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EDITORIAL

New International Guidelines for the Interpretation of the Electrocardiograph in Athletes: a "Traffic Light" Tool for Maximising Diagnostic Specificity

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While sudden cardiac death (SCD) is devastating in all age groups, the death of a young athlete, especially those with a popular reputation or during a high-profile sporting event, is greatly magnified in the media. The incidence of sudden death in young athletes (≤35 years of age) is approximately 0.6–3.6 per 100,000 population per year, and most deaths are due to cardiovascular causes [1–3]. The cardiovascular screening of competitive athletes is mandated in some countries and recommended in others. Screening comprises a history and examination in the USA, while the addition of a 12-lead electrocardiograph (ECG) is recommended in most other countries and also by sporting bodies, such as the International Olympics Committee and the International bodies representing cycling, rowing, tennis and swimming, among others [4,5]. The aim of screening is to identify cardiomyopathies and channelopathies that may be asymptomatic but represent a potential trigger for exercise-related deaths. The most common underlying aetiology varies between reports from different countries; primary causes are hypertrophic cardiomyopathy (HCM; dominant in United States cohort [3,6]) or idiopathic left ventricular hypertrophy [6], arrhythmogenic right ventricular dysplasia (ARVC; dominant in Italian cohort [2]), dilated cardiomyopathy, myocarditis, channelopathies in the presence of a structural normal heart [7], aortic dissection and coronary artery disease (including coronary anomalies) [6].

Although prevention of SCD in the young is a major public health priority, the optimal strategy is an issue of considerable debate [8,9]. In a sport-loving country such as Australia, where sport participation is highest in our massive rural expanses and medical access is limited, athlete screening will require significant financial and infrastructure commitment [9]. Exercise is a known trigger and can unmask occult cardiac disease to precipitate SCD [8]. Thus, restriction from strenuous exercise of those athletes with structural heart disease may be a prudent intervention to prevent SCD, although the efficacy of this strategy is untested. Other interventions, including beta blockers for those with long-QT syndrome or defibrillator implantation in those with structural heart disease and high-risk features, may also have the potential to save lives. On the other hand, only a minority of athletes excluded from competitive sport would have been expected to suffer an event. For example, hypertrophic cardiomyopathy is associated with an increased risk of SCD but the majority of athletes diagnosed with HCM have an apical variant with few high-risk features [10]. Thus, the ratio of athletes excluded per life saved is likely to be high. Furthermore, sporting exclusion can have major psychosocial and financial repercussions for an aspiring athlete. As a result of these compelling arguments both for and against screening, debate rages as to whether screening should be recommended and what is the best strategy. It is unlikely that a randomised trial comparing outcomes in screened and unscreened populations will ever be conducted on sufficient scale and so hard answers are likely to remain elusive.

What is not contentious is that IF screening is to be undertaken then it should be done well. Part of the difficulty in accurately identifying structural heart disease is providing

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the tools and knowledge platform sufficient to minimise false diagnoses. Both false positive diagnoses (incorrectly labelling a healthy athlete with pathology) and false negative diagnoses (failing to identify signs of underlying pathology) can cause great harm. Evidence suggests that screening with history and examination alone has both poor specificity and sensitivity [8,11]; and, it could be argued that screening should either include a 12-lead ECG or not be done at all. In regard to interpretation of the 12-lead ECG, a European task force lead by Domenico Corrado of Italy recognised that an athlete's ECG required a different interpretation framework from that of the general population because of the high prevalence of ''abnormalities'' as a result of healthy physiological adaptation and body habitus common in athletes. The task force nominated a number of features (including sinus bradycardia and increased QRS voltages, amongst others) that were common, ''training related'', and almost always benign [12]. This was the first major step forward in increasing the specificity of an athlete's ECG interpretation, and a number of refinements have been made since to further reduce the proportion of athletes that would be required to undergo further testing on the basis of ECG characteristics.

An estimated 60% of the disorders associated with sudden cardiac arrest or death in young individuals may have detectable ECG abnormalities [13]. Although the ECG is an objective tool, it is subject to variable interpretation, even among cardiologists [14]. The optimal false positive rates are achieved when there are clear guidelines for the recognition of normal variants in athletes which may otherwise be perceived as abnormal, resulting in a cascade of cardiac investigations and, potentially inappropriate exclusion from sport.

In 2016, an international panel of experts assembled in Seattle, Washington, USA, under the chair of Jonathan Drezner for a weekend of debate and discussion aimed at creating the clearest and most accurate iteration of athletic ECG guidelines to date. It was noted that up to 30% of athletes were being identified as 'abnormal' on existing ECG interpretation criteria [15], and this served as an enormous barrier to the implementation of screening. Thus, a stated intention of the committee was to set pragmatic cut-offs that favoured specificity over sensitivity using the rapidly accumulating databases of athlete screening programs to test the impact of changes to definitions. The result was a comprehensive document published in the *Journal of the American College of Cardiology* [16] and the *European Heart Journal* [17], with a more comprehensive version in the *British Journal of Sports Medicine* [18].

As compared with previous criteria, the 2017 ''International criteria'' provide stricter criteria for most ECG abnormalities. For example, recognising that T-wave inversion in V1 and V2 is very common among endurance athletes [15], the new criteria require T-wave inversion to extend to V3 before being considered ''positive'' and a trigger for further investigation. The criteria recognise ethnic variability by including the common pattern of T-wave inversion in V1–V4 with preceding J-point elevation to be considered a normal variant in black athletes. Similarly, there is a relaxation of the long-QT cut-offs, and consideration of short-QT is no longer included as it was considered too rare to warrant consideration in a screening program. Some criteria that were previously considered abnormal have been moved to an in-between category of ''borderline findings'' that are considered abnormal and a trigger for further evaluation *only* if there are multiple criteria present on the ECG.

Thus, there is a ''traffic light'' approach to the ECG, succinctly summarised in Figure 1. Electrocardiograph features in the green are normal for an athlete, those in the red should be considered abnormal and prompt further evaluation while a ''yellow light'' can be normal in isolation or abnormal if there are multiple present (e.g., right axis deviation and right bundle branch block).

It is important to recognise that the ECG will not be able to detect anomalous coronary arteries, premature coronary atherosclerosis, and aortopathies which are important causes of sudden cardiac arrest/death in athletes, and may also miss early stages of cardiomyopathies. Although the aim of the International Criteria is to improve specificity while maintaining sensitivity (i.e., not to miss any underlying pathology), it stands to reason that some pathology will be overlooked as a result of making the criteria for an abnormal ECG more stringent. For example, as many as 11% of patients with arrhythmogenic right ventricular cardiomyopathy have T-wave inversion confined to V1 and V2 [19]. These patients would be missed with the new criteria. However, the expert panel in Seattle deemed that this was an acceptable compromise given that these ''abnormalities'' are extremely common amongst athlete populations.

There have already been some attempts to validate the new International Criteria. Professor Sanjay Sharma, one of the most active investigators leading the knowledge base underpinning athlete ECG interpretation, assessed the ECGs of 4,925 athletes according to four different criteria [20]. Relative to the European Society of Cardiology (ESC), Seattle and Refined criteria, the new International Criteria reduced the number of ''abnormal'' ECGs by 86%, 50% and 30% respectively (all $p < 0.0001$). All 15 athletes with pathology (6 with hypertrophic cardiomyopathy, 3 with long-QT and 6 with accessory pathway) were identified with each of the diagnostic criteria, suggesting that there was no decrement in sensitivity associated with the improvement in specificity. The authors estimated a marked reduction in the costs associated with each serious diagnosis, from $35,993 by the 2010 ESC criteria to $26,405 with the 2017 International Criteria. Furthermore, the same investigators reported that the inter-observer variability between eight cardiologists improved significantly with progressive iterations of athlete ECG interpretation guidelines from poor (kappa statistic [κ] = 0.15) to moderate (κ = 0.41) [21]. Reproducibility of ECG assessment remains a significant limitation of screening programs [14], but it would seem that criteria refinements have had the dual benefits of fewer false positive results and enhanced agreement between assessors.

When abnormalities are found suggestive of structural heart disease, an echocardiogram is a useful first step, but