Electrical and structural adaptations of the paediatric athlete’s heart: a systematic review with meta-analysis

Gavin McClean,1,2 Nathan R Riding,1 Clare L Arden,1,3,4 Abdulaziz Farooq,1 Guido E Pieles,5,6 Victoria Watt,7 Carmen Adamuz,7 Keith P George,2 David Oxborough,2 Mathew G Wilson1,2,8

ABSTRACT

Aim To describe the electrocardiographic (ECG) and echocardiographic manifestations of the paediatric athlete’s heart, and examine the impact of age, race and sex on cardiac remodelling responses to competitive sport.

Design Systematic review with meta-analysis.

Data sources Six electronic databases were searched to May 2016: MEDLINE, PubMed, EMBASE, Web of Science, CINAHL and SPORTDiscus.

Inclusion criteria (1) Male and/or female competitive athletes, (2) participants aged 6–18 years, (3) original research article published in English language.

Results Data from 14 278 athletes and 1668 non-athletes were included for qualitative (43 articles) and quantitative synthesis (40 articles). Paediatric athletes demonstrated a greater prevalence of training-related and training-unrelated ECG changes than non-athletes. Athletes ≥14 years were 15.8 times more likely to have inferolateral T-wave inversion than athletes <14 years. Paediatric black athletes had significantly more training-related and training-unrelated ECG changes than Caucasian athletes. Age was a positive predictor of left ventricular (LV) internal diameter during diastole, interventricular septum thickness during diastole, relative wall thickness and LV mass. When age was accounted for, these parameters remained significantly larger in athletes than non-athletes. Paediatric black athletes presented larger posterior wall thickness during diastole relative to PWd than Caucasian athletes. Paediatric male athletes also presented larger PWd than females.

Conclusions The paediatric athlete’s heart undergoes significant remodelling both before and during ‘maturational years’. Paediatric athletes have a greater prevalence of training-related and training-unrelated ECG changes than non-athletes, with age, race and sex mediating factors on cardiac electrical and LV structural remodelling.

INTRODUCTION

Regular and sustained intensive physical activity is associated with a number of electrophysiological,1 structural and functional cardiac adaptations,2 collectively referred to as the ‘athlete’s heart’. It is also well documented that race and sex significantly impact these manifestations of the adult athlete’s heart.3 4 While previous systematic reviews and meta-analyses have detailed the adult athlete’s heart phenotype,5 with some accounting for race and sex,6 data from paediatric (6–18 years) athletic populations are limited to original research, often restricted by inadequate sample sizes and heterogeneity to assess the impact of age, race and sex in tangent.

Sports academies are increasingly used by clubs and governing bodies alike to develop and nurture talented sports stars of the future. Consequently, there is increasing competitiveness, professionalism and training demands placed on the paediatric athlete during the maturational period. The International Olympic Committee, among others, has called for more diligence to safeguard the physiological development of the paediatric athlete.8–10 Performing a cardiac preparticipation evaluation (PPE) within paediatric populations is controversial due to a lack of international consensus as to when, how and who should undertake such examinations.11 12 While data from the USA indicate that paediatric black athletes are particularly susceptible to sudden cardiac death (SCD),13 there is a general lack of understanding as to which factors (eg, physical growth, race and sex) have the potential to increase the likelihood of generating a false-positive diagnosis and unnecessary disqualification from competitive sport. Consequently, the distinction between paediatric athlete’s heart and cardiac pathology associated with SCD is especially important for this population.

Therefore, the primary aim of this systematic review and meta-analysis was to describe the electrocardiographic (ECG), structural and functional manifestations of the paediatric athlete’s heart compared with that of age-matched non-athletes. The secondary aims were to determine the impact of an athlete’s chronological age, race and sex on cardiac remodelling responses to intensive competitive sport.

METHODS

This review was conducted and reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines.14

Literature searching

A systematic search was conducted using six electronic databases: (1) MEDLINE, (2) PubMed, (3) EMBASE, (4) Web of Science, (5) CINAHL and (6) SPORTDiscus. Databases were searched from inception to May 2016. Search terms were mapped to relevant MeSH terms or subject headings under four concepts:

1. ‘Paediatric’
2. ‘Athlete’

Terms within each concept were combined with the Boolean operator ‘OR’, and then concepts were combined with the ‘AND’ operator to produce the search strategy (online supplementary Appendix A). To supplement the electronic database searching, we hand searched reference lists of eligible articles, ePublication lists of key journals, and undertook citation tracking using Google Scholar (online supplementary Appendix B). All identified articles were imported into Endnote X4 for application of selection criteria (Thomson Reuters, California, USA).

Selection criteria
Titles and abstracts of potentially eligible articles were independently screened by two authors (GM and NRR) against the selection criteria. For articles where it was not immediately clear from the title and/or abstract whether they should be included, we obtained the full text for independent screening. Discrepancies were resolved via consensus discussion, with a third reviewer (MGW) consulted if consensus could not be reached.

Inclusion criteria were: (1) data reported for male and/or female competitive athletes, with or without competition to non-athletes; (2) all participants were aged 6–18 years old at the time of assessment; and (3) an original research article published in English language. We defined a competitive athlete as:

‘One who participates in an organised team or individual sport that requires regular competition against others as a central component, places a high premium on excellence and achievement, and requires some form of systematic (and usually intense) training’.

Participants not meeting this definition were classified as non-athletes. Articles were limited to English language owing to translation costs. Articles that did not document athlete age range were excluded because of the risk of including athletes >18 years. If ECG and/or echocardiographic outcome data were not reported, or if professional guidelines for data acquisition were not observed or cited, articles were also excluded.

Risk of bias assessment
We developed a 15-item risk of bias assessment checklist (online supplementary Appendix C), comprising items from Downs and Black’s ‘Assessment of Methodological Quality of Randomised and Non-Randomised Studies’ checklist, and a previously published athlete’s heart meta-analysis checklist. The purpose was to identify articles of low methodological quality that could bias results, with articles achieving ≤50% of total possible appraisal score, excluded from quantitative synthesis. Two reviewers (GM and NRR) independently assessed all included articles. Discrepancies were resolved via consensus discussion, and consistency was measured using an interclass correlation coefficient (ICC2,1).

Data extraction
All ECG and echocardiographic data were extracted by one reviewer (GM) using a predefined extraction form and reviewed by a second reviewer (NRR), with discrepancies resolved by consensus (online supplementary Appendix D). Data extraction included the calculated mean (SD) for continuous data and n for dichotomous data. If insufficient data were reported, corresponding authors were contacted to request additional data.
height velocity = 14; peak weight velocity = 14.3; peak leg length velocity = 14.4; 90% of adult stature = 13.9; 95% of adult stature = 14.9; genital stage IV = 14.6; and pubic hair stage IV = 15.1) and the onset of menarche within females (13.2 years).

Data were combined as per the Cochrane guidelines.30 If data were reported for the same participants in more than one article, the data were extracted from the article with the largest cohort size (with corresponding author’s confirmation). If an article reported multiple follow-ups, data were extracted from the latest visit (ie, longest follow-up). When standard deviation (SD) was not reported, it was imputed from the average SD, only using articles with ≥30 participants. To ensure that results were not subsequently biased, sensitivity analysis was conducted omitting imputed SD data. Statistical heterogeneity was examined using the I² index.31

RESULTS

Literature search

The literature search identified 2030 potentially eligible articles, of which 972 were duplicates. After application of the selection criteria, 43 articles remained for qualitative analysis and 40 remained for quantitative analysis (figure 1).

Risk of bias assessment

There was substantial agreement (71% (95% CI 49% to 84%)) between the reviewers for the risk of bias assessment (see online supplementary Appendix E). Most frequently, discrepancies occurred when assessing ‘professional guidelines’ and ‘missing data’ (77% (95% CI 61% to 88%)). Risk of bias scores ranged from 4 to 13 out of a maximum possible score of 15. No articles reported ‘power analysis’ or ‘intra-observer reliability’, with non-athlete ‘activity levels’ poorly described in 44%. Three articles were excluded from quantitative synthesis due to low methodological quality.

Demographic data

Data from 14 278 athletes (mean age 13.8 ± 1.3 years (range: 6–18)) and 1668 non-athletes (mean age 12.6 ± 0.6 years (7–18)) were extracted from 43 articles. There were no differences in age or BSA between paediatric athletes and non-athletes. Athletes competed in 30 different sports, with football (soccer) predominating (33%). There were more males and Caucasians, but proportionately distributed among both athletes and non-athletes. In two articles, sex was not reported. In 23 articles, race was not documented and in 29 articles maturational status was not reported (table 1).

Data management

Within the 40 articles that were quantitatively synthesised, 2 articles reported overlapping data from a group of 155 athletes, 2 articles reported overlapping data from a cohort of 158 athletes and 2 articles reported overlapping data from a cohort of 900 athletes. Four articles presented multiple follow-up data. Adjustments were made, to account for this in the meta-analysis (supplementary Appendix F).

ECG characteristics

Paediatric athlete versus paediatric non-athlete

Paediatric athletes had a significantly longer PR interval, and a significantly greater frequency of sinus bradycardia, first-degree atrioventricular (AV) block, incomplete right bundle branch block (IRBBB), voltage criteria for LVH and early repolarisation when compared with paediatric non-athletes (table 2). The prevalence of TWI ≥ 1 mm was similar between athletes and non-athletes (6.7% vs 5.9%). However, athletes were 12.7 times more likely to have deep TWI ≥ 2 mm in ≥ 2 contiguous leads (except leads III and aVR) than non-athletes (4.7% vs 0.3%). Athletes were 1.4 times more likely to have anterior TWI (6.5% vs 5.7%) and 1.5 times more likely to have extended
## Table 1  Article characteristics

<table>
<thead>
<tr>
<th>Author, year</th>
<th>n</th>
<th>Chronological ageMean (range)</th>
<th>Biological age</th>
<th>Race(C:B:O)</th>
<th>Sex(M:F)</th>
<th>Sport</th>
<th>n</th>
<th>Chronological ageMean (range)</th>
<th>Biological age</th>
<th>Race(C:B:O)</th>
<th>Sex(M:F)</th>
<th>ECG</th>
<th>ECHOcho</th>
</tr>
</thead>
<tbody>
<tr>
<td>Agrebi et al, 2015</td>
<td>24</td>
<td>13.9 (11–17)</td>
<td>NR</td>
<td>0:0:24</td>
<td>24:0</td>
<td>Handball</td>
<td>21</td>
<td>10.7 (9–12)</td>
<td>Prepubertal</td>
<td>21:0:0</td>
<td>21:0</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>Attisani et al, 2011</td>
<td>1865</td>
<td>13.7 (6–18)</td>
<td>NR</td>
<td>NR</td>
<td>1865:0</td>
<td>Soccer/gymnastics</td>
<td>Y</td>
<td>N</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bartkevičienė, 2015</td>
<td>167</td>
<td>14.8 (12–17)</td>
<td>NR</td>
<td>NR</td>
<td>167:0</td>
<td>Soccer</td>
<td>Y</td>
<td>N</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bessem et al, 2015</td>
<td>193</td>
<td>14.0 (10–19)</td>
<td>NR</td>
<td>134:29:30</td>
<td>193:0</td>
<td>Soccer</td>
<td>Y</td>
<td>N</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calo et al, 2015</td>
<td>2261</td>
<td>12.4 (8–18)</td>
<td>Peripubertal</td>
<td>2261:0:0</td>
<td>2261:0</td>
<td>Swimming</td>
<td>Y</td>
<td>N</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Csajági et al, 2015</td>
<td>18</td>
<td>13.7 (13–15)</td>
<td>Midpubertal</td>
<td>18:0:0</td>
<td>8:7</td>
<td>Swimming</td>
<td>Y</td>
<td>N</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dr. Paolo et al, 2012</td>
<td>216</td>
<td>16.1 (14–18)</td>
<td>NR</td>
<td>63:153:0</td>
<td>216:0</td>
<td>Soccer</td>
<td>Y</td>
<td>N</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dinu et al, 2010</td>
<td>40</td>
<td>12.7 (10–17)</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>Athletics</td>
<td>N</td>
<td>Y</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hauser et al, 2013</td>
<td>26</td>
<td>12.6 (7–17)</td>
<td>NR</td>
<td>NR</td>
<td>18:8</td>
<td>Triathlon</td>
<td>N</td>
<td>Y</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hoogsteen et al, 2011</td>
<td>66</td>
<td>17.5 (17–18)</td>
<td>NR</td>
<td>NR</td>
<td>66:0</td>
<td>Cycling</td>
<td>N</td>
<td>Y</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kinoshita et al, 2015</td>
<td>34</td>
<td>16.5 (16–17)</td>
<td>NR</td>
<td>0:0:34</td>
<td>0:34</td>
<td>Middle-long-distance runners</td>
<td>N</td>
<td>Y</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Koch et al, 2014</td>
<td>343</td>
<td>13 (10–15)</td>
<td>NR</td>
<td>NR</td>
<td>189:154</td>
<td>High-school athletes</td>
<td>Y</td>
<td>N</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Konopka et al, 2015</td>
<td>78</td>
<td>14.3 (12–17)</td>
<td>NR</td>
<td>78:0:0</td>
<td>64:14</td>
<td>Soccer, tennis, rowing</td>
<td>N</td>
<td>Y</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Madeira et al, 2008</td>
<td>21</td>
<td>15.9 (15–16)</td>
<td>NR</td>
<td>NR</td>
<td>21:0</td>
<td>Soccer, swimming</td>
<td>N</td>
<td>Y</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medved et al, 1986</td>
<td>72</td>
<td>10 (8–14)</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>Swimming</td>
<td>72</td>
<td>10 (8–14)</td>
<td>NR</td>
<td>NR</td>
<td>NS</td>
<td>N</td>
<td>Y</td>
</tr>
<tr>
<td>Migliore et al, 2012</td>
<td>2765</td>
<td>13.9 (8–18)</td>
<td>Peripubertal</td>
<td>2765:0:0</td>
<td>1914:851</td>
<td>18 sporting disciplines (invasion games/gymnastics/winter sports/horse riding/racket/tenendurance/combat)</td>
<td>Y</td>
<td>N</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Moafes, 1992</td>
<td>9</td>
<td>16.2 (14–17)</td>
<td>NR</td>
<td>NR</td>
<td>9:0</td>
<td>Basketball</td>
<td>Y</td>
<td>N</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Papadakis et al, 2009</td>
<td>1710</td>
<td>16 (14–18)</td>
<td>Postpubertal</td>
<td>1642:0:0</td>
<td>1414:291</td>
<td>15 sporting disciplines (invasion games/race/tenendurance/combat)</td>
<td>400</td>
<td>16 (14–18)</td>
<td>Postpubertal</td>
<td>385:0:0</td>
<td>330:70</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>Pavlik et al, 2001</td>
<td>165</td>
<td>14.7 (10–18)</td>
<td>NR</td>
<td>165:0:0</td>
<td>165:0</td>
<td>7 sporting disciplines (endurance/invasion games/weightlifting)</td>
<td>22</td>
<td>14.7 (10–18)</td>
<td>NR</td>
<td>22:0:0</td>
<td>22:0</td>
<td>N</td>
<td>Y</td>
</tr>
<tr>
<td>Pelà et al, 2015</td>
<td>138</td>
<td>14.3 (11–17)</td>
<td>NR</td>
<td>96:42:0</td>
<td>138:0</td>
<td>Soccer</td>
<td>Y</td>
<td>Y</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pelà et al, 2016</td>
<td>206</td>
<td>13.8 (11–17)</td>
<td>NR</td>
<td>206:0:0</td>
<td>158:48</td>
<td>Soccer</td>
<td>Y</td>
<td>Y</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Petridis et al, 2004</td>
<td>137</td>
<td>16.6 (15–18)</td>
<td>NR</td>
<td>NR</td>
<td>137:0</td>
<td>Swimming</td>
<td>N</td>
<td>Y</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rowland et al, 1994</td>
<td>10</td>
<td>12.2 (11–13)</td>
<td>Prepubertal</td>
<td>NR</td>
<td>10:0</td>
<td>Middle distance runners</td>
<td>18</td>
<td>11.3 (10–14)</td>
<td>Prepubertal</td>
<td>NR</td>
<td>18:0</td>
<td>Y</td>
<td>Y</td>
</tr>
<tr>
<td>Rowland et al, 1997</td>
<td>9</td>
<td>12.2 (9–15)</td>
<td>Early pubertal</td>
<td>NR</td>
<td>9:0</td>
<td>Cyclists</td>
<td>N</td>
<td>Y</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rowland et al, 2000</td>
<td>8</td>
<td>11.9 (10–13)</td>
<td>Early pubertal</td>
<td>NR</td>
<td>8:0</td>
<td>Cyclists and triathletes</td>
<td>39</td>
<td>12.2 (10–13)</td>
<td>Early pubertal</td>
<td>NR</td>
<td>39:0</td>
<td>N</td>
<td>Y</td>
</tr>
<tr>
<td>Schmied et al, 2009</td>
<td>155</td>
<td>16.4 (14–17)</td>
<td>NR</td>
<td>0:155:0</td>
<td>155:0</td>
<td>Soccer</td>
<td>Y</td>
<td>Y</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Continued
### Table 1

<table>
<thead>
<tr>
<th>Author, year</th>
<th>n</th>
<th>Chronological age range (n, mean)</th>
<th>Biological age (range)</th>
<th>Race (C:B:O)</th>
<th>Sex (M:F)</th>
<th>Sport</th>
<th>n</th>
<th>Chronological age Mean (range)</th>
<th>Biological age</th>
<th>Race (C:B:O)</th>
<th>Sex (M:F)</th>
<th>ECG</th>
<th>ECHOcho</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sharma et al, 1999</td>
<td>70</td>
<td>1000</td>
<td>15.7 (14–18)</td>
<td>Postpubertal</td>
<td>998:8:4</td>
<td>730:180</td>
<td>9 sporting disciplines</td>
<td>70</td>
<td>15.7 (14–18)</td>
<td>Postpubertal</td>
<td>998:8:4</td>
<td>730:180</td>
<td>9 sporting disciplines</td>
</tr>
<tr>
<td>Shi and Selig, 2005</td>
<td>49</td>
<td>13</td>
<td>15.3 (14–16)</td>
<td>NR</td>
<td>NR</td>
<td>13:0</td>
<td>Gymnastics/swimmers</td>
<td>54</td>
<td>15.3 (14–16)</td>
<td>NR</td>
<td>NR</td>
<td>13:0</td>
<td>Gymnastics/swimmers</td>
</tr>
<tr>
<td>Stoner, 1997</td>
<td>64</td>
<td>37</td>
<td>9.9 (7–11)</td>
<td>NR</td>
<td>NR</td>
<td>0:37</td>
<td>Athletics</td>
<td>22</td>
<td>9.9 (7–11)</td>
<td>NR</td>
<td>NR</td>
<td>0:37</td>
<td>Athletics</td>
</tr>
<tr>
<td>Telford et al, 1988</td>
<td>51</td>
<td>85</td>
<td>11.9 (11–12)</td>
<td>NR</td>
<td>NR</td>
<td>48:37</td>
<td>Basketball</td>
<td>31</td>
<td>11.9 (11–12)</td>
<td>NR</td>
<td>NR</td>
<td>48:37</td>
<td>Basketball</td>
</tr>
<tr>
<td>Valente-Dos-Santos et al, 2006</td>
<td>52</td>
<td>73</td>
<td>15.4 (15–17)</td>
<td>Skeletal age</td>
<td>96:0:0</td>
<td>98:0:0</td>
<td>Basketball</td>
<td>73</td>
<td>15.4 (15–17)</td>
<td>Skeletal age</td>
<td>96:0:0</td>
<td>98:0:0</td>
<td>Basketball</td>
</tr>
<tr>
<td>Vasiliauskas et al, 2006</td>
<td>65</td>
<td>62</td>
<td>13.6 (8–17)</td>
<td>NR</td>
<td>NR</td>
<td>62:0</td>
<td>Soccer</td>
<td>47</td>
<td>13.6 (8–17)</td>
<td>NR</td>
<td>NR</td>
<td>62:0</td>
<td>Soccer</td>
</tr>
<tr>
<td>Zdravkovic et al, 2010</td>
<td>66</td>
<td>94</td>
<td>12.9 (12–14)</td>
<td>NR</td>
<td>NR</td>
<td>94:0:0</td>
<td>Soccer</td>
<td>47</td>
<td>12.9 (12–14)</td>
<td>NR</td>
<td>NR</td>
<td>94:0:0</td>
<td>Soccer</td>
</tr>
</tbody>
</table>

B: black; C: Caucasian; F: female; M: male; N: no; NR: not reported; Y: yes.

### Table 2

#### ECG characteristics of paediatric athletes and paediatric non-athletes

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Athletes</th>
<th>Non-athletes</th>
<th>% Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>PR interval (ms)</td>
<td>148 (142–154)*</td>
<td>139 (136–141)</td>
<td>6.1</td>
</tr>
<tr>
<td>QRS duration (ms)</td>
<td>86 (84–88)</td>
<td>83 (79–86)</td>
<td>4</td>
</tr>
<tr>
<td>QTc duration (ms)</td>
<td>396 (391–400)</td>
<td>386 (375–398)</td>
<td>3</td>
</tr>
<tr>
<td>QRS axis (°)</td>
<td>70 (63.1–76.1)</td>
<td>70 (67.9–73.0)</td>
<td>0</td>
</tr>
</tbody>
</table>

#### Group 1 ECG patterns

| Sinus bradycardia (%) | 37.4 (17.6–59.7)† | 19.2 (16.6–21.9) | 2.5 (2.1–3.0) |
| Sinus arrhythmia (%) | 45.8 (35.7–56.0) | 21.0 (18.0–24.0) | 2.5 (2.1–3.0) |
| First-degree AV block (%) | 2.2 (0.8–4.2)† | 0.4 (0.1–1.1) | 4.6 (1.7–12.4) |
| Second-degree AV block (Morbitz type I) (%) | 0.2 (0.1–0.4) | 0.1 (0.0–0.2) | 4.6 (1.7–12.4) |
| Incomplete RBBB (%) | 25.8 (18.2–33.7)† | 7.6 (4.2–12.4) | 4.3 (3.5–5.6) |
| LVH (%) | 35.2 (26.0–45.0) | 24.1 (18.0–30.1) | 4.6 (1.7–12.4) |
| ER (%) | 37.1 (25.6–49.2) | 29.2 (17.2–43.0) | 4.6 (1.7–12.4) |

#### Group 2 ECG patterns

| TWI (≥1 mm) (%) | 6.7 (4.7–8.9) | 5.9 (2.2–11.2) | 1.1 (0.8–1.5) |
| Deep TWI (≥2 mm) (%) | 4.7 (2.3–8.1)† | 0.3 (0.0–1.8) | 4.6 (1.7–12.4) |
| Anterior (%) | 6.5 (2.9–11.3) | 5.7 (2.2–10.6) | 1.2 (0.9–1.5) |
| Extended anterior (%) | 1.4 (0.2–3.5) | 0.9 (0.2–2.2) | 1.2 (0.0–2.2) |
| Inferior (%) | 1.0 (0.3–2) | 0.0 | 1.0 (0.0–2.2) |
| Lateral (%) | 0.3 (0.05–0.6) | 0.0 | 1.0 (0.0–2.2) |
| Inferolateral (%) | 2.0 (1.0–3.3) | 0.0 | 1.0 (0.0–2.2) |
| ST segment depression (%) | 0.03 (0.003–0.08) | 0.0 | 1.0 (0.0–2.2) |
| Abnormal Q waves (%) | 0.1 (0.03–0.2) | 0.0 | 1.0 (0.0–2.2) |
| LAE (%) | 3.5 (0.4–9.5) | 0.0 | 1.0 (0.0–2.2) |
| RAE (%) | 5.9 (0.9–14.8) | 0.0 | 1.0 (0.0–2.2) |
| RAD (%) | 0.4 (1.0–9.0) | 0.0 | 1.0 (0.0–2.2) |
| RVH (%) | 9.8 (7.0–13.0) | 0.0 | 1.0 (0.0–2.2) |
| Ventricular pre-excitation (%) | 0.6 (0.2–1.1) | 0.0 | 1.0 (0.0–2.2) |

Note: Continuation...
Impact of paediatric athlete age

Paediatric athletes ≥14 years had a significantly longer QRS duration and a significantly greater frequency of sinus bradycardia and voltage criteria for LVH than athletes <14 years (table 3). Athletes ≥14 years were 1.3 times more likely to have TWI than athletes <14 years (6.9% vs 5.4%). Athletes <14 years were 1.2 times more likely to have anterior TWI than athletes ≥14 years (6.7% vs 5.4%). Athletes ≥14 years were 3.1 times more likely to have extended anterior TWI (1.7% vs 0.5%), and 15.8 times more likely to have inferolateral TWI (2.5% vs 0.1%) than athletes <14 years.

### Impact of paediatric athlete race

Black paediatric athletes had a significantly greater frequency of sinus bradycardia, first-degree AV block, IRBBB, voltage criteria for LVH and early repolarisation compared with Caucasian athletes (table 4). Black athletes were 4 times more likely to have TWIs (23.4% vs 5.9%) and 2.6 times more likely to have deep TWIs (10.6% vs 4.2%) than Caucasian athletes. Furthermore, black athletes were 2.9 times more likely to have anterior TWI (12.2% vs 4.2%), 36 times more likely to have extended anterior TWI (10.8% vs 0.3%) and 6.5 times more likely to have inferolateral TWI (8.2% vs 1.3%) than Caucasian athletes. Finally, black athletes were 5 times more likely to have abnormal Q waves (0.5% vs 0.1%) and 2.9 times more likely to have left atrial enlargement (LA) (5.7% vs 2.0%) when compared with Caucasian athletes.

### Echocardiographic patterns

Paediatric athletes versus paediatric non-athletes

Athletes had a significantly greater LVId (LVIDd +8.2%), LVID during systole (LVSDd +14.2%), IVSd (IVSd +12.9%), PWTd (PWTd +12.2%), RWT (RWT +5.6%), LVM (+27.6%) and left atrial diameter (LAD) (+12.3%) than non-athletes (table 5). One per cent of athletes (95% CI 0.3 to 2.3, 5 articles; n=4460) had LVH (LV wall thickness >12 mm). LVH was not observed in non-athletes. There were no significant differences in cardiac functional parameters between athletes and non-athletes. Using imputed SDs did not influence the results.

### Impact of age: paediatric athletes versus paediatric non-athletes

Age was a positive predictor of LVId, IVSd, PWTd, RWT and LVM in athletes and non-athletes (p≤0.001). After accounting for age, athletes had greater LVId, IVSd, RWT and LVM (p≤0.05) than non-athletes.

### Impact of paediatric athlete age

Paediatric athletes ≥14 years had a significantly greater LVId (LVIDd +13.5%), LVID during systole (LVSDd +15.9%), IVSd (IVSd +15.2%), PWTd (+21.3%), LVM (+38.7%), aortic root (+14.2%) and LAD (+15.6%) than athletes <14 years (table 6). With the exception of E/A ratio (+13.6% greater in athletes ≥14 years), there were no statistical differences with regard to left ventricle function.

### Impact of paediatric athlete race

Black athletes had a significantly greater PWTd (+12.4%) and LAD (+13.4%) than Caucasian athletes (table 7). Prevalence of LVH (LV wall thickness >12 mm) was 17.1 times greater among black (2 articles, n=319) than Caucasian athletes (3 articles; n=3318) (7.1% vs 0.4%).

### Impact of paediatric athlete sex

Male athletes had a significantly larger IVSd (+9.2%) than female athletes (table 8). Prevalence of LVH was 2.6 times greater in male than female athletes (4 articles, n=2184) (23% vs 9%).

---

**Table 3 ECG characteristics of paediatric athletes: impact of age**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>≥14 years</th>
<th>&lt;14 years</th>
<th>% Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>PR interval (ms)</td>
<td>151 (140–162) (99%)</td>
<td>142 (137–147) (58%)</td>
<td>8 (6%)</td>
</tr>
<tr>
<td>QRS duration (ms)</td>
<td>92 (91–93) * (4; 1991) (59%)</td>
<td>74 (70–82) (3; 77) (88%)</td>
<td>18 (18%)</td>
</tr>
<tr>
<td>QTc duration (ms)</td>
<td>377 (354–400) (5; 4205) (99%)</td>
<td>394 (375–412) (3; 77) (6%)</td>
<td>17 (9%)</td>
</tr>
<tr>
<td>QRS axis* (°)</td>
<td>76 (73–78) (4; 2816) (89%)</td>
<td>74.7 (71.4–88.0) (2; 63) (75%)</td>
<td>2 (2%)</td>
</tr>
</tbody>
</table>

**Group 1 ECG patterns**

<table>
<thead>
<tr>
<th>Sinus bradycardia (%)</th>
<th>61.3 (46.3–75.3)</th>
<th>18.8 (12.1–26.7)</th>
<th>6.6 (4.1–10.7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVH (%)</td>
<td>48.0 (36.4–59.5)</td>
<td>20.7 (9.3–35.1)</td>
<td>3.4 (2.2–5.5)</td>
</tr>
</tbody>
</table>

**Group 2 ECG patterns**

<table>
<thead>
<tr>
<th>TWI (≥1 mm) (%)</th>
<th>6.9 (3.7–10.9)† (6; 5051) (96%)</th>
<th>5.4 (0.2–16.9) (2; 1272) (92%)</th>
<th>1.3 (1.0–1.7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior (%)</td>
<td>5.4 (4.4–9.4)† (7; 6757) (98%)</td>
<td>6.7 (3.0–16.6) (3; 2516) (78%)</td>
<td>1.2 (1.0–1.5)</td>
</tr>
<tr>
<td>Extended anterior (%)</td>
<td>1.7 (0.4–4.0) (4; 3823) (94%)</td>
<td>0.5 (0.1–3.0) (2; 1257) (68%)</td>
<td>3.1 (1.4–6.6)</td>
</tr>
<tr>
<td>Infarolateral (%)</td>
<td>2.5 (1.0–4.6)† (5; 3710) (89%)</td>
<td>0.1 (0.01–0.4) (2; 1272) (90%)</td>
<td>15.8 (3.9–63.9)</td>
</tr>
</tbody>
</table>

---

* Data are presented as mean or percentage (95% CI) (number of articles; number of participants) (heterogeneity).
* p<0.01, significantly greater or more prevalent in athletes≥14 years than in athletes<14 years.
† p<0.001, significantly greater or more prevalent in athletes≥14 years than in athletes<14 years.
‡ p<0.05, significantly greater or more prevalent in athletes<14 years than in athletes≥14 years.
Anterior: V1–V3; extended anterior: V1–V4; inferolateral: leads II–aVF–V4–V6/aVL; NC, non-computable; LVH, left ventricular hypertrophy; TWI, T-wave inversion.

---

**Table 2 Continued**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Athletes</th>
<th>Non-athletes</th>
<th>% Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Short QT interval (%)</td>
<td>0.4 (0.02–1.1) (4; 4108) (81%)</td>
<td>0.3 (3; 834)</td>
<td>NC</td>
</tr>
<tr>
<td>Brugada-like ER (%)</td>
<td>0.2 (0.03–0.4) (5; 7079) (0%)</td>
<td>NC</td>
<td></td>
</tr>
</tbody>
</table>

Data are presented as mean or percentage (95% CI) (number of articles; number of participants) (heterogeneity).
greater among male (5 articles; n=4028) than female athletes (2 articles; n=432) (1.2% vs 0.4%).

**DISCUSSION**

In the first systematic review with meta-analysis investigating the ECG, structural and functional manifestations of the paediatric athlete’s heart, we found that (1) paediatric athletes had a greater prevalence of training-related and training-unrelated ECG changes than non-athletes; (2) while the overall prevalence of TWI remained similar, the distribution and magnitude differed; (3) paediatric athletes had larger echocardiographic-derived LV dimensions than non-athletes, even after accounting for age; (4) paediatric black athletes had increased levels of training and training-unrelated ECG findings (particularly TWI); and finally (5) paediatric black athletes had increased levels of training and alterations in some ECG parameters. Two case reports by Parekh et al. (2013) and Litt et al. (2013) have reported malignant arrhythmias treated with defibrillation in African-American football players aged 13 and 14 years, respectively. The authors hypothesized that these young athletes had increased levels of training and training-unrelated ECG findings (particularly TWI). However, the authors also noted that the athletes had a greater prevalence of training-unrelated ECG findings (particularly TWI) compared to non-athletes. This finding is consistent with the findings of the current study, which found a greater prevalence of TWI in paediatric black athletes compared to non-athletes. The authors suggested that this may be due to the higher prevalence of black athletes in the current study compared to the study by Parekh et al. (2013) and Litt et al. (2013). However, the authors also noted that the black athletes in the current study had increased levels of training compared to the non-athletes, which may contribute to the increased prevalence of TWI.

### Table 4  ECG characteristics of paediatric athletes: impact of race

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Black (10–18 years)</th>
<th>Caucasian (18–18 years)</th>
<th>% Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>PR interval (ms)</td>
<td>161 (146–177) (2; 196) (91.6%)</td>
<td>141 (135–148) (3; 2529) (95%)</td>
<td>12</td>
</tr>
<tr>
<td>QTc duration (ms)</td>
<td>86 (82–90) (3; 525) (94.9%)</td>
<td>92 (88–95) (4; 3232) (98.3%)</td>
<td>-7</td>
</tr>
<tr>
<td>QTc duration (ms)</td>
<td>394 (387–401) (3; 525) (95%)</td>
<td>398 (392–403) (4; 3232) (98.4%)</td>
<td>-1</td>
</tr>
</tbody>
</table>

**Group 1 ECG patterns**

- Sinus bradycardia (%): 38.2 (18.6–60.1) (3; 525) (95%) vs. 29.3 (10.9–52.2) (5; 6197) (99%) (1.5 (1.3–1.8) *p≤0.001, significantly greater or more prevalent in black than Caucasian athletes.
- First-degree AV block (%): 11.4 (6.9–16.5) (2; 483) (65%) vs. 11.0 (2.5–25.0) (4; 5991) (92%) (11.6 (8.0–17.0) *p≤0.05, significantly greater in athletes than non-athletes.
- Incomplete RBBB (%): 22.1 (13.1–32.7) (3; 525) (83%) vs. 21.1 (15.0–27.9) (5; 6197) (97%) (1.1 (0.9–1.3) *p≤0.01, significantly greater in athletes than non-athletes.
- LVH (%): 60.0 (11.0–98.3) (3; 525) (99%) vs. 28.1 (20.2–36.7) (5; 6197) (97%) (3.9 (3.3–4.7) *p≤0.001, significantly greater in athletes than non-athletes.
- ER (%): 74.3 (41.0–96.6) (3; 525) (98%) vs. 31.0 (17.4–46.5) (5; 6197) (99%) (6.4 (5.2–7.9) *p≤0.001, significantly greater in athletes than non-athletes.

**Group 2 ECG patterns**

- TWI (≥1 mm) (%): 23.4 (19.8–27.1) (3; 512) (69%) vs. 5.9 (5.3–6.6) (5; 5263) (71%) (4.0 (3.3–4.8) *p≤0.001, significantly greater in athletes than non-athletes.
- Deep TWI (≥2 mm) (%): 10.6 (5.5–17.2) (3; 512) (73%) vs. 4.2 (0.7–10.4) (4; 3936) (97%) (2.6 (1.9–3.4) *p≤0.001, significantly greater in athletes than non-athletes.
- Anterior (%): 12.2 (6.2–16.9) (3; 512) (43%) vs. 4.2 (3.0–5.6) (4; 3936) (25%) (2.9 (2.2–3.8) *p≤0.001, significantly greater in athletes than non-athletes.
- Anterior extended (%): 10.8 (7.8–14.2) (3; 358) (0%) vs. 0.3 (0.03–0.8) (3; 3298) (25%) (36 (18–71) *p≤0.001, significantly greater in athletes than non-athletes.
- Inferolateral (%): 8.2 (6.0–10.7) (3; 512) (95%) vs. 1.3 (0.3–3.1) (4; 3936) (0%) (6.5 (4.5–9.3) *p≤0.001, significantly greater in athletes than non-athletes.
- Abnormal Q waves (%): 0.5 (0.2–2.0) (3; 525) (95%) vs. 0.1 (0.04–0.3) (4; 6135) (19%) (5.0 (1.3–19.3) *p≤0.001, significantly greater in athletes than non-athletes.
- LVEF (%) 56.0 (53.9–58.1) (10; 1915) (98%) vs. 57.1 (54.6–59.5) (10; 1915) (98%) (-1.1 (−3.6) *p≤0.001, significantly greater in athletes than non-athletes.
- LVM (g) 135.7 (122.2–149.1) (3; 525) (99%) vs. 129.2 (115.9–142.5) (5; 6197) (99%) (6.5 (4.5–9.3) *p≤0.001, significantly greater in athletes than non-athletes.
- ER (%): 74.3 (41.0–96.6) (3; 525) (98%) vs. 31.0 (17.4–46.5) (5; 6197) (99%) (6.4 (5.2–7.9) *p≤0.001, significantly greater in athletes than non-athletes.

**Data are presented as mean or percentage (95% CI) (number of articles; number of participants) (heterogeneity).**

*p≤0.05, significantly greater or more prevalent in black than Caucasian athletes.

**Table 5  Echocardiographic patterns of paediatric athletes and paediatric non-athletes**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Athletes</th>
<th>Non-athletes</th>
<th>% Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVIDd (mm)</td>
<td>47.3 (46.2–48.3)</td>
<td>43.4 (41.7–45.1)</td>
<td>8.2</td>
</tr>
<tr>
<td>LVIDs (mm)</td>
<td>29.6 (28.4–30.8)</td>
<td>25.4 (24.8–26.0)</td>
<td>14.2</td>
</tr>
<tr>
<td>IVSd (mm)</td>
<td>8.5 (8.2–8.8)</td>
<td>7.4 (7.1–7.8)</td>
<td>12.9</td>
</tr>
<tr>
<td>LVOTd (mm)</td>
<td>8.2 (7.8–8.6)</td>
<td>7.2 (6.6–7.8)</td>
<td>12.2</td>
</tr>
<tr>
<td>RVWT (mm)</td>
<td>0.36 (0.34–0.37)</td>
<td>0.34 (0.33–0.35)</td>
<td>5.6</td>
</tr>
<tr>
<td>LVM (g)</td>
<td>135.7 (122.4–149.1)</td>
<td>98.2 (84.6–111.8)</td>
<td>27.6</td>
</tr>
</tbody>
</table>

**Data are mean (95% CI) (number of studies; number of participants) (heterogeneity).**

*p≤0.001, significantly greater in athletes than non-athletes.

**ECC characteristics of the paediatric athlete**

This study confirms that regular and prolonged physical training is associated with a high prevalence of bradycardia, repolarisation changes, atrial enlargement and ventricular hypertrophy in paediatric athletes. However, the magnitude, prevalence and distribution of such changes are dependent on the chronological age of the paediatric athlete. Similar to adult athletes, race impacted ECG remodelling in the paediatric athlete. 

---

Black pediatric athletes had significantly more training-related changes, anterior, extended anterior, inferolateral and deep TWIs, in addition to Q waves and LAE compared with Caucasian athletes.

**TWI in the paediatric athlete: impact of age and race**

Inverted T waves may represent the only sign of an inherited heart muscle disease even in the absence of any other features or before structural changes in the heart can be detected.63 Yet, until complete formation of adult ventricular mass, TWIs may persist across leads V1–V3 within the paediatric population, owing to right ventricular dominance.72 Our findings from over 9000 pediatric athletes and over 800 pediatric non-athletes support this notion, with a relatively high, but similar prevalence of anterior TWI (V1-V3) observed in both athletes and non-athletes (6.5% vs 5.7%), respectively, suggesting that this is a maturational trait largely not resultant of athletic training. The slightly higher prevalence of anterior TWI in athletes versus non-athletes also suggests that regular exercise may exacerbate or prolong the presence of juvenile TWI. Nevertheless, pediatric athletes were 12.7 times more likely to present with deep TWI (≥2 mm) than non-athletes. Deep TWI (≥2 mm) in the precordial leads is a major concern as these ECG alterations are a recognised manifestation of hypertrophic cardiomyopathy and arrhythmogenic right ventricular cardiomyopathy.73

TWIs are uncommon among adult Caucasian athletes. Conversely, African/Afro–Caribbean black athletes have a higher prevalence of TWI as well as more striking repolarisation changes and magnitude of voltage criteria for LVH than Caucasian athletes of similar age and size participating in identical sports.14,74 Similar to their adult counterparts,2 we found that black pediatric athletes are 4 times more likely to exhibit any TWI and 36 times more likely to exhibit extended ante-
When is anterior TWI normal?
Recently updated international recommendations for 12-lead ECG interpretations in athletes recommends that TWI ≥1 mm in depth in two contiguous anterior leads (V2–V4) is abnormal (with the exception of TWI confined to leads V1–V4 in black athletes and leads V1–V3 in all athletes aged <16 years) and should prompt further evaluation for underlying structural heart disease. Our data support this recommendation, demonstrating a significantly reduced prevalence of anterior TWI (V1–V3) in athletes ≥14 years likely as a consequence of maturation. Based on current evidence, TWI in the anterior leads (V1–V3) in paediatric athletes <14 years of age (or prepubertal athletes) should not prompt further evaluation in the absence of symptoms, signs or a family history of cardiac disease.

Our data also support the observation that, like their adult counterparts, paediatric black athletes were 3 times more likely to have anterior TWI (V1–V3) and 36 times more likely to have extended anterior TWI (V1–V4) when compared with Caucasians. In adult black athletes, it is recognised that anterior TWI is a normal variant when preceded by J-point elevation and convex ST segment elevation, unlike in arrhythmogenic right ventricular cardiomyopathy where the J point and/or ST segment is usually isoelectric or depressed prior to TWI. Appreciating the J point and preceding ST segment may help differentiate between physiological adaptation and cardiomyopathy in athletes with anterior TWI affecting leads V3 and/or V4, and may prove to be especially useful in athletes of mixed race. A recent study compared black and Caucasian healthy athletes against hypertrophic cardiomyopathy and arrhythmogenic right ventricular cardiomyopathy patients, all of whom had anterior TWI. Within athletes, the combination of J-point elevation ≥1 mm and TWI confined to leads V1–V4 excluded hypertrophic cardiomyopathy or arrhythmogenic right ventricular cardiomyopathy with 100% negative predictive value, regardless of race. Conversely, anterior TWI associated with minimal or absent J-point elevation (<1 mm) may reflect a cardiomyopathy. Such detailed investigations have yet to be extended to the paediatric athletic population.

Inferior and/or lateral TWI warrants investigation
We were surprised by the high prevalence of inferolateral TWI in both black (8.5%) and Caucasian (1.3%) paediatric athletes. It is unlikely that all such athletes harbour a sinister cardiomyopathy and may represent a racial variant in black athletes. Despite this, lateral lead TWI should be viewed with caution. We recently investigated 155 athletes presenting with pathological TWI with clinical examination, ECG, echocardiography, exercise testing, 24 hours Holter ECG and cardiac magnetic resonance. Cardiac disease was established in 44.5% of athletes (81% hypertrophic cardiomyopathy). Inferior and/or lateral TWI were the most commonly observed ECG abnormalities (83.9%) and were largely isolated findings without other ECG abnormalities (43.2%). In our experience, regardless of an increased frequency after 14 years and a higher prevalence in paediatric black athletes, inferolateral TWI should be considered pathological in all cases until proven otherwise. While exclusion from competitive sport is not warranted in the asymptomatic paediatric athlete without a family history of SCD and normal secondary examinations, annual follow-up is essential to ascertain possible disease expression.

LV morphology of the paediatric athlete
While most adult athletes have LV structural changes that are considered physiological, there are a small proportion who develop pronounced morphological changes that overlap with phenotypic expressions of cardiac pathology associated with SCD. Several groups have produced algorithms to aid in this differentiation. Data for these algorithms primarily derive from 5 large echocardiographic studies examining 5053 elite, predominately male adult athletes; 134 (2.7%) had a maximal wall thickness ≥12 mm (of which 27 (0.5%) athletes had a maximum wall thickness of ≥13 mm). In absolute terms and regardless of an athlete’s BSA, the upper limit of physiological hypertrophy for adult male athletes is considered ≥13 mm for maximal wall thickness and ≥65 mm for LVId.

Despite undergoing significant changes in anthropometry during maturation, paediatric athletes have significantly larger cardiac diameters, wall thicknesses and LVM than non-athletes even after adjusting for age. From 4460 paediatric athletes analysed, just 1.1% presented with a maximal wall thickness ≥12 mm, although a maximal wall thickness of 15 mm was documented in one study. A pooled mean LVId of 47 mm (<14 years: 44.2 mm vs ≥14 years: 51 mm) is similar to upper limits previously observed among patients with paediatric hypertrophic cardiomyopathy (48 mm). Thus, such adult upper limit criteria may not be applicable to the paediatric athlete. Regardless of race, values above these should be viewed with suspicion in paediatric athletes, particularly if the athlete also presents with cardiac symptoms, a family history of SCD and/or an abnormal ECG. Given the widely recognised impact of chronological age and somatic growth on paediatric echocardiographic variables, it is our suggestion that Z scores (which account for the effects of body size and chronological age) are instead used for differential diagnosis when normative data are available, as previously suggested within paediatric-specific echocardiographic guidelines.

Impact of chronological age on LV remodelling
Cardiac enlargement increased with chronological age, as demonstrated by our meta-regression as well as by others, and helps to explain the heterogeneity observed within this dataset. After accounting for age (using meta-regression), paediatric athletes had greater LV morphology than paediatric non-athletes, demonstrating the potent stimulus exercise has on cardiac structure. These changes appeared to be exaggerated during the pubertal growth stage, suggesting a potential role of hormonal factors in cardiac remodelling. We recognise that while chronological age is a linear factor, growth and maturation are not, and thus maturational status for children of the same chronological age can differ dramatically. The assessment of maturational status was conducted among only 14 of the 43 (33%) articles included for qualitative synthesis, and relied largely on assessment by the Tanner Scale (79%), regarded to be inappropriate by many due to obvious child protection concerns. In our experience, clinical interpretation of cardiac PPE data should be governed by biological age rather than chronological age. According to the International Olympic Committee consensus statement on youth athletic development, skeletal age is the most useful estimate of maturity status and can be used from childhood into late adolescence. However, this can only be confirmed by radiological hand–wrist imaging. Since this is not widely available in most cardiological units, alternative simple measures such as percentage of predicted mature (adult) height at the time of observation may provide an estimate of
Review

maturity status. However, care is warranted, as (1) predicted mature (adult) height only demonstrates moderate concordance with classifications of maturity status, based on skeletal age, and (2) historical height data of the patient is required to rule out sudden growth spurts.

Impact of race on LV remodelling

Data from the USA indicate that pediatric black athletes are particularly susceptible to SCD, and therefore, the distinction between athlete’s heart and cardiac pathology is of particular relevance in this group. Consistent with previous observations in adults, we found that pediatric black athletes had increased LVH in response to chronic training loads compared with Caucasian athletes. This change is consistent with a concentric remodelling pattern. Furthermore, the likelihood of LVH was 17.1 times greater among black when compared with Caucasian athletes. We speculate that these race-specific manifestations of the athlete’s heart are the result of haemodynamic influences, specifically greater peripheral vascular resistance and a smaller nocturnal decline in blood pressure.

Impact of sex on LV remodelling

The last 3 decades have witnessed an exponential rise in the number of females participating in high-level competitive sport. Consistent with observations among adults, we found a reduced LVH response to chronic training loads in female athletes compared with males. This might be due to hormonal differences and lower testosterone concentrations. However, the relative differences of sex across maturational years are yet to be fully elucidated among paediatric athletes. Females reach complete pubertal development at an earlier chronological age and thus we may expect such relative differences between female and male athletes to be smaller during the early stages of pubertal development.

Limitations

A high statistical heterogeneity ($I^2$) was observed; this may be because it was not possible to stratify data according to biological age, race or sex due to inconsistent methodology and designs implemented within the observational studies included. Because of this, a random-effects meta-analysis model was adopted to provide a more conservative pooled estimate. Activity levels of our non-athlete cohort are unknown and thus they may not actually be sedentary; however, in all cases, participants did not meet classification criteria for a competitive athlete.

While we used the 2010 ESC recommendations for interpretation of the 12-lead ECG, at the time of publication, it was not intended to be used in athletes ≤12 years old. We recently observed that the 2014 ‘Refined Criteria’ for ECG interpretation in athletes outperformed both the 2013 Seattle Criteria and the 2010 ESC recommendations, by significantly reducing the number of false-positive ECGs in Arabic, Black and Caucasian adult athletes while maintaining 100% sensitivity for serious pathological cardiac remodelling. Again, however, all three ECG criteria are only applicable for adult athletes and not paediatric athletes. Thus, for paediatric cardiac PPEs, the attending cardiologist or sports medicine physician is left with the conundrum of which criteria should be used for ECG interpretation. Recently, published International recommendations for ECG interpretation in athletes do account for age and race, respectively. TWI in the anterior leads (V1–V3) in adolescent athletes <16 years of age (or prepubertal athletes) and black adult athletes with J-point elevation and convex ST segment elevation followed by TWI in V2–V4 would now not prompt further evaluation in the absence of symptoms, signs or a family history of cardiac disease. But in most non-black athletes age ≥16 years, anterior TWI beyond lead V2 would prompt further evaluation given the potential overlap with arrhythmogenic right ventricular cardiomyopathy.

Finally, echocardiographic data were largely limited to left ventricle structural variables, owing to insufficient data available for synthesis. Such limitations highlight the importance of further research in the paediatric athlete extending to other chambers of the heart, and beyond load-dependent measurements of cardiac function (ejection fraction or fractional shortening) towards tissue Doppler imaging and myocardial deformation (strain) imaging.

Conclusion

Similar to adult athletes, pediatric athletes had a greater prevalence of training-related and training-unrelated ECG changes than non-athletes. Significant cardiac remodelling in paediatric athletes occurs both before and during their ‘maturational years’, with race and sex significantly impacting on the pattern of remodelling observed. The results demonstrate the importance of adjusting for age when assessing LV morphology in paediatric athletes, while consideration of an athlete’s race and sex is further required when differentiating between physiological and pathological cardiac remodelling.

What is already known?

- Chronic training loads are associated with a number of electrophysiological, structural and functional cardiac adaptations in adult athletes.
- Race and sex significantly impact on the cardiac remodelling of the adult athlete’s heart.
- Paediatric athletes undergo significant growth and maturational changes, but unlike known musculoskeletal changes, there is limited information regarding how the heart may adapt to training before, during and after puberty.

What are the new findings?

- Paediatric athletes were up to 13 times more likely to have deep T-wave inversion (TWI) (>2 mm) than age-matched non-athletes.
- Paediatric athletes ≥14 years of age were up to 16 times more likely to have inferolateral TWI (warranting further investigation) than athletes <14 years.
- Paediatric black athletes were up to 36 times more likely to have extended anterior TWI (leads V1–V4) than Caucasians.
- Even after accounting for age, left ventricular structural parameters were larger among paediatric athletes than paediatric non-athletes.

Contributors

GM, NRR, CLA and MGW contributed to the conception and design of the review. GM applied the search strategy. GM, NRR and MGW applied the selection criteria. GM and NRR completed assessment of risk of bias. GM, CLA and AF analysed the data. All authors contributed to data interpretation. GM and MGW wrote the manuscript. NRR, CLA, AF, GEP, VW, CA, KPG and DO critically revised the manuscript for important intellectual content. GM and MGW are responsible for the overall content as guarantors.

Competing interests

None declared.

Open Access This is an Open Access article distributed in accordance with the Creative Commons Attribution Non Commercial (CC BY-NC 4.0) license, which permits others to distribute, remix, adapt, build upon this work non-commercially.

REFERENCES


11. Vetter VL. Electrocardiographic screening of all infants, children, and teenagers should be performed. Circulation 2014;130:688–97.


20. Sokolow L, Lyon TR. The ventricular complex in left ventricular hypertrophy as obtained by unipolar precordial and limb leads. Am Heart J 1940;37:161–86.


64 Sterner JE. Cardiac dimensions gymasts and swimmers. Biol Sport 1997;14:115–25.
Electrical and structural adaptations of the paediatric athlete’s heart: a systematic review with meta-analysis

Gavin McClean, Nathan R Riding, Clare L Ardern, Abdulaziz Farooq, Guido E Pieles, Victoria Watt, Carmen Adamuz, Keith P George, David Oxborough and Mathew G Wilson

Br J Sports Med published online March 31, 2017

Updated information and services can be found at: http://bjsm.bmj.com/content/early/2017/03/30/bjsports-2016-097052

These include:

References
This article cites 107 articles, 38 of which you can access for free at: http://bjsm.bmj.com/content/early/2017/03/30/bjsports-2016-097052#ref-list-1

Open Access
This is an Open Access article distributed in accordance with the Creative Commons Attribution Non Commercial (CC BY-NC 4.0) license, which permits others to distribute, remix, adapt, build upon this work non-commercially, and license their derivative works on different terms, provided the original work is properly cited and the use is non-commercial. See: http://creativecommons.org/licenses/by-nc/4.0/

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to: http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to: http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to: http://group.bmj.com/subscribe/