n = 1), and the one with concentric hypertrophy (athlete’s heart) would have been missed without echocardiography of all players.

Other findings
Supporting Information Table S5 relates the present prevalence of abnormal findings to other studies: T-wave inversions and ST elevations ≥1 or 2 mm divided in different groups, Q waves categorized as 25% of the ensuing R wave or ≥4 mm in two or more leads, and LVH with associated non-voltage criteria.

Discussion
The prevalence of abnormal ECGs in professional soccer players in Norway was reduced from 29.3% to 11.2% when the specified ESC recommendations were substituted by the new Seattle criteria. The main differences were accounted for by the reduction in T-wave inversion, right atrial enlargement, right ventricular hypertrophy, and intraventricular conduction abnormalities (Fig. 2). Using the Seattle criteria, common and training-related ECG changes increased from 65.1% to 83.8%, mainly driven by an increased prevalence of incomplete right bundle branch block and first-degree AV block. The only case of concentric hypertrophy diagnosed by echocardiography alone was assessed as athlete’s heart, and all players with abnormal ECGs according to the ESC’s recommendations only were normal.

ECG findings according to the ESC and Seattle criteria
The great benefit of the new Seattle criteria is their detailed description of each ECG finding with corresponding reference value for abnormality and recommended follow-up (Drezner et al., 2013). Hence, comparison with other studies is simplified. In a recent study applying the new Seattle criteria on 16- to 35-year-old Australian elite athletes of both sexes recruited from >15 sporting disciplines, Brosnan et al. (2013) found 4.5% with abnormal ECGs. We found more than twice as many abnormal ECGs using the same criteria. There are several explanations for this. First, our athletic population consisted only of men playing soccer, both factors associated with more abnormal ECG findings (Pelliccia et al., 2000). Second, body position or exact placement of ECG electrodes are not reported by Brosnan et al. nor are specifications of their ECG device. The muscular filter setting to 40 Hz in our study may have broadened the QRS complexes. Manual ECG ruler without magnifier measures only to the nearest 10 ms and 0.1 mV. In our study all digital samples were displayed, and the on-screen calipers measured durations and amplitudes down to 2 ms and 0.05 mV, respectively. Hence, small deflections as T-wave inversions ≥1.0 mm and Q waves ≥3.0 mm were easier to detect (Berge et al., 2014).
Third, the visual assessment both on paper and on-screen has subjective components: where to start and end a measurement and in choice of lead. The correlation between visual analyses of the QRS duration in the average PQRST complex in lead II and computerized measurements in the SGC was moderate. According to the principle of first wave onset and latest offset, intervals derived from the SGC will always be greater than in individual leads, explaining the higher prevalences of intraventricular conduction delay in our study (Kligfield et al., 2007). Consequently, redefinitions are required for SGC measurements (Kligfield et al., 2007). Still, computer-derived pathological measurements should always be visually assessed to check for technical errors. Forth, choice of reference values: in lack of detailed descriptions and reference values in the ESC recommendations (Corrado et al., 2010), we chose to add the specifications from Uberoi et al. while Brosnan et al. chose other reference values. These differences had special impact on the prevalence of long and short QT interval, 0.5% vs 2.3% and 0.7% vs 4.3%, respectively, and on the prevalence of common and training-related ECG findings as first-degree AV block (Supporting Information Table S6), and right atrial enlargement, which was not mentioned at all in the Seattle criteria. Moreover, the huge difference in prevalence of early repolarization might be explained by the confusing definition of J point (Supporting Information Table S7) in addition to different reference values (Froelicher, 2012). Earlier participation in screening will also influence the results: the prevalence of abnormal findings will be lower in Italy where many athletes already are excluded owing to mandatory cardiac screening since 1982, and higher in Norway after the very first cardiac screening.

The prevalence of abnormal ECGs in other studies varies from 1.8% to 35% (Supporting Information Table S6). Pathologic T-wave inversions and Q waves, which account for the majority of abnormal ECG findings in our study, vary from 0% to 22.8% and 0% to 3.0%, respectively, due to different reference values in other studies. Different reference values in our study could have reduced the prevalence of pathologic T-wave inversions to 0.9% and Q waves to 0% (Supporting Information Table S5).

**Recommended follow-up**

Mandatory echocardiography revealed one player with symmetric concentric hypertrophy and a normal ECG. The cardiologists assessed the findings as athlete’s heart and he continued to play soccer. If the lower limit for heart rate had been increased from 30 to 35 bpm, he would have been referred to follow-up. The other two players with heart rate < 35 bpm had type II second-degree AV block. Riding et al. (2013) found no athletes with hypertrophic cardiomyopathy by echocardiography alone. The two players with left ventricle hypertrophy due to hypertension would have been detected if players with repeated BP ≥ 160/100 mmHg were referred for follow-up. Hypertension as the only manifested reason for restricted activity and treatment after screening underscores the need for proper follow-up of young athletes with high BP (Berge et al., 2013a). Because all players underwent echocardiography, auscultation was not performed. It might, however, have detected some among the nine players with grade I–II valve insufficiencies. No abnormal echocardiogram was found in any of the 106 players with abnormal ECGs according to the ESC’s recommendations only, slightly indicating that the Seattle criteria increase the specificity related to echocardiographic findings without increasing the number of false-negative ECG findings.

**Differences related to ethnicity and sporting discipline**

We have presented all abnormal ECG findings related to ethnicity, but because of a small number of black players, we do not want to draw any conclusions. Anyway, the results are difficult to compare with other investigations, because some studies lack definition of ethnicity (Magalski et al., 2008; Le et al., 2010), while other studies use self-definition through questionnaires (Basavarajaiah et al., 2008a; Papadakis et al., 2011; Gati et al., 2013). Interestingly, none of these studies describe how they define athletes descending from parents with different ethnicity. Bhopal (2004) recommended that researchers should explain their understanding of the concepts of race or ethnicity and the classification they use. The prevalence of LVH in black athletes is usually reported to be high (Wilson et al., 2012; Schmied et al., 2013). Variations in cardiovascular diseases between African-born and Caribbean-born black people in New York exceed those between black and white people (Agymang et al., 2005). Hence, environmental factors as birthplace and migration might also influence development of LVH.

Professional American football players who play at positions that require sprinting and similar bursts of physical activity associated with abrupt elevations in heart rate, like our outfield players, showed more abnormal ECGs than players at positions with lower levels of activity (Magalski et al., 2008). This might contribute to higher prevalence of abnormal ECG findings in our athletes, compared with studies of athletes from other sporting disciplines.

**Family history, symptoms, and ECG findings**

There were no significant differences in family history of cardiovascular disease or symptoms during sports activity between players with abnormal vs normal ECGs. To assess retrospectively if the players “yes” or “no” should have warranted further follow-up was not possible. It has previously been reported that 13.0% of the anonymous
responders had been afraid of losing their license to play soccer (Solberg et al., 2012). This might have led to underreporting in the present as well as other studies (Wilson et al., 2012; Gati et al., 2013). Papadakis et al. (2011) describe no serious cardiac symptoms among 904 black athletes. On the other side, Wilson et al. (2008) refer the highest prevalence of positive family history of heart disease in 29.9% of junior athletes, even though cut-off for young age in family members was < 35 years, in contrast to usually 50–55 years. This high score is probably owing to that participants were encouraged to ask their first- and second-degree relatives if the family cardiac disease history was unknown to them initially.

Limitations
Our results are representative for professional male soccer players in Norway, but not necessarily for athletes in other sporting disciplines and with different age and sex. Our application of on-screen measurements of SGC and averaged PQRST complexes cannot be compared with results from traditional measurements on paper; however, the method used in the present study gives significantly less variability (Kligfield et al., 2007).

The regional ethical committee did not permit results of recommended follow-up examinations to be part of the study, consequently leaving the exact number of players with false positive or negative ECGs unknown. Lack of long-term follow-up data makes it difficult to say whether or not the players who had abnormal ECG according to the ESC-based screening, but passed the ECG-test based on the Seattle criteria, could safely continue athletic activity, or belong to a group at higher than normal cardiovascular risk.

More players would have been recommended for follow-up, if symptoms related to exercise or family history of cardiovascular disease had been systematically assessed during the screening situation.

Perspectives
We have shown a marked reduction in ECG findings classified as abnormal when applying the Seattle criteria instead of the specified ESC recommendations in professional male soccer players. However, mandatory echocardiography in all soccer players seems unnecessary. None of the players with abnormal ECGs only according to the ESC’s recommendations had abnormal echocardiograms. We therefore consider the new Seattle criteria as an important step to reduce the burdens and costs of follow-up that are required in athletes’ screening programs (Le et al., 2010; Borjesson & Dellborg, 2011; Mayer et al., 2012). However, further revisions of the Seattle criteria are needed as the prevalence of abnormal ECG findings is still high, especially in black athletes in other studies (Sheikh et al., 2014). In future studies factors related to participants, the ECG recordings, and technical specifications have to be thoroughly described to facilitate comparisons between studies.

Key words: ECG criteria, screening, electrocardiography, echocardiography, sudden cardiac death, soccer, football, Scandinavia.

Acknowledgements
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Supporting information
Additional Supporting Information may be found in the online version of this article at the publisher’s web-site:

Fig. S1. ECG findings from preparticipation screening of 587 athletes: numbers and percentages (%) of patterns suggestive of cardiomyopathy according to the European Society of Cardiology’s (ESC) classification of 12-lead ECG pattern with specifications from Uberoi et al. compared with the new Seattle criteria for ECG interpretations in athletes.

Fig. S2. ECG findings from preparticipation screening of 587 athletes: numbers and percentages (%) of patterns suggestive of primary electrical disease according to the European Society of Cardiology’s (ESC) classification of 12-lead ECG pattern with specifications from Uberoi et al. compared with the new Seattle criteria for ECG interpretations in athletes.

Fig. S3. ECG findings from preparticipation screening of 587 athletes: numbers and percentages (%) of common and training-related changes according to the European Society of Cardiology’s (ESC) classification of 12-lead ECG pattern with specifications from Uberoi et al. compared with the new Seattle criteria for ECG interpretations in athletes.

Table S1. Reference values for patterns suggestive of cardiomyopathy according to the European Society of Cardiology’s (ESC) classification of 12-lead ECG pattern with specifications from Uberoi et al. compared with the new Seattle criteria for ECG interpretations in athletes.

Table S2. Reference values for patterns suggestive of primary electrical disease according to the European Society of Cardiology’s (ESC) classification of 12-lead ECG pattern with specifications from Uberoi et al. compared with the new Seattle criteria for ECG interpretations in athletes.

Table S3. Reference values for patterns suggestive of normal training-related ECG findings according to the European Society of Cardiology’s (ESC) classification of 12-lead ECG pattern with specifications from Uberoi
et al. compared with the new Seattle criteria for ECG interpretations in athletes.

**Table S4.** Median ECG measurements in 587 professional male soccer players in Norway.

**Table S5.** Other ECG findings in 587 male professional male football players in Norway and showing how slightly different reference values would have altered the number of normal and abnormal ECG findings.

**Table S6.** Prevalence of variables in other studies with greatest discrepancy to prevalence of the same variables in our preparticipation screening study of 587 professional male football players in Norway.

**Table S7.** Reference values for abnormality of variables with greatest discrepancies in prevalence to variables in our preparticipation screening study of 587 professional male football players in Norway.

**References**


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