Table 3 Selected studies providing normative data for left ventricular end-diastolic internal dimension in competitive athletes

Author[ref.]	Imaging tech.	Athlete type	Age (y)	W/M, (*n*)	Women, mean ± SD (max.)	Men, mean ± SD (max.)
Spirito[53]	TTE	C (Italian, national)	22 ± N/A	209/738	–	53 ± 5 (66)*
Finocchiaro[54]	TTE	C (British, regional/ national)	22 ± 6	600/644	49 ± 4 (–)	54 ± 5 (–)
Pelliccia[40]	TTE	C (Italian, national)	21 ± 5	600/738	49 ± 4 (11)	54 ± 4 (66)
Weiner[21]	TTE	C (American, university)	19 ± 3	197/300	47 ± 7 (55)	54 ± 6 (62)
Pelliccia[55]	TTE	C (Italian, national)	24 ± N/A	352/957	48 (66)	55 (70)
Pluim[56]	TTE	S (meta-analysis)	18-40	–/544	–	52 ± 1 (–)
Pluim[56]	TTE	E (meta-analysis)	18-40	–/413	–	54 ± 1 (–)
Engel[57]	TTE	C (American, pro-basketball)	26 ± 4	–/526	–	57 ± 0.2 (71)
Krysztofiak[58]	TTE	C (Polish, pediatric)	12 ± 5	327/464	–	44 ± 1 (60)*
Makan[59]	TTE	C (British, pediatric)	16 ± 1	236/664	–	51 ± 4 (60)*
Weiner[37]	TTE	C (American, university, football)	19 ± 1	–/113	–	53 ± 4 (60)
Prakken[60]	CMR†	E (Dutch, national)	26 ± 6	33/46	55 ± 4 (63)	60 ± 4 (68)
Luijkx[61]	CMR‡	E (Dutch, national)	27 ± 5	24/57	55 ± 3.9 (63)	60 ± 4 (67)

C, Combined endurance and strength training physiology; *CMR*, Cardiac magnetic resonance imaging; *E*, endurance training physiology; *M*, Men; *Max.*, Maximal values recorded; *S*, strength training physiology; *TTE*, transthoracic echocardiography; *W*, women.
Data reflect measurements of the left ventricular end-diastolic major dimension (mm) obtained from a parasternal long-axis transthoracic echocardiographic view unless otherwise specified.
*Denotes cardiac data inclusive of both men and women.
†Data reflect maximum end-diastolic dimension as measured from a 4-chamber view.
‡Data reflect maximal end-diastolic dimension as measured from a short-axis view.

high-intensity and high-volume team-based training showed a phasic remodeling response with distinct acute adaptations, including increases in LV chamber size, early diastolic function, and systolic twist followed by a chronic phase of adaptation characterized by increasing wall thickness and regression in LV twist.[49] Finally, genetics appear to be a determinant of EICR. Studies examining polymorphisms within genes coding for proteins of the renin-angiotensin-aldosterone axis found that the angiotensin converting enzyme-deletion/deletion (DD) polymorphism was associated with more LV hypertrophy than the insertion/insertion (II) polymorphism during 10 weeks of exercise training in military recruits.[50] Specific polymorphisms of the angiotensinogen gene have also been associated with LV remodeling.[51] In addition, familial hypertension, a complex polygenic trait, was shown to be associated with both the magnitude and geometry of exercise-induced concentric LV remodeling in youthful normotensive endurance-trained athletes.[52] The integration of prior exercise exposure and genetics into the interpretation of clinical imaging will require further studies clarifying their impact on myocardial structure and function.

C. Left Ventricular Adaptations

Dilation of the LV is common and should be considered as a normal finding in endurance CA (Table 3). LV end-diastolic dimensions, as measured by TTE, in a large group ($n = 1,309$) of Italian elite CA representing 38 different sports varied from 38 to 66 mm in women (mean = 48 mm) and from 43 to 70 mm in men (mean = 55 mm).[55] LV end-diastolic diameter was ≥55 mm in 45% and ≥60 mm in 14% of this cohort. Thus, use of the recommended upper limits of normality of 55-58 mm[62] would render approximately 40% of male CA in this study as abnormal. In a US-based study of approximately 500 university CA, approximately 25% exceeded sex-specific recommended limits for LV end-diastolic diameter.[21] However, it is noteworthy that the majority of CA in these two studies had LV chamber dimensions within normal limits, indicating that not all CA demonstrate LV dilation. Thus, the use of "cut-off" values for LV end-diastolic diameter or volume, to either establish or exclude the presence of pathologic cardiomyopathy, is not recommended.

Mild thickening of LV walls, either with or without concomitant LV chamber dilation, may develop in CA (Table 4). Balanced LV wall thickening and chamber dilation (i.e., eccentric LV hypertrophy as defined by increased LV mass and a relative wall thickness <0.42) is common among CAs who engage in endurance sports with concomitantly highly levels of isotonic and isometric loads such as rowing and cycling (Figure 4). In contrast, mild isolated LV wall thickening (i.e., concentric LVH as defined by increased LV mass and a relative wall thickness ≥0.42) may be seen in athletes who participate in strength-based activities with no significant isotonic component, including weight lifting and American-style football (Figure 5).[33,34] Functional implications of concentric LVH stimulated by EICR, the same variant of hypertrophy that accompanies pathologic forms of ventricular pressure overload such as hypertension and aortic stenosis, remain incompletely understood. Longitudinal studies of male American-style football players showed that the development of concentric LVH was associated with relative impairments both of early diastolic relaxation velocity,[34] and systolic function,[67] raising uncertainty about the adaptive nature of this form of EICR. LV wall thickening attributable to EICR, regardless of whether it occurs in an eccentric or concentric morphology, rarely leads to measurements that exceed 12-13 mm in Caucasian CA. In 947 elite Italian CA, only a small number