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Prevention of sudden cardiac death in athletes: balancing safety, sustainability, and access to cardiovascular screening

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Abstract

Sudden cardiac death (SCD) in athletes remains a major clinical and public health concern, raising complex questions regarding the optimal balance between safety, sustainability, and equitable access to cardiovascular screening. Two contrasting paradigms are currently adopted worldwide: the mandatory and standardized Italian model and the voluntary, history-based approach predominantly used in the United States. The Italian system, which includes routine electrocardiography and exercise testing, has been associated with a marked reduction in SCD incidence but is burdened by high costs, false-positive findings, and organizational challenges. Conversely, the US model limits resource utilization but is associated with higher mortality rates and significant inequalities in access to preventive care. Using competitive basketball as a representative high-risk sport model, this narrative review summarizes the principal cardiac conditions associated with SCD. It discusses the strengths and limitations of existing screening strategies. We propose a hybrid, risk-based screening approach that integrates targeted diagnostic testing with continuous clinical surveillance, emergency preparedness, and structured education programs. Attention is given to the role of physiotherapists and allied health professionals as daily clinical observers within multidisciplinary teams. A multidimensional preventive strategy—combining selective screening, professional training, emergency action planning, and shared decision-making—may represent a sustainable and ethically sound model for further reducing SCD among athletes while preserving the right to sport.

Introduction

Sudden cardiac death (SCD) in youth sports is a profoundly tragic event that strikes young individuals who appear to be in perfect health, often without any recognizable premonitory symptoms.[1] This dramatic occurrence is characterized by its total unpredictability and the strong emotional and media impact it garners when notable and widely reported cases emerge, such as those of Davide Astori or Christian Eriksen, which have brought public and professional attention to the urgent need to develop and implement robust and effective preventive strategies. Basketball represents a particularly relevant model for the study of SCD in athletes. Epidemiological studies have reported a relatively higher incidence of SCD in basketball players compared with several other sports, particularly among male competitive athletes. This increased risk may be related to the combination of high-intensity intermittent exertion, elevated adrenergic stimulation, and substantial hemodynamic load.[2]

In addition, the typical anthropometric characteristics of basketball players—such as increased height and body mass—contribute to greater cardiovascular demand and may facilitate both physiological cardiac remodeling and the potential unmasking of underlying cardiomyopathies. These features make basketball a valuable clinical model for analyzing mechanisms, risk stratification, and preventive strategies for SCD. The interaction between sport-specific physiological stress and underlying cardiac conditions is particularly relevant in basketball, where repeated bursts of high-intensity activity and rapid hemodynamic fluctuations may act as triggers for malignant arrhythmias in susceptible individuals. The physical and physiological demands imposed by basketball place it among the sports with the highest relative risk for acute cardiac events. Furthermore, the specific anthropometric and functional characteristics of basketball players —such as high average stature, often significant muscle mass, and the consistent volume of work they are subjected to during training and games—contribute to amplifying both the hemodynamic demands and metabolic requirements on the circulatory system. For these reasons, basketball constitutes a highly significant and representative model for the analysis of risk factors associated with SCD, offering a useful clinical and sporting context to study mechanisms, prevention, and targeted surveillance measures. Epidemiological studies have shown that basketball is among the sports with the highest reported incidence of SCD, particularly in male competitive athletes.[3] The combination of high-intensity intermittent exercise, large body size, and substantial hemodynamic load may contribute to both physiological cardiac remodeling and the potential unmasking of underlying cardiomyopathies.

Main causes of sudden cardiac death in athletes[4]

Hypertrophic cardiomyopathy

Hypertrophic cardiomyopathy (HCM) is the most common genetic heart disease, with an estimated prevalence of 1:500 in the general population (Table 1).[5] It is caused by autosomal dominant mutations in sarcomeric genes, particularly *MYH7* and *MYBPC3*.[5,6] Pathophysiology includes diastolic dysfunction, myocardial ischemia, left ventricular outflow tract obstruction, and ventricular arrhythmias. Myocyte disarray and fibrosis create an arrhythmogenic substrate that can lead to SCD, especially during intense exertion. In basketball, the alternation of isometric and dynamic efforts, pressure peaks, and elevated heart rate can aggravate the intraventricular pressure gradient and trigger arrhythmias. ESC and AHA/ACC guidelines advise against competitive sports participation for athletes with symptomatic or high-risk HCM.[7,8] The condition is a central target of screening programs.[9]

Arrhythmogenic right ventricular cardiomiopathy

Arrhythmogenic right ventricular cardiomiopathy (ARVC) is a hereditary cardiomyopathy characterized by fibro-fatty replacement of the myocardium, predominantly in the right ventricle.[10] It is caused by mutations in desmosomal genes (*PKP2*, *DSP*, *DSG2*), with autosomal dominant transmission and variable penetrance. Intense physical exercise accelerates disease progression and precipitates the onset of symptoms.[11] Ventricular arrhythmias, syncope, and SCD are the most frequent clinical manifestations. Diagnosis is based on the revised Task Force Criteria (Padua Criteria),[12] which integrate ECG data, imaging, genetics, and family history. Cardiac MRI is the reference diagnostic tool for analyzing structural progression.[13] Furthermore, vigorous physical activity has been observed to impair myocardial function in patients with ARVC.[14]

Hereditary channelopathies

Channelopathies are primary electrical disorders without structural alterations caused by mutations affecting cardiac ion channels or proteins involved in calcium handling. These abnormalities alter cardiac action potential duration or intracellular calcium cycling, creating a substrate for malignant ventricular arrhythmias. In catecholaminergic polymorphic ventricular tachycardia (CPVT), mutations in the *RYR2* gene impair the normal regulation of calcium release from the sarcoplasmic reticulum.[15] During adrenergic stimulation, abnormal diastolic calcium leakage may trigger delayed afterdepolarizations and bidirectional or polymorphic ventricular tachycardia. The main hereditary channelopathies associated with SCD in athletes include long QT syndrome (involving genes such as *KCNQ1*, *KCNH2*, and *SCN5A*), Brugada syndrome (most commonly associated with *SCN5A*), and CPVT (typically involving *RYR2* and *CASQ2*).[16]

In basketball, high adrenergic stimulation can trigger arrhythmias, particularly in LQT1 and CPVT, whereas Brugada syndrome is more commonly associated with arrhythmic risk at rest or during fever. The association between syncope and the pathogenic p.Arg420Gln variant in the *RYR2* gene has also been documented.[1] Resting ECG may be normal, making further diagnostic evaluation essential in selected cases.[17] Exercise stress testing is particularly useful in CPVT and LQT1, where ventricular arrhythmias are often provoked by adrenergic stimulation. Holter monitoring may help identify intermittent ventricular ectopy or nonsustained ventricular tachycardia. In suspected Brugada syndrome with nondiagnostic baseline ECG, pharmacological challenge with sodium channel blockers such as flecainide or ajmaline may unmask a diagnostic type 1 ECG pattern. These diagnostic tools should be applied within an integrated clinical framework, especially in athletes presenting exertional syncope, palpitations, or a relevant family history.

Congenital coronary artery anomalies

Congenital coronary artery anomalies represent the second leading cause of SCD in athletes.[18] Malignant variants, such as anomalous origin from the opposite sinus with an interarterial course, can cause myocardial ischemia and arrhythmias during exertion.[19] Dynamic compression between the aorta and pulmonary artery is the main mechanism. Diagnosis requires advanced imaging (CCTA, MRI, IVUS) and a functional stress evaluation.[20-22] Standard ECG and echocardiogram are often normal.

Cardiovascular screening Italy *vs.* United States

The Italian model

In Italy, the sports medical examination has played a central and mandatory role since 1982, regulated by legislation that establishes fitness for competitive sports practice.[23] This protocol includes a thorough history collection aimed at identifying personal and family risk factors, a complete physical examination with cardiac assessment, a resting ECG performed with

standardized criteria, stress testing to evaluate the cardiovascular response to exercise, and spirometric investigations.[24] The systematic implementation of this approach has been associated with a significant decrease in mortality from SCD among athletes, with data showing a reduction from approximately 3.6 to 0.4 cases per 100,000 athletes per year.[2] This result is attributable to the screening's ability to identify silent cardiac anomalies and predisposing conditions before they manifest in acute events. However, the Italian model presents practical and organizational limitations: the national routine generates high costs, requires specialized resources and dedicated facilities, and can lead to waiting times and logistical burdens for sports clubs and families. Furthermore, extended screening carries a non-negligible risk of false positives (abnormal ECGs) estimated at around 5-10%,[25] resulting in the need for additional investigations, possible temporary restrictions on activity, and psychological impact on athletes and their families. Cardiovascular screening may also have important psychological and social implications. False-positive findings or temporary restriction from sports participation can generate anxiety, uncertainty, and, in some cases, social stigma, particularly in young athletes. For this reason, screening programs should incorporate clear communication strategies, ensuring that athletes and their families understand the meaning and limitations of diagnostic findings. When appropriate, psychological support and counseling should be considered as part of a comprehensive preventive approach. In addition, disparities in access to cardiovascular screening remain a critical issue, particularly in healthcare systems where screening is not universally implemented. Socioeconomic factors, availability of specialized centers, and organizational differences may limit access to appropriate evaluation and follow-up. Future strategies should aim to improve equity through structured, accessible, and potentially publicly supported screening programs. These aspects are essential to ensure that prevention strategies remain not only effective but also ethically and socially sustainable. From a screening perspective, sports such as basketball, characterized by high dynamic and adrenergic load, may particularly benefit from structured cardiovascular evaluation.

The US model

In the United States, cardiovascular screening for athletes is predominantly voluntary and not uniformly federally regulated, largely relying on professional recommendations and local practices. The American Heart Association proposes a targeted approach based on focused history and clinical physical examination, excluding the routine use of resting ECG and stress tests as mass screening tools.[26] This paradigm reduces immediate costs and limits the number of specialist examinations required but is associated with an estimated incidence of SCD higher than in contexts with more intensive screening, with reported values around 1-2 cases per 100,000 athletes per year.[3] The US model also faces issues of equity and consistency: access to health check-ups is unequal, depending on insurance, the resources of schools and local sports organizations, and the variability of clinical practices between regions. The lack of standardization can lead to fragmented screening, underdiagnosis of at-risk conditions, and differences in the follow-up of at-risk athletes.

The ethical paradox

The Italian screening model has been associated with a measurable reduction in SCD; however, it also implies substantial healthcare expenditure. From a health economics perspective, evaluation of screening programs ideally requires cost-effectiveness analyses, often expressed in terms of quality-adjusted life years. Such analyses remain complex in the context of sports eligibility screening because they involve not only medical outcomes but also broader societal considerations related to participation in voluntary recreational or competitive activities. In the US, money is saved, but a higher mortality rate is accepted. Individual freedom translates into a collective waiver of

prevention. A hybrid model, based on risk algorithms and selective testing, could offer a balance (Table 2).[27]

The physiotherapist as a cardiac sentinel

Physiotherapists and other allied health professionals may play a supportive observational role within multidisciplinary teams by identifying symptoms or performance changes that warrant medical evaluation. However, diagnostic assessment and eligibility decisions remain the exclusive responsibility of qualified physicians. Monitoring workload and exercise tolerance is essential.[28,29] Specific training in sports cardiology is recommended. Their responsibilities include: i) functional observation and early reporting; ii) education of athletes and families; iii) interdisciplinary collaboration;[30] iv) participation in emergency action plans (EAPs);[31] v) basic life support and defibrillation (BLSD) training and automated external defibrillator (AED) use.

Complementary preventive strategies

An effective strategy for reducing SCD in athletes cannot be limited to diagnostic screening; it must include a coordinated package of educational, organizational, infrastructural, and governance interventions that work synergistically to increase awareness, improve response readiness, and reduce response time to acute events.[32]

Sudden cardiac death awareness campaigns

- Campaign objectives: inform the public, athletes, and sports operators about the existence, causes, and early warning signs of SCD; dismantle myths and misconceptions; promote proactive behaviors.
- Target and communication channels: direct specific messages to students, amateur and competitive athletes, coaches, club managers, parents, and school staff; use digital channels and media.
- Effective content testimonials, information on how to recognize syncope, chest pain, or unusual dyspnea, invitations to participate in evaluation programs, and BLSD training.

Education for athletes, coaches, and parents

- Structured training programs: design educational paths modulated by age and role.
- Key training content: recognition of warning signs, management of cardiovascular emergencies in the field, principles of first aid and cardiopulmonary resuscitation, AED use, and importance of workload control.
- Parents and sports organizations: play a fundamental role in prevention. Families should be encouraged to report episodes of syncope, unexplained seizures, or a family history of sudden death. Sports clubs should promote a culture of cardiovascular safety by facilitating access to screening programs and ensuring the presence of trained personnel and AED devices.

Emergency action plans and regular simulations

- EAP design: every facility and team must have a written plan that defines roles, communication flows, emergency transport routes, and responsibilities during a cardiac arrest.[31]
- Practical drills and simulations: organize regular simulations involving athletes, coaches, physiotherapists, service personnel, and, when possible, territorial emergency services.

Automated external defibrillator availability and accessibility

In Italy, the availability of AEDs in sports facilities has been mandatory since the Balduzzi Decree and subsequent regulations. Therefore, emergency preparedness programs described in this

manuscript should be interpreted as reinforcement and optimization of existing legal requirements rather than as novel recommendations.

- Strategic defibrillator distribution: ensure easily accessible AEDs in every sports facility, in visible locations outside rooms.
- Maintenance and functional check: schedule periodic inspections, battery, and electrode replacement.

Ethics and medico-legal responsibility

Ethical and medico-legal management of at-risk athletes

In Italy, the issuance of fitness-to-play clearance constitutes a formal medical act that carries precise legal and professional implications; the sports physician is called to evaluate not only the athlete's state of health but also the legal consequences of their clinical decisions.

Shared decision-making and the role of the team

Cardiovascular screening may also have relevant psychological implications. False-positive findings or temporary sport disqualification can generate anxiety, uncertainty, and stigma among young athletes. For this reason, screening programs should include clear communication strategies, ensuring that athletes and families understand the meaning of diagnostic findings and the difference between precautionary restrictions and definitive diagnoses. When necessary, psychological support and counseling should be considered. Shared decision-making today represents a modern, ethically sound, and clinically responsible response to the dilemmas that arise in managing at-risk athletes. Actively involving the athlete, family, and healthcare team fosters an integrated view of the problem and facilitates necessary emotional and practical support.

Role of the physiotherapist and continuous observation

The physiotherapist, due to their daily proximity to the athlete, can play a crucial role in the diagnostic and decision-making process. Their constant clinical observation allows for the detection of functional changes, subtle symptoms, or performance variations that might be missed during sporadic medical check-ups.

Documentation, responsibility, and genetic uncertainty

Accurate and complete documentation is essential to protect both the patient and the healthcare professional. In many cases, especially when dealing with rare genetic conditions or atypical phenotypes, guidelines are not unequivocal, and the literature offers limited evidence, increasing the margin of diagnostic uncertainty. In this context, the team's responsibility extends to the ability to critically interpret available data.

Emerging technologies in cardiovascular screening

Artificial intelligence is increasingly applied to automated ECG interpretation. Machine-learning algorithms trained on large datasets of athletes may improve the differentiation between physiological adaptations and pathological abnormalities, potentially reducing false-positive rates and unnecessary downstream testing. However, current evidence remains heterogeneous, and clinical validation in real-world screening settings is still required before widespread adoption.

Wearable devices, including smartwatches and chest-based sensors, enable continuous or intermittent monitoring of heart rhythm during training and daily activities. These tools may facilitate the detection of previously unrecognized arrhythmias or abnormal heart rate responses. Nevertheless, their use is limited by issues related to signal accuracy, false positives, and the need

for clinical confirmation of recorded events. Therefore, wearable technologies should be considered complementary rather than alternative to standard clinical evaluation.
Advanced genetic testing, particularly next-generation sequencing, allows simultaneous analysis of multiple genes associated with inherited cardiomyopathies and channelopathies. In selected high-risk athletes or families with suspected inherited cardiac disease, genetic testing may contribute to earlier identification of pathogenic variants and improved risk stratification. However, the interpretation of variants of uncertain significance remains a major challenge, requiring integration with clinical and imaging data.
Overall, these technologies may support a more personalized and risk-based approach to screening, but their implementation should be guided by clinical judgment, cost-effectiveness considerations, and adherence to current guideline recommendations. Future prospective studies are needed to define the clinical impact of these technologies within structured screening programs.

Operational recommendations

- Adoption of mandatory and repeated cardiovascular screenings, with stress testing and advanced imaging.
- Specialized training for physiotherapists in recognizing cardiac signs.
- Implementation of emergency action plans and widespread distribution of AEDs in all sports facilities.
- Promotion of a shared prevention culture among athletes, families, and healthcare professionals.
- Development of hybrid screening models based on risk algorithms and personalized stratification.

Practical implementation of the hybrid model

The proposed hybrid model aims to integrate the universal coverage of the Italian system with the cost-conscious approach of the U.S. model, while remaining adaptable to different healthcare settings. This model should be interpreted as a conceptual and operational framework rather than a direct alternative to existing national regulations. In practical terms, a two-step risk-based strategy can be implemented. First-level screening should be performed in all competitive athletes and includes standardized personal and family medical history, physical examination, and resting 12-lead ECG interpreted according to international athlete-specific criteria. This approach ensures broad preventive coverage while maintaining feasibility and accessibility. Second-level evaluation should be reserved for athletes presenting predefined risk indicators, including abnormal ECG findings, exertional syncope or unexplained palpitations, family history of SCD or inherited cardiomyopathy, abnormal response during exercise testing, and known or suspected pathogenic genetic variants. In these selected individuals, advanced investigations may include cardiac magnetic resonance imaging, prolonged ECG monitoring (Holter or wearable devices), exercise testing, and targeted genetic analysis when appropriate. These criteria are consistent with current international recommendations and reflect commonly accepted clinical red flags in sports cardiology.
This selective escalation of diagnostic intensity allows for optimization of resource allocation, reducing unnecessary testing while maintaining a high level of safety. From a health economics perspective, such an approach may improve the cost-effectiveness of screening programs by concentrating resources on higher-risk populations. As a practical example, in a competitive basketball team, all athletes may undergo baseline ECG screening before the season. Only those with abnormal findings or relevant clinical history would proceed to advanced testing, such as cardiac MRI or extended rhythm monitoring. Importantly, in countries such as Italy, any modification of screening strategies must remain aligned with existing medico-legal frameworks

and national recommendations (*e.g.*, COCIS: *Comitato Organizzativo Cardiologico per l'Idoneità allo Sport*), which currently define the standard of care for sports eligibility certification.

Methodological limitations

This article represents a narrative review and therefore has inherent methodological limitations. The literature was not selected through a predefined systematic search strategy, and study inclusion was based on relevance, clinical significance, and conceptual contribution. Therefore, this approach may be subject to selection bias and may not fully capture the entire body of available evidence. In addition, the absence of quantitative synthesis limits the ability to provide pooled estimates or direct comparisons between different screening strategies. Furthermore, some of the considerations presented—particularly those related to the proposed hybrid model—are based on conceptual reasoning and integration of existing knowledge rather than prospective validation studies. Future research, including systematic reviews and prospective studies, will be essential to better define the effectiveness, cost-benefit profile, and real-world applicability of different cardiovascular screening strategies in athletes.

Conclusions

Effective prevention requires a system that combines targeted screening, dynamic surveillance, professional training, and adequate organizational resources. Comparative analysis of different screening paradigms may help inform future policy discussions regarding the balance between prevention, sustainability, and access to sport, while respecting the specific regulatory frameworks governing each country through hybrid models that merge the coverage and standardization of the Italian model with the flexibility and attention to economic sustainability of the US model. Investing in the training of physiotherapists, the availability of advanced diagnostic tools, and the organization of operational multidisciplinary teams represents the most promising strategy to reduce the incidence of SCD among athletes. Athlete safety cannot be entrusted to a single diagnostic moment; it must be built day by day, through observation, education, and collaboration. SCD is preventable,[33] but only if addressed with competence, responsibility, and a systemic vision.

References

1. Emery MS, Kovacs RJ. Sudden cardiac death in athletes. JACC Heart Fail 2018;6:30-40.
2. 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.
3. Maron BJ, Haas TS, Ahluwalia A, et al. Demographics and epidemiology of sudden deaths in young competitive athletes: from the United States National Registry. Am J Med 2016;129:1170-7.
4. Harmon KG, Asid IM, Maleszewski JJ, et al. Incidence, cause, and comparative frequency of sudden cardiac death in NCAA athletes: a decade in review. Circulation 2015;132:10-9.
5. Marian AJ, Braunwald E. Hypertrophic cardiomyopathy: genetics, pathogenesis, clinical manifestations, diagnosis, and therapy. Circ Res 2017;121:749-70.
6. Christian S, Cirino A, Hansen B, et al. Diagnostic validity and clinical utility of genetic testing for hypertrophic cardiomyopathy: a systematic review and meta-analysis. Open Heart 2022;9:e001815.
7. Arbustini E, Protonotarios A, Gimeno JR, et al. 2023 ESC guidelines for the management of cardiomyopathies. Eur Heart J 2023;44:3503-626.
8. Ommen SR, Mital S, Burke M, et al. 2020 AHA/ACC guideline for the diagnosis and treatment of patients with hypertrophic cardiomyopathy. J Am Coll Cardiol 2020;76:e159-240.

9. Corrado D, Basso C, Schiavon M, Thiene G. Screening for hypertrophic cardiomyopathy in young athletes. N Engl J Med 1998;339:364-9.
10. Krahn AD, Wilde AAM, Calkins H, et al. Arrhythmogenic right ventricular cardiomyopathy. JACC Clin Electrophysiol 2022;8:533-53.
11. James CA, Bhonsale A, Tichnell C, et al. Exercise increases age-related penetrance and arrhythmic risk in ARVC-associated desmosomal mutation carriers. J Am Coll Cardiol 2013;62:1290-7.
12. Corrado D, Perazzolo Marra M, Zorzi A, et al. Diagnosis of arrhythmogenic cardiomyopathy: the Padua criteria. Int J Cardiol 2020;319:106-14.
13. Mast TP, James CA, Clakins H, et al. Evaluation of structural progression in arrhythmogenic right ventricular dysplasia/cardiomyopathy. JAMA Cardiol 2017;2:293-302.
14. Saberniak J, Hasselberg NE, Borgquist R, et al. Vigorous physical activity impairs myocardial function in patients with arrhythmogenic right ventricular cardiomyopathy. Eur J Heart Fail 2014;16:1337-44.
15. Stępień-Wojno M, Ponińska J, Biernacka EK, et al. A recurrent exertional syncope and sudden cardiac arrest in a young athlete with known pathogenic p.ARG420GLN variant in the RYR2 gene. Diagnostics 2020;10:435.
16. Tobert KE, Bos JM, Garmany R, Ackerman MJ. Return-to-play for athletes with long QT syndrome or genetic heart diseases predisposing to sudden death. J Am Coll Cardiol 2021;78:594-604.
17. Zorzi A, Vessella T, De Lazzari M, et al. Screening young athletes for diseases at risk of sudden cardiac death: role of stress testing for ventricular arrhythmias. Eur J Prev Cardiol 2020;27:311-20.
18. Gentile F, Castiglione V, de Caterina R. Coronary artery anomalies. Circulation 2021;144:983-96.
19. Gräni C, Kaufmann PA, Windecker S, Buechel RR. Diagnosis and management of anomalous coronary arteries with a malignant course. Interv Cardiol Rev 2019;14:83-7.
20. Xie Z, Zou J, Zhu H, Bu H. Case report: anomalous origin of the right coronary artery from the left sinus of valsalva with aortic dissection. Front Cardiovasc Med 2022;9:900803.
21. Angelini P. Is echocardiography adequate to identify the severity of anomalous coronary arteries? JACC Cardiovasc Imaging 2016;9:898-9.
22. Tso JV, Cantu SM, Kim JH. Case series of coronary artery anomalies in athletes: challenges in clinical management and sports eligibility. JACC Case Rep 2022;4:1074-9.
23. 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.
24. Fritsch P, Ehringer-Schetitska D, Dalla Pozza R, et al. Cardiovascular pre-participation screening in young athletes: recommendations of the Association of European Paediatric Cardiology. Cardiol Young 2017;27:311-20.
25. Ebadi AA. Student athlete ECGs: case examples of normal and abnormal sport physical ECGs. Prog Pediatr Cardiol 2022;65:101519.
26. Mitten MJ, Maron BJ, Zipes DP. Eligibility and disqualification recommendations for competitive athletes with cardiovascular abnormalities: task force 15: legal aspects. J Am Coll Cardiol 2015;66:2447-50.
27. Anderson CL, Munawar S, Reilly L, et al. How functional genomics can keep pace with VUS identification. Front Cardiovasc Med 2022;9:900431.

28. Bourdon PC, Cardinale M, Murray A, et al. Monitoring athlete training loads: consensus statement. Int J Sports Physiol Perform 2017;12:S2161-70.
29. West AS, Clubb J, Torres-Ronda L, et al. More than a metric. Int J Sports Med 2021;42:300-6.
30. Bosch B, Mansell H. Interprofessional collaboration in health care: lessons to be learned from competitive sports. Can Pharm J 2015;148:176-9.
31. Casa DJ, Guskiewicz KM, Anderson SA, et al. National Athletic Trainers' Association position statement: preventing sudden death in sports. J Athl Train 2012;47:96-118.
32. Kerr ZY, Gildner P, Parker SK, et al. Sport culture and communication among middle school athletes, parents, and staff: a qualitative study. PLoS One 2023;18:e0282252.
33. Madias C, Maron BJ, Alsheikh-Ali A, et al. Commotio cordis. Indian Pacing Electrophysiol J 2007;7:235-9.

Table 1. Main causes of sudden cardiac death in athletes.

Category	Primary etiology	Gene(s) involved	Pathophysiological mechanism	Key diagnostic tools
Structural cardiomyopathies	Hypertrophic cardiomyopathy	*MYH7*, *MYBPC3*	Asymmetric hypertrophy, fibrosis, and adrenergically triggered arrhythmias	ECG, echocardiography, MRI, genetic testing
Arrhythmogenic cardiomyopathy	Arrhythmogenic right ventricular cardiomyopathy	*PKP2*, *DSG2*, *DSP*	Fibro-fatty myocardial replacement, exercise-induced arrhythmic risk	ECG, echocardiography, MRI, genetic testing
Channelopathies	Long QT syndrome, Brugada syndrome, CPVT	*KCNQ1*, *SCN5A*, *RYR2*	Ion channel dysfunction, catecholamine-triggered arrhythmias	ECG, exercise test, genetic analysis
Congenital coronary anomalies	Abnormal origin or interarterial course	–	Ischemia due to coronary compression during exertion	CT coronary angiography, cardiac MRI
Acquired/ mechanical causes	Myocarditis, commotio cordis	–	Post-viral fibrosis or blunt chest trauma	MRI, biomarkers, clinical history

Table 2. Comparison of screening paradigms in Italy, the US, and the hybrid proposal.

Feature	Italy (mandatory)	U.S. (voluntary)	Proposed hybrid
SCD mortality	≈0.4/100,000	≈1–2/100,000	Reduced, cost-balanced
Access/equity	Universal	Variable	Targeted universal
False positives	5–10%	Lower	Minimized via algorithm
Cost	Public burden	Private	Shared
Diagnostic yield	Moderate	Low	Optimized for risk groups