Improving the interpretation of the athlete’s electrocardiogram

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This editorial refers to ‘Clinical significance of electrocardiographic right ventricular hypertrophy in athletes: comparison with arrhythmogenic right ventricular cardiomyopathy and pulmonary hypertension’1, by A. Zaidi et al., on page 3649 and ‘Should axis deviation or atrial enlargement be categorised as abnormal in young athletes? The athlete’s electrocardiogram: time for re-appraisal of markers of pathology’2, by S. Gati et al., on page 3641.

Pre-participation screening based on a 12-lead electrocardiogram (ECG) is effective in identifying athletes with potentially lethal cardiovascular disease and saves lives by disqualifying them from competitive sports activity.1–3 However, concerns have been raised regarding the cost-effectiveness of pre-participation ECG screening which are predominantly related to the high number of false positives, mandating further and expensive investigations to exclude an underlying heart disease.4 Cardiac adaptation and remodelling resulting from sustained athletic training (‘athlete’s heart’) induce ECG changes (Figure 1) that may mimic those seen in heart muscle diseases causing sudden cardiac death (SCD) in the young, such as hypertrophic cardiomyopathy (HCM).5 Recent consensus documents have provided modern criteria for interpretation of the athlete’s ECG, which were based on a better definition of physiological vs. abnormal ECG changes.6–9 According to the European Society of Cardiology (ESC) consensus document,7 common ECG changes due to cardiac adaptation to physical exertion (Group 1) should not cause alarm and the athlete should be allowed to participate in competitive sports without additional evaluation. On the other hand, further diagnostic work-up should be reserved for the subset of athletes with uncommon and not sports-related ECG changes (Group 2), which potentially reflect an underlying heart disease and an increased risk of SCD. This modern approach to ECG interpretation has proven potentially reflect an underlying heart disease and an increased risk of SCD. This modern approach to ECG interpretation has proven to lower the traditionally high number of false positives and to reduce unnecessary and expensive investigations, predominantly among the large cohort of athletes with a physiological increase of QRS voltages.7,8,10 However, whether and to what extent the increased specificity alters the ECG sensitivity for detection of potentially dangerous cardiovascular conditions remain to be established. Ideally, a correct interpretation of an ECG parameter should aim to reduce the number of false-positive results (‘save money’) but to maintain screening sensitivity to avoid missing potentially dangerous cardiovascular conditions (‘save lives’).11 This can be achieved by assessing the true prevalence of specific ECG abnormalities in athletes vs. diseases that predispose to SCD. Therefore, new studies providing data on the relative prevalence of ECG changes, either in isolation or in combination, in the athlete’s heart vs. heart diseases are more than welcome.

A recent study by Calore et al.12 compared the ECG tracings of 247 HCM patients with those of 133 highly trained athletes with augmented left ventricular (LV) mass and found that ECG abnormalities of HCM and athletes’ hearts overlap marginally. Specifically, the pattern of isolated increase of QRS voltages (Sokolow–Lyon index: SV1 + RV5 or RV6, whichever is larger, > 35 mm) was found in only 2% of HCM patients, compared with in 40% of athletes. The combination of an isolated increase of QRS voltages and non-voltage criteria (i.e. atrial enlargement, QRS axis deviation, complete bundle branch block, ST-segment or T-wave abnormalities, and pathological Q wave) showed a sensitivity of 96% and a specificity of 52% in identifying HCM. When the pattern of isolated increase of QRS voltages was interpreted as normal, non-voltage ECG criteria alone resulted in a substantial increase of specificity to 92%, with an expected considerably favourable impact on screening cost-effectiveness, and a slight reduction of sensitivity to 93%. The most frequent ECG abnormalities associated with HCM were repolarization abnormalities, such as T-wave inversion and/or ST-segment depression (82% of cases), while the vast majority of highly trained athletes either showed a normal ECG or exhibited pure voltage criteria of LV hypertrophy, in the absence of other depolarization/repolarization abnormalities.

Now Zaidi et al.13 and Gati et al.14 have provided new and important data on the relative prevalence of some Group 2 ECG abnormalities

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in healthy athletes compared with patients with cardiomyopathy. The first study\textsuperscript{13} demonstrated that voltage-based criteria for right ventricular (RV) hypertrophy (i.e. Sokolow–Lyon index = RV\textsubscript{1} + SV\textsubscript{5} or SV\textsubscript{6} $>$ 10.5 mm) are not so uncommon in athletes (12%) and reflect the electrical manifestations of RV adaptation to sustained athletic training, similarly to physiological voltage criteria for LV hypertrophy. Voltage criteria for RV hypertrophy were more common in athletes than in controls and were identified almost exclusively in male athletes, who show more profound physiological cardiac adaptations than their female counterparts; moreover, voltage RV hypertrophy (in isolation or in combination with right axis deviation $>$ 120\textdegree) was not associated with cardiac pathology in asymptomatic athletes. In comparison, none of the cardiomyopathic or pulmonary hypertensive study patients exhibited voltage RV hypertrophy without additional ECG abnormalities. On the basis of these findings, it is reasonable to conclude, in agreement with the authors, that isolated voltage RV hypertrophy is representative of the normal spectrum of physiological cardiac adaptations resulting from regular exercise training. The second study\textsuperscript{14} reported a low prevalence of axis deviation and atrial enlargement in isolation both in athletes and in patients with HCM, i.e. left axis deviation (LAD) 1.5\% vs. 1.7\%, respectively; and left atrial enlargement (LAE) 2.1\% vs. 3.5\%, respectively. Exclusion of axis deviation and atrial enlargement in isolation from the Group 2 ECG abnormality would increase the specificity for HCM from 90\% to 94\%, with a reduction in the sensitivity from 91\% to 89.5\%.

The findings of this latter study largely overlap with those found in the subanalysis of the studies by Migliore et al.\textsuperscript{5} and Calore et al.\textsuperscript{12} on the relative prevalence of isolated LAD and LAE in two Italian athletic populations, which included, respectively, 2765 adolescents undergoing pre-participation screening (mean age = 14 years) and 133 highly trained athletes with echocardiographically augmented LV mass (mean age = 27 years), vs. 247 HCM patients. The prevalence of isolated LAD in the two populations of athletes was 0.5\% and 2\%, respectively, and the prevalence of isolated LAE 0\% and 1\%, respectively; in comparison, among HCM patients, the prevalence of LAD and LAE in isolation was 1\% and 2\%, respectively, and that of LAD and LAE in association with voltage criteria for LV hypertrophy 0.5\% and 1\%. Accordingly, in the subanalysis of the study of Calore et al., exclusion of LAD and LAE (either in isolation or in association with voltage criteria for LV hypertrophy) from the Group 2 ECG abnormalities would increase the specificity for HCM from 52\% to 55\%, with a reduction in the sensitivity from 96\% to 92\%.

These new and consistent data from the UK and Italy raise the need for a revision of the criteria for ECG interpretation in the athlete’s heart, also taking into account the true prevalence of an isolated

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**Figure 1** Physiological basis of electrocardiogram (ECG) changes in the athlete’s heart. Participation in sports activity and regular physical training are associated with adaptive morphofunctional and neuroautonomic cardiovascular remodelling (‘athletes heart’) which results in physiological ECG changes. Electrocardiographic and echocardiographic features of a 31-year-old triathlete. (A) ECG recordings (V1 to V3 leads) showing sinus bradycardia (45 bpm), incomplete right bundle branch block (RBBB), and early repolarization. (B) Long-axis parasternal and four-chamber views showing an increase of both left and right ventricular dimension (left ventricular end-diastolic dimension = 84 mL/m\textsuperscript{2} and right ventricular end-diastolic dimension = 77 mL/m\textsuperscript{2} with mild hypertrophy of the anterior ventricular septum (12 mm) and posterior free wall (11 mm), and an augmented left ventricular (LV) mass index (168 g/m\textsuperscript{2}). (C) Doppler diastolic waveform consistent with a normal pattern of LV filling. AV, atrioventricular; RV, right ventricular.
ECG abnormality in a healthy population of athletes vs. cardiomyopathic patients. A diagram illustrating a proposed revision of the classification of ECG abnormalities in the athlete is reported in (Figure 2). By analogy with isolated QRS voltage criteria for LV hypertrophy, voltage RV hypertrophy is added to Group 1 which includes training-related ECG abnormalities, much more common in the athlete’s heart than in cardiomyopathy. The ECG abnormalities of Group 2 (uncommon and unrelated to training) are further divided into ‘major’, i.e. those that are uncommon in the athlete’s heart but frequent in cardiomyopathy (e.g. T-wave inversion), and ‘minor’, i.e. those that are infrequent in both athletes and patients with cardiomyopathy (e.g. atrial enlargement).

According to ESC recommendations,7 ‘major’ Group 2 ECG abnormalities, which are often observed in patients with heart disease, should trigger additional clinical work-up of the athlete to exclude an underlying pathological disorder. On the other hand, Group 1 ECG abnormalities, which are considered an electrical marker of physiological heart adaptation to physical exercise, do not require further evaluation in the absence of symptoms and/or a positive family history. In this regard, the small loss of sensitivity by interpreting the ECG pattern of isolated QRS voltage criteria for LV hypertrophy as normal can be reasonably justified in the context of a mass screening because of the concomitant substantial improvement of specificity (by 50%), with ensuing enormous cost savings.12

Recommendations for management of athletes showing the new subset of ‘minor’ Group 2 ECG changes, such as atrial enlargement and axis deviation, are open to debate. The authors from the UK suggest excluding these patterns from the abnormal category of ECG changes which warrant further cardiovascular evaluation, with the aim of improving the specificity and cost-effectiveness of the screening process.14 However, we should recognize that ignoring

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**Figure 2** Proposed revision of criteria for electrocardiogram (ECG) interpretation in the athlete. Group 1 includes training-related ECG abnormalities which are more common in the athlete’s heart than in cardiomyopathy. Uncommon and training-unrelated ECG abnormalities of Group 2 are divided into ‘major’, i.e. uncommon in the athlete’s heart but frequent in cardiomyopathy, and ‘minor’, i.e. infrequent in both athletes and patients with cardiomyopathy (see text for further explanation). †Sokolow–Lyon voltage criteria. AV, atrioventricular; LBBB, left bundle branch block; LV, left ventricular; RBBB, right bundle branch block; RV, right ventricular.
such ‘minor’ ECG abnormalities at pre-participation screening, while impacting the specificity marginally (by 3—4%), leads to a parallel decrease of ECG screening sensitivity (by 2—4%) that is a not negligible reduction of the power to identify potentially lethal cardiomyopathy in the athlete. In assessing the double-edged duality of ECG preparticipation screening—cost vs. effectiveness—it is critical to consider that prevention is not about saving money, it is about saving lives.4,11,15

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References