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The electrocardiographic manifestations of athlete’s heart and their association with exercise exposure

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Abstract
Objective: The aim of this study was to define the minimum amount of exercise per week (‘current exposure’) and the total amount of exercise (‘lifetime exposure’) needed to lead to the electrocardiographic changes fitting athlete’s heart. Methods: All the pre-participation screenings (including electrocardiograms (ECGs)) from collegiate athletes performed at University Sports Medical Center in 2013 and 2014 were collected. Data on height, weight, sex, age, current sport(s) participation and lifetime sport(s) participation were collected. Current exposure was categorised into 0–3, 3–6, 6–10 and >10 hours/week. Lifetime sport exposure was divided into five categories: 0–1000, 1001–2000, 2001–3000, 3001–4000 and >4000 hours. Results: The study population consisted of 1229 athletes (current exposure) and 1104 athletes (lifetime exposure). Current sport exposure: There was a significant increase in training-related ECG changes in the category 3–6 vs. <3 hours/week. When looking at individual parameters, we found an association with a significant difference in sinus bradycardia and QRS voltage (<3 vs. 3–6 hours/week) and first-degree AV-block (<3 vs. >10 hours/week). Lifetime sport exposure: There was an increase in training-related ECG changes that reached significance at an exposure >3000 hours. When looking at individual parameters, we found an association with a significant difference in sinus bradycardia (0–1000 vs. 2001–3000), QRS voltage (0–1000 vs. 3001–4000) and first-degree AV-block (0–1000 vs. >4000). Conclusion: A minimum of ≥3 hours/week of current exposure and a lifetime exposure of >3000 hours is needed to lead to the electrocardiographic changes fitting athlete’s heart.

Keywords: Athlete, electrocardiogram, exercise, heart

Highlights
• We recommend the use of ≥3 hours/week practising sports as a minimum value for the (electrocardiographic) development of an athlete’s heart.
• A minimum of >3000 hours of lifetime exposure is needed to lead to the electrocardiographic changes fitting athlete’s heart.
• Sinus bradycardia appears to be an early sign of the development of athlete’s heart.
• First-degree AV-block appears to occur as a late sign of the development of athlete’s heart.

Introduction
Exposure to exercise will lead to cardiac adaptations known as athlete’s heart. These adaptations have been frequently described by many authors, including Prior and La Gerche, who published an overview article on athlete’s heart in 2012 (Prior & La Gerche, 2012). As reported in the Seattle criteria, electrophysiological adaptations can also occur when the heart is exposed to exercise. These adaptations are described in Table I of an article on training-related electrocardiogram (ECG) changes (Drezner, Ackerman, et al., 2013). While generally speaking the association between athlete’s heart and exposure to exercise is clear, there is a paucity of data on the intricacies of this association. In fact, to date only two published articles give us some
insight. Fagard described the significant lowering of resting heart rate, increase in peak Vo2 and increase in left ventricular mass in 127 males when they performed more than 3 hours of exercise per week (Fagard, 2003). Ten years later, Drezner, Fischbach, et al. (2013) elaborated on ‘normal’ electrocardiographic findings in athletes. They posit that a minimum of 4 hours per week of regular and long-term participation in exercise is required for these electrical manifestations to become apparent, although no literature references are provided for this cut-off point. This arbitrary number of hours per week therefore seems more of an expert opinion than a scientifically proven statement. The next part of the statement, that the exercise should be regular and long-term, also lacks scientific confirmation, and moreover long-term and regular are poorly defined and give room for various interpretations.

The aim of this study was to define the minimum amount of exercise per week needed (‘current exposure’) for the electrical manifestations of athlete’s heart to become apparent. We also wanted to define the total amount of exercise (‘lifetime exposure’) needed to lead to the electrocardiographic changes fitting athlete’s heart.

### Materials and methods

#### Study design

When a student wants to participate in a collegiate sports education program in the Dutch city of Groningen, medical clearance is required. This involves a pre-participation cardiovascular screening according to the Lausanne recommendations. All pre-participation screenings were performed at University Sports Medical Center (USMC) in Groningen. All such screenings performed at USMC in 2013 and 2014 were collected. The study conformed with the principles defined in the Helsinki Declaration and the institutional ethics committee.

#### Subject and sports characteristics

Data on height, weight, sex, age, current sports participation (type of sport classified into static and dynamic (as described by (Levine et al., 2015) and hours/week), lifetime sports participation (type of sport classified into static and dynamic (as described by Levine et al. (2015), hours/week, number of years) were collected using biometric measurements and a questionnaire.

#### Electrocardiogram

A standard 12-lead resting ECG (Wech Allyn Cardi-oPerfect software V.1.6.4) was recorded. The ECG was analysed by the principal investigator (BB) for the presence of training-related ECG changes as described by Drezner, Ackerman, et al. (2013) in the Seattle criteria.

#### Data analysis of exposure

The following categories for current sport exposure were used: First, the proposed cut-off point of 4 hours per week for regular and long-term participation in exercise was used. This was based on the recommendation of Drezner, Fischbach, et al. (2013) that a minimum of 4 hours per week of regular and long-term participation in exercise is required for these electrical manifestations to become apparent. This cut-off point was used to define the minimum amount of exercise per week needed (‘current exposure’). The next category was the number of hours per week for lifetime sports participation, which was divided into four categories: 0–1000 hours, 1001–2000 hours, 2001–3000 hours, and 3001–4000 hours. The last category was for those who had participated in sports for more than 4000 hours. These categories were used to define the total amount of exercise (‘lifetime exposure’) needed to lead to the electrocardiographic changes fitting athlete’s heart.

### Table I. Population demographics.

<table>
<thead>
<tr>
<th>Current exposure</th>
<th>&lt;3 hours/week</th>
<th>3–6 hours/week</th>
<th>6–10 hours/week</th>
<th>&gt;10 hours/week</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Numbers</td>
<td>113 (9%)</td>
<td>510 (41%)</td>
<td>446 (38%)</td>
<td>140 (11%)</td>
<td>1229</td>
</tr>
<tr>
<td>Sex (% male)</td>
<td>49% male</td>
<td>71% male</td>
<td>78% male</td>
<td>66% male</td>
<td>70% male</td>
</tr>
<tr>
<td>Age (years)</td>
<td>19.4</td>
<td>18.8</td>
<td>19.1</td>
<td>19.1</td>
<td>19.0</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>175</td>
<td>179</td>
<td>180</td>
<td>179</td>
<td>179</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>66.1</td>
<td>69.6</td>
<td>71.9</td>
<td>72.8</td>
<td>70.6</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>21.4</td>
<td>21.8</td>
<td>22.2</td>
<td>22.6</td>
<td>22.0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Lifetime exposure</th>
<th>0–1000 hours</th>
<th>1001–2000 hours</th>
<th>2001–3000 hours</th>
<th>3001–4000 hours</th>
<th>&gt;4000 Hours</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Numbers</td>
<td>251 (23%)</td>
<td>209 (19%)</td>
<td>236 (21%)</td>
<td>225 (20%)</td>
<td>183 (17%)</td>
<td>1104</td>
</tr>
<tr>
<td>Sex (% male)</td>
<td>59% male</td>
<td>64% male</td>
<td>75% male</td>
<td>84% male</td>
<td>75% male</td>
<td>71% male</td>
</tr>
<tr>
<td>Age (years)</td>
<td>19.2</td>
<td>18.7</td>
<td>18.3</td>
<td>18.9</td>
<td>19.4</td>
<td>18.9</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>178</td>
<td>178</td>
<td>180</td>
<td>180</td>
<td>179</td>
<td>179</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>69.6</td>
<td>69.9</td>
<td>70.7</td>
<td>71.5</td>
<td>71.9</td>
<td>70.6</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>21.9</td>
<td>22.0</td>
<td>21.8</td>
<td>22.0</td>
<td>22.5</td>
<td>22.0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Sport type as defined by Levine et al. (2015) (total n = 1365)</th>
<th>A1</th>
<th>A2</th>
<th>A3</th>
<th>B1</th>
<th>B2</th>
<th>B3</th>
<th>C1</th>
<th>C2</th>
<th>C3</th>
<th>Multiple sport categories</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>8</td>
<td>1</td>
<td>125</td>
<td>24</td>
<td>1</td>
<td>29</td>
<td>444</td>
<td>34</td>
<td>25</td>
<td>674 (49%)</td>
</tr>
</tbody>
</table>

(0.6%) (0.1%) (9.2%) (1.8%) (0.1%) (2.1%) (32.5%) (2.5%) (1.8%) (49%)
hours/week of Drezner et al. and Fagard was compared to a cut-off point of 3 hours/week (Drezner, Fischbach, et al., 2013; Fagard, 2003). This cut-off point was based on practical experience in the Netherlands. When looking at soccer (the major sport in the Netherlands), we can define four categories of soccer players based on their participation and playing level. In the first category, soccer players only play in a recreational (social) team and participate in a maximum of one match (90 min) and one practice session (60 min) per week. This means that they exercise a maximum of 2.5 hours/week, hence the cut-off point of 3 hours/week. The second category is 3–6 hours/week, with the upper limit chosen by defining the second category of soccer players as low-level competitive first team players who play one weekly match (90 min) and practice a maximum of twice a week (90 min per practice). This leads to an average exposure of 4.5 hours/week. Because the next (third) category of soccer players who play at a high regional/national level practice at least three times and play one match weekly, 6 hours/week was chosen as the lower limit. The final cut-off value is 10 hours/week, defining the fourth category; this was chosen because when assuming a 90-min duration of every exercise session, athletes who reach >10 hours/week are exercising on a daily basis.

Lifetime sport exposure was calculated using the following formula:

\[
(Sport\text{ I years of participation} \times \text{hours/week} \times 52
+ (\text{Sport\text{ II years of participation} \times \text{hours/week} \times 52})
\ldots\text{etc} = \text{lifetime amount of sport exposure (in hours)}.
\]

Lifetime sport exposure was divided into five categories based on the range of exposure found: 0–1000 hours, 1001–2000 hours, 2001–3000 hours, 3001–4000 hours and >4000 hours.

**Statistical analyses**

The statistical analysis was performed using IBM SPSS Statistics V.22.0. To evaluate significant differences amongst the categories, Crosstabs with the Pearson Chi-Square test was used. A \( p \)-value < .05 was considered significant.

### Results

The total study population consisted of 1436 student athletes. Athletes who had not completely filled out the sport exposure questionnaires were excluded from this study. Of these 1436 athletes, 1229 (86%) had completely filled out the current sport exposure questions questionnaire and 1104 (77%) the lifetime sport exposure questionnaire. Almost half of the athletes participated in multiple sport types and could not be classified into static or dynamic sport categories (Levine et al., 2015). For the population demographics per category, see Table I.

#### Current sport exposure (hours/week)

The comparison between 3 hours/week vs. 4 hours/week is shown in Table II, and the crosstabs for current sport exposure are provided in Table III. When looking at the categories, we see that in the <3 hours/week category 51% of athletes already show at least one training-related ECG change and 17% have two or more training-related ECG changes. Still, there was a significant increase in the proportion of athletes showing training-related ECG changes in category 3–6 hours/week compared to category <3 hours/week.

When looking at the individual parameters, we found an association with a significant difference in sinus bradycardia and QRS voltage. Both showed a significant increase between <3 hours/week and 3–6 hours/week sport exposure. First-degree AV-block also showed an association, but the difference for this criterion was significant only between <3 hours/week and >10 hours/week. None of the other criteria showed an association with current sport exposure.

#### Lifetime sport exposure (hours)

The crosstabs for the lifetime sport exposure are provided in Table IV. When looking at the categories, we see that in the first category (0–1000 hours) 63% of athletes already showed at least one training-related ECG change and 22% had two or more training-related ECG changes. We nonetheless found a clear association between

<table>
<thead>
<tr>
<th>Table II</th>
<th>&gt;3 hours/week vs. ≥4 hours/week.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt;3 hours/week</td>
</tr>
<tr>
<td>≥1 Training-related ECG changes</td>
<td>50.9%a</td>
</tr>
<tr>
<td>≥2 Training-related ECG changes</td>
<td>17.0%a</td>
</tr>
</tbody>
</table>
lifetime sport exposure and training-related ECG changes – the higher the exposure, the higher the proportion of athletes with ECG changes. The increase in ECG changes reached significance at an exposure >3000 hours.

When looking at the individual parameters, we found an association with a significant difference in sinus bradycardia and QRS voltage. Sinus bradycardia showed a significant increase between 0–1000 hours and 2001–3000 hours of sport exposure, and QRS voltage showed a significant increase between 0–1000 and 3001–4000 hours of sport exposure. First-degree AV-block also showed an association, but the difference for this criterion was significant only between 0–1000 hours and >4000 hours of sport exposure. None of the other criteria showed an association with lifetime sport exposure.

Discussion

The aim of this study was to define the minimum amount of ‘current’ exercise needed for the electrical manifestations of athlete’s heart to become apparent. We also wanted to determine the total amount of exercise (‘lifetime exposure’) needed to
lead to the electrocardiographic changes fitting athlete’s heart.

Both with current and lifetime sport exposure, athletes who are or have been the least active already have a high prevalence of electrocardiographic manifestations of athlete’s heart. This same pattern is found in the literature, when athletes are compared to sedentary subjects. Both Sharma et al. and Papadakis et al. show that, even though athletes had a significantly higher prevalence of training-related ECG changes, the sedentary controls also showed a prevalence for individual parameters of around 20% (Papadakis et al., 2009; Sharma et al., 1999). It is therefore difficult use a clear cut-off point beyond which one can speak of athlete’s heart. Instead, the development of athlete’s heart, at least in terms of ECG changes, seems more of a gradual process and not an all-or-nothing phenomenon.

Current sport exposure: 3 hours/week is a better cut-off point than 4 hours/week

If we tried to define a minimum (current) level of sport exposure that leads to a significant increase in training-related ECG changes, our study shows that ≥3 hours/week would be a better cut-off point than ≥4 hours/week, the value posted by Drezner, Fischbach, et al. (2013) and Fagard (2003).

Table II shows that in our population, when using ≥3 hours/week the increase in prevalence of training-related ECG changes was higher than ≥4 hours/week. In addition, we feel that ≥3 hours/week is more clinically useful, as it better discriminates between sedentary and occasionally active individuals vs. athletes who want to compete. We therefore advocate using ≥3 hours/week as a cut-off value instead of ≥4 hours/week.

Lifetime exposure: >3000 hours is a cut-off point

When being exposed to over 3000 sporting hours (lifetime exposure), the prevalence of training-related ECG changes significantly increased compared to <3000 hours, with 76% having at least one training-related ECG change and 35% having two or more training-related ECG changes. This is still a long way from the elite athletes described by Wasfy et al. (2015). They studied a cohort (n = 330) of elite rowers and found that 94% had at least one training-related ECG change and 74% had two or more training-related ECG changes. When looking at these differences, we can argue that our athletic population is a competitive but not (yet) an elite athletic population. Hence to get the first significant increase in training-related ECG changes, you have to practice for at least >3000 hours, but to get to the elite level the amount of training hours required will be much higher.

Individual training-related ECG findings

Interestingly, not all training-related ECG changes showed the same association with exposure to exercise. Sinus bradycardia, QRS voltage criteria and incomplete RBBB (trend) showed a similar pattern, a higher category (lifetime or current exposure) being associated with a gradual increase in the amount of training-related ECG changes. By contrast, the prevalence of first-degree AV-block only seemed to increase significantly at the highest exercise level. Early repolarisation, sinus arrhythmia and junctional/atrial rhythm showed no association with exposure to exercise.

In the setting of exposure to exercise, sinus bradycardia, sinus arrhythmia, first-degree AV-block, early repolarisation and junctional/atrial rhythm are all considered signs of increased vagal tone (Drezner, Fischbach, et al., 2013). However, when looking at our results, not all of these signs of presumed increased vagal tone appear at the same pace. Sinus bradycardia, for instance, seems to occur relatively quickly after being exposed to exercise, while first-degree AV-block seems to occur only after being exposed to a high amount of exercise. We could therefor speculate that sinus bradycardia is an early sign of the development of athlete’s heart and that the late appearance of a first-degree AV-block could be a sign of structural and possible irreversible changes after being exposed to prolonged and intense exercise.

The other signs showed no association with exposure to exercise. Reasons for this could be that the association will only become apparent in the elite athletic population; that the association would only become visible when we compare this population to a sedentary population with no exposure to exercise; that the expected association with exercise is influenced by other variables, such as ethnicity or age; or that the numbers in this study are too small to see any association. For the lack of association with early repolarisation, an additional explanation could be that early repolarisation seems to be a dynamic process that is in part directly linked to exercise. This theory is supported by the findings of Noseworthy et al., who showed that early repolarisation is more common at times of peak fitness (Noseworthy et al., 2011). This also fits with the results of a yet-unpublished study by our group, where after a 9-month training period in a cohort of novice rowers...
the prevalence of early repolarisation in male rowers was 10% higher.

QRS voltage and incomplete RBBB findings are both indirect signs of increased cardiac chamber size (Drezner, Fischbach, et al., 2013). QRS voltage showed an association with exposure to exercise, a higher category of lifetime or current exposure being associated with a higher prevalence of QRS voltage. It thus appears that although the relation between isolated QRS voltages and LVH is somewhat problematic, the isolated QRS voltage criterion is a valid ECG marker of athlete’s heart.

Although the association between exercise exposure and the incomplete RBBB criterion was not significant, a clear trend is visible. The trend is comparable to the isolated QRS voltage criterion. The association may have become significant had we used a larger population.

**Limitations**

Results of this study should be interpreted with some caution because the exposure data was collected retrospectively through a questionnaire. This might have resulted in a recall bias. It is however very difficult to collect lifetime exposure data of a big cohort in any other way. We were also unable to differentiate between different sport types. It would have been very interesting to see what the effect of strength (static load), endurance (dynamic load) and mixed sport types would have been on training-related ECG changes (Brosnan et al., 2014; Levine et al., 2015; Prior & La Gerche, 2012). Because almost half of the athletes practiced (or had been practicing) more than one type of sport, we were not able to separate them into the different types, therefore the categories were too small for analyses.

The importance of ethnical differences and training-related ECG changes has been described by many authors (Calore et al., 2015; Fernandez & Thompson, 2014; Grace, Duvenage, & Jordaan, 2015; Masica, Maron, & Krovetz, 1972; Riding et al., 2013; Sharma, 2003; Sheikh et al., 2013; Sheikh & Sharma, 2014; Wilson et al., 2012). Since over 98% of the athletes in this cohort are of Caucasian West-European ethnicity, these results cannot be extrapolated to other ethnicities. Further research into the susceptibility to training-related ECG changes among different ethnicities is warranted.

A final limitation is the rather low number of highly exposed (elite) athletes. As already mentioned above, it could be that some of the training-related ECG changes only become apparent in this elite population.

**Conclusion**

This study shows that the development of athlete’s heart in terms of ECG changes is a gradual process. Still, as a practical recommendation, we would advise the use of ≥3 hours/week practising sports as a minimum value. We also showed that a lifetime exposure of ≥3000 hours of exercise is necessary to lead to a significant increase in the prevalence of the electrocardiographic changes fitting athlete’s heart. To attain the amount of training-related ECG changes seen in elite athletes, much more exposure is necessary. In terms of individual changes, sinus bradycardia appears to be an early sign of the development of athlete’s heart, while first-degree AV-block appears to occur as a late sign, possibly reflecting structural remodelling rather than functional adaptation.

**Perspective**

This study is one of the first to describe in more detail the association between exercise and the electrical manifestations of athlete’s heart. The study shows that when using a cut-off point to determine whether someone’s ECG is to be considered an athlete’s ECG, a minimum of ≥3 hours/week of exercise is necessary. It also shows that a more differentiated approach to the electrical manifestations of athlete’s heart based on exercise exposure is warranted.

**Disclosure statement**

No potential conflict of interest was reported by the authors.

**References**


