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Debate: challenges in sports cardiology; US versus European approaches

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INTRODUCTION (BRUCE HAMILTON)

For practitioners working with elite athletes, the field of sports cardiology provides clinical, academic, administrative and fiscal challenges. These challenges are exemplified and reinforced by the lack of consistency and consensus both in the literature and academic presentations. Through the presentation of a series of clinical questions, this debate attempts to 'cut to the chase' on cardiovascular issues relevant to the clinician dealing with elite athletes. In so doing, we hope to crystallize some of the most important elements of the complex cardiological management of elite athletes, in a concise, readable format. Frequently over the last 10 years, many of the controversies in this field have been (rightly or wrongly) presented in a Europe versus USA paradigm. We have chosen to test whether there really are polarised views across the Atlantic, by deliberately pitting specialists from the USA against those from the UK. Professors Levine and Thompson are both internationally recognised sports cardiologists, with immense academic and clinical credibility, and who will represent the 'US approach'. Professor Whyte and Doctor Wilson are cardiac physiologists with a wealth of experience in the testing, evaluation and screening of elite athletes, and who have equally impressive academic credibility and for the purposes of this debate, they will be representing the 'European approach'. To initiate this process, each team was required to provide a concise answer (circa 200–300 words) to a series of five clinical conundrums. Subsequently, each team had the opportunity to provide a rebuttal to the opposing team's answers, and the following reflects the consolidation of those answers.

QUESTION 1

A popular debate in the literature and the conference podium is the clinical utility of the ECG in the preparticipation screening of athletes. If one assumes that the overall cost:benefit of cardiac screening for athletes remains to be determined, is it now established that the ECG adds diagnostic value to the preparticipation cardiac screening of otherwise healthy athletes and should be a prerequisite for screening programmes?

PDT and BDL

Every debate must specify the topic for debate. Our side readily concedes that an ECG increases the diagnosis of multiple *potentially* fatal cardiac conditions including atrio-ventricular accessory pathways, right

ventricular cardiomyopathy, Brugada's syndrome, long QT syndrome (LQTS) and hypertrophic cardiomyopathy (HCM). Indeed, taking any other position is foolish since the diagnosis of several of these conditions can only be made with an ECG. We are also not debating whether or not an ECG adds to the cost of screening. Of course it does and to argue otherwise would again be foolish. The only topic worthy of debate is whether or not the addition of an ECG to screening does more good than harm thereby justifying any required expenditures. Screening proponents rarely recognise the potential harm of a screening intervention, but such harms exist as discussed in a recent *JAMA* editorial on cancer screening titled 'The harms of screening'.¹ The editorial correctly notes that the reason most position stands are cautious about recommending screening is because screening can result in iatrogenic disease. We have concerns that ECG screening will increase the number of athletes falsely diagnosed with disease (because the diagnosis of many of these conditions is imperfect) as well as the number of athletes correctly diagnosed with real disease who would never have experienced a cardiac event, but who will receive overly aggressive or inappropriate treatment. Until there is proof that such harm does not occur with ECG screening, there is absolutely no justification for routinely recommending or performing such treatment. This is also obvious because the primary rule of medicine is 'first, do no harm'.

GPW and MGW

There is consensus agreement from both the American College of Cardiology/American Heart Association and the European Cardiology Society that compelling justification exists for cardiovascular preparticipation screening on medical, ethical and legal grounds.^{2 3} While agreement on the implementation of screening may exist, there remains a difference in opinion regarding the methods employed to assess cardiovascular risk between the UK and USA. In line with the entire preparticipation screening debate, the North Americans base their exclusion of a resting ECG on a complete absence of empirical data, simply critiquing European data. This is puzzling to us, considering that some 30 years ago, data from the USA⁴ clearly demonstrated that in a cohort of 78 patients with HCM who had either died suddenly or had aborted cardiac arrest, where previous ECGs were available, 96% presented an abnormal ECG. The Italians further reported on 49 athletes

identified as harbouring a disease associated with sudden cardiac death and demonstrated that only 14 (29%) presented cardiac symptoms before the time of death.⁵ Our own group reported on nine asymptomatic athletes from a cohort of 2720 young individuals (an incidence of 0.3% in line with current consensus) of whom none would have been identified without resting ECG.⁶ To avoid appearing Euro-centric, a recent study from Harvard provides strong support for an enhanced sensitivity and negative predictive accuracy with the (their words) 'mandatory' inclusion of ECG.⁷ It is clear that the resting ECG significantly improves the sensitivity of preparticipation screening, and despite the likely protestations of Professors Levine and Thompson, it is clear that some in the North American community believe it should be included as well.⁷⁻⁹ In summary, a preparticipation screening programme without a resting ECG is frankly a waste of time and money.

PDT and BDL Question 1 rebuttal

We agree with Professors Whyte and Wilson that ECG screening increases the sensitivity to detect HCM and the other potentially fatal conditions noted in our treatise, but that is not the point of this debate. The point of the debate is simple: Is there conclusive evidence that ECG screening does more good than harm? Such evidence is required before such testing is *mandated* for all athletes. Physicians should manage individual patients as appropriate for the clinical situation, and all concerned parents should be allowed to accept the risks as well as the benefit of ECG screening if they choose, but mandating testing for a population requires a higher level of justification. It is obvious that such evidence does not exist; if it did, there would be no debate. In the absence of conclusive data on the risks and benefits of screening, it is just as likely, and we believe more likely, that mandated screening would create more iatrogenic disease and deaths than lives saved. The onus is on Professors Whyte and Wilson to prove that this is not so.

GPW and MGW Question 1 rebuttal

Professors Thompson and Levine concede that the ECG improves the diagnosis of inherited cardiac conditions—this should be debate over! Yet to accuse us of failing to recognise the potential harm ECG screening may cause, is countered by their unwillingness to recognise that the fact that the downstream cascade of the US screening model consistently increases the number of false-positive athletes requiring further testing (harming the athlete and their healthcare system), and as we and others have demonstrated, increases the number of potential false-negative athletes provided with incorrect medical reassurance. To not engage in research to provide an answer to this conundrum is in direct opposition to their premise; 'do no harm'. Rarely has so much resistance been based on so little evidence!

QUESTION 2

A frequently cited reason for cardiac screening of elite athletes is the negative effect that a Sudden Cardiac Death of a high-profile athlete may have on participation and therefore health of the population as a whole. However, limited evidence exists to support this hypothesis. Furthermore, the exact incidence of sudden cardiac death (SCD) is unknown. In light of the dearth of empirical evidence are we able to support or refute the mandating of cardiac screening in high-level athletes with confidence?

PDT and BDL

We agree that there is a dearth of empirical evidence on the risks of athletic participation. We are also unaware of evidence that the death of a high-profile athlete reduces athletic participation, particularly recreational activities that are used by the largest amount of the population to optimise health, but since Professors Whyte and Wilson are concerned enough about the risks of athletic competition to mandate ECGs for all athletes, they may consider any decrease in athletic participation as a life-saving intervention. It is this absence of data that is the core of our argument. Without data documenting that ECG screening saves more lives than it harms, the prudent approach is to 'do no harm'. So why do any screening at all, even the limited screening recommended by the American Heart Association? Again, there are no concrete data supporting that this approach works, but such screening can detect conditions such as aortic stenosis that were historical, but not current,¹⁰ frequent causes of exercise-related death, suggesting that athletes with this condition are somehow detected and excluded. Furthermore, the death rate among athletes in the USA achieved without mandated ECG screening approximates that of other locations where ECG screening is mandated,¹¹ suggesting that the history and physical examination alone may be as effective as other approaches in identifying athletes who are most likely to die during sporting activities. A number of studies note the greater number of athletes with potentially fatal conditions detected by the ECG component of a screening programme. We are not convinced that all such athletes are helped and not hurt by this effort.

GPW and MGW

No negative 'measurable' effect on sporting participation rates for the general population following the SCD or aborted cardiac arrest of an elite athlete can be given. However, it is evident that for the 35 000 spectators and estimated 200 million TV viewers, watching the live (6 min!) cardiac resuscitation of the England Under-21 soccer captain during the recent Tottenham Hotspur versus Bolton Wanderers FA cup clash, found it particularly disturbing. Debate surrounding the incidence of sudden cardiac death in young athletes has been the major impediment to progress in finding an international consensus on preparticipation screening.¹² The central problem facing this debate is, with the exception of the Italians, the abject lack of systematic national registries (this includes the UK as well as the USA). However, particularly irksome in this debate is the perpetual diatribe from scientists who continue to publish their argument in highly reputable cardiology journals for a vanishingly small number of sudden cardiac deaths on newspaper reports and internet searches.¹³ This long-standing acrimonious debate was established by an early study from Van Camp *et al*,¹⁴ who postulated an incidence of sudden cardiac death in the USA of <20 per year. A more recent perusal of American newspapers has re-defined this number to 66 per year (0.6 deaths per 100 000 person-years)¹⁵; a figure similar to an earlier study from the same group examining sudden cardiac death in Minnesota (0.5 deaths per 100 000/year).¹⁶ Studies based on registries from the Italians suggest a rate of 2.1 deaths per 100 000/year;¹⁷ however, the introduction of screening reduced this number to a figure similar to that of the North Americans of 0.4 deaths per 100 000/year. Even the North Americans agree that the true incidence of sudden death in young athletes will only be achieved through mandatory centralised reporting.¹⁸ It is clearly time for an international embargo on guessing the incidence of sudden death and

to recognise the fact that thumbing the sports pages does not replace registries! We should all focus our efforts on other, more important, areas of the debate.

PDT and BDL Question 2 rebuttal

We disagree with Professors Whyte and Wilson that 'debate surrounding the incidence of sudden cardiac death in young athletes has been the major impediment to progress in finding an international consensus on preparticipation screening'. The major impediment to consensus is the lack of data evaluating the benefits and *harms* of mandated screening. For reasons unclear to us, Professors Whyte and Wilson fail to discuss the potential harms of screening. Unnecessary atrioventricular nodal ablations resulting in heart block, cardiac perforation and tamponade, and unnecessary defibrillators placed for incorrect diagnoses, including a lifetime of generator changes, pocket infections, endocarditis and inappropriate shocks are not televised. If they were, there would be a robust cry against mandated screening, but neither side of the argument should be settled by anecdotes or emotion. It should be noted that the specific athlete cited by Professors Whyte and Wilson who unfortunately suffered a cardiac arrest on the field witnessed by stadium and TV crowds had been screened with an ECG at least three times prior to this episode. Moreover, we would suggest that it is equally disturbing to watch TV images of car crashes, natural disasters and the deaths of young people from any cause. Professor Whyte and Dr Wilson find it 'irksome' that 'scientists ... publish their argument in highly reputable cardiology journals for a vanishingly small number of sudden cardiac deaths on newspaper reports and internet searches'. Others might find it irksome that scientists would recommend an expensive and potentially dangerous mandated screening programme without recognising the medical, psychological and financial risks of this recommendation. Still others might find it irksome that manuscripts published in 'highly reputable cardiology journals' after peer review are discounted so readily.

GPW and MGW Question 2 rebuttal

We repeat: it is simply not good enough to critique the Italian 'registry' of sudden deaths, and then provide counter evidence for not screening with ECG, based upon newspaper reports and internet search engines. This is exemplified by the use of reference 11 within which the US data are garnered from media reports. The absence of evidence argument is simply not a robust argument. It is clear that more evidence is required and centralised registries are the only answer: media reports do not replace registries!

QUESTION 3

Many sporting federations are being encouraged to perform cardiac screening on their athletes prior to an international sporting event. However, it appears many individuals (trainers, physiotherapists, sports medicine physicians, nurses, etc) are involved in performing the initial cardiac screening evaluation. Do you believe that there is enough emphasis placed on this element of their training to allow for the safe first-line screening of athletes?

PDT and BDL

We would discourage sporting federations from performing such screenings and would suggest that they cease such activities before they hurt an athlete physically or injure an athlete's career unnecessarily. We are aware of a National Basketball Association player whose salary was markedly limited by the diagnosis of HCM, a diagnosis, made by well-trained experts, which remarkably disappeared after he stopped playing. We are also aware of a

South American soccer player whose several million dollar salary was voided because of a markedly abnormal ECG, which never led to a conclusive diagnosis even when he was evaluated by exercise experts. Such events, albeit anecdotal, suggest that an athlete is at potential risk from screening regardless of who performs the screening. Who does the screening is a trivial and really moot point. What is important is whether or not there are data to justify such screening. There are not.

GPW and MGW

When debating the implementation and efficacy of preparticipation screening there is an important caveat; the inclusion of a resting ECG requires specialist sports cardiology expertise to optimise sensitivity. Indeed, this also applies to the administration of a family history and symptom questionnaire which requires specific training and expertise. While it may be undertaken by a specialist cardiac nurse, in our laboratories, it is always second checked by the attending sports cardiologist. This is the antithesis of the North American approach where questionnaires are sometimes performed by non-specialists without appropriate training including: physical therapists; nurses; and even coaches! The primary driving force of this non-specialist approach is cost-saving; however, given the pre-existing poor sensitivity of questionnaire and physical examination alone (established in answer 1 above), the use of non-specialists merely exacerbates the problem. Preparticipation cardiovascular screening of an elite athlete in the hands of a non-specialist is not only a waste of money but an unethical and immoral act of false reassurance. At present, there is little evidence for the provision of formal training in this area with only sporadic and unstructured educational opportunities for clinicians and healthcare workers to engage in the advancement of knowledge and skills (this includes ECG interpretation).¹⁹ Unless there is a significant emphasis placed on training and education, effective preparticipation screening is doomed to failure before it has begun.

PDT and BDL Question 3 rebuttal

We both see athletes with ECG 'abnormalities' detected on cardiac screening by competent physicians and admit that the diagnosis of what is normal versus what is abnormal is a challenge even after our combined 50 years of experience in clinical cardiology. The idea that education alone will eliminate false positives is simply naïve.

GPW and MGW Question 3 rebuttal

It would appear that Professor Levine and Thompson are suggesting that if the experts get it wrong, then there is no value in undertaking screening because of the potential harm to the athlete. Apart from the Magalski paper on NFL players²⁰ and Harmon *et al*²¹ paper on the incidence of SCD within the National Collegiate Athletic Association (NCAA), why has the world never seen any 'pooled' epidemiological screening data from the major US sporting federations National Football League (NFL), National Basketball Association (NBA), National Hockey League (NHL), Major League Baseball (MLB), Major League Soccer (MLS), etc). The world knows clubs in these leagues screen, as their insurance companies often mandate it before underwriting the player's salary. How many players were diagnosed, how many false positives, cost of the programme, number of problem cases, who performed the screening, etc... Let's work together and confirm the flaws in the system. Perhaps the North Americans should come off the fence and engage in research to assist in the provision of answers to these, and other, difficult questions.

QUESTION 4

HCM presents in many guises. The diagnostic pathway now involves numerous investigations including ECG, echocardiography and cardiac MRI, substantially increasing our understanding of the pathophysiological staging of the disorder. Despite this, in some cases definitive diagnosis may be difficult to ascertain (ie, The 'Grey Zone') and/or stratifying individual risk absent thereby limiting the ability to make clear recommendations. On this basis, how confidently can we disqualify elite athletes with inherited cardiac disease from professional sports?

PDT and BDL

It is sometimes difficult to separate disease from the myocardial adaptations that occur with exercise training, but more vexing is being certain of the prognosis for an asymptomatic otherwise healthy athlete even with unequivocal HCM. Clinicians assume that an asymptomatic athlete with HCM is at risk for an exercise-related cardiac event. This assumption is based on the poor prognosis noted when HCM was first identified, and on the observation that HCM is the dominant cause of sports-related cardiac deaths in the USA. What is becoming increasingly clear, however, is that HCM may affect 1 in 500 individuals²² and that most of these individuals with HCM do not die suddenly during physical activity. Consequently, exercise risk for a person with HCM may not be related to HCM alone, but to accompanying conditions. For example, 9 of 15 runners dying with HCM had concomitant conditions that could have increased their risk of death.²³ Also vexing is the possibility that exercise may positively influence the HCM genotype. In a murine model of HCM produced by inducing myosin heavy chain mutations, an exercise programme before HCM pathology appeared reduced fibrosis, myocyte disarray and markers of myocyte hypertrophy, whereas exercise after hypertrophy was established, reduced myocyte disarray, but not fibrosis.²⁴ Both results, at least in a murine model, suggest that exercise may have some value in individuals with HCM. Whether or not this is true, is not the subject of this debate, and is used solely to reinforce the lesson that many tenets in medicine that appear clear and logical today will be proved foolish in the future. At present, we do not even know that restricting exercise in an HCM patient improves their prognosis.

GPW and MGW

The area of risk stratification is the most problematic and poorly understood area in the entire preparticipation screening debate. The risk stratification of non-athletic patients harbouring the common diseases associated with sudden cardiac death in sport is poorly described. Our understanding of risk is exacerbated when intense training and competition is imposed on a carrier. Guidelines from both the ACC 36th Bethesda Conference and European Society of Cardiology recommend that athletes with unequivocal or 'probable' cardiomyopathy abstain from competitive sport and vigorous training with the exception of low-intensity activities.^{25 26} However, in the absence of a definitive diagnosis, a conservative approach with education, close observation and regular (annual) follow-up is the approach adopted by most UK specialist sports cardiologists. We recognise that SCD related to HCM is rare²⁷ and probably less than 1% per year. The risk is marginally higher in sporting individuals. An individual with HCM may therefore in fact have spent a lifetime participating in sport only to have the condition diagnosed later in life.²⁸ In the case of the 'grey zone' athlete with mild abnormalities but not diagnostic of a cardiomyopathy, the sporting eligibility

decision is always underpinned by the athletes family history of SCD and past or present personal symptoms, both of which are strong predictors of risk, abate derived from a diverse population of patients with cardiomyopathy; not athletes. Enhancing our understanding of risk stratification requires large-scale, multi-national research programmes to help establish the natural history of these diseases and establish robust disqualification algorithms. Classically, those antagonistic to preparticipation screening use the area of risk and disqualification, alongside cost (both to the healthcare system and the athletes earnings), to justify their non-committal to screening; this, despite the absence of any empirical data to support their argument.

PDT and BDL Question 4 rebuttal

We are not 'antagonistic to preparticipation screening'. For example, we believe that screening underlies the decrease in deaths from aortic stenosis in young athletes. It also identifies athletes who have a family history of sudden death, report syncope during exercise, have neck, back or joint problems that have not been adequately addressed, manifest findings of Marfan syndrome or are being abused at home. In sum, there are many reasons to 'screen' athletes beyond the myopia of cardiologists. We will also not be 'antagonistic' to the use of the ECG in screening athletes once there are data to justify its use and to reassure us that the harm does not outweigh the benefit. The key issue is that the ECG identifies both more real and more spurious disease, exposing those athletes with falsely positive ECGs to additional tests, medical interventions and potential side effects. We are not concerned with the costs of ECG screening, but with its potential harm.

GPW and MGW Question 4 rebuttal

Data from our histopathology colleagues demonstrate that of 118 athletes referred for pathological assessment to ascertain the precise aetiology of SCD, 81% died either during or immediately after exercise,²⁹ albeit small there is undeniably a link between phenotypic expression, continued intensive physical activity and sudden death. We also state that without a 'definitive' diagnosis, a conservative approach with education, close observation and regular follow-up is the optimal strategy. There is an alternative, North America approach; which is to 'bury one's head in the sand'. We anticipated that Professors Thompson and Levine would cite the Hippocratic oath; 'do no harm' in their defence to most of these conundrums. However, taking a lacklustre approach to active engagement in empirical research to seek answers to these and other difficult questions would appear to contravene this basic ethical tenant.

QUESTION 5

Many international sporting bodies now provide guidelines for the screening of athletes. However, none of these sporting bodies provide guidelines as to how to manage a positive result. If an elite athlete is diagnosed as being gene positive for an inherited cardiac disease, but is asymptomatic, phenotype negative with a negative family history of sudden death, what is the medico-legal and ethical responsibility (and how may that differ from the attending cardiologist or International Federation Medical Committee) with regard to care for this athlete who has met the qualifying criteria for an event such as the Olympic Games?

PDT and BDL

We would argue that genetically screening athletes for cardiovascular disease in the absence of specific reasons and for any

other reason than to facilitate a diagnosis is even more abhorrent than ECG screening. It is far too early in our understanding of cardiovascular genetics, epigenetics and post-translational gene modification to make recommendations to athletes regarding their eligibility for participation based on genetic testing alone. The medical-legal and ethical responsibility of any clinician dealing with an athlete with any positive screening test is to provide the best medical care available to that athlete. This includes explaining to the athlete that sometimes our diagnostic abilities exceed our ability to prognosticate. Medical decisions should be evidence based and when the evidence is lacking, the decision should be interpolated from the available evidence. The athlete's care should also be cognizant of the athlete's wishes. But in all dealings with athletes, the first injunction is to do no harm to that athlete's psychological state, career and health without concrete evidence that our recommendations provide more good than harm.

GPW and MGW

Currently no universal consensus exists to support recommendations for disqualification based on genotype positive–phenotype negative athletes, and thus we do not support a 'blanket approach' for genetic testing for all troublesome cases. It is true that for now, we do not know the natural history of those with myopathic ECGs or those who are genotype positive but demonstrate no phenotypic evidence of cardiomyopathy. Their risk of an adverse event relating to sport is likely considerably lower than those with phenotypic disease and hence very low (significantly less than 0.5% per year). To that end, the general consensus is (1) to allow full sporting participation in these cases unless there is a malignant family history or suspicious symptoms, that is, documented arrhythmia and/or syncope, and (2) to seek the expert opinion of a cardiologist with knowledge in sports cardiology and inherited cardiac disease 'before' genetic testing was undertaken. We have examples of asymptomatic elite athletes with negative family histories of SCD, who demonstrate abnormal ECGs suggestive of a cardiomyopathy, and are diagnosed with subtle and mild apical HCM. They have been cleared to compete in Olympic sport and have been event free for a number of athletic years thus far. Their event risk is ultralow based on all available long-term apical HCM follow-up. But it is worth noting that the genotype-positive diagnosis still carries the important message regarding family screening, as the same gene may manifest itself differently or more aggressively in other family members. From a medico-legal perspective the approach should be able to provide the athlete (parent/guardian where indicated) with the detailed results and highlight the absence of information in giving any recommendations. In addition to regular follow-up, full sporting participation should be accompanied by education of the athlete, their family and, where consent is granted, their sport. This later group is an area of much contention given the potential implications for contractual agreements and selection. In the absence of mandatory screening programmes, the results of clinical investigations remain confidential; it is this area which provides a major dilemma for attending cardiologists and team doctors who find themselves responsible to both the athlete and their club/sport. As with all other areas of this debate, the dearth of data can only be addressed through consensus agreement underpinned by research.

PDT and BDL Question 5 rebuttal

We disagree that 'the dearth of data can only be addressed through consensus agreement underpinned by research'. We suggest that data, and only data, can lead to consensus.

Consequently, failure to reach consensus is due to insufficient data. In the absence of data, first do no harm.

GPW and MGW Question 5 rebuttal

We generally agree with all of what Professors Thompson and Levine state. At present screening is based on phenotype not genotype. No data exist to support disqualification on genotype alone. To compound this deficiency in genotype diagnosis, the provision of gene testing is sporadic, expensive and invariably a fishing exercise. At present, the list of disease-associated mutations is not complete and is unlikely to ever achieve 100% sensitivity. It is our belief, along with that of the Bethesda guidelines, that genes can be inherited but not expressed. Accordingly, genetic testing must not be approached lightly, and should always be undertaken after the consultation with a specialist cardiologist, and after the athlete has provided informed consent following extensive consultation with a specialist genetic counsellor.³⁰

CONCLUSION (BRUCE HAMILTON)

Many years ago, from the sports physician perspective, it seemed we had sports cardiology pretty much 'sorted out'. However, over the last 10 years concern has been mounting, particularly with sports physicians working with elite athletes, at the lack of consensus within sports cardiology—which directly impacts upon our management of the elite athlete. Our hope in preparing this manuscript was that by posing relevant questions to appropriate specialists, we may short-circuit some of the complex academic arguments. As anticipated, this debate has again highlighted some of the many challenges that remain within the burgeoning field of sports cardiology.

We deliberately and provocatively titled this debate, 'USA versus European Approaches', but post hoc realised that we unwittingly confounded this underlying theme by pitting two physiologists against two clinical sports cardiologists. This confounder made itself abundantly clear in the first response of Professors Thompson and Levine, whereupon they highlighted the edict of 'first do no harm'. This statement will resound with clinicians reading this, and is an overlying principle upon which this discussion must, by necessity focus. Based on this medical/ethical message, a strong argument is presented that in the absence of appropriate evidence regarding the (suspected) negative consequences of mandated cardiac screening, either with or without electrocardiography, the application of both should be discarded. Unfortunately, it is clear that the data are not available to argue the relative merits of the opposing considerations of false positives (associated with cardiac screening) and the false negatives (associated with no screening or screening without an ECG) in an evidence-based manner. As a result, and appropriately, we must fall back on medical ethics as a guide. Paradoxically however, the fact that we lack the data (that realistically may only be generated via appropriate screening programmes) to provide consensus on this important issue may provide the imperative required to re-evaluate the application of 'first do no harm', with a view to the long-term benefits to future generations of athletes. As has been illustrated from review of breast cancer screening programmes, effective evaluation of the merits of the programme is only possible after sustained, long-term and careful evaluation of both positive and negative outcomes.³¹ It would be outrageous that if in 20 years time these same, currently unanswerable questions are similarly debated without adequate data.

It is clear from the discussion above, that (in a rare moment of harmony) all participants agree that any form of cardiac screening (if it were to happen) requires appropriately trained and skilled personnel. Somewhat surprisingly, it appears that

even appropriate education, specialist training and years of experience does not prevent the inappropriate management of some athletes. While one must acknowledge the positive steps being undertaken by organisations such as the IOC medical commission in the provision of education in cardiac screening and ECG interpretation, the conclusion of our colleagues above is disconcerting when one considers the risks involved.

Unfortunately, it seems that we remain unable to adequately stratify the risk of sports participation, even for those athletes with the most common condition associated with sudden cardiac death in sport, HCM. While there are guidelines in place, it seems that these are based on pragmatic consensus, rather than cold hard facts. Furthermore, as outlined above, the application of specified guidelines depends on diagnostic determinations such as 'unequivocal', 'probable' or 'definitive' relying on the subjective interpretation of the same specialists, who were unfortunately condemned above. Reductionists among us may like the idea of utilising genomic studies to provide the answers we are lacking clinically, but our protagonists all seem to agree, that we are a long way from being able to appropriately utilise genetics to assist us. We all agree that appropriate cardiological evaluation of athletes is important. This debate between eminent and experienced specialists in this field has highlighted that there remains a divergent opinion regarding the merits and dangers of both mandated cardiac screening and the content thereof. There is however consensus that there are insufficient data to allow for an evidence-based answer to either this or other sports cardiology conundrums.

The questions presented to the debaters reflect the daily challenges faced by practitioners working with elite athletes. For sports medicine practitioners and policy creators, Professors Thompson and Levine present a strong argument for not mandating cardiac screening. However, for clinicians dealing with individual elite athletes upon whom they may be required to perform personalised screening evaluations, Professor Whyte and Doctor Wilson present a strong case for inclusion of the ECG. Ultimately, the question we must answer is, how do we appropriately create an evidence base which is able to answer these challenging issues, such that our next generation of colleagues are not repeating the same questions—and that the next generation of athletes are not subject to the same level of uncertainty?

Finally, Professors Levine, Thompson, Whyte and Dr Wilson are to be commended and thanked for the passionate and informed responses provided. While there are clearly differences in approach and emphasis which encouraged conflict, it was in the spirit of competition that the opposing teams took a combative approach to the others position. Indeed, it is worth noting that all the participants have the same end goal, are working together in research to achieve that goal, and importantly, remain friends!

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REFERENCES

1. **Woolf SH**, Harris R. The harms of screening: new attention to an old concern. *JAMA* 2012;**307**:565–6.
2. **Maron BJ**, Thompson PD, Ackerman MJ, *et al.* Recommendations and considerations related to preparticipation screening for cardiovascular abnormalities in competitive athletes: 2007 update: a scientific statement from the American Heart Association Council on Nutrition, Physical Activity, and Metabolism: endorsed by the American College of Cardiology Foundation. *Circulation* 2007;**115**:1643–55.
3. **Corrado D**, Pelliccia A, Bjornstad HH, *et al.* Cardiovascular pre-participation screening of young competitive athletes for prevention of sudden death: proposal for a common European protocol. Consensus Statement of the Study Group of Sport Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology. *Eur Heart J* 2005;**26**:516–24.
4. **Maron BJ**, Roberts WC, Epstein SE. Sudden death in hypertrophic cardiomyopathy: a profile of 78 patients. *Circulation* 1982;**65**:1388–94.
5. **Corrado D**, Basso C, Schiavon M, *et al.* Screening for hypertrophic cardiomyopathy in young athletes. *N Engl J Med* 1998;**339**:364–9.
6. **Wilson MG**, Basavarajiah S, Whyte GP, *et al.* Efficacy of personal symptom and family history questionnaires when screening for inherited cardiac pathologies: the role of electrocardiography. *Br J Sports Med* 2008;**42**:207–11.
7. **Baggish AL**, Hutter AM Jr., Wang F, *et al.* Cardiovascular screening in college athletes with and without electrocardiography: a cross-sectional study. *Ann Intern Med* 2010;**152**:269–75.
8. **Le VV**, Wheeler MT, Mandic S, *et al.* Addition of the electrocardiogram to the preparticipation examination of college athletes. *Clin J Sport Med* 2010;**20**:98–105.
9. **Myerburg RJ**, Vetter VL. Electrocardiograms should be included in preparticipation screening of athletes. *Circulation* 2007;**116**:2616–26; discussion 2626.
10. **Maron BJ**, Thompson PD, Puffer JC, *et al.* Cardiovascular preparticipation screening of competitive athletes. A statement for health professionals from the Sudden Death Committee (clinical cardiology) and Congenital Cardiac Defects Committee (cardiovascular disease in the young), American Heart Association. *Circulation* 1996;**94**:850–6.
11. **Maron BJ**, Haas TS, Doerer JJ, *et al.* Comparison of U.S. and Italian experiences with sudden cardiac deaths in young competitive athletes and implications for preparticipation screening strategies. *Am J Cardiol* 2009;**104**:276–80.
12. **Drezner J**, Plum B, Engebretsen L. Prevention of sudden cardiac death in athletes: new data and modern perspectives confront challenges in the 21st century. *Br J Sports Med* 2009;**43**:625–6.
13. **Steinvil A**, Chundadze T, Zeltser D, *et al.* Mandatory electrocardiographic screening of athletes to reduce their risk for sudden death proven fact or wishful thinking? *J Am Coll Cardiol* 2011;**57**:1291–6.
14. **Van Camp SP**, Bloor CM, Mueller FO, *et al.* Nontraumatic sports death in high school and college athletes. *Med Sci Sports Exerc* 1995;**27**:641–7.
15. **Maron BJ**, Doerer JJ, Haas TS, *et al.* Sudden deaths in young competitive athletes: analysis of 1866 deaths in the United States, 1980–2006. *Circulation* 2009;**119**:1085–92.
16. **Maron BJ**, Gohman TE, Aeppli D. Prevalence of sudden cardiac death during competitive sports activities in Minnesota high school athletes. *J Am Coll Cardiol* 1998;**32**:1881–4.
17. **Corrado D**, Basso C, Rizzoli G, *et al.* Does sports activity enhance the risk of sudden death in adolescents and young adults? *J Am Coll Cardiol* 2003;**42**:1959–63.
18. **Maron BJ**. National electrocardiography screening for competitive athletes: feasible in the United States? *Ann Intern Med* 2010;**152**:324–6.
19. **Drezner JA**, Asif IM, Owens DS, *et al.* Accuracy of ECG interpretation in competitive athletes: the impact of using standardised ECG criteria. *Br J Sports Med* 2012;**46**:335–40.
20. **Magalski A**, Maron BJ, Main ML, *et al.* Relation of race to electrocardiographic patterns in elite American football players. *J Am Coll Cardiol* 2008;**51**:2250–5.
21. **Harmon KG**, Asif IM, Klossner D, *et al.* Incidence of sudden cardiac death in national collegiate athletic association athletes. *Circulation* 2011;**123**:1594–600.
22. **Maron BJ**, Gardin JM, Flack JM, *et al.* Prevalence of hypertrophic cardiomyopathy in a general population of young adults. Echocardiographic analysis of 4111 subjects in the CARDIA study. Coronary artery risk development in (young) adults. *Circulation* 1995;**92**:785–9.
23. **Kim JH**, Malhotra R, Chiampas G, *et al.* Cardiac arrest during long-distance running races. *N Engl J Med* 2012;**366**:130–40.
24. **Konhilas JP**, Watson PA, Maass A, *et al.* Exercise can prevent and reverse the severity of hypertrophic cardiomyopathy. *Circ Res* 2006;**98**:540–8.
25. **Maron BJ**, Ackerman MJ, Nishimura RA, *et al.* Task Force 4: HCM and other cardiomyopathies, mitral valve prolapse, myocarditis, and Marfan syndrome. *J Am Coll Cardiol* 2005;**45**:1340–5.
26. **Pelliccia A**, Fagard R, Bjornstad HH, *et al.* Recommendations for competitive sports participation in athletes with cardiovascular disease: a consensus document from the Study Group of Sports Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology. *Eur Heart J* 2005;**26**:1422–45.
27. **Maron BJ**, Casey SA, Haas TS, *et al.* Hypertrophic cardiomyopathy with longevity to 90 years or older. *Am J Cardiol* 2012;**109**:1341–7.
28. **Wilson MG**, Chandra N, Papadakis M, *et al.* Hypertrophic cardiomyopathy and ultra-endurance running—two incompatible entities? *J Cardiovasc Magn Reson* 2011;**13**:77.
29. **de Noronha SV**, Sharma S, Papadakis M, *et al.* Aetiology of sudden cardiac death in athletes in the United Kingdom: a pathological study. *Heart* 2009;**95**:1409–14.
30. **Charron P**, Arad M, Arbustini E, *et al.* Genetic counselling and testing in cardiomyopathies: a position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. *Eur Heart J* 2010;**31**:2715–26.
31. **Raftery J**. Possible net harms of breast cancer screening: updated modeling of Forrest report. *BMJ* 2011;**343**:d7627.