International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Scholarly Publisher
RS Global Sp. z O.O.
ISNI: 0000 0004 8495 2390

Dolna 17, Warsaw,
Poland 00-773
+48 226 0 227 03
editorial_office@rsglobal.pl

ARTICLE TITLE	THE TRANSFORMATION OF CARDIOVASCULAR SCREENING IN ATHLETES: THE MULTIMODAL ROLE OF ARTIFICIAL INTELLIGENCE IN DIFFERENTIATING CARDIAC PATHOLOGIES FROM PHYSIOLOGICAL ADAPTATION
DOI	https://doi.org/10.31435/ijitss.4(48).2025.4651
RECEIVED	16 November 2025
ACCEPTED	23 December 2025
PUBLISHED	29 December 2025
LICENSE	The article is licensed under a **Creative Commons Attribution 4.0 International License.**

THE TRANSFORMATION OF CARDIOVASCULAR SCREENING IN ATHLETES: THE MULTIMODAL ROLE OF ARTIFICIAL INTELLIGENCE IN DIFFERENTIATING CARDIAC PATHOLOGIES FROM PHYSIOLOGICAL ADAPTATION

Weronika Kozak *(Corresponding Author, Email: kozakweronikaa@gmail.com)*
University of Warmia and Mazury, Olsztyn, Poland
ORCID ID: 0009-0009-5605-2794

Aleksandra Grygorowicz
Medical University of Warsaw, Warsaw, Poland
ORCID ID: 0009-0002-7729-8178

Klaudia Baran
Medical University of Warsaw, Warsaw, Poland
ORCID ID: 0009-0004-9599-792X

Michał Ględa
Medical University of Lodz, Lodz, Poland
ORCID ID: 0009-0003-8148-160X

Michał Szyszka
Lower Silesian Center for Oncology, Pulmonology and Hematology, Wrocław, Poland
ORCID ID: 0009-0002-5307-1753

Weronika Radecka
Cardinal Stefan Wyszyński University, Warsaw, Poland
ORCID ID: 0009-0007-1116-398X

Agnieszka Szreiber
University of Warmia and Mazury, Olsztyn, Poland
ORCID ID: 0009-0006-3432-8284

Karol Grela
Medical University of Warsaw, Warsaw, Poland

Karolina Nowacka
Medical University of Gdańsk, Gdańsk, Poland
ORCID ID: 0009-0004-4933-755X

Kamil Jabłoński
Medical University of Silesia, Katowice, Poland
ORCID ID: 0009-0003-4682-2405

Anna Woźniak
Lazarski University, Warsaw, Poland
ORCID ID: 0009-0000-5065-6313

Karol Śliwa
University of Warmia and Mazury, Olsztyn, Poland
ORCID ID: 0009-0006-4181-9778

ABSTRACT

Purpose: The primary objective of this review is to evaluate the efficacy, clinical applications, and current limitations of Artificial Intelligence (AI) and Machine Learning (ML) in diagnosing cardiovascular diseases (CVD) among competitive athletes. Specifically, this study addresses the critical diagnostic challenge of differentiating benign physiological adaptations known as "athlete's heart" from potentially lethal pathologies, including cardiomyopathies and channelopathies, to prevent sudden cardiac death.
Materials and Methods: A systematic literature search was conducted across PubMed, Scopus, and Web of Science databases covering the period from 2000 to 2025. The review identified and synthesized 48 key studies utilizing AI algorithms—specifically deep learning applied to electrocardiography (AI-ECG) and automated imaging analysis (Echocardiography, CMR)—for the detection of Hypertrophic Cardiomyopathy (HCM), Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC), valvular anomalies, and inherited channelopathies. Diagnostic performance metrics were analyzed to compare AI methodologies against standard clinical criteria.
Results: Deep learning models applied to ECG demonstrate superior sensitivity (>90%) in detecting occult cardiomyopathies compared to traditional methods , while AI-enhanced imaging significantly improves the reproducibility of tissue characterization. AI algorithms, such as those analyzing phonocardiograms, show efficacy comparable to echocardiography in detecting valvular heart disease.
Conclusions: AI represents a paradigm shift in sports cardiology, offering potential for scalable and cost-effective screening. However, widespread clinical implementation is currently hindered by the "black box" nature of algorithms and the scarcity of large, athlete-specific training datasets. Future deployment requires explainable AI models validated on diverse athletic cohorts.

KEYWORDS

Cardiology, Athlete’s Heart, Artificial Intelligence, Sudden Cardiac Death, Cardiovascular Screening

CITATION

Weronika Kozak, Aleksandra Grygorowicz, Klaudia Baran, Michał Ględa, Michał Szyszka, Weronika Radecka, Agnieszka Szreiber, Karol Grela, Karolina Nowacka, Kamil Jabłoński, Anna Woźniak, Karol Śliwa. (2025). The Transformation of Cardiovascular Screening in Athletes: The Multimodal Role of Artificial Intelligence in Differentiating Cardiac Pathologies from Physiological Adaptation. *International Journal of Innovative Technologies in Social Science*. 4(48). doi: 10.31435/ijitss.4(48).2025.4651

Introduction

Cardiovascular diseases remain one of the leading causes of morbidity and mortality in young competitive athletes, with conditions such as hypertrophic cardiomyopathy (HCM), arrhythmogenic right ventricular cardiomyopathy (ARVC), valvular heart diseases**,** congenital coronary artery anomalies (CCAA), myocarditis, and inherited channelopathies representing the most frequent substrates of exercise-related sudden cardiac death. (Bhatia et al., 2022; Campuzano et al., 2012; Cotet et al., 2025; Gräni, 2018; Schwartz et al., 2017; Ullal et al., 2016)Despite major advances in sports cardiology, the early identification of these disorders continues to pose substantial challenges. Physiological cardiac remodeling induced by intensive training—characterized by chamber dilation, augmented left ventricular mass, increased vagal tone, and hemodynamic adaptations—often mimics or obscures pathological findings.(Augustine & Howard, 2018; Henning, 2024; Lauschke & Maisch, 2009; Palermi et al., 2023) As a result, traditional diagnostic tools such as electrocardiography (ECG) and echocardiography may have limited specificity and sensitivity in differentiating benign adaptive features from early or subtle manifestations of cardiovascular disease.(Cavarretta et al., 2024; D’Ascenzi et al., 2021)

Cardiac magnetic resonance (CMR), advanced echocardiographic techniques, and genetic testing have significantly improved diagnostic accuracy; however, their routine use in large athletic populations is constrained by cost, accessibility, and the need for specialist interpretation.(D’Ascenzi et al., 2021) Moreover,

several high-risk cardiovascular conditions—such as concealed long QT syndrome, early-stage ARVC, or congenital coronary anomalies—may remain undetected despite comprehensive evaluation.(Bos et al., 2021)

In recent years, artificial intelligence (AI) has emerged as a transformative tool with the potential to enhance cardiovascular screening and diagnostics.(Smaranda et al., 2024) Deep learning algorithms applied to ECG, cardiac imaging, auscultation, and wearable device data have demonstrated the ability to detect subtle morphological, electrical, or hemodynamic patterns that are often imperceptible to clinicians.(Zhou et al., 2025) Early studies suggest that AI-enhanced ECG can identify HCM even when the resting ECG appears normal, distinguish physiological from pathological hypertrophy, and improve detection of inherited arrhythmia syndromes.(Croon et al., 2025; Jiang et al., 2024; Ko et al., 2020) Similarly, AI-powered image analysis in echocardiography and CMR has shown promise in automated quantification of ventricular function, myocardial deformation, scar burden, and valvular abnormalities—yielding highly reproducible measurements with reduced inter-observer variability.(Bourfiss et al., 2023; Duffy et al., 2022; Fahmy et al., 2022; Monopoli et al., 2025)

Despite these advances, significant limitations persist. Many AI models are developed using datasets derived from general clinical populations, which may not accurately represent the unique physiological characteristics of trained athletes.(Bellfield et al., 2022) Furthermore, rarer conditions—such as congenital valvular defects or ARVC—lack large, well-annotated datasets suitable for robust training and validation of machine learning systems. As a result, the generalizability of AI-based tools to athletic cohorts remains incompletely established.(Aljehani et al., 2023; Jone et al., 2022; Kübler et al., 2021)

This article provides a comprehensive overview of the current and emerging role of artificial intelligence in the diagnosis of cardiovascular diseases relevant to competitive athletes. It examines the capabilities and limitations of AI-based methods across key clinical entities—including hypertrophic cardiomyopathy, ARVC, congenital valvular heart disease, congenital coronary anomalies, myocarditis, and inherited channelopathies—while highlighting the diagnostic challenges inherent to the athletic population. Particular emphasis is placed on multimodal AI approaches that integrate ECG, imaging, and physiological signals, as well as on the prerequisites for safe and effective clinical implementation in sports medicine.(Zhou et al., 2025)

Methodology

To compile this comprehensive overview, a systematic literature review was conducted. The PubMed, Scopus, and Web of Science databases were searched covering the period from January 2000 to February 2025. The search strategy employed a combination of Medical Subject Headings (MeSH) and text keywords, including: “Cardiology”, “Athlete’s Heart”, “Artificial Intelligence”, “Sudden Cardiac Death”, “Cardiovascular Screening”.

1. Investigated the pathophysiology of exercise-related cardiac remodeling and its differentiation from pathology (e.g., HCM, ARVC, DCM).
2. Evaluated the performance of AI algorithms (CNNs, deep learning) applied to ECG, Echocardiography, CMR, or digital auscultation.
3. Addressed specific diagnostic challenges in athletic populations, such as the "gray zone" of hypertrophy or repolarization anomalies.

Studies were selected based on their methodological rigor and relevance to the specific clinical entities discussed (HCM, ARVC, CCAA, Valvular Disease, Myocarditis, and Channelopathies). A total of 48 key references were synthesized to assess the diagnostic accuracy (sensitivity, specificity, AUC) and clinical limitations of current AI tools.

Results

1. Hypertrophic Cardiomyopathy

Hypertrophic cardiomyopathy (HCM) in athletes represents one of the most significant challenges in modern sports medicine. This condition, characterized by abnormal thickening of the myocardial walls, is the most common cause of sudden cardiac death in young, physically active individuals.(Malhotra & Sharma, 2017; Ullal et al., 2016) Early identification of HCM is crucial; however, in athletes, conventional diagnostic methods are frequently insufficient because the physiological adaptations induced by intensive training may resemble the disease phenotype.(Henning, 2024; Kübler et al., 2021; Lauschke & Maisch, 2009) In recent years, interest in artificial intelligence (AI) techniques has been steadily increasing, as they hold the potential to facilitate the differentiation of hypertrophic cardiomyopathy from the so-called “athlete’s heart,” thereby improving the diagnostic process and enhancing athlete safety.(Siontis et al., 2024)

Pathophysiological Mechanisms

From a pathophysiological perspective, HCM is fundamentally distinct from the hemodynamic adaptations observed in the "athlete's heart." It is primarily a genetic disorder of the cardiac sarcomere, most commonly inherited in an autosomal dominant pattern caused by mutations in genes encoding contractile proteins, such as β-myosin heavy chain (*MYH7*) and cardiac myosin-binding protein C (*MYBPC3*).(Malhotra & Sharma, 2017)

The critical histological hallmark that differentiates HCM from physiological remodeling is myocyte disarray—a chaotic architectural arrangement of cardiomyocytes—accompanied by increased interstitial fibrosis and intramural coronary arteriole dysplasia (small vessel disease). (Henning, 2024; Lauschke & Maisch, 2009) While athletic training induces a harmonic, reversible increase in myocyte size with preserved cellular alignment, HCM presents with structural disorganization that disrupts normal electrical propagation.

This combination of cellular disarray and replacement fibrosis creates a heterogeneous myocardial substrate. This substrate facilitates re-entrant electrical circuits, thereby predisposing individuals to malignant ventricular tachyarrhythmias, which are the primary mechanism of sudden cardiac death in these athletes. (Bhatia et al., 2022; Fahmy et al., 2022) Anatomically, this often manifests as asymmetric hypertrophy (predominantly affecting the interventricular septum), which may lead to dynamic left ventricular outflow tract (LVOT) obstruction and diastolic dysfunction—features rarely observed in pure physiological hypertrophy. (Augustine & Howard, 2018)

HCM vs. Athlete's Heart – Diagnostic Challenges

The athlete's heart undergoes several physiological adaptations in response to systematic training, including mild left ventricular hypertrophy, chamber enlargement, and resting bradycardia. Although physiological, these changes may mimic the appearance of HCM, particularly in the early stages of the disease or in cases of mild hypertrophy. Distinguishing physiological adaptation from pathology remains a major challenge.(Augustine & Howard, 2018; Lauschke & Maisch, 2009) Traditional diagnostic tests such as ECG and echocardiography do not always provide conclusive information—especially in endurance athletes, in whom myocardial wall thickness may reach borderline values.(Cavarretta et al., 2024; Henning, 2024)

Importance of Early Diagnosis and Associated Risks in Athletes

In some athletes, the first manifestation of HCM may be sudden cardiac death, typically during intense physical exertion.(Bhatia et al., 2022) For this reason, sports federations worldwide recommend routine screening that includes ECG and echocardiography.(D'Ascenzi et al., 2021). Difficulties occur when it is challenging to differentiate physiological adaptation from structural pathology, which may require advanced diagnostic tools such as cardiac magnetic resonance imaging (CMR) or genetic testing. These are precisely the situations in which artificial intelligence is assuming an increasingly important role.(Baba Ali et al., 2024; Lauschke & Maisch, 2009)

AI in HCM Diagnostics – New Opportunities

Artificial intelligence—particularly deep learning techniques—has been transforming cardiac diagnostics in recent years. One of the first areas in which AI demonstrated notable effectiveness is ECG analysis. AI-ECG algorithms trained on hundreds of thousands of recordings can detect subtle patterns imperceptible to the human eye. Studies from institutions such as the Mayo Clinic have shown that AI can identify HCM with very high sensitivity and specificity, even when a standard ECG appears normal.(Ko et al., 2020)

Another rapidly developing area is AI-based interpretation of cardiac imaging. Deep learning algorithms used in echocardiography and CMR can precisely assess wall thickness, chamber volumes, myocardial motion, and detect scarring or fibrosis.(Fahmy et al., 2022) In athletes, AI can assist in differentiating the uniform, symmetrical hypertrophy typical of the athlete's heart from the asymmetric hypertrophy characteristic of HCM. Importantly, these algorithms provide highly reproducible analyses, reducing inter-observer variability.(Bellfield et al., 2022; Duffy et al., 2022; Yu et al., 2022)

Mobile Diagnostics and the Future of Screening

With the advancement of portable medical technologies such as smartwatches and mobile electrocardiographs, ECG recordings can now be obtained during routine training activities. Integrating these data with AI algorithms may enable continuous monitoring and early detection of concerning abnormalities. Ongoing clinical studies are investigating whether AI analyzing single-lead ECG recordings (e.g., from a smartwatch) can detect HCM and differentiate it from training-related physiological adaptations.(Croon et al., 2025)

2. Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is a genetic disorder of the myocardium characterized by fibrofatty replacement of cardiac muscle, impaired right ventricular wall motion, and a propensity for malignant arrhythmias. (Campuzano et al., 2012) In athletes, the diagnosis of ARVC is particularly challenging, as physiological adaptations to intensive training—such as right ventricular cavity enlargement or repolarization changes—may mimic pathological features, creating a diagnostic "grey zone."(Campuzano et al., 2012) In this context, AI-based technologies capable of analyzing both ECG signals and cardiac imaging (CMR, echocardiography) may offer additional tools for early disease detection and risk stratification.

Pathophysiological Mechanisms and the Impact of Exercise

From a pathophysiological standpoint, ARVC is predominantly a disease of the **desmosome**, a protein complex responsible for mechanical cell-to-cell adhesion. In the majority of cases, the condition is driven by mutations in genes encoding desmosomal proteins, such as plakophilin-2 (*PKP2*) or desmoglein-2 (*DSG2*), which compromise the structural integrity of the intercalated discs.(Corrado et al., 2017)

The athletic population is uniquely vulnerable due to the interaction between this genetic substrate and mechanical stress. Intensive physical exertion increases right ventricular wall stress, which disrupts these fragile desmosomal connections, triggering myocyte necrosis and an inflammatory response.(Campuzano et al., 2012) This process leads to the hallmark fibrofatty replacement of the myocardium, which creates a substrate for re-entrant ventricular arrhythmias. Crucially, molecular studies suggest that "electrical remodeling"—characterized by the downregulation of gap junction proteins (Connexin 43)—often precedes macroscopic structural damage.(Haq et al., 2024) This dissociation explains why electrical instability may manifest on the ECG (and potentially be detected by AI) before overt structural defects are visible on standard cardiac imaging.

AI-Enhanced Electrocardiography as an Emerging Diagnostic Modality for ARVC

One of the earliest and most significant examples of the translational application of artificial intelligence in ARVC diagnostics is the study describing the "AI-enhanced ECG"—a deep-learning model capable of detecting ARVC-related features from a standard 12-lead electrocardiogram. Haq et al. demonstrated that a neural network trained on datasets of patients with confirmed ARVC can distinguish affected individuals from controls with good diagnostic performance and may serve as a rule-out biomarker in clinical populations. This study suggests that AI-ECG algorithms are capable of identifying subtle morphological and temporal patterns that are either imperceptible or inconsistently recognized by human readers.(Haq et al., 2024)

Machine Learning–Enhanced CMR for Automated Assessment of Right Ventricular Dysfunction in ARVC

Cardiac magnetic resonance imaging (CMR) remains the gold standard for the assessment of right ventricular morphology and function, as well as for the identification of tissue characteristics in ARVC. In this field, studies have increasingly applied machine learning techniques for the automated quantification of myocardial motion, regional wall motion abnormalities, and strain tracking. For example, research on deep learning–based automated strain analysis has demonstrated significant differences in peak strain parameters between patients with ARVC and healthy controls, suggesting the potential for automated detection of pathological right ventricular dyskinesia. Such algorithms enhance the objectivity of regional wall motion assessment—an aspect that is critical for the diagnosis of ARVC.(Bourfiss et al., 2022, 2023)

Limitations in Data Availability for AI-Based ARVC Research in Athletes

It should be emphasized, however, that current knowledge has limitations. ARVC is a relatively rare disease, which hampers the collection of large, balanced training datasets—particularly those including athletes exhibiting physiological right ventricular remodeling.(Aljehani et al., 2023) Nevertheless, it is important to acknowledge the limitations of the current evidence. ARVC is a relatively rare condition, which complicates the assembly of large, well-balanced training datasets—especially those incorporating athletes with physiological right ventricular remodeling. (Kübler et al., 2021)

3. Congenital Valvular Heart Disease

Congenital valvular heart disease (CVHD) constitutes a significant portion of developmental cardiac disorders. Despite advances in prenatal and pediatric diagnostics, many valvular defects remain undiagnosed or overlooked—particularly in adults or athletes whose hearts have undergone hemodynamic adaptations due to intensive physical activity.(Viera et al., 2024) In this context, artificial intelligence (AI)–based technologies provide promising tools that may facilitate early detection and management of CVHD.

Pathophysiological Mechanisms and Hemodynamic Impact

The pathophysiology of CVHD in athletes is defined by the interaction between altered valve mechanics and the high-flow hemodynamic state induced by exercise. The most common entity is the bicuspid aortic valve (BAV), affecting 1–2% of the population. In BAV, the abnormal leaflet architecture generates turbulent blood flow and eccentric shear stress on the aortic wall, predisposing the athlete not only to valvular stenosis or regurgitation but also to aortopathy and potential dissection. (Jone et al., 2022; Viera et al., 2024)Under resting conditions, mild valvular defects may remain hemodynamically silent. However, during intense physical exertion, cardiac output can increase five-fold (>30 L/min). In the presence of valvular stenosis (e.g., aortic or pulmonary), this increased flow necessitates supra-physiological intraventricular pressures to maintain perfusion, leading to concentric hypertrophy and potential subendocardial ischemia.(Palermi et al., 2023) Conversely, in regurgitant lesions (e.g., mitral valve prolapse), the "volume load" of exercise compounds the regurgitant volume, accelerating ventricular dilation and increasing wall tension. Over time, these mechanical stressors can trigger maladaptive remodeling, fibrosis, and electrical instability, distinguishing the pathological substrate from benign athletic adaptation. (Jone et al., 2022)

AI-Enhanced Auscultation for the Detection of Left-Sided Valvular Heart Disease

Recent years have provided substantial evidence that AI can effectively support the diagnosis of valvular heart disease, both acquired and congenital. In a groundbreaking study published in 2025, researchers employed an “AI stethoscope” (an electronic stethoscope combined with a machine learning algorithm) which, after collecting data from 514 patients, was able to automatically identify left-sided valvular heart disease. In the testing phase, the algorithm achieved reasonable sensitivity and specificity (sensitivity ~70%, specificity ~74%, AU-ROC ~0.76), suggesting that a simple, rapid, and cost-effective method—analogous to auscultation—could serve as a screening tool.(Zhou et al., 2025)

Deep Learning in ECG and Multimodal Imaging

Another promising direction involves systems based on conventional electrocardiography (ECG). A 2025 study demonstrated that AI-ECG models could predict the risk of developing clinically significant valvular regurgitation (mitral, aortic, or tricuspid) by leveraging large datasets of paired ECG and echocardiography recordings, encompassing nearly one million ECG-echo pairs. This approach opens the possibility that, in athletic populations where echocardiography or CMR is not routinely performed, AI-ECG could serve as a cost-effective screening tool.(Liang et al., 2025; Lin et al., 2024)

In the realm of cardiac imaging, AI has also demonstrated substantial potential. Recent studies indicate that AI algorithms can automatically segment valvular structures, analyze their anatomy and function, and assist in classifying patients according to their risk of disease progression. This represents a significant advance, as the assessment of valvular defects is becoming less dependent on operator experience and more objective and reproducible.(Huang et al., 2023; Monopoli et al., 2025)

Moreover, research on digital auscultation and heart sound analysis using deep learning shows that AI can match or even surpass the performance of clinicians. In the referenced study, the algorithm detected moderate-to-severe aortic stenosis with a sensitivity of 93.2%, whereas the expert annotators demonstrated sensitivities ranging from 82.5% to 97.5% depending on the reader. In the case of mitral regurgitation, the algorithm achieved a sensitivity of 66.2%, compared with 58.6% to 82.8% for the clinicians.(Chorba et al., 2021)

Diagnostic Limitations in the Athletic Population

Despite promising results, the application of AI to valvular heart disease—particularly in specialized populations such as athletes—presents important challenges and limitations. First, most available studies focus on acquired valvular disorders (e.g., degenerative regurgitation, calcification) rather than congenital defects, meaning that algorithms may not be optimized to detect subtle developmental anomalies characteristic of CVHD.(Jone et al., 2022)

Second, logistical and epidemiological challenges exist: congenital valvular defects are relatively rare in adult populations, making it difficult to assemble large, well-annotated training datasets that include physically active individuals or athletes. Without appropriately diverse data, machine learning models are prone to overfitting or limited generalizability.(Jone et al., 2022)

Finally, cardiac adaptations in athletes (remodeling, chamber enlargement, hemodynamic changes) may mask valvular abnormalities or produce variable echocardiographic appearances, complicating accurate classification. In such cases, a multimodal approach—integrating AI-ECG, AI-auscultation, and AI-echo/CMR—may prove crucial.(Palermi et al., 2023)

4. Cardiomyopathies and Myocarditis

Cardiomyopathies—including hypertrophic cardiomyopathy, dilated cardiomyopathy, and arrhythmogenic right ventricular cardiomyopathy—and myocarditis are major causes of sudden cardiac death and exercise restrictions in young and middle-aged athletes. (Cotet et al., 2025)Their diagnostic workup relies on a combination of clinical history, electrocardiography, echocardiography, and advanced cardiac imaging, particularly cardiac magnetic resonance (CMR), while indeterminate cases may require histopathological confirmation through endomyocardial biopsy.(Cotet et al., 2025; Rapezzi et al., 2013)

Pathophysiological Mechanisms: Inflammation vs. Adaptation

The pathophysiology of myocarditis and non-hypertrophic cardiomyopathies differs fundamentally from the physiologic hypertrophy of the athlete's heart. Myocarditis is primarily an inflammatory disease of the myocardium, typically triggered by viral infection (e.g., Coxsackievirus, Parvovirus B19, or SARS-CoV-2). The disease progresses through three phases: acute viral injury, immunological activation, and—in some cases—chronic fibrosis.(Tschöpe et al., 2021)

In athletes, this process is critically dangerous because intense physical exertion during the acute phase can exacerbate viral replication and enhance the inflammatory response, leading to extensive myocyte necrosis and accelerated arrhythmogenesis. This results in myocardial edema and, subsequently, replacement fibrosis (scarring), which serves as a permanent substrate for re-entrant ventricular tachycardia.(Cotet et al., 2025; Łajczak & Jóźwik, 2024)

In contrast, Dilated Cardiomyopathy (DCM) is often caused by cytoskeletal or sarcomeric gene mutations (e.g., *TTN*, *LMNA*) leading to cardiomyocyte elongation, wall thinning ("eccentric hypertrophy"), and depressed systolic function. Unlike the reversible chamber dilation seen in endurance athletes (where ejection fraction remains normal or slightly lower at rest but recruits normally during exercise), pathological DCM is characterized by reduced contractile reserve and interstitial fibrosis.(Rapezzi et al., 2013)

AI-Enhanced Multimodal Diagnostics

The Role of AI in CMR and Tissue Characterization Cardiac magnetic resonance (CMR) remains the gold standard for non-invasive tissue characterization, utilizing the updated Lake Louise Criteria to detect myocardial edema (T2-weighted imaging) and fibrosis (Late Gadolinium Enhancement, LGE). Artificial intelligence has significantly enhanced this modality. Deep learning algorithms are now capable of automating the segmentation of the myocardium and quantifying LGE burden with precision that matches or exceeds human experts. For instance, AI models described by Zhang et al. (2024) and Wang et al. (2024) automate the measurement of T1 and T2 mapping values, allowing for the objective detection of diffuse fibrosis or subtle edema that might be missed by visual inspection alone. This "AI-CMR" approach is particularly valuable in distinguishing the physiological T1 mapping values of a trained athlete from the pathological elevation seen in early diffuse fibrosis. (Shyam-Sundar et al., 2024; Wang et al., 2024; Zhang et al., 2024)

Multimodal Integration (ECG + Imaging) Beyond imaging, AI systems have demonstrated capabilities in analyzing 12-lead ECG signals (AI-ECG) to detect subtle repolarization abnormalities associated with early cardiomyopathy. Multimodal models integrating AI-ECG, AI-auscultation, and AI-echo/CMR theoretically enhance the accuracy of differentiating physiological athletic adaptation from pathology by cross-referencing electrical anomalies with structural data. (Łajczak & Jóźwik, 2024)

Diagnostic Limitations in Athletic Cohorts

Despite these technological strides, real-world implementation remains constrained by several factors. First, the physiological cardiac adaptations to training—such as chamber enlargement and increased vagal tone—can mimic the "dilated" phenotype of DCM or the inflammatory changes of myocarditis, confusing AI classifiers trained on non-athletic populations. (Baba Ali et al., 2024; Smaranda et al., 2024)Furthermore, the low prevalence of acute myocarditis and specific cardiomyopathies in athletic cohorts creates a data scarcity problem. This leads to a lack of robust, athlete-specific training datasets, increasing the risk of overfitting and reducing the generalizability of AI tools to the unique physiology of the elite athlete. (Zhang et al., 2024) Consequently, current AI applications serve as decision-support tools rather than autonomous diagnostic systems.

5. Congenital Coronary Artery Anomalies

Congenital coronary artery anomalies (CCAA) encompass abnormal origin, course, or departure from the typical anatomical configuration of the coronary arteries.(Lau et al., 2023) In athletes, CCAA represents one of the most deceptive causes of sudden cardiac death (SCD), as resting 12-lead ECGs are frequently normal. The most clinically relevant variants include an anomalous origin with an interarterial course (between the aorta and pulmonary artery) or an acutely angulated take-off. (Basso et al., 2000; Pérez-Pomares et al., 2016)

Pathophysiological Mechanisms: Dynamic Ischemia

Unlike atherosclerotic coronary disease, the pathophysiology of CCAA in athletes is functional and dynamic. The primary mechanism of ischemia is mechanical compression of the anomalous vessel during strenuous physical exertion. In the high-risk variant—Anomalous Coronary Artery from the Opposite Sinus (ACAOS) with an interarterial course—the vessel runs between the aorta and the pulmonary artery. (Gräni, 2018)

During intense exercise, the expansion of the aortic root and pulmonary trunk can extrinsically compress the anomalous vessel. Furthermore, these anomalies often feature a slit-like ostium (an acute take-off angle) and a proximal intramural course (running within the aortic wall). As cardiac output and stroke volume increase, the aortic wall stretches, causing the slit-like orifice to collapse in a valve-like manner, severely compromising coronary blood flow precisely when myocardial oxygen demand is maximal. This acute mismatch leads to extensive myocardial ischemia and lethal ventricular arrhythmias. (Angelini, 2007; Basso et al., 2000)

AI-Enhanced Imaging and Diagnostics

Diagnostic evaluation relies primarily on anatomic imaging—most commonly coronary computed tomography angiography (CCTA) or invasive coronary angiography. (Neves et al., 2015) In this domain, Artificial Intelligence is emerging as a powerful adjunct. Deep learning algorithms applied to CCTA can now perform automated vessel segmentation and centerline extraction, significantly reducing the time required for post-processing and reconstruction. (Lau et al., 2023)

AI models are being trained to automatically detect the origin of coronary arteries and classify their course (e.g., retroaortic vs. interarterial) with high accuracy. This "AI-reader" capability acts as a safety net, flagging potential anomalies in large datasets that might be overlooked by human readers focused on other findings (e.g., calcium scoring). (Lau et al., 2023)

Clinical Implications and Diagnostic Limitations

Despite these technological advances, significant limitations persist. The lack of large, athlete-specific cohorts with confirmed CCAA limits the training data for AI models, meaning current algorithms are largely derived from general clinical populations. (Lau et al., 2023) Moreover, while AI can identify anatomy, it currently struggles to predict the functional significance of a lesion (i.e., whether it causes ischemia), which still requires stress testing or fractional flow reserve (FFR) assessment.

From a practical standpoint, in suspected "malignant" CCAA variants (e.g., the left coronary artery originating from the right sinus with an interarterial course), restriction of competitive activity is advised until a full diagnostic workup is completed. Decisions regarding return-to-play or surgical correction should be made in specialized centers, as AI tools currently serve only as anatomical detectors, not functional risk stratifiers. (Tso et al., 2020)

6. Long QT Syndrome and Other Channelopathies

Long QT syndrome (LQTS) and other cardiac channelopathies (e.g., Brugada syndrome, catecholaminergic polymorphic ventricular tachycardia [CPVT]) are inherited disorders of ion channel function leading to abnormal cardiac repolarization and an increased susceptibility to torsades de pointes and other life-threatening ventricular arrhythmias.(Schwartz et al., 2017)

Pathophysiological Mechanisms: Ion Channels and Adrenergic Stress

The pathophysiology of channelopathies is defined by the failure of transmembrane ion currents to maintain electrical stability, particularly under stress. In Long QT Syndrome (LQTS), mutations typically cause a "loss of function" in potassium channels or a "gain of function" in sodium channels. This prolongs the action potential duration (APD) and delays repolarization.(Wilde et al., 2022)

In the athletic context, the mechanism of repolarization reserve is critical. Normally, during intense exercise, sympathetic stimulation (adrenergic surge) shortens the QT interval to match the rapid heart rate. In athletes with LQTS (particularly type 1, LQT1), the impaired potassium channels current prevents this necessary shortening. This maladaptation leads to a paradoxical prolongation of the QT interval during tachycardia, resulting in Early Afterdepolarizations (EADs). These EADs can trigger "R-on-T" phenomena,

precipitating polymorphic ventricular tachycardia (Torsades de Pointes) and sudden cardiac death. (Priori et al., 2013; Schwartz et al., 2017)

Similarly, in CPVT, the pathology lies in intracellular calcium handling (mutations in RyR2). Adrenergic stress causes calcium leak from the sarcoplasmic reticulum, triggering delayed afterdepolarizations (DADs) exclusively during exertion, often in structurally normal hearts.(Priori et al., 2013)

AI-Enhanced Diagnostics and Genotype Prediction

Standard diagnostic assessment includes a 12-lead ECG with QTc measurement, yet up to 25–40% of genotype-positive patients may present with a normal resting QTc ("concealed LQTS"). (Schwartz et al., 2009) Here, Artificial Intelligence has shown transformative potential.

Deep learning models applied to raw 12-lead ECG voltage data have demonstrated the ability to detect these concealed cases. A landmark study utilizing a convolutional neural network (CNN) achieved an AUC of 0.90 in identifying patients with congenital LQTS, even when standard QTc measurements were within normal limits (<450 ms). Furthermore, AI models have shown the capacity to distinguish between specific genetic subtypes (e.g., LQT1 vs. LQT2) based on subtle T-wave morphological patterns that are difficult for human experts to quantify.(Bos et al., 2021; Jiang et al., 2024) These tools could theoretically serve as a scalable "pre-genetic" screening method.

Clinical Implications and Limitations

Despite these capabilities, robust validation in large, independent athlete cohorts is needed before AI-ECG can be adopted for routine pre-participation screening.(Drezner et al., 2017) The primary limitation is that most AI models are trained on clinical populations (often symptomatic patients from genetic registries), and their performance in the general athletic population—characterized by vagal-induced repolarization changes—remains less established.(Bos et al., 2021)

Practically, in athletes with suspected LQTS, decisions regarding competitive participation should remain comprehensive, considering QTc duration, history of syncope, genotype, and treatment status (e.g., β-blocker adherence). AI-ECG tools currently support early triage but should not replace evaluation by an electrophysiology specialist.(Johnson & Ackerman, 2013; Wilde et al., 2022)

Detailed Characterization of Algorithm Performance in Key Studies

The application of artificial intelligence in sports cardiology demonstrates tangible benefits across four primary clinical domains: hypertrophic cardiomyopathy (HCM), arrhythmogenic right ventricular cardiomyopathy (ARVC), valvular heart disease, and channelopathies.

1. Hypertrophic Cardiomyopathy (HCM) vs. Athlete's Heart

The primary challenge in this domain is differentiating physiological left ventricular hypertrophy (LVH) in athletes from mild phenotypes of HCM.

- AI-ECG Analysis: Studies utilizing convolutional neural networks (CNNs) have demonstrated the superiority of AI over traditional interpretation. A model developed by Ko et al. (2020) based on 12-lead ECG analysis achieved an AUC of 0.96 in detecting HCM, identifying the disease even in patients with clinically normal ECGs according to standard criteria.(Ko et al., 2020)
- Imaging (CMR and Echocardiography): In cardiac imaging, radiomic techniques (texture analysis) allow for the detection of interstitial fibrosis invisible to the human eye. Fahmy et al. (2022) demonstrated that deep learning algorithms applied to cardiac magnetic resonance (CMR) can automatically quantify late gadolinium enhancement (LGE) with high reproducibility, which is critical for sudden cardiac death risk stratification.(Fahmy et al., 2022)

2. Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)

The diagnosis of ARVC is complicated by the physiological enlargement of the right ventricle observed in endurance athletes.

- AI-ECG: The model described by Haq et al. (2024) demonstrated the ability to differentiate patients with ARVC from healthy controls based on subtle depolarization and repolarization changes often overlooked by clinicians.(Haq et al., 2024)
- CMR Automation: Research by Bourfiss et al. (2022, 2023) proved that automatic segmentation of the right ventricle and feature-tracking strain analysis by AI eliminate inter-observer variability, a major source of misdiagnosis when relying on the Padua Criteria.(Bourfiss et al., 2022, 2023)

3. Valvular Heart Disease and Auscultation

Recent advancements in digital auscultation and AI-ECG pave the way for cost-effective screening.

- Digital Stethoscope: In a study by Zhou et al. (2025), an AI algorithm analyzing phonocardiogram (PCG) data was utilized to detect left-sided valvular heart disease. This study employed a machine learning

algorithm integrated with an electronic stethoscope ("AI stethoscope") on a cohort of 514 patients. In the testing phase, the model achieved an Area Under the Receiver Operating Characteristic curve (AU-ROC) of 0.76 for the general detection of left-sided valvular heart disease. Detailed efficacy analysis demonstrated a sensitivity of 70.00% and specificity of 73.68% for identifying left-sided VHD. It is worth noting that higher sensitivities for specific defects, such as 93.2% for moderate-to-severe aortic stenosis, have been reported in related deep learning studies (e.g., Chorba et al., 2021)) referenced by the authors.(Chorba et al., 2021; Zhou et al., 2025)

- ECG Prediction: Liang et al. (2025) demonstrated that AI can predict the presence of significant aortic or mitral regurgitation based solely on ECG waveforms, potentially serving as a triage tool prior to referral for echocardiography. (Liang et al., 2025)

4. Channelopathies (Long QT Syndrome)

Standard QTc measurements often fail in patients with "concealed" Long QT Syndrome (LQTS).

- A study by Bos et al. (2021) from the Mayo Clinic showed that a neural network could identify patients with congenital LQTS even when their resting QTc was within normal limits (<450 ms), achieving an AUC of 0.90. This represents a breakthrough in detecting athletes at risk of sudden cardiac death (SCD) who would otherwise pass standard screening.(Bos et al., 2021)

Table 1. Summary of diagnostic performance of selected AI models in cardiology (based on analyzed sources)

Clinical Entity	AI Modality	Key Source Study	Performance Metric (AUC / Sensitivity)	Clinical Application
HCM	12-lead AI-ECG	(Ko et al., 2020)	AUC: 0.96	Detection of HCM in "normal" ECGs
Concealed LQTS	12-lead AI-ECG	(Bos et al., 2021)	AUC: 0.90	Identification of LQTS genotype with normal QTc
ARVC	AI-ECG	(Haq et al., 2024)	High precision (see text)	Differentiation of ARVC from RV adaptation
Valvular Disease	Digital Stethoscope	(Zhou et al., 2025)	Sensitivity: ~70% (overall), >90% (severe AS)	Mass screening in sports
Fibrosis (LGE)	CMR Deep Learning	**(Fahmy et al., 2022)**	High correlation with experts	Automated SCD risk assessment

Discussion

Bridging the Diagnostic "Gray Zone" The central challenge in sports cardiology remains the differentiation of physiological adaptation ("athlete's heart") from pathological remodeling. As highlighted by Henning (2024) and Lauschke & Maisch (2009), the overlap in structural features—such as left ventricular hypertrophy and chamber dilation—creates a diagnostic "gray zone" where traditional criteria often lack specificity. (Henning, 2024; Lauschke & Maisch, 2009)

This review confirms that AI represents a paradigm shift in addressing this ambiguity. By analyzing non-linear relationships in data, AI models can identify microstructural and electrical signatures of disease that precede macroscopic remodeling.

Enhanced Sensitivity via Deep Learning A recurring finding across clinical entities is the superior sensitivity of Deep Learning compared to standard clinical markers.

- In Electrocardiography (AI-ECG): The work by Bos et al. (2021) demonstrates that AI can detect "invisible" pathologies, such as concealed Long QT Syndrome or early-stage HCM, from a standard 12-lead ECG with AUCs exceeding 0.90. This suggests that AI extracts sub-threshold morphological data (e.g., subtle T-wave variances) that human readers miss.(Bos et al., 2021)
- In Imaging (AI-CMR/Echo): AI eliminates inter-observer variability, a critical flaw in manual assessment. Automated segmentation and tissue characterization (e.g., LGE quantification or strain analysis) provide objective metrics for risk stratification in ARVC and HCM. (Bourfiss et al., 2023; Fahmy et al., 2022)

The Promise of Multimodal Integration Current literature suggests that the future of screening lies in multimodal integration. Single-modality assessments are often insufficient; for instance, anatomic imaging in CCAA identifies the lesion but not the ischemia (Tso et al., 2020).

Combining AI-ECG (electrical substrate), AI-Stethoscope (hemodynamic turbulence), and AI-Imaging (structural fibrosis) could theoretically construct a holistic "Digital Twin" of the athlete's heart, offering a higher predictive value for Sudden Cardiac Death than any single test. (Palermi et al., 2023; Zhou et al., 2025). Key components include:

- AI-ECG: analyzing the electrical substrate.
- AI-Stethoscope: detecting hemodynamic turbulence.
- AI-Imaging: quantifying structural fibrosis.

Critical Limitations: The Data Gap Despite the high performance of AI in *silico* studies, clinical translation to sports medicine is hindered by significant data biases.

1. Generalizability: Most high-performing algorithms (e.g., for HCM or LQTS) were trained on general clinical populations or disease registries, not on elite athletes (Bellfield et al., 2022). The unique vagal tone and repolarization patterns of athletes may lead to false positives in models not calibrated for this specific physiology.

2. Scarcity of Rare Conditions: Diseases like ARVC and Congenital Coronary Artery Anomalies (CCAA) are rare, preventing the aggregation of the massive datasets required to train robust Deep Learning models without overfitting (Aljehani et al., 2023; Lau et al., 2023).

3. "Black Box" Nature: The lack of explainability in neural networks remains a barrier. In the context of disqualifying a young athlete from competition, clinicians require interpretable features rather than opaque probability scores. (Smaranda et al., 2024).

Ethical and Legal Implications of AI in Sports Qualification

The deployment of AI in pre-participation screening introduces complex bioethical dilemmas. The primary concern is the potential for unwarranted disqualification ("false positives"). Algorithms trained on general populations may misinterpret athlete-specific adaptations—such as pronounced sinus bradycardia or early repolarization—as pathological, leading to unnecessary exclusion from competition. This carries profound consequences for a young athlete's career, scholarship opportunities, and mental health.

Conversely, false-negative results in high-risk conditions like ARVC could lead to preventable sudden cardiac death, raising significant legal questions regarding liability. If an AI tool clears an athlete who subsequently suffers a cardiac arrest, does liability rest with the physician, the algorithm developer, or the sports organization? Current consensus suggests that AI must remain a "Clinical Decision Support System" (CDSS) rather than an autonomous gatekeeper. The final decision on return-to-play must involve shared decision-making between the expert cardiologist and the athlete, utilizing AI as an adjunctive data point rather than a definitive verdict.

Conclusions

1. Transformative Screening Potential: Artificial Intelligence, particularly AI-ECG, has demonstrated the ability to detect occult cardiovascular diseases (such as concealed LQTS and early HCM) with significantly higher sensitivity than traditional manual interpretation. This positions AI as a powerful, cost-effective "triage" tool for mass pre-participation screening.

2. Objectivity in Imaging: AI-enhanced imaging (CMR and Echocardiography) provides highly reproducible, automated quantification of myocardial strain, fibrosis, and wall thickness, reducing the subjectivity inherent in the assessment of the "athlete's heart."

3. Pathophysiological Insight: AI models can identify disease-specific patterns—such as electrical remodeling in ARVC or micro-architectural disarray in HCM—that reflect the underlying pathophysiology (e.g., desmosomal disruption or sarcomeric mutation) before overt structural damage occurs.

4. Necessity for Athlete-Specific Validation: To ensure safety and prevent unwarranted disqualifications, AI models must be validated on large, diverse cohorts of elite athletes. Current models trained on general populations risk misinterpreting physiological adaptations (e.g., vagal bradycardia) as pathological.

5. Decision Support, Not Replacement: At the current stage of technological maturity, AI should serve as a Clinical Decision Support System (CDSS) for the sports cardiologist, facilitating early detection and standardized risk stratification, but not replacing the comprehensive clinical judgment required for return-to-play decisions.

Disclosures
Funding Statement: The article did not receive any funding.
Institutional Review and Board Statement: Not applicable.
Informed Consent Statement: Not applicable.
Data Availability Statement: Not applicable.
Conflict of Interest Statement: No conflicts of interest to declare.

REFERENCES

1. Aljehani, A., Kew, T., Baig, S., Cox, H., Sommerfeld, L. C., Ensam, B., Kalla, M., Steeds, R. P., & Fabritz, L. (2023). Characterisation of patients referred to a tertiary-level inherited cardiac condition clinic with suspected arrhythmogenic right ventricular cardiomyopathy (ARVC). *BMC Cardiovascular Disorders*, *23*(1), 14. https://doi.org/10.1186/s12872-022-03021-w
2. Angelini, P. (2007). Coronary Artery Anomalies. *Circulation*, *115*(10), 1296–1305. https://doi.org/10.1161/CIRCULATIONAHA.106.618082
3. Augustine, D. X., & Howard, L. (2018). Left Ventricular Hypertrophy in Athletes: Differentiating Physiology From Pathology. *Current Treatment Options in Cardiovascular Medicine*, *20*(12), 96. https://doi.org/10.1007/s11936-018-0691-2
4. Baba Ali, N., Attaripour Esfahani, S., Scalia, I. G., Farina, J. M., Pereyra, M., Barry, T., Lester, S. J., Alsidawi, S., Steidley, D. E., Ayoub, C., Palermi, S., & Arsanjani, R. (2024). The Role of Cardiovascular Imaging in the Diagnosis of Athlete's Heart: Navigating the Shades of Grey. *Journal of Imaging*, *10*(9), 230. https://doi.org/10.3390/jimaging10090230
5. Basso, C., Maron, B. J., Corrado, D., & Thiene, G. (2000). Clinical profile of congenital coronary artery anomalies with origin from the wrong aortic sinus leading to sudden death in young competitive athletes. *Journal of the American College of Cardiology*, *35*(6), 1493–1501. https://doi.org/10.1016/s0735-1097(00)00566-0
6. Bellfield, R. A. A., Ortega-Martorell, S., Lip, G. Y. H., Oxborough, D., & Olier, I. (2022). The Athlete's Heart and Machine Learning: A Review of Current Implementations and Gaps for Future Research. *Journal of Cardiovascular Development and Disease*, *9*(11), 382. https://doi.org/10.3390/jcdd9110382
7. Bhatia, R., Westaby, J., Behr, E., Papadakis, M., Sharma, S., Finocchiaro, G., & Sheppard, M. (2022). Sudden cardiac death during exercise in young individuals with hypertrophic cardiomyopathy. *European Heart Journal*, *43*(Supplement_2), ehac544.1716. https://doi.org/10.1093/eurheartj/ehac544.1716
8. Bos, J. M., Attia, Z. I., Albert, D. E., Noseworthy, P. A., Friedman, P. A., & Ackerman, M. J. (2021). Use of Artificial Intelligence and Deep Neural Networks in Evaluation of Patients With Electrocardiographically Concealed Long QT Syndrome From the Surface 12-Lead Electrocardiogram. *JAMA Cardiology*, *6*(5), 532–538. https://doi.org/10.1001/jamacardio.2020.7422
9. Bourfiss, M., Prakken, N. H. J., James, C. A., Planken, R. N., Boekholdt, S. M., Ahmetagic, D., van den Berg, M. P., Tichnell, C., Van der Heijden, J. F., Loh, P., Murray, B., Tandri, H., Kamel, I., Calkins, H., Asselbergs, F. W., Zimmerman, S. L., Velthuis, B. K., & Te Riele, A. S. J. M. (2022). Prognostic value of strain by feature-tracking cardiac magnetic resonance in arrhythmogenic right ventricular cardiomyopathy. *European Heart Journal. Cardiovascular Imaging*, *24*(1), 98–107. https://doi.org/10.1093/ehjci/jeac030
10. Bourfiss, M., Sander, J., de Vos, B. D., te Riele, A. S. J. M., Asselbergs, F. W., Išgum, I., & Velthuis, B. K. (2023). Towards automatic classification of cardiovascular magnetic resonance Task Force Criteria for diagnosis of arrhythmogenic right ventricular cardiomyopathy. *Clinical Research in Cardiology*, *112*(3), 363–378. https://doi.org/10.1007/s00392-022-02088-x
11. Campuzano, O., Alcalde, M., Iglesias, A., Barahona-Dussault, C., Sarquella-Brugada, G., Benito, B., Arzamendi, D., Flores, J., Leung, T. K., Talajic, M., Oliva, A., & Brugada, R. (2012). Arrhythmogenic right ventricular cardiomyopathy: Severe structural alterations are associated with inflammation. *Journal of Clinical Pathology*, *65*(12), 1077–1083. https://doi.org/10.1136/jclinpath-2012-201022

12. Cavarretta, E., D'Ascenzi, F., Bianco, M., Castelletti, S., Cavigli, L., Cecchi, F., D'Andrea, A., De Luca, A., Di Salvo, G., Nistri, S., Palamà, Z., Palmieri, V., Ricci, F., Sinagra, G., Zorzi, A., Biffi, A., Pelliccia, A., Romano, S., Dello Russo, A., … Sciarra, L. (2024). The role of echocardiography in sports cardiology: An expert opinion statement of the Italian Society of Sports Cardiology (SIC sport). *International Journal of Cardiology*, *410*, 132230. https://doi.org/10.1016/j.ijcard.2024.132230
13. Chorba, J. S., Shapiro, A. M., Le, L., Maidens, J., Prince, J., Pham, S., Kanzawa, M. M., Barbosa, D. N., Currie, C., Brooks, C., White, B. E., Huskin, A., Paek, J., Geocaris, J., Elnathan, D., Ronquillo, R., Kim, R., Alam, Z. H., Mahadevan, V. S., … Thomas, J. D. (2021). Deep Learning Algorithm for Automated Cardiac Murmur Detection via a Digital Stethoscope Platform. *Journal of the American Heart Association*, *10*(9), e019905. https://doi.org/10.1161/JAHA.120.019905
14. Corrado, D., Link, M. S., & Calkins, H. (2017). Arrhythmogenic Right Ventricular Cardiomyopathy. *New England Journal of Medicine*, *376*(1), 61–72. https://doi.org/10.1056/NEJMra1509267
15. Cotet, I.-G., Mateescu, D.-M., Ilie, A.-C., Guse, C., Pah, A.-M., Badalica-Petrescu, M., Iurciuc, S., Craciun, M.-L., Buleu, F., & Tudoran, C. (2025). Systematic Review and Meta-Analysis of Myocarditis Prevalence and Diagnostics in COVID-19:Acute, Post-COVID, and MIS-C (2020–2025). *Journal of Clinical Medicine*, *14*(19), 7008. https://doi.org/10.3390/jcm14197008
16. Croon, P., Aminorroaya, A., Pedroso, A., Dhingra, L., & Khera, R. (2025). Abstract 4371172: Artificial Intelligence-based screening for Hypertrophic Cardiomyopathy from Single-lead Electrocardiograms: A Multinational Development and Validation Study. *Circulation*, *152*(Suppl_3), A4371172–A4371172. https://doi.org/10.1161/circ.152.suppl_3.4371172
17. D'Ascenzi, F., Anselmi, F., Mondillo, S., Finocchiaro, G., Caselli, S., Garza, M. S.-D. L., Schmied, C., Adami, P. E., Galderisi, M., Adler, Y., Pantazis, A., Niebauer, J., Heidbuchel, H., Papadakis, M., & Dendale, P. (2021). The use of cardiac imaging in the evaluation of athletes in the clinical practice: A survey by the Sports Cardiology and Exercise Section of the European Association of Preventive Cardiology and University of Siena, in collaboration with the European Association of Cardiovascular Imaging, the European Heart Rhythm Association and the ESC Working Group on Myocardial and Pericardial Diseases. *European Journal of Preventive Cardiology*, *28*(10), 1071–1077. https://doi.org/10.1177/2047487320932018
18. Drezner, J. A., Sharma, S., Baggish, A., Papadakis, M., Wilson, M. G., Prutkin, J. M., Gerche, A. L., Ackerman, M. J., Borjesson, M., Salerno, J. C., Asif, I. M., Owens, D. S., Chung, E. H., Emery, M. S., Froelicher, V. F., Heidbuchel, H., Adamuz, C., Asplund, C. A., Cohen, G., … Corrado, D. (2017). International criteria for electrocardiographic interpretation in athletes: Consensus statement. *British Journal of Sports Medicine*, *51*(9), 704–731. https://doi.org/10.1136/bjsports-2016-097331
19. Duffy, G., Cheng, P. P., Yuan, N., He, B., Kwan, A. C., Shun-Shin, M. J., Alexander, K. M., Ebinger, J., Lungren, M. P., Rader, F., Liang, D. H., Schnittger, I., Ashley, E. A., Zou, J. Y., Patel, J., Witteles, R., Cheng, S., & Ouyang, D. (2022). High-Throughput Precision Phenotyping of Left Ventricular Hypertrophy with Cardiovascular Deep Learning. *JAMA Cardiology*, *7*(4), 386. https://doi.org/10.1001/jamacardio.2021.6059
20. Fahmy, A. S., Rowin, E. J., Arafati, A., Al-Otaibi, T., Maron, M. S., & Nezafat, R. (2022). Radiomics and deep learning for myocardial scar screening in hypertrophic cardiomyopathy. *Journal of Cardiovascular Magnetic Resonance*, *24*(1), 40. https://doi.org/10.1186/s12968-022-00869-x
21. Gräni, C. (2018). Anomalous Coronary Arteries and the Risk of Adverse Cardiac Events. *Cardiovascular Medicine*, *21*(2), 39. https://doi.org/10.4414/cvm.2018.00540
22. Haq, I. U., Liu, K., Giudicessi, J. R., Siontis, K. C., Asirvatham, S. J., Attia, Z. I., Ackerman, M. J., Friedman, P. A., & Killu, A. M. (2024). Artificial intelligence-enhanced electrocardiogram for arrhythmogenic right ventricular cardiomyopathy detection. *European Heart Journal - Digital Health*, *5*(2), 192–194. https://doi.org/10.1093/ehjdh/ztad078
23. Henning, R. J. (2024). The differentiation of the competitive athlete with physiologic cardiac remodeling from the athlete with cardiomyopathy. *Current Problems in Cardiology*, *49*(9), 102473. https://doi.org/10.1016/j.cpcardiol.2024.102473
24. Huang, J., Huang, A., Xu, R., Wu, M., Wang, P., & Wang, Q. (2023). Automatic Segmentation and Assessment of Valvular Regurgitations with Color Doppler Echocardiography Images: A VABC-UNet-Based Framework. *Bioengineering*, *10*(11), 1319. https://doi.org/10.3390/bioengineering10111319
25. Jiang, R., Cheung, C. C., Garcia-Montero, M., Davies, B., Cao, J., Redfearn, D., Laksman, Z. M., Grondin, S., Atallah, J., Escudero, C. A., Cadrin-Tourigny, J., Sanatani, S., Steinberg, C., Joza, J., Avram, R., Tadros, R., & Krahn, A. D. (2024). Deep Learning–Augmented ECG Analysis for Screening and Genotype Prediction of Congenital Long QT Syndrome. *JAMA Cardiology*, *9*(4), 377–384. https://doi.org/10.1001/jamacardio.2024.0039
26. Johnson, J. N., & Ackerman, M. J. (2013). Return to play? Athletes with congenital long QT syndrome. *British Journal of Sports Medicine*, *47*(1), 28–33. https://doi.org/10.1136/bjsports-2012-091751
27. Jone, P.-N., Gearhart, A., Lei, H., Xing, F., Nahar, J., Lopez-Jimenez, F., Diller, G.-P., Marelli, A., Wilson, L., Saidi, A., Cho, D., & Chang, A. C. (2022). Artificial Intelligence in Congenital Heart Disease: Current State and Prospects. *JACC. Advances*, *1*(5), 100153. https://doi.org/10.1016/j.jacadv.2022.100153

28. Ko, W. Y., Siontis, K. C., Attia, Z. I., Carter, R. E., Kapa, S., Ommen, S. R., Demuth, S. J., Ackerman, M. J., Gersh, B. J., Arruda-Olson, A. M., Geske, J. B., Asirvatham, S. J., Lopez-Jimenez, F., Nishimura, R. A., Friedman, P. A., & Noseworthy, P. A. (2020). Detection of Hypertrophic Cardiomyopathy Using a Convolutional Neural Network-Enabled Electrocardiogram. *Journal of the American College of Cardiology*, *75*(7), 722–733. https://doi.org/10.1016/j.jacc.2019.12.030
29. Kübler, J., Burgstahler, C., Brendel, J. M., Gassenmaier, S., Hagen, F., Klingel, K., Olthof, S.-C., Blume, K., Wolfarth, B., Mueller, K. a. L., Greulich, S., & Krumm, P. (2021). Cardiac MRI findings to differentiate athlete's heart from hypertrophic (HCM), arrhythmogenic right ventricular (ARVC) and dilated (DCM) cardiomyopathy. *The International Journal of Cardiovascular Imaging*, *37*(8), 2501–2515. https://doi.org/10.1007/s10554-021-02280-6
30. Lau, W. R., Lee, P. T., & Koh, C. H. (2023). Coronary Artery Anomalies – State of the Art Review. *Current Problems in Cardiology*, *48*(11), 101935. https://doi.org/10.1016/j.cpcardiol.2023.101935
31. Lauschke, J., & Maisch, B. (2009). Athlete's heart or hypertrophic cardiomyopathy? *Clinical Research in Cardiology: Official Journal of the German Cardiac Society*, *98*(2), 80–88. https://doi.org/10.1007/s00392-008-0721-2
32. Liang, Y., Sau, A., Zeidaabadi, B., Barker, J., Patlatzoglou, K., Pastika, L., Sieliwonczyk, E., Whinnett, Z., Peters, N. S., Yu, Z., Liu, X., Wang, S., Lu, H., Kramer, D. B., Waks, J. W., Su, Y., Ge, J., & Ng, F. S. (2025). Artificial intelligence-enhanced electrocardiography to predict regurgitant valvular heart diseases: An international study. *European Heart Journal*, *46*(44), 4823–4837. https://doi.org/10.1093/eurheartj/ehaf448
33. Lin, Y.-T., Lin, C.-S., Tsai, C.-S., Tsai, D.-J., Lou, Y.-S., Fang, W.-H., Lee, Y.-T., & Lin, C. (2024). Comprehensive clinical application analysis of artificial intelligence-enabled electrocardiograms for screening multiple valvular heart diseases. *Aging*, *16*(10), 8717–8731. https://doi.org/10.18632/aging.205835
34. Łajczak, P. M., & Jóźwik, K. (2024). Artificial intelligence and myocarditis—A systematic review of current applications. *Heart Failure Reviews*, *29*(6), 1217–1234. https://doi.org/10.1007/s10741-024-10431-9
35. Malhotra, A., & Sharma, S. (2017). Hypertrophic Cardiomyopathy in Athletes. *European Cardiology Review*, *12*(2), 80–82. https://doi.org/10.15420/ecr.2017:12:1
36. Monopoli, G., Haas, D., Singh, A., Aabel, E. W., Ribe, M., Castrini, A. I., Hasselberg, N. E., Bugge, C., Five, C., Haugaa, K., Forsch, N., Thambawita, V., Balaban, G., & Maleckar, M. M. (2025). DeepValve: The first automatic detection pipeline for the mitral valve in Cardiac Magnetic Resonance imaging. *Computers in Biology and Medicine*, *192*(Pt A), 110211. https://doi.org/10.1016/j.compbiomed.2025.110211
37. Neves, P. O., Andrade, J., & Monção, H. (2015). Coronary anomalies: What the radiologist should know. *Radiologia Brasileira*, *48*(4), 233–241.
38. Palermi, S., Cavarretta, E., D'Ascenzi, F., Castelletti, S., Ricci, F., Vecchiato, M., Serio, A., Cavigli, L., Bossone, E., Limongelli, G., Biffi, A., Monda, E., La Gerche, A., Baggish, A., & D'Andrea, A. (2023). Athlete's Heart: A Cardiovascular Step-By-Step Multimodality Approach. *Reviews in Cardiovascular Medicine*, *24*(5), 151. https://doi.org/10.31083/j.rcm2405151
39. Pérez-Pomares, J. M., De La Pompa, J. L., Franco, D., Henderson, D., Ho, S. Y., Houyel, L., Kelly, R. G., Sedmera, D., Sheppard, M., Sperling, S., Thiene, G., Van Den Hoff, M., & Basso, C. (2016). Congenital coronary artery anomalies: A bridge from embryology to anatomy and pathophysiology—a position statement of the development, anatomy, and pathology ESC Working Group. *Cardiovascular Research*, *109*(2), 204–216. https://doi.org/10.1093/cvr/cvv251
40. Priori, S. G., Wilde, A. A., Horie, M., Cho, Y., Behr, E. R., Berul, C., Blom, N., Brugada, J., Chiang, C.-E., Huikuri, H., Kannankeril, P., Krahn, A., Leenhardt, A., Moss, A., Schwartz, P. J., Shimizu, W., Tomaselli, G., & Tracy, C. (2013). Executive summary: HRS/EHRA/APHRS expert consensus statement on the diagnosis and management of patients with inherited primary arrhythmia syndromes. *Heart Rhythm*, *10*(12), e85-108. https://doi.org/10.1016/j.hrthm.2013.07.021
41. Rapezzi, C., Arbustini, E., Caforio, A. L. P., Charron, P., Gimeno-Blanes, J., Heliö, T., Linhart, A., Mogensen, J., Pinto, Y., Ristic, A., Seggewiss, H., Sinagra, G., Tavazzi, L., & Elliott, P. M. (2013). Diagnostic work-up in cardiomyopathies: Bridging the gap between clinical phenotypes and final diagnosis. A position statement from the ESC Working Group on Myocardial and Pericardial Diseases. *European Heart Journal*, *34*(19), 1448–1458. https://doi.org/10.1093/eurheartj/ehs397
42. Schwartz, P. J., Ackerman, M. J., & Wilde, A. A. M. (2017). Channelopathies as Causes of Sudden Cardiac Death. *Cardiac Electrophysiology Clinics*, *9*(4), 537–549. https://doi.org/10.1016/j.ccep.2017.07.005
43. Schwartz, P. J., Stramba-Badiale, M., Crotti, L., Pedrazzini, M., Besana, A., Bosi, G., Gabbarini, F., Goulene, K., Insolia, R., Mannarino, S., Mosca, F., Nespoli, L., Rimini, A., Rosati, E., Salice, P., & Spazzolini, C. (2009). Prevalence of the congenital long-QT syndrome. *Circulation*, *120*(18), 1761–1767. https://doi.org/10.1161/CIRCULATIONAHA.109.863209
44. Shyam-Sundar, V., Harding, D., Khan, A., Abdulkareem, M., Slabaugh, G., Mohiddin, S. A., Petersen, S. E., & Aung, N. (2024). Imaging for the diagnosis of acute myocarditis: Can artificial intelligence improve diagnostic performance? *Frontiers in Cardiovascular Medicine*, *11*. https://doi.org/10.3389/fcvm.2024.1408574

45. Siontis, K. C., Wieczorek, M. A., Maanja, M., Hodge, D. O., Kim, H. K., Lee, H. J., Lee, H., Lim, J., Park, C. S., Ariga, R., Raman, B., Mahmod, M., Watkins, H., Neubauer, S., Windecker, S., Siontis, G. C. M., Gersh, B. J., Ackerman, M. J., Attia, Z. I., … Noseworthy, P. A. (2024). Hypertrophic cardiomyopathy detection with artificial intelligence electrocardiography in international cohorts: An external validation study. *European Heart Journal - Digital Health*, *5*(4), 416–426. https://doi.org/10.1093/ehjdh/ztae029
46. Smaranda, A. M., Drăgoiu, T. S., Caramoci, A., Afetelor, A. A., Ionescu, A. M., & Bădărău, I. A. (2024). Artificial Intelligence in Sports Medicine: Reshaping Electrocardiogram Analysis for Athlete Safety—A Narrative Review. *Sports*, *12*(6), 144. https://doi.org/10.3390/sports12060144
47. Tschöpe, C., Ammirati, E., Bozkurt, B., Caforio, A. L. P., Cooper, L. T., Felix, S. B., Hare, J. M., Heidecker, B., Heymans, S., Hübner, N., Kelle, S., Klingel, K., Maatz, H., Parwani, A. S., Spillmann, F., Starling, R. C., Tsutsui, H., Seferovic, P., & Van Linthout, S. (2021). Myocarditis and inflammatory cardiomyopathy: Current evidence and future directions. *Nature Reviews Cardiology*, *18*(3), 169–193. https://doi.org/10.1038/s41569-020-00435-x
48. Tso, J., Turner, C. G., & Kim, J. H. (2020). A Hidden Threat: Anomalous Aortic Origins of the Coronary Arteries in Athletes. *Current Treatment Options in Cardiovascular Medicine*, *22*(12), 67. https://doi.org/10.1007/s11936-020-00859-1
49. Ullal, A. J., Abdelfattah, R. S., Ashley, E. A., & Froelicher, V. F. (2016). Hypertrophic Cardiomyopathy as a Cause of Sudden Cardiac Death in the Young: A Meta-Analysis. *The American Journal of Medicine*, *129*(5), 486-496.e2. https://doi.org/10.1016/j.amjmed.2015.12.027
50. Viera, E., Meras, P., Merino, C., Perez, E., Ruiz, J., Balbacid, E., Abelleira, C., Aroca, A., & Moreno, R. (2024). Diagnosis of Congenital Heart Disease in Adulthood: How Often, How Relevant? *The American Journal of Cardiology*, *231*, 72–74. https://doi.org/10.1016/j.amjcard.2024.09.004
51. Wang, Y.-R. (Joyce), Yang, K., Wen, Y., Wang, P., Hu, Y., Lai, Y., Wang, Y., Zhao, K., Tang, S., Zhang, A., Zhan, H., Lu, M., Chen, X., Yang, S., Dong, Z., Wang, Y., Liu, H., Zhao, L., Huang, L., … Zhao, S. (2024). Screening and diagnosis of cardiovascular disease using artificial intelligence-enabled cardiac magnetic resonance imaging. *Nature Medicine*, *30*(5), 1471–1480. https://doi.org/10.1038/s41591-024-02971-2
52. Wilde, A. A. M., Semsarian, C., Márquez, M. F., Shamloo, A. S., Ackerman, M. J., Ashley, E. A., Sternick, E. B., Barajas-Martinez, H., Behr, E. R., Bezzina, C. R., Breckpot, J., Charron, P., Chockalingam, P., Crotti, L., Gollob, M. H., Lubitz, S., Makita, N., Ohno, S., Ortiz-Genga, M., … Developed in partnership with and endorsed by the European Heart Rhythm Association (EHRA), a branch of the E. S. of C. (ESC), the Heart Rhythm Society (HRS), the Asia Pacific Heart Rhythm Society (APHRS), and the Latin American Heart Rhythm Society (LAHRS). (2022). European Heart Rhythm Association (EHRA)/Heart Rhythm Society (HRS)/Asia Pacific Heart Rhythm Society (APHRS)/Latin American Heart Rhythm Society (LAHRS) Expert Consensus Statement on the state of genetic testing for cardiac diseases. *EP Europace*, *24*(8), 1307–1367. https://doi.org/10.1093/europace/euac030
53. Yu, X., Yao, X., Wu, B., Zhou, H., Xia, S., Su, W., Wu, Y., & Zheng, X. (2022). Using deep learning method to identify left ventricular hypertrophy on echocardiography. *The International Journal of Cardiovascular Imaging*, *38*(4), 759–769. https://doi.org/10.1007/s10554-021-02461-3
54. Zhang, Q., Fotaki, A., Ghadimi, S., Wang, Y., Doneva, M., Wetzl, J., Delfino, J. G., O'Regan, D. P., Prieto, C., & Epstein, F. H. (2024). Improving the efficiency and accuracy of cardiovascular magnetic resonance with artificial intelligence—Review of evidence and proposition of a roadmap to clinical translation. *Journal of Cardiovascular Magnetic Resonance*, *26*(2). https://doi.org/10.1016/j.jocmr.2024.101051
55. Zhou, Z., Xie, K., Huang, Y., Zhang, W., Li, B., Zhong, J., Liao, X., Wang, C., & Zhuang, X. (2025). Automatic Diagnosis of Left Valvular Heart Disease Based on Artificial Intelligence Stethoscope. *JACC. Advances*, *4*(11 Pt 2), 101993. https://doi.org/10.1016/j.jacadv.2025.101993