

GUIDELINES AND RECOMMENDATIONS

# A guideline update for the practice of echocardiography in the cardiac screening of sports participants: a joint policy statement from the British Society of Echocardiography and Cardiac Risk in the Young

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## Abstract

Sudden cardiac death (SCD) in an athlete is a rare but tragic event. In view of this, pre-participation cardiac screening is mandatory across many sporting disciplines to identify those athletes at risk. Echocardiography is a primary investigation utilized in the pre-participation setting and in 2013 the British Society of Echocardiography and Cardiac Risk in the Young produced a joint policy document providing guidance on the role of echocardiography in this setting. Recent developments in our understanding of the athlete's heart and the application of echocardiography have prompted this 2018 update.

### Key Words

- ▶ cardiac screening
- ▶ athletes heart
- ▶ echocardiography

## Introduction

### Update to 2013 guidelines

The first British Society of Echocardiography (BSE) and Cardiac Risk in the Young (CRY) guidelines for the role of echocardiography in the cardiac screening of sports participants were published in 2013 and have

been implemented in screening of athletes throughout the United Kingdom. Since its release there have been important additions to the evidence base, which have advanced our understanding of the athlete's heart (AH)

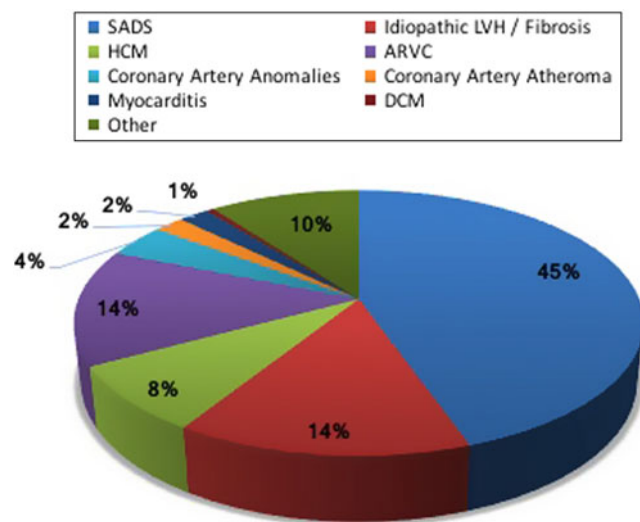
and the role of diagnostic testing. This includes new international guidelines on the athlete's electrocardiogram (ECG) (1) and further insight into the multi-factorial nature of the phenotypic expression of the AH (2, 3). Consequently, the BSE and CRY consider it timely to provide an updated version of the 2013 guidelines.

### Which conditions to look out for?

This BSE document is aimed at providing guidance for the use of echocardiography in screening young athletes (ages 14–35 years) for inherited and congenital cardiac disease and is endorsed by CRY. Sudden cardiac death (SCD) in young athletes although relatively rare has a devastating impact on the individual, their family and the wider community. The causes of SCD in the athletic population have been reported extensively in the literature with Fig. 1 highlighting relevant conditions from a UK registry of young athletes (4). Although in contemporary studies sudden arrhythmic death syndrome (SADS) predominates, structural heart disease, potentially detectable by echocardiography, still comprise many SCD cases in young and athletic individuals (5, 6). Cardiac screening is aimed at identifying these conditions to reduce the risk of SCD.

### The 12-lead electrocardiogram should be the first investigation

Current European recommendations (7) for cardiac screening of the athlete state that following a detailed questionnaire (including any symptoms or family history)



**Figure 1**  
Causes of sudden cardiac death.

and brief examination, the 12-lead electrocardiogram (ECG) should be the primary investigation. The ECG should be interpreted in accordance with International Consensus guidelines (1) (Fig. 2). Those with 2 or more 'Borderline ECG Findings' or ANY 'Abnormal ECG Findings' require further investigation.

### A full standard echocardiographic assessment should be performed

The specific sporting/screening organisation will decide whether their athletes require a routine transthoracic echocardiogram (TTE) as a first-line investigation within the standard screening process. In the case of an ECG-only screening, TTE is recommended as a second-line investigation in those athletes with an abnormal ECG, cardiovascular symptoms, abnormal physical examination findings or a family history of sudden death under the age of 40 years. Regardless of whether TTE is a first- or second-line investigation, it should be performed according to the BSE Minimum Dataset for a Standard Transthoracic Echocardiogram in an Adult (8) and should also consider recommendations made in the Supplementary Protocols for (i) Comprehensive Assessment of the Right Heart and (ii) the Assessment of Diastolic Function. Details of where and how to measure specific echo parameters are given in these three protocols, which are available online at [www.bsecho.org](http://www.bsecho.org). There are however additional elements that may be considered optional in non-athletes that become highly relevant in athletes to ensure a comprehensive evaluation. For example, the assessment of the coronary arteries to exclude coronary artery anomalies. If there is evidence of an abnormality, other supplementary guidelines may then become relevant and should be utilized. For example, the application of this protocol may identify right ventricular (RV) enlargement and possible dysfunction, and it would therefore be pertinent to then utilize the ARVC protocol to obtain further diagnostic information.

### Type of athletic activity should be known

It is important to use the BSE normative values based on sex and age but take account of the type of sporting activity performed. All echocardiographers involved in cardiac screening of athletes should understand the physiological adaptation in cardiac structure and function to regular exercise, which can be variable depending on the type and volume of exercise training. All cardiac chambers may enlarge (9). Physiological adaptation of the left ventricle usually falls within the constraints of

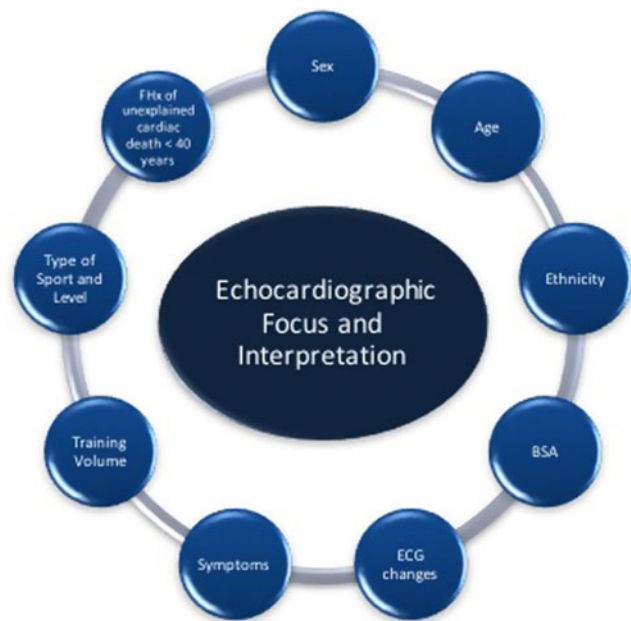


**Figure 2** ECG athlete interpretation.

normal geometry although eccentric hypertrophy may occur especially in athletes who engage in high dynamic sports (10). A relatively recent systematic review and meta-analysis (9) highlights ranges for trained male athletes and demonstrates that greater LV chamber size is likely in endurance athletes compared to those that have a resistance focus. Resistance activity can be defined as

**Table 1** Sporting examples demonstrating specific MET values for determining exercise intensity (12).

Sporting discipline	Metabolic equivalent (MET)
Soccer	10.0
Running (6 mph)	10.0
Running (7.5 mph)	12.5
Running (10.9 mph)	18.0
Cycling (>20 mph) racing	16.0
Cycling (<10 mph) leisure	4.0
Cricket	5.0
Rugby	10.0
Tennis (singles)	8.0
Hockey	8.0
Boxing	12.0
Golf	4.5
Rowing (competitive)	12.0
Swimming (leisure)	6.0
Swimming (competitive)	10.0



**Figure 3** Pre-echocardiographic information.

**Table 2** Considerations for each of the specific pre-examination factors.

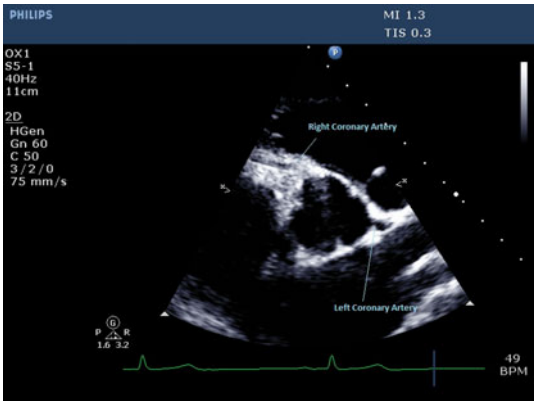
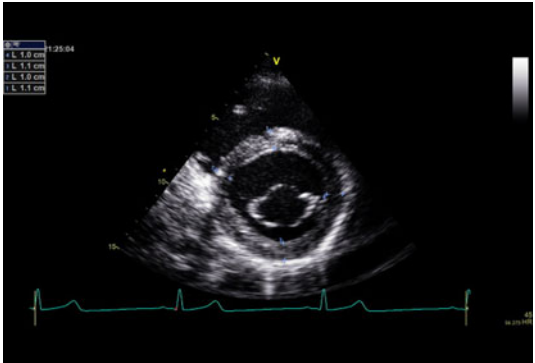
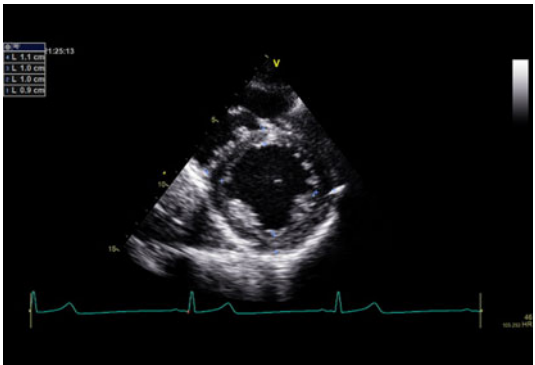
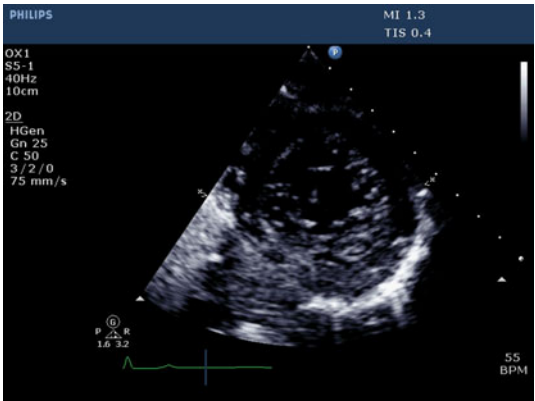
Sex	Cardiac chamber dimensions in female athletes rarely fall outside of the established 'normal range'. If they do, further investigation is required (10, 13, 14, 15) It is more common for male athletes to demonstrate a degree of eccentric remodeling of all cardiac chambers (10, 15)
Age	Highly trained junior athletes still develop cardiac remodeling in response to physiological conditioning, but this is often at a lower magnitude than in senior athletes (16, 17) That aside, where structural values fall outside the BSE 'normal range' functional assessment is key
Ethnicity	LV (18) and RV (19) cavity sizes are similar between African/Afro-Caribbean and white athletes; however, wall thicknesses and LA size are often larger in the African/Afro-Caribbean athlete (19, 20) Any wall thickness measurement with a value greater than 13 mm in white male athletes (or greater than 11 mm in white female athletes) or greater than 15 mm (20) in African/Afro-Caribbean male athletes (or 13 mm (21) in African/Afro-Caribbean female) requires further investigation There is a lack of data pertaining to the structure and function of Asian athletes although there is no significant difference in ECG findings between West Asian and Caucasian athletes (22). The lack of available data on Asian ethnicity suggests that standard criteria as applied to Caucasian athletes should be utilized until further data is available
Body surface area (BSA)	The relationship between body size and chamber dimensions is well established (23, 24) and therefore all chamber dimensions should be indexed for body surface area. That aside, cardiac adaptation to exercise involves eccentric hypertrophy beyond what may be attributable to body composition alone (9, 23) In the extremes of height and weight (BSA > 2.3 m <sup>2</sup> ) non-indexed LV wall thickness and diastolic diameter should not exceed 15 mm and 65 mm respectively (25)
Symptoms	A positive history including exertional chest pain, syncope or near-syncope, irregular heartbeat or palpitations, shortness of breath or fatigue and in particular exertional symptoms should direct the echocardiographer to closely assess for potential causes of SCD (4) (Fig. 1). Symptoms are non-specific and therefore it is important to ensure all possible causes are excluded. That aside it is important to be aware that exertional chest pain may direct further evaluation for coronary anomalies while syncope may be related to arrhythmogenic substrate such as ARVC or HCM or to outflow obstruction
ECG changes	The type of ECG changes that are present on an athlete's ECG will further guide the focus of the examination. For example T-wave inversion in leads V1–V3 is one of the hallmarks of ARVC and should lead to a more focused assessment of the right heart (26). On the contrary inferolateral T-wave inversion is more frequently present in HCM and should prompt a detailed LV assessment (1)
Training volume/level	Elite athletes are likely to demonstrate a greater degree of physiological cardiac adaptation than those athletes who train at a much lower intensity (27)
Sporting type	It is apparent that specific sporting disciplines create a specific stimulus that directs the degree of chamber enlargement (28, 29). Endurance athletes (cyclists, rowers, long distance runners) are likely to have a greater adaptation of all chambers than athletes who engage in sport of a combined stimulus (soccer, tennis, hockey) or strength (powerlifting, wrestling, judo) (9, 23, 30, 31, 32)

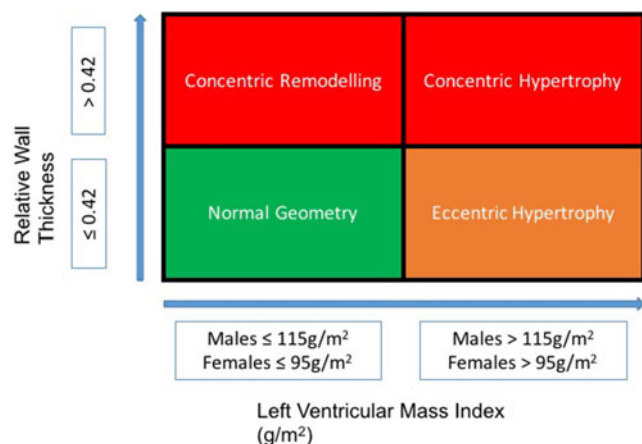
anaerobic isometric exercise at incremental workloads of >30% maximal voluntary contraction and includes sporting disciplines such as martial arts, wind-surfing and weight-lifting. Endurance activity can be defined as aerobic isotonic dynamic exercise at incremental workloads of 70–90% of maximum heart rate and includes sporting disciplines such as long and middle distance running, swimming or cycling, soccer and basketball. It is important to note that many sporting disciplines involve a combination of resistance and endurance exercise (boxing, rugby, rowing, American football) and therefore there is likely to be an overlap in ranges. The Mitchells classification provides information on specific sporting disciplines relative to the static and dynamic components and may serve as a useful guide in this regard (11).

### Amount of athletic activity should be known

When screening patients for inherited cardiac disease due to a family history, the referring physician/echocardiographer should establish the patient's level of physical activity. The total volume of training can be defined as (volume = intensity × duration) or Metabolic Equivalent Test (MET-h/week = METS × duration). An example of a select range of sporting disciplines and their specific METS is highlighted in Table 1. In summary, low-intensity exercise is defined as corresponding to 1.8–2.9 METS, moderate intensity is defined as corresponding to 3–6 METS and high-intensity exercise is defined as >6 METS.

**Table 3** Additional image acquisition.

View and modality	Explanatory note	Image
PSAX AV level (2D)	<ol style="list-style-type: none"> <li>(1) Identify coronary ostia. The left and right ostia usually originate from their respective aortic sinuses</li> <li>(2) Ensure origin is at sinus level</li> <li>(3) Identify proximal courses and exclude aberrant vessel, especially malignant course between great vessels (aorta and pulmonary artery) (33)</li> </ol>	 <p>PHILIPS MI 1.3 TIS 0.3 OX1 SS-1 40Hz 11cm 2D HGen Gn 60 C 50 3/2/0 75 mm/s P A R 1.0 3.2 49 BPM</p>
PSAX BASAL LV level (2D)	<p>LV wall thicknesses should be measured from the maximum dimension at end diastole from:</p> <ol style="list-style-type: none"> <li>(1) Anterior septum</li> <li>(2) Inferior septum</li> <li>(3) Posterior/Inferolateral wall</li> <li>(4) Lateral/Anterolateral wall</li> </ol>	 <p>1:25:04 L 1.0 cm L 1.1 cm L 1.0 cm L 1.1 cm</p>
PSAX MID LV level (2D)	<p>LV wall thicknesses should be measured from the maximum dimension at end diastole from:</p> <ol style="list-style-type: none"> <li>(1) Anterior septum</li> <li>(2) Inferior septum</li> <li>(3) Posterior/Inferolateral wall</li> <li>(4) Lateral/Anterolateral wall</li> </ol>	 <p>1:25:13 L 1.1 cm L 1.0 cm L 1.1 cm L 0.9 cm</p>
PSAX MID to apical level (2D)	<ol style="list-style-type: none"> <li>(1) Excess LV trabeculations is a common finding in athletes (34)</li> <li>(2) LV hypert trabeculation is more prevalent in black athletes</li> <li>(3) Red-flags – thinned compacted layer &lt;5mm and regional wall motion abnormality in the region of excess trabeculation. Further imaging is advised to exclude Left Ventricular Non-Compaction (LVNC) Cardiomyopathy</li> </ol>	 <p>PHILIPS MI 1.3 TIS 0.4 OX1 SS-1 40Hz 10cm 2D HGen Gn 25 C 50 3/2/0 75 mm/s P A R 1.0 3.2 55 BPM</p>



**Figure 4**  
Defining LV geometry.

## Echocardiographic examination

### Pre-echocardiographic information

The aim of the TTE is to differentiate physiological adaptation from pathological abnormality where possible. Therefore, it is important that the echocardiographer understands the main pathological conditions that may be found and must be aware of normal variation in response to exercise. The extent and nature of physiological cardiac adaptation in the AH is based on several factors and an attempt should be made to obtain information on each of these before the TTE is performed. This should include the list of information presented in Fig. 3. Although a standard minimum dataset is recommended for all echocardiograms, prior knowledge of these demographics and ECG findings will help to focus the examination, aid interpretation of findings and contribute to the subsequent management of the athlete (Table 2).

### Echocardiographic examination

The following protocols should be strictly adhered to so as to exclude pathology:

- BSE Minimum Dataset.
- BSE protocol for the assessment of LV diastolic function.
- BSE protocol on the assessment of the right heart with a focus on ARVC.
- BSE protocol for HCM.
- BSE protocol for DCM.

In addition, the following image acquisition should be made (Table 3).

LV geometry should be determined using a combination of LV mass indexed to BSA (LVMI) and relative wall thickness

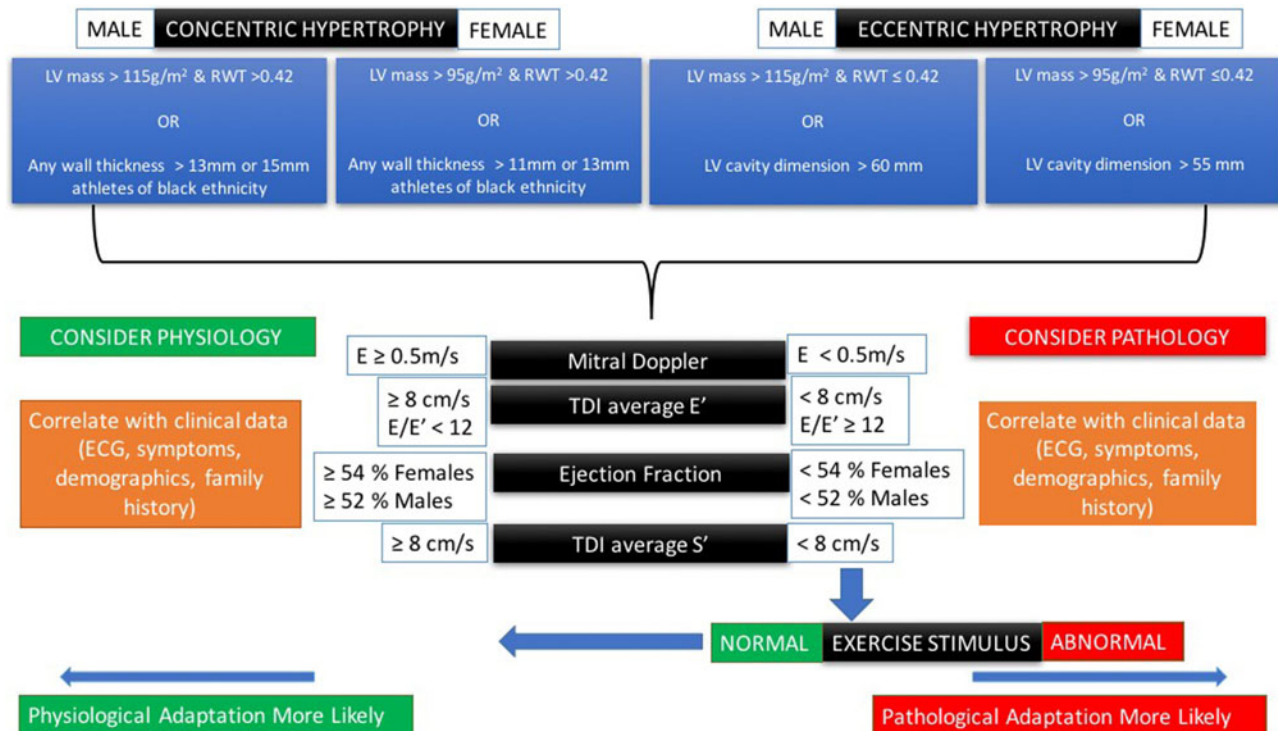
**Table 4** Upper limits for LV and RV.

Parameter	Male athlete	Female athlete
LV dimension diastole (mm)	64	57
LV interventricular septal thickness (mm)	13	11
LV posterior wall thickness (mm)	13	11
LV mass (g)	263	243
RVOT <sub>1</sub> (mm)	44	
RVOT <sub>2</sub> (mm)	41	
RVD <sub>1</sub> (mm)	49	
RVD <sub>2</sub> (mm)	44	
RVD <sub>3</sub> (mm)	92	

(RWT) (35). LVMI is calculated as per BSE guidelines (5) and RWT is calculated by summing septal and posterior wall thickness in diastole and dividing into the LV diastolic cavity dimension. LV geometry can be reported as ‘normal’ (normal RWT and normal LVMI), ‘concentric remodeling’ (increased RWT with normal LVMI), ‘concentric hypertrophy’ (increased RWT and increased LVMI) or ‘eccentric hypertrophy’ (normal RWT with increased LVMI) according to published criteria (Fig. 4).

These new guidelines raise the potential diagnostic role of exercise testing in the athlete who presents with borderline to reduced ventricular systolic and/or diastolic function. The physiological mechanisms for this are based upon the enlarged chamber requiring minimal contractility to maintain an adequate stroke volume at rest. During exercise, the AH can generate a much greater reserve than in the non-athlete and allows for the increased stroke volumes that are required for optimal cardiac output. This is demonstrated by an increase in contractility and early diastolic filling as determined by EF, TDI and Doppler (trans-mitral E wave, trans-mitral A wave, LVOT VTI, Septal S', E' and A' and Lateral S', E' and A'). A similar finding can be seen in the RV. In patients with DCM, the diseased myocardium is unable to generate sufficient reserve and contractility in response to an exercise stimulus, and contractility and filling do not improve or even deteriorate.

The type of exercise stimulus has not been clearly defined. A short isometric exercise e.g. sit ups or squats or a supine cycle ergometer causing an increase of 50% in heart rate may aid to differentiate AH from DCM. Unpublished data suggest that an increase of >11% in ejection fraction is a normal response in the athlete. There are less data available on RV response to exercise but anecdotal evidence would suggest a similar pattern to that of the LV i.e. increased RVFAC and TDI in athletes and a blunted response in those with pathological adaptation.



**Figure 5**  
Algorithm when left-sided parameters suggest abnormal geometry.

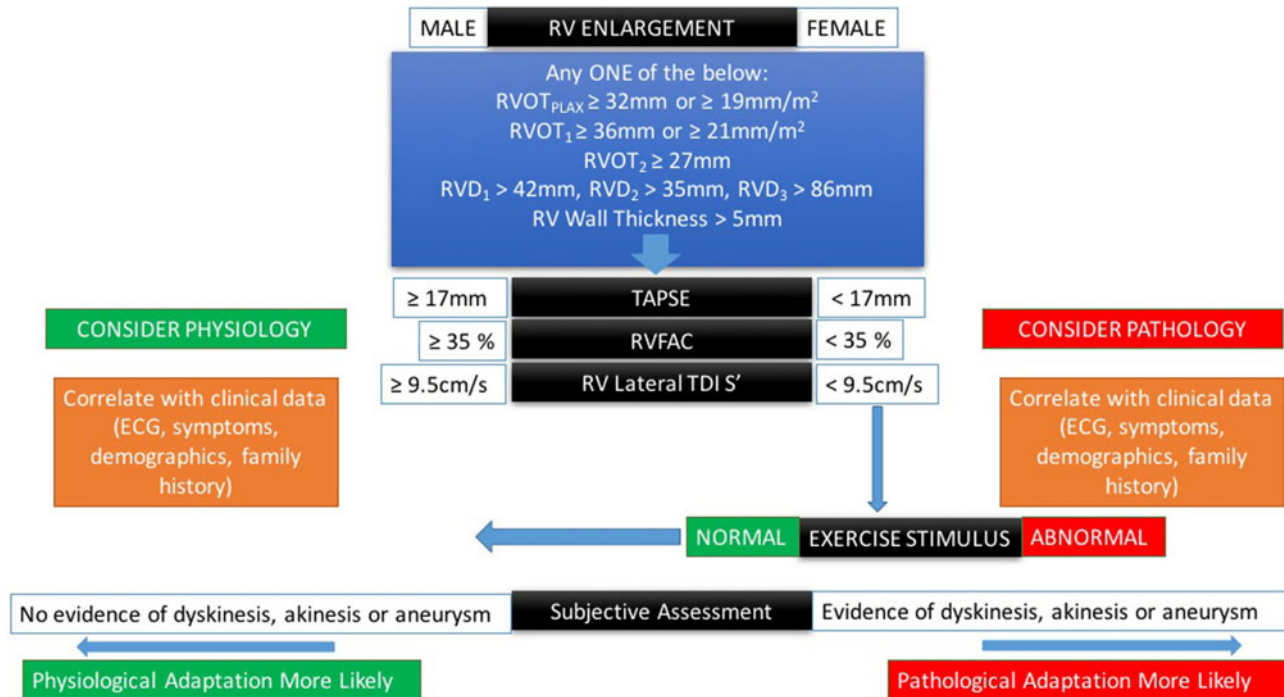
### Data interpretation

Due to the multi-factorial nature of the AH, it is difficult to provide an all-encompassing normal range. Data from key studies and meta-analyses for LV size and function in female and male athletes respectively (9, 10, 13, 36) have been included (Table 4). Table 4 also provides non-gender specific cut-offs for RV structure obtained from three large studies (19, 23, 30). It is important to acknowledge that body size directly influences cardiac size and in the extremes of body size, values above the proposed upper limits may be observed (20). In view of this, the data in both the tables and the diagnostic algorithms should only be used to guide subsequent assessment rather than providing definitive diagnostic information.

The following algorithms highlight possible interpretation of the athlete's echocardiogram in the absence of normal LV or RV geometry (Figs 5 and 6). Definitions of concentric and eccentric hypertrophy have been generated based on the use of LVMI and RWT and/or absolute non-indexed cavity size or wall thickness. The cavity cut-off for subsequent use of the algorithm has been based on non-athletes as a safety net to reduce the risk of false-negative results. In addition, a reporting template for pre-participation screening is provided in Supplementary Fig. 1

### Abbreviations

AH	Athletic heart
ARVC	Arrhythmogenic right ventricular cardiomyopathy
AV	Aortic valve
BSA	Body surface area
DCM	Dilated cardiomyopathy
EDV	End diastolic volume
EF	Ejection fraction
HCM	Hypertrophic cardiomyopathy
IVSd	Interventricular septal (thickness) in diastole
LA	Left atrium
LBBS	Left bundle branch block
LV	Left ventricle
LVd	Left ventricular diameter in diastole
LVH	Left ventricular hypertrophy
LVMI	Left ventricular mass index
LVNC	Left ventricular non-compaction
LVPWd	Left ventricular posterior wall (thickness) in diastole
MET	Metabolic equivalent test
PSAX	Parasternal short axis
RA	Right atrium
RBBS	Right bundle branch block
RV	Right ventricle
RVD	Right ventricular diameter
RVOT	Right ventricular outflow tract
RWT	Relative wall thickness
SADS	Sudden arrhythmic death syndrome
SV	Stroke volume
TDI	Tissue Doppler imaging
TTE	Transthoracic echocardiography



**Figure 6**  
Algorithm when right-sided parameters suggest abnormal geometry.

(see section on [supplementary data](#) given at the end of this article).

## Definitions

**Eccentric hypertrophy:** cardiac chamber response to increased preload manifesting as a larger cavity and a proportional increase in wall thickness to offset an elevated wall stress. The quantitative values to define eccentric hypertrophy are presented in [Fig. 4](#).

**Concentric hypertrophy/remodeling:** cardiac chamber response to increased afterload manifesting as an increase in wall thickness (of any walls) without a concomitant increase in cavity dimension. The quantitative values to define concentric hypertrophy and remodeling are also presented in [Fig. 4](#).

**Sudden arrhythmic death syndrome:** SCD with structurally normal heart on post-mortem, with no abnormality identified on macroscopic and histological evaluation and negative toxicology screening.

**Idiopathic left ventricular hypertrophy:** cardiomyocyte hypertrophy with or without fibrosis in the absence of myocyte disarray.

**Idiopathic left ventricular fibrosis:** isolated myocardial fibrosis.

## Supplementary data

This is linked to the online version of the paper at <https://doi.org/10.1530/ERP-17-0075>.

## Declaration of interest

The authors declare that there is no conflict of interest that could be perceived as prejudicing the impartiality of this guideline.

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