International Journal of Cardiology 410 (2024) 132230

Contents lists available at ScienceDirect

International Journal of Cardiology

journal homepage: www.elsevier.com/locate/ijcard

The role of echocardiography in sports cardiology: An expert opinion statement of the Italian Society of Sports Cardiology (SIC sport)

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ARTICLE INFO

Keywords:
Athletes
Echocardiography
Pre-participation screening
Sports cardiology
Cardiomyopathy
Sudden cardiac death
Heart valve disease

ABSTRACT

Transthoracic echocardiography (TTE) is routinely required during pre-participation screening in the presence of symptoms, family history of sudden cardiac death or cardiomyopathies <40-year-old, murmurs, abnormal ECG findings or in the follow-up of athletes with a history of cardiovascular disease (CVD). TTE is a cost-effective first-line imaging modality to evaluate the cardiac remodeling due to long-term, intense training, previously known as the athlete's heart, and to rule out the presence of conditions at risk of sudden cardiac death, including cardiomyopathies, coronary artery anomalies, congenital, aortic and heart valve diseases. Moreover, TTE is useful for distinguishing physiological cardiac adaptations during intense exercise from pathological behavior due to an underlying CVD.

In this expert opinion statement endorsed by the Italian Society of Sports Cardiology, we discussed common clinical scenarios where a TTE is required and conditions falling in the grey zone between the athlete's heart and

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https://doi.org/10.1016/j.ijcard.2024.132230
Received 10 March 2024; Received in revised form 9 May 2024; Accepted 3 June 2024
Available online 8 June 2024

underlying cardiomyopathies or other CVD. In addition, we propose a minimum dataset that should be included in the report for the most common indications of TTE in sports cardiology clinical practice.

1. Introduction

The pre-participation screening (PPS) primarily aims to identify pathologies at risk for sudden cardiac death (SCD) and represents an important step forward in primary prevention of the active population. In Italy, PPS has been based on family and medical history, physical examination, 12-lead resting ECG and stress ECG since 1982 [1]. In the presence of anomalies or suspicious findings, the sports medicine physician asks for second-line investigations, including transthoracic echocardiography (TTE), 24-h ECG monitoring, coronary computed tomography angiography (CCTA), cardiac magnetic resonance (CMR), nuclear imaging test, genetic testing and so on, in a step-by-step multimodality approach [2,3]. The criteria for ECG interpretation in athletes have been revised from 2005 to 2017 and have nowadays increased the diagnostic accuracy, thus reducing the number of false positive exams, and the need for additional testing. However, echocardiography remains the most required investigation performed in addition to the standard PPS, given its ability to non-invasively identify structural heart diseases, being an accessible, low-cost, and repeatable imaging technique [4,5]. Other than the minimum 2D dataset of a standard TTE, additional echocardiographic parameters in sports cardiology and sports medicine are needed, such as visualization of the coronary artery ostia and additional advanced techniques, including three-dimensional (3D), stress echo and speckle-tracking echocardiography (STE), that are particularly useful to evaluate an athlete with a suspicion of a subclinical cardiovascular (CV) disease.

This document, endorsed by the Italian Society of Sports Cardiology (SIC sport), aims to enhance the appropriate criteria for requiring an echocardiogram by the sports medicine physician. Contemporary indications for echocardiography in sports cardiology are resumed in Fig. 1. We aim to present the "12-lead ECG guided approach" to echocardiogram in Table 1, where we provided the essential echocardiographic parameters needed based on the clinical suspicious and the on the ECG finding at the 12-lead resting ECG. Last but not least, we aim to propose a specific dataset for the echocardiographic report in sports cardiology (Table 2), pointing out the essential parameters that should always be provided when evaluating competitive athletes with a clear indication for TTE. In doubtful cases we suggest also additional and advanced echocardiographic parameters that may be helpful in discriminating athletes falling in the grey zone between physiological and pathological remodeling.

2. Physiological determinants of the athlete's heart

The physiological cardiac adaptation to long-lasting exercise, previously known as the "athlete's heart", can be clearly defined by TTE. The training-induced physiological remodeling primarily depends on the type of sport, the intensity, the training volume per week, and the years of continued sports practice [6]. Cardiac adaptation is proportional to the amount of hemodynamic stress, the total duration and frequency of training and is partially reversible after a 3–6-month detraining period [2]. Competitive athletes involved in endurance sports develop a predominant increase in biventricular and biatrial dimensions with eccentric LV hypertrophy, due to the volume overload associated with the high cardiac output. Conversely, athletes practicing mainly static/isometric exercise develop a relative increase in the LV wall thickness due to arterial pressure overload without developing a substantial LV hypertrophy [6,7].

The values and upper limits of normality for the definition of LV remodeling in athletes have been extensively evaluated by TTE, about 15% of endurance athletes showed LV end-diastolic diameter > 60 mm in the presence of normal diastolic and systolic indexes. The maximum thickness of the interventricular septum (IVS) was <13 mm in most of the Caucasian athletes, whereas only 3% had a septal thickness between 13 and 16 mm [8]. Individual characteristics such as sex, ethnicity, and environmental factors play an important role. Indeed, cardiac

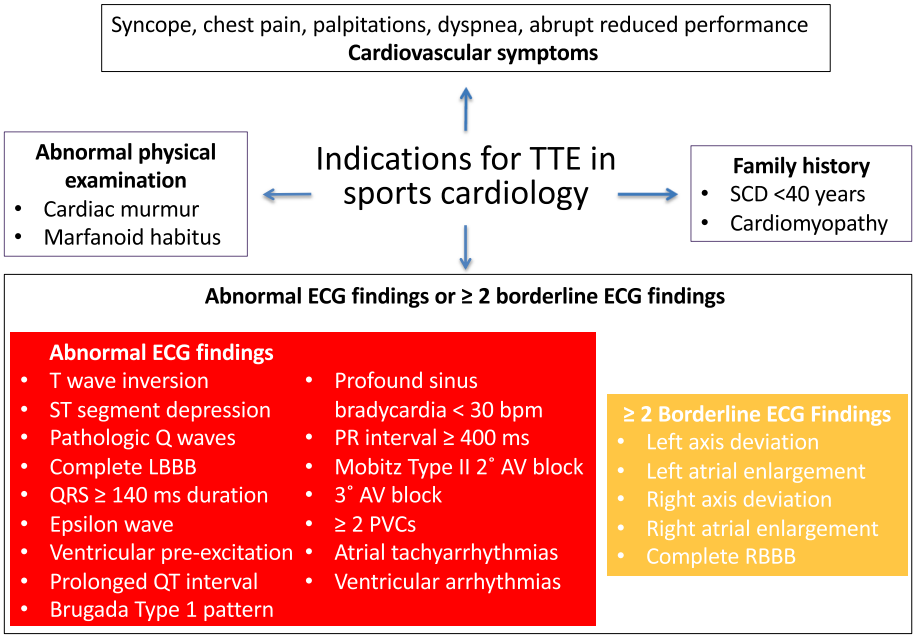

Fig. 1. Indications for echocardiography in sports cardiology. Criteria for defining abnormal and borderline ECG findings are defined as on Sharma S et al. [4]. AV: atrio-ventricular; ECG: electrocardiogram; LBBB: left bundle branch block; PVCs: premature ventricular contractions; RBBB: right bundle branch block; SCD: sudden cardiac death; TTE: trans-thoracic echocardiography.

Table 1
The "12-lead ECG guided approach" to echocardiogram. In the suspicion of a specific disease based on the possible findings at basal ECG, additional echocardiographic parameters are required; if available, advanced echocardiographic parameters are useful to complete the diagnostic management. AF: atrial fibrillation; ARVC: arrhythmogenic right ventricular cardiomyopathy; AV: atrioventricular; CHD: congenital heart disease; DCM: dilated cardiomyopathy; HCM: hypertrophic cardiomyopathy; LA: left atrium; LAE: left atrial enlargement; LBBB: left bundle branch block; LV: left ventricular; LVOT: left ventricular outflow tract; MAD: mitral annular disjunction; PVM: mitral valve prolapse; QTc: corrected QT interval; RBBB: right ventricular bundle branch block; RV: right ventricular; RVFAC: right ventricular fractional area change; SAM: systolic anterior motion; sPAP: systolic pulmonary artery pressure; ST: sino-tubular; TAPSE: tricuspid annular plane systolic excursion; TDI: tissue Doppler imaging; VAs: ventricular arrhythmias; WMAs: wall motion abnormalities.

Rule out	Possible basal ECG findings	Required additional echo parameters	Advanced echo parameters, if needed and/or available
HCM	Increased QRS voltage for LV or RV hypertrophy, pathologic Q waves, T wave flattening/ inversion in inferior, infero-lateral, lateral, LAE+ other borderline ECG criteria, VAs	LV maximal wall thickness, LA volume index, LV volume and function, LVOT gradient/obstruction, mitral valve morphology/SAM, LV diastolic function, hypertrabeculation, papillary muscles hypertrophy, apical aneurisms, sPAP	LV global longitudinal strain
ARVC	T-wave inversion in V1-V3, in athletes>16 years old (in the absence of complete RBBB and not preceded by J-point/ST-segment elevation), epsilon wave, low QRS voltage in limbs leads, RV conduction disturbances (in the absence of RBBB), dual morphology LBBB VAs	RV WMAs, RV outflow tract dimensions in long and short axis, basal and medial RV diameter in apical view, RVFAC, TDI-derived S′ wave velocity, TAPSE	RV longitudinal strain (particularly RV free wall strain) and 3D RVEF
ALVC	Negative T-waves in left precordial leads (V4-V6) (in the absence of complete LBBB), low QRS voltage in limbs leads, dual morphology RBBB VAs, complex RBBB VAs	Global LV systolic dysfunction, regional WMAs, with or without LV dilatation	LV longitudinal strain and 3D LVEF
DCM	Increased QRS voltage for LV or RV hypertrophy, Q waves, T-wave inversion, LBBB, low QRS voltage in limbs leads, LAE, RAE, AV block, AF	Wall thickness, indexed LV volumes and function, indexed LV mass, diastolic function, TAPSE, TDI-derived S′ wave velocity	LV global longitudinal strain, 3D LV volumes and LVEF
Myocarditis/ Pericarditis	Non-specific ST segment and T waves flattening/ inversion, AV block, long QTc, VAs	LV and RV ventricular function, LV and RV WMAs, sPAP, diastolic function	3D LV volumes and LVEF, LV and RV global longitudinal strain
PVM	T-wave inversion in the inferior leads; long QTc; ST segment elevation; RBBB VAs, superior and inferior axis with QRS < 130 msec; atypical RBBB VAs and QRS ≥130 ms	Mitral valve morphology (Barlow, bileaflet involvement) and function (regurgitation, mean gradient), sPAP, LV dimensions and volumes, MAD, LA dimensions and index volume, mitral TDI S′ wave (Pickelhaube sign)	3D LV volumes and LVEF, LV global longitudinal strain
BAV	Increased QRS voltage for LV hypertrophy, T-wave flattening/ inversion, borderline ECG criteria	Aortic valve morphology and function (peak and mean gradient; regurgitation); LVOT, aortic bulb, ST junction and ascending aorta diameters; trans-isthmic aortic gradient; LV volumes and function; coronary ostia	3D LV volumes and LVEF
CHD	Pathological and borderline ECG criteria	LV and RV ventricular function, WMAs, sPAP, aorta dimensions	3D LV volumes and LVEF, LV and RV global longitudinal strain

remodeling is less pronounced in women than men, even for index values (mean IVS thickness in women = 9 mm; upper limit = 12 mm) and abnormal ECG findings have been reported as more prevalent than in men [9]. Moreover, electrical and structural adaptations appear more evident in black than Caucasian athletes. In particular, Afro-Caribbean athletes are characterized by similar cavity size but a disproportionate wall thickening compared with Caucasians (>12 mm in 18% of cases), while only 2% of Asiatic and <1% of Arabic athletes showed a maximum IVS thickness > 13 mm [10]. Conversely, eccentric LV remodeling was more common in Japanese athletes with an enlarged LV cavity when corrected for body surface area (BSA) compared to Afro-Caribbean and Caucasian athletes. Finally, the starting age and the cumulative years of exercise exposure may have a role in cardiac remodeling.

3. Evaluation of coronary artery ostia

The prevalence of an anomalous origin of coronary arteries (AOCAs) ranges between 0.1 and 1% in adults and paediatric population [11]. The anomalous aortic origin of the left coronary artery (AOLCA) with inter-arterial intramural course seems to bring the higher risk of adverse cardiac events [11,12] due to the compression of the first-tract intramural course by the systolic expansion of the aorta [13,14]. Other anomalies, such as a coronary artery arising from a "wrong" sinus but positioning anteriorly to the pulmonary artery (pre-pulmonic) or posteriorly to the aorta (posterior/retroaortic), are generally considered benign [11]. Even if most of the subjects with AOCA are asymptomatic, they may report symptoms as chest pain/discomfort, cold sweating, syncope/near syncope during or shortly after effort [12,15]. The stress ECG may be completely normal and only occasionally shows ECG changes as ST segment depression, with/without the occurrence of negative T-waves or premature ventricular beats (PVBs) [12,15]. TTE is a useful, reliable method, especially in young athletes, characterized by good acoustic windows and a longer diastolic phase (due to the exercise-induced sinus bradycardia), for non-invasive in vivo detection of the coronary ostia and first tracts [15](Fig. 2). The best echocardiographic projection for the coronary ostia is the parasternal short-axis view (SAX) at the level of the aortic root, and it is not uncommon to observe AOCA

Table 2
Essential echocardiographic parameters that should be included in all reports. Specific additional parameters are required based on the clinical indication to perform an echocardiographic exam in sports cardiology. Advanced echocardiographic parameters may not be widely available, if available should be included in specific conditions at risk of sudden cardiac death. CW: continuous wave; IVS: interventricular septum; LA: left atrial; LVOT: left ventricular outflow tract; PW: pulsed wave; RVOT: right ventricular outflow tract; TDI: tissue Doppler imaging.

Structure	Essential parameters	Additional parameters	Advanced parameters
Left Ventricle	End-diastolic and end-systolic diameters	End-diastolic and end-systolic indexed volumes (Simpson's biplane)	3D end-diastolic and end-systolic volumes
	IVS and posterior wall thickness	Simpson's biplane ejection fraction	3D ejection fraction
	Ejection fraction, Regional wall motion		Global longitudinal strain (GLS)
Right Ventricle	Basal and/or mid-cavity diameter	RVOT diameters (in long and short axis)	3D end-diastolic and end-systolic volumes
	Tricuspid annular plane systolic excursion	Fractional area change	3D ejection fraction
	TDI-derived tricuspid lateral annular systolic velocity (s' velocity)	Wall thickness and wall motion abnormalities	Global and free wall longitudinal strain
Left atrium	End-systolic area	End-systolic volume index	LA strain
	End-systolic antero-posterior diameter		
Right atrium	End-systolic area	End-systolic volume index	
Thoracic Aorta	Sinus of Valsalva diameter	LVOT diameter	
	Ascending aorta diameter	Sino-tubular junction diameter	
	Aortic arch size	Trans-isthmic gradient	
Coronary artery	Ostia	Course of the first tracts (right artery and left coronary trunk)	
Hemodynamic and valve function	PW Doppler transmitral flow	PW Doppler LVOT flow	
	TDI mitral medial and lateral annular velocities	CW Doppler Pulmonic flow	
	CW aortic flow		Pulmonary veins ostia and flow
	Systolic pulmonary artery pressure	CW Doppler Tricuspid regurgitation velocity	Sovrahepatic veins flow
	Aortic and mitral valve morphology; Eventual valve regurgitation and/or stenosis (graded as mild, moderate or severe)	Prolapsing scallop, in case of prolapse	3D valve morphology, particularly in case of valve dysfunction
Pericardium	Absence/presence of pericardial effusion	Hemodynamic characterization of pericardial effusion/constrictive pericarditis, if present. Pericardial appearance, including thickness	
Inferior vena cava	Diameter and collapse	Estimated right atrial pressure	Sovrahepatic veins flow

in the presence of a bicuspid aortic valve [16]. When, after a careful search, coronary arteries are not visualized in their correct position, a potential anomaly should be ruled out. When an AOCA is suspected, since there are several anatomical variants, it is mandatory to report the suspicious AOCA, in particular in high-risk conditions, such as the presence of an AOCA intramural course (usually with a faster diastolic flow than the other coronary arteries) or an acute angle take-off, and to recommend further multimodality imaging work-up, including CCTA or CMR in younger subjects in order to solve the diagnostic conundrum [17]. The evaluation of a normal origin of coronary arteries should always be indicated in the final echocardiographic report (Table 2), given its relevance for sports eligibility and appropriate clinical management.

4. Common clinical scenarios in sports cardiology

4.1. Arrhythmias

TTE represents a fundamental step for a correct work-up and stratification of tachyarrhythmias in athletes, both supraventricular and ventricular, especially if TTE is guided by assessment of ECG abnormalities and qualitative characteristics of PVBs [18]. Therefore, in presence of ECG abnormalities and/or arrhythmias, those data should be shared with the physician performing TTE. Primarily, 12-lead resting ECG can raise the suspicion of a concealed genetic, syndromic or metabolic cardiomyopathy [19]. In case of a doubtful pathological LV hypertrophy, TTE is extremely useful to define LV thickness and mass, dimensions and volumes and systo-diastolic function, as well as aortic sizes and atrial dimensions, particularly in hypertensive heart disease [20]. In athletes with supraventricular arrhythmias, particularly atrial fibrillation, TTE should evaluate atrial size, volumes and function and to rule out heart valve disease. In the suspicion of arrhythmogenic right ventricular cardiomyopathy (ARVC), assessment of the RV dimensions, regional wall motion abnormalities (WMAs), and functional indexes appear crucial for a reliable report. Table 1 summarizes the TTE work-up in sports cardiology based on the abnormal or borderline finding identified at the 12-lead resting ECG.

4.2. T-wave inversion

Anterior T-wave inversion (TWI) represents a normal finding in asymptomatic children <16 years old and usually disappears with pubertal development, the so called juvenile T-wave pattern [21,22]. Similarly, TWI confined to V1-V4 may be considered a normal finding, particularly in black athletes, when preceded by J-point/ST-segment elevation and when the clinical suspicion is low (no symptoms, negative family history of SCD/cardiomyopathy, no arrhythmias on exercise testing, etc.…) [4]. In case of doubt, a comparison with previous ECG tracings could be very helpful in directing the echocardiographer in imaging acquisition. However, TWI is a recognized hallmark of ARVC or HCM, and TTE represents the first-line imaging tool to address further tests, including the indication to CMR. Therefore, a careful echocardiographic evaluation should include assessment of biventricular dimensions and function and rule out LV or RV WMAs. In the presence of extended anterior TWI or inferolateral TWI, TTE should exclude the presence of pathological hypertrophy, which may be segmental and confined to specific segments, such as the LV apex. Notably, even if imaging techniques are completely normal in athletes with TWI, indication to CMR should be considered in the individual case and a strict follow-up with continuous clinical surveillance is recommended in these

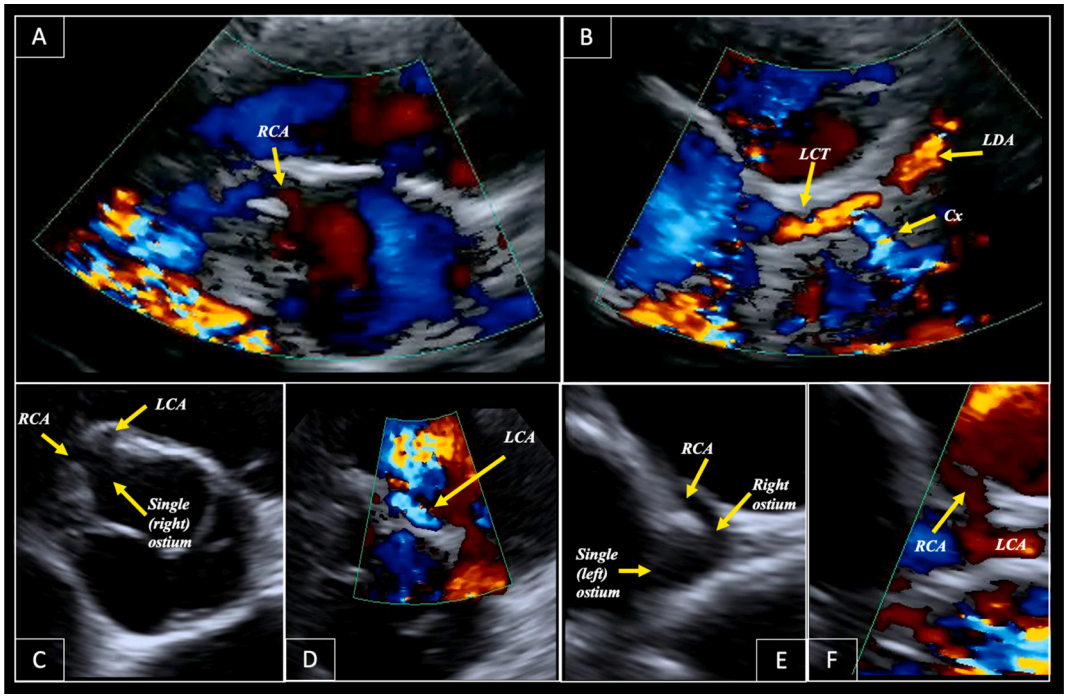

Fig. 2. Evaluation of coronary artery by transthoracic echocardiography. Panel A: the right coronary artery (RCA) ostium and first tract are visualized arising from the right coronary sinus of Valsalva. The left coronary trunk (LCT), left descending artery (LDA) and circumflex artery (Cx) are nicely depicted in Panel B. Panel C shows the anomalous aortic origin of the left coronary artery: both the left coronary artery (LCA) and the right coronary artery (RCA) originate from a common right ostium in the right coronary sinus. The use of color Doppler clearly identify the interarterial intramural course of the LCA in Panel D. Panel E shows the anomalous aortic origin of the RCA from the left coronary sinus and the interatrial course of the RCA (Panel F).

individuals because ECG abnormalities may represent the initial expression of underlying cardiomyopathies that may not be evident until many years later and may ultimately be associated with adverse outcomes [23]. CMR can be an additional exam in highly suspicious cases.

TWI can also be the expression of myocarditis or channelopathy, such as long QT syndrome (LQTS). Finally, in master athletes, the presence of TWI may express ischemic heart disease [24]. In this case, a careful assessment of LV function and WMAs is required, although additional provocative tests, including CCTA, stress echocardiography, stress CMR and SPECT are useful to exclude or confirm the presence of coronary artery disease.

4.3. Channelopathies

TTE is primarily indicated to confirm the absence of structural abnormalities in patients affected with LQTS or Brugada syndrome. Regional mechanical alterations were first noted using M-mode in LQTS patients and associated with arrhythmic events [25]. However, the advent of TDI and STE techniques has expanded the role of TTE, demonstrating that mechanical dispersion is more prominent in symptomatic LQTS versus asymptomatic LQTS patients and is absent in healthy individuals [26,27]. Similarly to the mechanical dispersion, the electromechanical window can also identify patients with increased arrhythmic risk [26,27].

In Brugada Syndrome (BrS), the increasing evidence of cardiac structural abnormalities and common genetic substrate with ARVC has progressively led to the concept of the two being the expression of a wider disease and, in some cases, the overlapping syndrome [28]. Therefore, in BrS patients, particular attention should be spent on the study of the RV to detect regional WMAs, and STE can be useful to further differentiate between the Brugada pattern and RBBB [29].

4.4. Syncope and near-syncope symptoms

The prevalence of syncope is very low in the young athletic population (~6%), with most syncope spells being unrelated to exercise [30]. Nevertheless, athletes with syncope represent a particular clinical challenge as the potential causes of their spells can range from conditions with a benign prognosis to CV disorders associated with SCD. Indeed, it is useful to classify athletes with syncope into three distinct subgroups based on the syncope timing: unrelated to exercise, exertional or post-exertional. After initial history, physical examination, and resting ECG, appropriate diagnostic tests should be selected by risk stratification considering low- and high-risk clinical features (Supplementary Table 1) [31].

In the setting of syncope *unrelated to exercise* and low-risk features, ancillary diagnostic imaging, including TTE, has a relatively low diagnostic yield and should not be routinely used for syncope assessment. In patients with *post-exertional* spells and low-risk features, vaso-vagal and orthostatic hypotension are the most common causes of syncope, usually exacerbated by venous pooling during exercise, volume depletion, dehydration and orthostasis and by peripheral vasodilation as a physiologic response to heat production (heat syncope) [32]. In selected individuals with post-exertional syncope and intermediate-to-high risk profile, TTE can be recommended as a second-tier non-invasive test useful to rule out a concealed cardiac pathology and to assess intravascular volume status.

Exertional syncope is a red flag and a high-risk feature raising concern for underlying structural heart disease. Syncope during (or immediately after) exercise is possibly the only symptom preceding sudden death. In this context, echocardiography is highly recommended to diagnose structural CV abnormalities and should include imaging of the origin and proximal course of the coronary arteries [15].

Exercise stress echocardiography can be further considered for dynamic assessment of latent LV outflow tract obstruction in HCM or subaortic stenosis, pulmonary artery pressure in subjects with heart

valve disease, congenital heart disease, or suspected pulmonary hypertension, and for evaluation of LV diastolic function and biventricular systolic function if there is suspicion of an underlying cardiomyopathy.

5. How to image the heart by echocardiography for the suspicion of an underlying cardiovascular disease

5.1. Hypertrophic cardiomyopathy

HCM is diagnosed when the LV wall thickness is ≥15 mm and is not explained solely by loading condition [19]. Lesser degrees of wall thickening (13–14 mm) can also be suspicious for HCM in presence of family and personal history, genetic findings and ECG abnormalities consistent with HCM. The diagnosis of HCM requires a multi-parametric evaluation, TTE evaluation should include the description of LV hypertrophy, its distribution, localization, and severity, as well as mitral valve (MV) apparatus abnormalities [33]. A full description of the 4-heart chambers' volumes and function should be included in the TTE report [2]. Pathological LV hypertrophy is usually asymmetric, even with the involvement of few LV segments, while most athletes with physiological LV remodeling show symmetric and homogeneous LV hypertrophy [2,8,34]. HCM commonly shows a reduced (<43 mm) or normal LV cavity size, while the physiologic LV eccentric hypertrophy in athletes is usually associated with an enlarged LV cavity (end-diastolic diameter > 54 mm), particularly in male endurance athletes [2,33,35]. Moreover, the athlete's heart is characterized by a harmonic and balanced increase of all cardiac chambers, while a non-harmonic, disproportionate remodeling potentially suggests pathological LV hypertrophy (as also in hypertensive heart disease) [36]. Ejection fraction is typically increased in patients with HCM. Conversely, myocardial longitudinal velocities on TDI and deformation parameters on STE are often reduced and may be abnormal even before HCM phenotype is fully developed [37,38]. Additional morphological abnormalities, including papillary muscle and mitral leaflet abnormalities may be present. The systolic anterior motion of the mitral valve apparatus may provoke LV outflow tract obstruction, which should always be evaluated at rest and during Valsalva maneuver, in the standing position or during exercise test by Doppler echocardiography [39].

Competitive athletes can show left atrial (LA) enlargement that can overlap with atrial dilation observed in HCM patients [40]. However, the enlarged atria are characterized by normal reservoir function and myocardial stiffness in athletes and associated with enlarged LV cavity, while HCM patients with increased LA volumes show a small/normal LV cavity and lower values of LA reservoir function, as assessed by peak atrial longitudinal strain at STE [40,41]. LA enlargement in athletes is a benign adaptation to the increased volume overload induced by intensive training, while LA dilatation in HCM is multifactorial, mostly related to LV diastolic dysfunction and/or mitral regurgitation(MR) [18,41]. Indeed, diastolic function is usually abnormal in HCM sedentary patients, while athletes with physiological LV hypertrophy usually demonstrate a supernormal diastolic function with normal estimated filling pressures on TDI [42,43]. Of note, in athletes with HCM, normal indices of diastolic pulsed wave Doppler (PW) transmitral filling can be observed, but reduction of septal e' velocity by TDI may be present even in the early stage of the disease [33,42,43]. However, a few athletes with sarcomeric HCM, due to pathogenic variants and mild concentric LV hypertrophy, may also show a relatively increased LV cavity size, normal diastolic LV and atrial function, then the precise diagnosis relies on additional tests [44,45] (Fig. 3, Table 1). TEE can underestimate (or even did not detect) hypertrophy limited to the antero-lateral free wall, posterior interventricular septum or LV apex, while CMR identifies LV and RV involvement in all segments; additional abnormalities of the mitral valve apparatus and papillary muscles; but also the presence of late gadolinium enhancement [46].

5.2. Dilated cardiomyopathy

Dilated cardiomyopathy (DCM) is defined by the presence of LV or biventricular dilatation (>2 z-scores above population mean values) and systolic dysfunction (LVEF<50%) in the absence of abnormal loading conditions or coronary artery disease sufficient to cause global systolic impairment [19]. Beyond the classic phenotype, the clinical spectrum of the disease also includes LV dilatation in the absence of reduced LVEF (i.e. isolated LV dilatation) and reduced LVEF in the absence of LV cavity dilatation (i.e. hypokinetic non-dilated cardiomyopathy) [47]. These conditions are generally considered different expressions of the disease

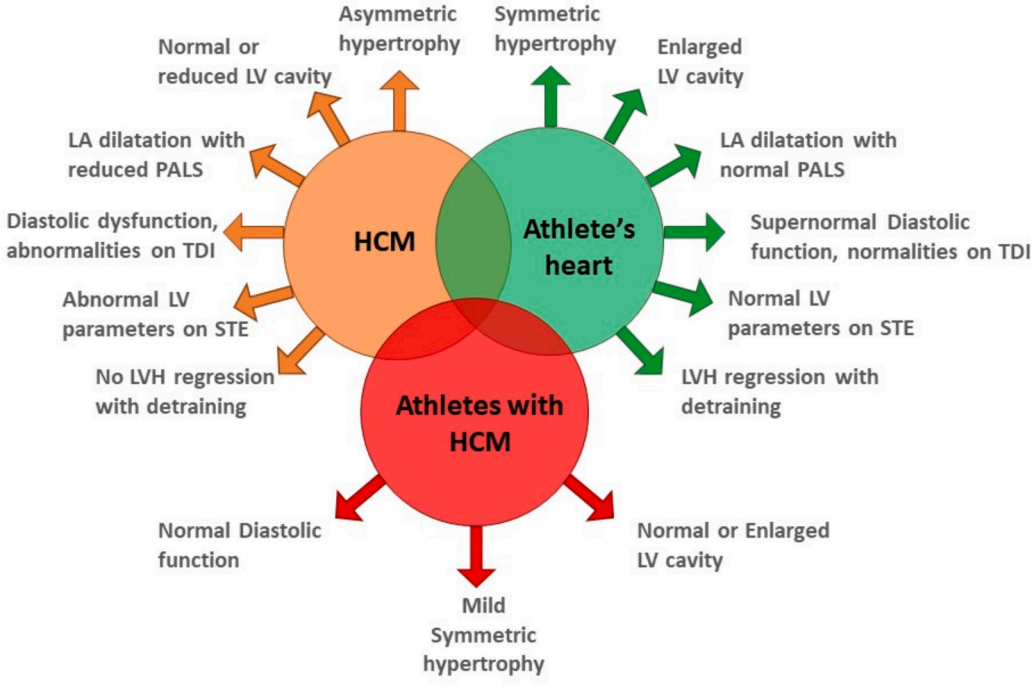

Fig. 3. Differences between the specific features of the athlete's heart, hypertrophic cardiomyopathy (HCM) and athletes with HCM.

and suggestive of a cardiomyopathy in the context of a family history of DCM. However, it should be emphasized that these entities may represent a diagnostic issue in the differentiation from the athlete's heart. As a first-line imaging modality, TTE should systematically report LV diameters and volumes, LV wall thickness, presence of WMAs and systolic function [19]. Furthermore, regional LV WMAs with coronary artery distribution may suggest the presence of ischemic heart disease. Diastolic dysfunction, LA dilatation, RV dysfunction, and mitral and tricuspid functional regurgitation are supportive for diagnosing DCM instead of athlete's heart. Moreover, in case of mildly reduced LV function at rest (EF 50% to 55%) exercise echocardiography may help in differential diagnosis. An impaired LV STE (i.e. GLS $< -17\%$) and reduction of LV contractile reserve at stress echocardiography (i.e. an increase of LVEF<11% and peak LVEF <63%) favor the diagnosis of DCM instead of physiological remodeling [48,49]. Second-line imaging modalities should be targeted to the suspected aetiology [48] and indication to CMR should be considered, due to the a decisive role of CMR in the diagnostic pathway by identifying a non-ischemic scar [19].

5.3. Arrhythmogenic right ventricular cardiomyopathy

The diagnosis of ARVC is challenging and requires a comprehensive analysis that relies on several clinical parameters in addition to cardiac imaging, including a detailed personal and family history and ECG interpretation [50]. The echocardiographic analysis should report a combination of regional RV WMAs and global RV dilation/dysfunction. Specific criteria for the appropriate interpretation of echocardiographic RV dimensional data have been published [51]. While RV dilatation is one of the typical features of the athlete's heart, the cut-off values proposed as diagnostic criteria were derived from studies on the general population. The degree of RV dilatation in the athlete should be interpreted considering the type of sports practiced, the training volume and intensity per week, and the years of sports practice [52,53]. Indeed, athletes engaged in endurance sports, particularly those requiring a combination of static and dynamic exercise such as rowing or canoeing, exhibit the greatest RV dimensional remodeling, while athletes engaged in strength sports, such as weight-lifting, exhibited the lowest degree of RV remodeling [52]. The presence of RV dysfunction with WMAs is uncommon and absent in the athlete's heart [51]. The assessment of RV WMAs requires specific expertise. In suspected cases of ARVC, dedicated acoustic windows should be performed to identify these anomalies, such as the modified parasternal long-axis view (LAX) and the subcostal view. At the same time, 3D echocardiography may accurately measure RV volumes and ejection fraction, and STE may characterize myocardial deformation [54], further contributing to the differential diagnosis between athlete's heart and ARVC [51,55](Table 1). In addition, CMR is very helpful to better define the RV dilatation, including RV outflow tract dilatation and rounded RV apex, the presence of RV WMAs and quantification of the ejection fraction, as well as the presence of RV/LV LGE [55].

5.4. Arrhythmogenic left ventricular cardiomyopathy

The current 2023 ESC guidelines [19] define non-dilated LV cardiomyopathy as the presence of non-ischaemic LV scarring or fatty replacement in the absence of LV dilatation, with or without global or regional WMAs. More recently, a European task force proposed specific diagnostic criteria for arrhythmogenic left ventricular cardiomyopathy (ALVC), with the aim to improve the diagnosis of the left variant of "non-ischemic" myocardial scarring predisposing to ventricular electrical instability [55]. If ALVC is suspected based on ECG abnormalities or PVBs morphology [18], TTE should report all morpho-functional abnormalities, if any, adding the relevant data on LV and RV dimensions, volumes and function, systolic pulmonary artery pressures (PAP), atrial dimension and geometry. Echocardiography may demonstrate the presence of wall thinning or irregular wall profiles, LV WMAs or LV dysfunction (Table 1), and advanced techniques such as TDI and STE can help in identifying subclinical myocardial dysfunction, but in an early-concealed form, the echocardiography may be still normal, and the diagnosis supported by the presence of a LV scar requires CMR with LGE. In addition, electroanatomic mapping may confirm diagnosis by the presence of low voltages suggestive for structural myocardial abnormalities [55,56].

5.5. Excessive trabeculation of the left ventricle (LV non-compaction)

LV non-compaction is characterized by the presence of a double-layered myocardial wall, with a thicker non-compacted (NC) subendocardial layer characterized by pronounced trabeculations and a thin compacted (C) epicardial layer [57,58]. Several physiological conditions characterized by volume overload, such as eccentric physiologic remodeling in athletes and pregnancy, are associated with excessive trabeculation, without evidence of a pathologic conditions (i.e., LV hypertrabeculation), but a LV non-compaction phenotype can also be found associated with other primary cardiomyopathies (i.e. DCM, HCM, ARVC, etc.). Therefore, at present, excessive trabeculation should be considered a phenotypic trait [19], which can occur in isolation and do not require any additional exam. In addition, when excessive trabeculation appears in coexistence with other characteristics such as LV WMAs, LV systolic and diastolic dysfunction, reduced LV GLS, reduced LV contractile reserve during exercise and an abnormal tissue characterization at CMR (i.e. presence of LGE), it supports the diagnosis of a primary heart muscle disease [57,58]. Therefore, only in the suspicion of a primary heart muscle disease, the identification of a pathogenic genetic mutation in sarcomeric, cytoskeletal, mitochondrial, desmosomal, storage and ion channel genes can reinforce the diagnosis [45].

5.6. Myocarditis and pericarditis

Myocarditis is an inflammatory disease of the heart that may occur because of infections, immune system activation, or exposure to toxic substances, with a wide clinical spectrum of presentations, ranging from minor symptoms to heart failure, arrhythmias, cardiogenic shock and SCD [59].

Athletes with clinically suspected myocarditis should undergo a standard TTE to assess cardiac chamber size, regional and global function, WMAs, and pericardium [60]. A broad spectrum of findings can be observed, spanning from minor LV asynergies to global systolic dysfunction, usually in the presence of normal or mildly increased mid-apical LV size, increased LV wall thickness, and abnormal myocardial echogenicity [61]. Diastolic dysfunction (i.e. restrictive filling pattern), ventricular thrombi and RV dysfunction could also be observed [61]. Quantitative evaluation of regional and global LV function through STE should be performed whenever feasible since it can unmask subclinical contractility abnormalities and systolic dysfunction [62]. Moreover, it was reported a correlation between regional strain values and presence and extent of LGE detected by CMR [62]. Follow-up TTE and CMR are relevant to verifying LV systolic function's normalization and assessing residual oedema and/or LGE [63].

In case of pericarditis, TTE should describe pericardial thickness and the presence of pericardial effusion but also identify LV WMAs and global systolic dysfunction in the presence of coexisting myocardial involvement [64]. The evaluation of septal motion and respiratory changes in transmitral, transtricuspid and suprahepatic vein flow, as well as TDI, are useful in specific settings, such as constrictive pathophysiology [64]. Even if follow-up TTE is generally not recommended in non-complicated forms, we consider reasonable a revaluation before the resumption of exercise [63].

5.7. Evaluation of the athlete with a congenital heart disease (CHD)

Since 2013, the European recommendations for physical activity in

adolescents and adults with CHD have been based on haemodynamic and electrophysiological parameters [65], moving away from specific anatomical defects, which do not correlate to exercise-associated risks. The evaluation is based on five parameters: (I) ventricular function, (II) pulmonary artery pressure, (III) aortic dimensions, (IV) presence of arrhythmias, and (V) arterial oxygen saturation at rest and during exercise. Echocardiography has a pivotal role in assessing three out of five of those parameters, ideally in centers dedicated to the evaluation of CHD (Supplementary Fig. 1).

5.7.1. Assessment of ventricular structure and function

Functional assessment by echocardiography should follow published guidelines [66,67], considering sex, age, ethnicity, BSA, family history, symptoms, type of sport, and sport volume. Because of the highest frame rate compared with all the other imaging techniques, echocardiography is the best technique to detect and unmask dynamic and fixed obstructions. The echocardiographic report should always provide LV end-diastolic and end-systolic diameters, wall thickness and volumes, BSA-index LV mass, and LV ejection fraction measured by biplane Simpson's method. In expert centers, it is recommended to include LV global longitudinal strain (GLS), excluding segments where a patch has been placed, and 3D LV volumes assessment, including 3D ejection fraction [66].

Echocardiography can measure RV diameters and function. For the RV morphological assessment diameters, both RV end-diastolic diameters at the inlet and at the outlet portion must be provided [67]. RV free wall thickness from parasternal long-axis view or from subcostal view can be measured. RV function should be assessed, including fractional area change (FAC), TAPSE and myocardial peak systolic velocity of the tricuspid valve annulus (RV TDI S′ wave). In centers with experience in advanced echocardiography and CHD, RV STE parameters and 3D RV ejection fraction should be included in the report [66]. In patients with specific CHD, where coronary abnormalities are more frequent, either because of the original cardiac abnormality (cono-truncal diseases, coronary artery fistula, anomalous left coronary artery from the pulmonary artery, etc.) or because of the surgical repair (arterial switch repair, Ross operation, etc.) visualization of the coronary arteries and functional test (exercise stress echo) should be part of the assessment.

5.7.2. Assessment of pulmonary artery pressure

TTE is usually sufficient to evaluate the systolic PAP in individuals with tricuspid regurgitation (TR). Pulmonary hypertension (PH) is excluded when TR velocity is $\leq$2.8 m/s, and no additional echocardiographic variables suggestive of PH are present. Thus, TR velocity should always be included in the report. In cases where TR velocity cannot be sampled, the use of saline contrast can be helpful to obtain a better Doppler trace. In the absence of a good TR trace, the sampling of pulmonic regurgitation can be used to estimate mean and diastolic PAP. Indirect echo signs of PH should also be included in the evaluation (LV eccentricity index >1.1, RVOT acceleration time < 105 ms and/or notching) [68]. In cases where a high index of suspicion for PH persists, particularly when restriction from some or all competitive sports is contemplated, right heart catheterization should be performed [69].

5.8. Aortic diameters

Aortic diameters in athletes should be measured by TTE using standard methodology [67,70](Supplementary Fig. 2). Serial measurements are needed in case of aortic dilatation because the rate of increase in aortic diameter is important for risk stratification [71]. While absolute and indexed aortic diameters are useful, z-scores may ultimately be used to define normal values ($-2 < z > +2$) and reported in the final echocardiographic report, particularly when the aorta is dilated [72,73]. In case of borderline or pathological values, additional cross-sectional imaging by CT or CMR and regular follow-up are required [74].

Aortic root dilatation in repaired tetralogy of Fallot (TOF) or pulmonary atresia ventricular septal defect patients is a frequent finding in about 1/3 of such patients in different studies [75]. For these patients, an index based on an observed to expected aortic root diameter ratio > 1.5, should be considered rather than the absolute value [75]. Although aortic aneurysm is common in TOF patients, progressive aortic dilation is uncommon and aortic dissection is very rare in TOF patients with significant aortic aneurysms who did not undergo aortic surgery, rendering it an exceedingly rare complication [75]. Thus, aortic root dilatation in such patients should be interpreted with caution by expert centers on CHD.

Exercise echocardiography is a sensitive tool to unmask significant pathology in CHD patients [76,77]. This modality is particularly useful in the presence of borderline lesions or residual defects to assess their impact on hemodynamics and ventricular function during exercise.

5.9. Bicuspid aortic valve

The congenital BAV is a common condition (0.5–2% of the adult general population) with a heterogeneous, incompletely defined genetic background [16,78–80]. Similarly, the natural history of BAV is characterized by a significant heterogeneity of its valvular and aortic phenotypic expressions and associated disorders [81]. The echocardiographic assessment of the valvulo-aortopathy of the BAV syndrome is then aimed at (i) detecting the BAV and characterizing its phenotype; (ii) assessing the presence and severity of valvular hemodynamic impairment; (iii) measuring the thoracic aorta (TA), as dilatation is the most prevalent clinical expression of BAV-related aortopathy; (iv) ruling-out commonly associated congenital disorders [76]. Aortic coarctation, in particular, is present in 7–10% of adults with BAV, whereas BAV is detected in up to 60% of patients with coarctation and should be, thus, systematically ruled-out in BAV patients [82].

Whatever the imaging modality, BAV is diagnosed in short-axis views at the base of the heart, demonstrating, in systole, the existence of only 2 commissures delimitating only 2 cusps [78](Supplementary Fig. 3). Recently, a consensus statement has provided a classification approach covering the entire spectrum of the disease [78]. The 3 BAV phenotypes are: (1) the so-called "fused BAV" (90–95% of BAV) with 3 sinuses of Valsalva and 3 subgroups, based on the fusion of the right-left cusps (70–80%), the right and non-coronary cusps (20–30%), and left and non-coronary cusps (3–6%). A fibrous ridge between the fused cusps (raphe) is frequent, though it may become detectable by TTE over time. (2) the "2-sinus BAV" (5–7%) with only 2 sinuses of Valsalva and 2 subgroups characterized by the lateral-lateral, or anteroposterior orientation. (3) The "partial fusion BAV" (or *form fruste*), whose prevalence is actually undetermined [79,80,83].

Aortic dilatation is the most common manifestation of BAV aortopathy [79,83]. Its prevalence varies according to the definition of normality and the age of the patient, resulting in a significant increase in lifetime risk of acquiring aortic dilatation compared to the general population [84]. Aortic dilatation can occur in any region of the whole TA [79,85]. There are, however, 2 major forms of aortic dilatation in BAV [79,85]: (1) the ascending phenotype (70%), preferentially located beyond the sinotubular junction, and (2) the root phenotype (20%) preferentially located at the sinuses of Valsalva, possibly involving also the ventriculo-aortic junction.

5.10. Mitral valve prolapse

The prevalence of mitral valve prolapse (MVP) in athletes is not different from the general population, ranging from 0.6 to 2.5% [86], but it is mandatory to accurately identify the presence of MVP, as a systolic displacement of one or both mitral valve (MV) leaflets $\geq$2 mm above the plane of the mitral annulus in the sagittal view [87] to avoid overdiagnosis. Under the umbrella of degenerative MR, a plethora of conditions are included, ranging from mitral valve prolapse (MVP) due to Barlow's disease to fibroelastic deficiency. While thickened leaflets

with excessive tissue, MV annular dilatation and chordal thickening/elongation characterize Barlow's disease, in fibroelastic deficiency, the leaflets and chords appear thinner and elongated. Bi-leaflet Barlow's disease was associated with a malignant and potentially arrhythmogenic phenotype. Therefore, qualitative analysis of mitral valve (MV) morphology and the definition of the prolapsing scallop(s) should be included in the report [88,89]. In addition, the presence or absence of mitral annular disjunction (MAD), which is a systolic separation between the ventricular myocardium and the mitral annulus supporting the posterior leaflet (anteriorly, the presence of the mitro-aortic fibrous continuity prevents MAD), should be described [86](Fig. 4). The presence of MR is easily identified qualitatively using the color Doppler and the flow jet area, but a more semi-quantitative or quantitative approach is needed to grade the severity of MR when more than a small, central jet is identified [90]. In addition, data related to LV end-systolic and end-diastolic dimensions and volumes, LA volume index and LV ejection fraction are mandatory in the report and should be referred to the type, intensity and years of sports practice. Even if a severe MR can be asymptomatic and well-tolerated, severe MR is characterized by excess mortality and risk of SCD irrespectively of MV morphology [91]. It is pivotal to rule out possible symptoms related to a significant MR or an arrhythmogenic MVP, such as palpitations, reduced performance, dyspnea at effort and unexplained pre-syncope or syncope [92]. Stress echocardiography can be valuable in athletes to evaluate the impact of MR during effort and reduced performance or symptoms at effort in apparently asymptomatic athletes. On the other side, features of malignant arrhythmogenic MVP should be ruled out irrespective of the hemodynamic impact of the MR [86,88]. In athletes with MVP and complex ventricular arrhythmias at rest or during exercise, CMR with LGE is particularly useful for identifying focal myocardial fibrosis and better defining LV volumes, function and the hemodynamic impact of MR [18,87].

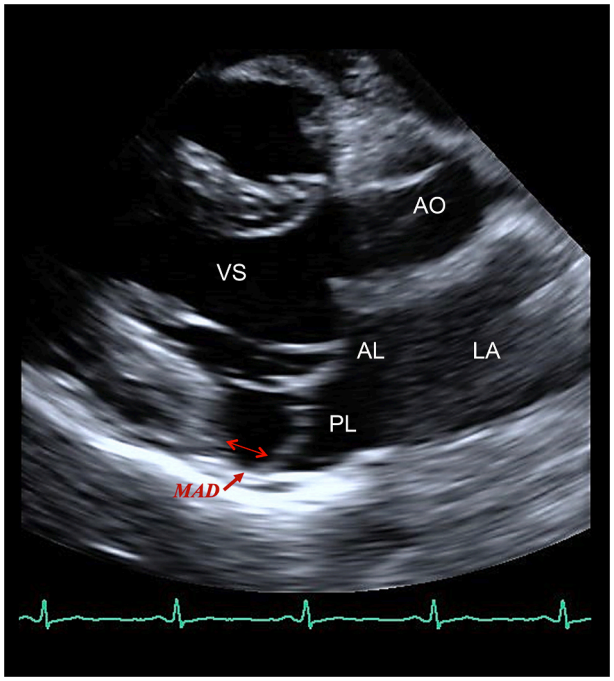

Fig. 4. In the parasternal long axis view the presence of mitral annular disjunction (MAD) is appreciated (red arrow) as a detachment between the posterior myocardium and the posterior mitral annulus. The two mitral leaflets are visible. AL: anterior leaflet; AO: aortic root; LA: left atrium; LV: left ventricle; PL: posterior leaflet.

6. Conclusion

Echocardiography is an invaluable imaging technique in sports cardiology to evaluate the physiological adaptations and to rule out CV disease at risk for SCD and to follow up athletes with CV abnormalities. TTE is more powerful when a comprehensive report, with the appropriate data, is provided. Therefore the indication for the exam should be clearly identified, and the report should conclude or require additional examinations related to the specific clinical indication (Table 2). Even if echocardiography is not used as a screening exam, its availability, cost-effectiveness and acceptability make it the perfect first-line exam when indicated.

CRediT authorship contribution statement

Elena Cavarretta: Writing – review & editing, Writing – original draft, Conceptualization. **Flavio D'Ascenzi:** Writing – original draft, Conceptualization. **Massimiliano Bianco:** Writing – original draft. **Silvia Castelletti:** Writing – original draft. **Luna Cavigli:** Writing – original draft. **Franco Cecchi:** Writing – review & editing. **Antonello D'Andrea:** Writing – original draft. **Giovanni Di Salvo:** Writing – review & editing, Writing – original draft. **Stefano Nistri:** Writing – original draft. **Zefferino Palamà:** Writing – review & editing, Writing – original draft. **Vincenzo Palmieri:** Writing – review & editing, Writing – original draft. **Fabrizio Ricci:** Writing – original draft. **Gianfranco Sinagra:** Writing – review & editing, Writing – original draft. **Alessandro Biffi:** Writing – review & editing. **Antonio Pelliccia:** Writing – review & editing. **Silvio Romano:** Writing – review & editing. **Antonio Dello Russo:** Writing – review & editing, Writing – original draft, Conceptualization. **Paolo Zeppilli:** Writing – review & editing. **Giampiero Patrizi:** Writing – review & editing. **Luigi Sciarra:** Writing – review & editing, Conceptualization.

Declaration of competing interest

The authors report no relationships that could be construed as a conflict of interest.

All authors have seen and approved the manuscript being submitted, have contributed significantly to the work, attest to the validity and legitimacy of the data and its interpretation, and agree to its submission to the International Journal of Cardiology.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijcard.2024.132230.

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