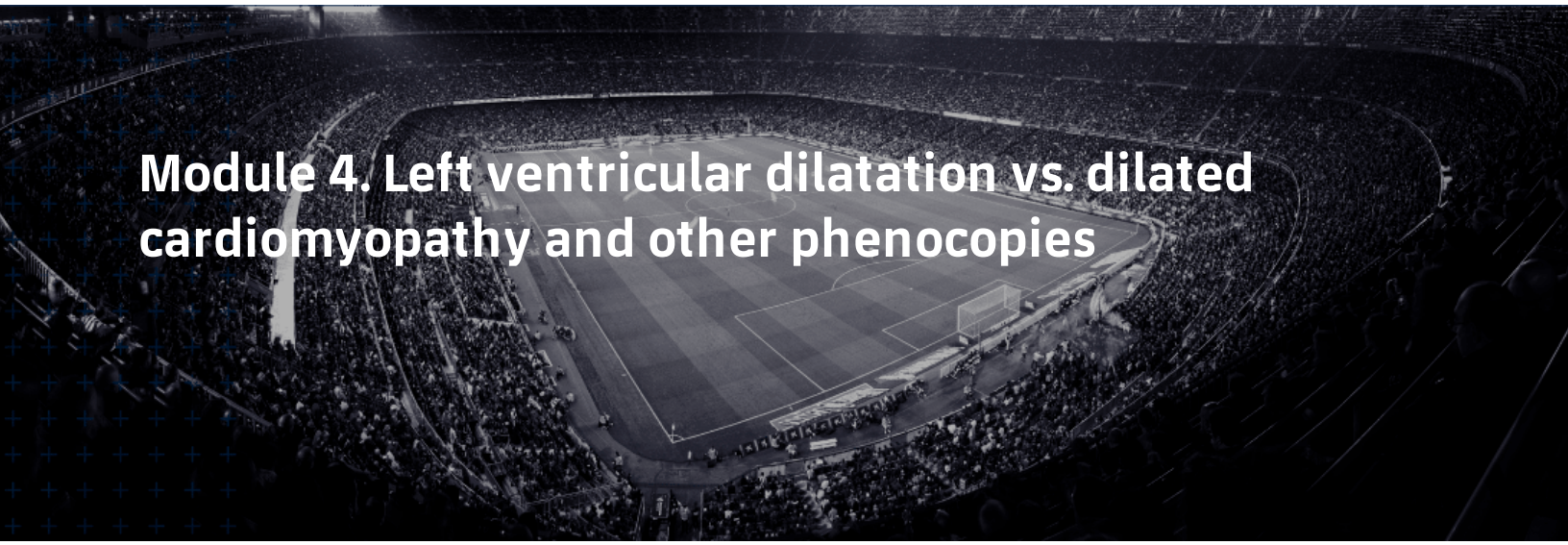

# Module 4. Left ventricular dilatation vs. dilated cardiomyopathy and other phenocopies

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# Introduction: The athlete's heart

The “athlete's heart” is a concept that encompasses the functional and morphological physiological changes that the heart undergoes in response to high-intensity training and that promotes increased performance, usually in the context of sporting competition (Prior & La Gerche, 2012). It was first described by Henschen in the late 19th century based on clinical examination and the recognition of cardiac cavities dilation and bradycardia in highly trained athletes.

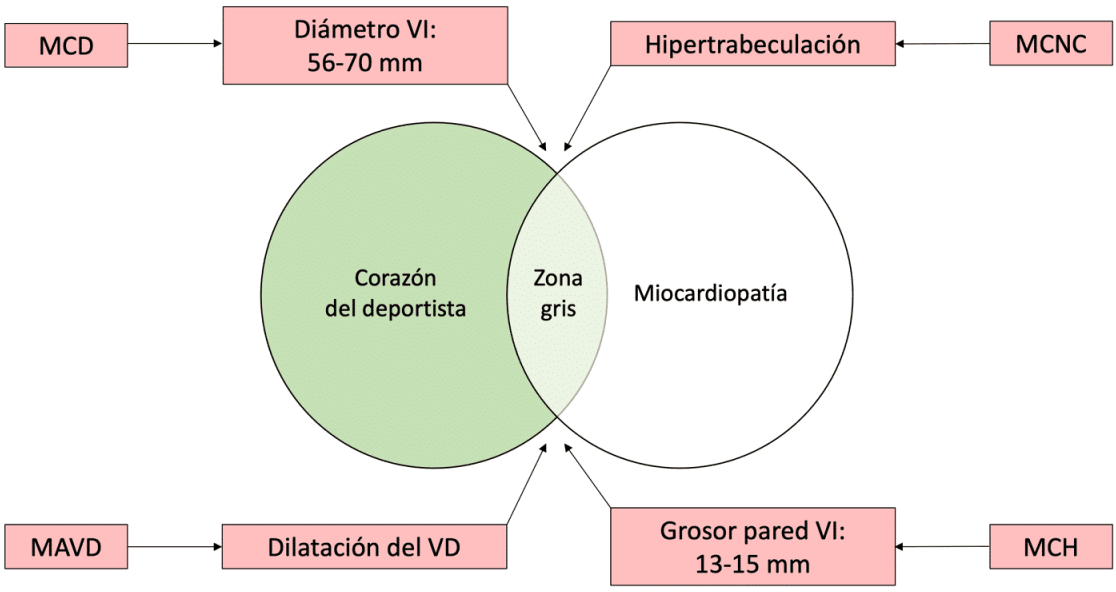
The cardiovascular response varies depending on the sport, as physiological demands differ. Most sports involve a combination of static and dynamic exercise in varying proportions, but, in general, endurance sports require greater dynamic load (isotonic) on the muscles while strength sports tend to demand greater static load (isometric) on various muscle groups (Mitchell et al., 2005). This, in turn, explains why in endurance sports, the heart is predominantly exposed to volume overload while in strength sports, it is subjected to greater pressure overload.

Over the years, it has been observed that the changes in the athlete's heart, secondary to volume or pressure overload in the cardiac chambers, lead to complex adaptive cardiac remodeling. This can result in slight dilation of the left or right cardiac cavities, a modest increase in left ventricular wall thickness and an increase in left ventricular trabeculations. Such remodeling can sometimes be difficult to differentiate in early stages from other conditions, such as dilated cardiomyopathy (DCM), arrhythmogenic right ventricular dysplasia (ARVD), hypertrophic cardiomyopathy (HCM) or non-compaction cardiomyopathy (NCCM), respectively (Abulí et al., 2020; Kübler et al., 2021; Maron & Maron, 2017) (Figure 1).

Accurate differentiation between expected adaptive remodeling secondary to sports practice and early-stage cardiomyopathy is critical in athletes, particularly elite athletes, while a false negative in diagnosing cardiomyopathy can delay diagnosis and treatment, potentially leading to disease progression. Furthermore, the management of these athletes changes drastically if a cardiomyopathy is diagnosed, as these conditions are known causes of sudden death in young people and athletes (Emery & Kovacs, 2018) and, therefore, provide sufficient grounds to recommend withdrawal from competitive sports (Maron et al., 2015). On the other hand, a false positive diagnosis of cardiomyopathy in an athlete could unnecessarily deprive them of continuing in elite sports, with potential consequences for their quality of life, mental health, and even economic situation (Maron et al., 2015).

In this chapter, we review how to establish an accurate differential diagnosis between adaptive cardiac remodeling secondary to sport, with particular focus on left ventricle (LV) dilatation and hypertrabeculation, and dilated and non-compaction cardiomyopathies.

**Figure 1. Differential diagnosis between athlete's heart and cardiomyopathy**




Source: Maron & Maron, 2017, p. 6.

The gray zone represents the overlap between the athlete's heart and cardiomyopathies.

**VI:** Spanish acronym for left ventricle (LV)

**MCD:** Spanish acronym for dilated cardiomyopathy (DCM)

**MCNC**: Spanish acronym for non-compaction cardiomyopathy (NCCM)

**MAVD**: Spanish acronym for right ventricle arrhythmogenic cardiomyopathy (RVAC)

**VD:** Spanish acronym for right ventricle (RV)

**MCH:** Spanish acronym for hypertrophic cardiomyopathy (HCM)

CONTINUE

# Unit 4.1. Left ventricle remodeling in the athlete's heart

When assessing left ventricular (LV) remodeling in the heart, it is important to consider parameters such as LV dilation and function, an increase in trabeculae and the presence and pattern of fibrosis. In addition, factors such as age, gender and training load should also be taken into account, as these variables significantly impact the cardiac remodeling of athletes. It has been shown that adaptive cardiac changes are less common in younger individuals and in women (Sanz de la Garza et al., 2017a), and the greater the training load is, the more pronounced cardiac remodeling is (Sanz de la Garza et al., 2017b).

Firstly, regarding LV dilation, in an extensive meta-analysis published in 2000 by Pluim et al. (2000) changes in LV structure were examined and it was demonstrated that endurance athletes exhibit a more pronounced increase in wall thickness, as well as an increase in LV end-diastolic diameter. Similarly, Pelliccia et al. (1999) presented a large study involving 1,309 elite athletes from various sports disciplines and found that up to 15% of male endurance athletes had elevated LV end-diastolic diameter values (→60 mm), within the range

compatible with potential dilated cardiomyopathy. No athlete had an LV end-diastolic diameter >70 mm. Subsequent studies have corroborated that exercise-induced LV remodeling in endurance athletes is characterized by LV cavity enlargement with a slight increase in wall thickness, resulting in eccentric hypertrophy (Utomi et al., 2013; Caselli et al., 2011).

Furthermore, in a recently published meta-analysis where multiple studies were reviewed, including apparently healthy competitive athletes assessed by cardiac magnetic resonance imaging (CMR) with a mean age of 31 ± 8 years, normal values for LV size and function in athletes have been proposed (indexed LV end-diastolic volume of 111 ± 4 ml/m2 and ejection fraction of 59 ± 1%). Specifically, in this meta-analysis, 27 studies involving 983 male athletes (25% endurance athletes) were evaluated, and larger ventricular volumes were found in athletes compared to the general population. Moreover, endurance athletes exhibited larger volumes and also an increase in LV mass (D'Ascenzi et al., 2019).

Secondly, regarding functional changes in the LV secondary to sports practice, most studies provide evidence that LV systolic function is preserved, with athletes generally exhibiting left ventricular ejection fraction (LVEF) values similar to those of the general population (D'Ascenzi et al., 2019; Tahir et al., 2018). An exception to this may be endurance athletes with markedly dilated LVs, who may have a lower-than-normal LVEF, suggesting that in these more dilated

hearts, less vigorous contraction is required to maintain a normal stroke volume at rest.

Thirdly, increased trabeculation in the LV has been described as a common finding, especially in athletes of African descent or Afro-Caribbean descent, as an adaptive response to high training loads. In an echocardiographic study that included 1,146 athletes from various sports disciplines and 415 healthy controls of the same age, it was reported that hypertrabeculation was present in 18.3% of the athletes (29% Black vs. 16% Caucasian), with 8.1% of them meeting the diagnostic criteria for NCCM (Gati et al., 2013). Therefore, hypertrabeculation is another important parameter to consider in the evaluation of LV remodeling in athletes.

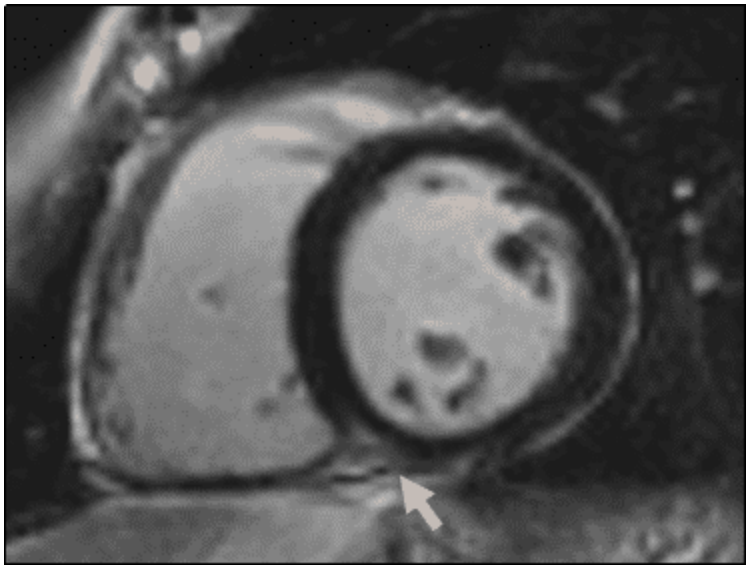
Fourth and finally, another factor to consider in the evaluation of the LV is the presence of myocardial fibrosis, which can be assessed non-invasively by CMR using T1-enhanced sequences post-gadolinium. The most typical enhancement pattern (as a surrogate marker of myocardial fibrosis) described in healthy, asymptomatic athletes is at the inferior insertion point of the interventricular septum, also known as the hinge point. In a study that included 93 young high-level endurance athletes (36 ± 6 years) and 72 controls, myocardial fibrosis was assessed by CMR, and it was found that the prevalence of enhancement at the insertion point was 37.6% in athletes and 2.8% in the general population ($p < 0.001$) (Domenech-Ximenos et al., 2020a). The presence of this enhancement pattern was not

associated with greater remodeling of either the LV or RV, and it is believed to be another benign feature of remodeling in the athlete's heart (Figure 2). However, in the literature, other enhancement patterns have been described in athletes, typically in older individuals, such as subepicardial enhancement, which is suggestive of scarring from prior myocarditis (Bohm et al., 2016), or subendocardial or transmural enhancement, which is associated with an old myocardial infarction (Breuckmann et al., 2009). These enhancement patterns, on the other hand, are known to potentially have a malignant behavior, as these scars, especially if extensive, can serve as an arrhythmic substrate during exercise (Chandra et al., 2013; Van der Schoor et al., 2016).

All these changes in the athlete's heart have traditionally been considered reversible with detraining (Pelliccia et al., 2002), but several recent publications have questioned their benign nature, as a higher incidence of ventricular arrhythmias and sudden death has been detected in high-performance athletes compared to the general population (Harmon et al., 2015; Marijon et al., 2011). One of the strategies to reduce the incidence of cardiac events in this population is the establishment of cardiovascular screening in athletes to properly evaluate the athlete's heart. In such evaluations, it is essential to always integrate all information about the athletes, including race, gender, discipline and training load, in order to establish an accurate differential diagnosis between expected cardiac

changes secondary to sports practice and potential early stages of cardiomyopathies.

**Figure 2. Cardiac MRI**



Source: own source.

**Figure 2.** Cardiac MRI of a highly trained athlete showing a small focus of fibrosis at the inferior interventricular insertion point or hinge point in the late contrast enhancement sequences (arrow).

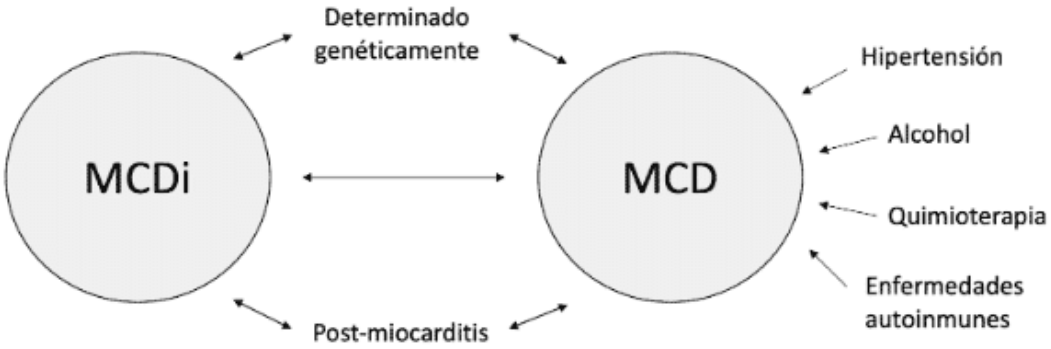
CONTINUE

# Unit 4.2. Dilated cardiomyopathy

Dilated cardiomyopathy is the most common form of cardiomyopathy and is a pathological entity characterized by LV dilatation or bi-ventricular dilatation accompanied by systolic dysfunction with reduced LVEF, in the absence of coronary artery disease, hypertension, valvular disease or congenital disease sufficient to account for the dysfunction (Elliott et al., 2008; Pinto et al., 2016). Prevalence is estimated to range from 1/2500 to 1/250 individuals (Hershberger et al., 2013).

Dilated cardiomyopathy is a complex entity that, though classically classified based on genetic causes or non-genetic causes, is now understood to involve interactions between genetic predisposition and various environmental factors (e.g. hypertension, alcohol use, infectious processes, chemotherapeutic treatments, etc.), which increase the likelihood of an individual actually developing the disease (Figure 3) (Merlo et al., 2018). Nevertheless, it is estimated that around 40% of cases of DCM are of genetic origin (with up to fifty different causative genes identified) (Hershberger et al., 2013).

**Figure 3. Characterization of the etiology of dilated cardiomyopathy**




Source: Merlo et al., 2017, p. 230.

**MCDi**: Spanish acronym for idiopathic dilated cardiomyopathy (IDCM)

**MCD**: Spanish acronym for dilated cardiomyopathy (DCM)

Difficulties in differentiating between a DCM and athlete's heart may arise, since, in early stages of DCM, both entities can share phenotypic characteristics. As explained in the previous section, some endurance athletes who are exposed to volume overload may exhibit LV dilation, which may or may not be associated with a reduced LVEF. Below, the parameters that can help differentiate between these two entities in electrocardiogram (ECG), echocardiography and CMR are outlined.

**Electrocardiogram**

The athletes' ECG can often show abnormalities related to adaptive, exercise-induced changes, such as an incomplete right bundle branch block [of the His bundle] or mild to moderate sinus bradycardia (Domenech-Ximenos et al., 2020b), and these are considered normal findings in an athlete's ECG. Patients with DCM may have a normal ECG in the early stages of the disease, and up to 30% of them may have a normal ECG even in more advanced stages.

It is important to note that a complete right bundle branch block is observed in up to 3% of athletes, and its presence or left axis deviation of the QRS, as an isolated finding, is not considered pathological and does not warrant completion of the diagnostic algorithm. In contrast, a complete left bundle branch block is very uncommon in the absence of pathology in athletes, and its identification requires ruling out an underlying structural pathology. Other findings that patients with DCM may exhibit, and which would not be expected in a healthy athlete's ECG, are the presence of low voltage QRS complexes, T-wave inversion, pathological Q waves, left atrial dilatation and premature ventricular contractions or atrial fibrillation.

**Echocardiography**

In echocardiography, LV end-diastolic diameter values greater than 60 mm associated with a reduced LVEF should raise suspicion of the

possibility that an athlete may have DCM. However, it is always important to perform a comprehensive assessment of the heart and consider that, if there is a harmonious LV dilation along with a slight RV and left atrial dilation, these findings may correspond to adaptive changes. On the other hand, if a disproportionate dilation of the LV relative to the RV is observed, it should be suspected that the findings may correspond to the early stages of DCM. Additionally, LV dilation in patients with DCM is often accompanied by mitral regurgitation (Merlo et al., 2018), which is usually absent or mild in athletes.

Another factor to take into account is the fact that some endurance athletes, who are exposed to significant volume overloads, may have LV dilatation accompanied by a slight reduction in LVEF at rest. Nevertheless, this corresponds to an adaptive response, and the reduction in LVEF in these athletes recovers and resolves during exercise. Therefore, in athletes who have LV end-diastolic diameters >60 mm whose LVEF is slightly reduced, performing an exercise echocardiography or exercise CMR to assess contractile reserve can help distinguish between physiological changes of the athlete's heart and the early stages of DCM (Galderisi et al., 2015; Millar et al., 2020). Athletes show a normal response to exercise, which includes an increase in contractility and left ventricular motility, unlike many patients with DCM, who often have little or no contractile reserve (i.e., no improvement in motility during an exercise stress test).

In echocardiography, myocardial strain can also be assessed, which involves studying the deformation of the myocardium in different spatial planes. However, in a study that included 148 competitive athletes, it was found that up to 37% of those with reduced LVEF had normal values of longitudinal myocardial strain while 58% of those with LVEF values within the normal range had reduced values of longitudinal myocardial strain (Flannery et al., 2017). In light of these results, it seems that the absolute value of strain may not be useful in distinguishing between adaptive response and early stages of pathology. However, it is important to assess the presence of pathological patterns, such as post-systolic thickening, as an indicator of pathology and to differentiate it from adaptation (Bijnens et al., 2009).

**Cardiac MRI**

Currently, CMR is the gold standard among non-invasive imaging tests for evaluating cardiac volumes and global and regional systolic function, as it provides high anatomical resolution with three-dimensional images, as well as more precise morphological and functional analyses than echocardiography, all without radiation.

On the one hand, as previously mentioned, exercise CMR can be useful for differentiating between physiological remodeling in athletes and the early stages of DCM. In this regard, Claessen et al. (2018) assessed ten highly trained healthy athletes with a slight LVEF

reduction and nine DCM patients with mild LVEF reduction using exercise CMR. The most relevant findings of the study were that patients with DCM showed little to no improvement in LVEF (5 ± 6%) while athletes with adaptive remodeling exhibited a significant improvement in LVEF (14 ± 3%). The authors of the study proposed an increase in LVEF with exercise >11.2% as a cutoff point to differentiate those individuals with physiological remodeling from those with early-stage DCM (sensitivity, 93%; specificity, 90%; AUC = 0.92, $p < 0.001$). However, it is important to note that performing exercise CMR with a bicycle suitable for use in an MRI room is available to only a few healthcare institutions worldwide, and those that do have it mainly use it for academic and research purposes.

Regarding CMR, sequences are available that allow for the non-invasive characterization of myocardial tissue, such as late contrast enhancement (LCE) sequences, which are a useful tool for detecting focal macroscopic myocardial fibrosis (Schelbert et al., 2010; Iles et al., 2015) and T1 mapping sequences, which enable the detection of interstitial or diffuse myocardial fibrosis and allow for the quantification of the myocardial extracellular space (Kammerlander et al., 2016; Messroghli et al., 2017).

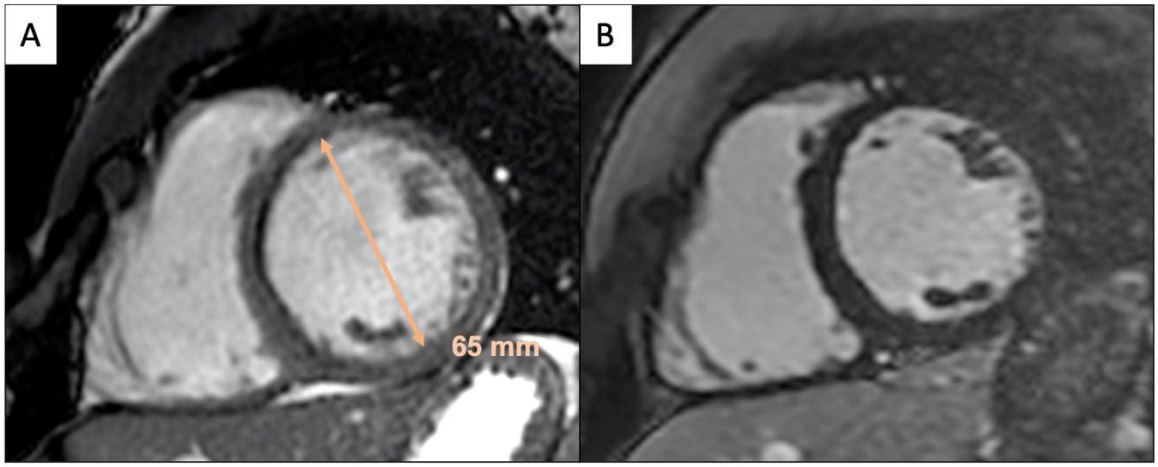
CMR is essential in the study of DCM, because, with LCE sequences, it is possible to reliably determine whether its etiology is ischemic or non-ischemic. Patients with DCM who have a subendocardial or transmural enhancement pattern following a coronary vascular

territory can be classified as having ischemic DCM (it is assumed that the LV dilation is secondary to adverse remodeling from a previous myocardial infarction). On the other hand, individuals with LCE showing a subepicardial pattern can be classified as having DCM secondary to a prior myocarditis event. However, other types of DCM secondary to other causes, or idiopathic DCM, may not show enhancement or may show mid-wall enhancement in the basal interventricular septum, which is non-specific in terms of etiology but is associated with a poor prognosis (Assomull et al., 2006). It is important to note that up to 60% of genetically tested DCM cases do not show LCE (Tayal et al., 2017), so the presence of LCE is not necessary for diagnosis. As for the possible presence of myocardial fibrosis in highly trained healthy athletes, the presence of small foci of fibrosis at the inferior interventricular insertion point has often been reported, although these have not been necessarily associated with worse remodeling (Domenech-Ximenos et al., 202a). In conclusion, the presence of LCE does not specifically help to differentiate between exercise-induced adaptive remodeling and early-stage DCM, but its distribution pattern is worth considering. For example, in individuals who engage in sports and have some LV dilation but only show LCE at the inferior interventricular insertion point, it is more likely that all changes are related to sports practice (Figure 4). Conversely, in individuals who engage in sports and have LV dilation but present with old scars from infarctions or myocarditis, especially if extensive, close periodic follow-up should be considered,

as the dilation could be secondary to adverse remodeling, indicating an early stage of DCM (Figure 5).

Finally, in patients where no LCE is observed that would allow differentiation between physiological remodeling and early stages of DCM, T1 mapping sequences can play a significant role. There is a study that demonstrated that patients with early-stage DCM had slightly higher native T1 values compared to athletes (1017 ± 42 ms vs 957 ± 32 ms, $p < 0.01$) (Mordi et al., 2016), and, while diagnosis cannot be based solely on these values, considering them along with other parameters may help guide the final diagnosis. At this point, it is worth mentioning that by taking into account native T1 values, post-contrast T1 values of the myocardium and blood, along with hematocrit levels, the myocardial extracellular volume can be estimated, that is, the space between the myocytes or myocardial cells (Flett et al., 2010). Some studies have shown that the increase in LV mass in athletes is the result of the expansion of the cellular compartment (myocyte hypertrophy), and, therefore, athletes may have lower extracellular volume values than the general population (McDiarmid et al., 2016). Thus, this is another variable to consider when evaluating these individuals, since, in DCM, low native T1 or extracellular space values would not be expected.

**Figure 4. MRI**




Source: own source.

**Figure 4.** Cardiac magnetic resonance images of a highly trained athlete showing a slight dilation of the left ventricle (end-diastolic diameter of 65 mm) in the short-axis cine-SSFP sequence (A), with normal systolic function and no pathological enhancement in the late contrast enhancement sequence (B).

**Figure 5. MRI**

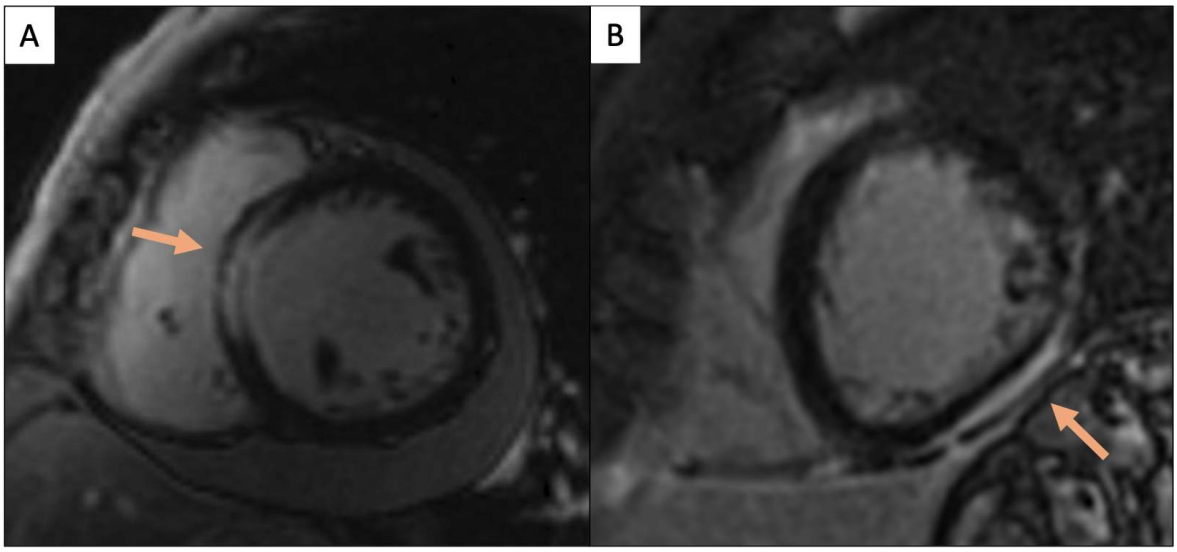



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**Figure 5.** Cardiac magnetic resonance images of two patients with dilated cardiomyopathy. (A) Patient with idiopathic dilated cardiomyopathy showing mid-wall enhancement in the basal interventricular septum (arrow), which is associated with a poor prognosis. (B) Patient with dilated cardiomyopathy showing a subepicardial fibrosis scar in the inferolateral midventricular segment of the left ventricle (arrow), related to a previous myocarditis scar.

## Genetic testing

With all the non-invasive imaging techniques discussed in the previous sections, in most cases, a correct differential diagnosis between the athlete's heart and dilated cardiomyopathy can be established. However, in some very specific cases, genetic testing may

be considered to identify possible mutations associated with dilation in cardiac cavities. The clinical scenarios in which genetic testing should be considered include athletes with a family history of dilated cardiomyopathy, especially if there are significant conduction abnormalities, disproportionate arrhythmic burden, or cases of sudden death in the family (Castelletti et al., 2022).

CONTINUE

# Unit 4.3. Non-compaction cardiomyopathy

NCCM is a rare cardiomyopathy that affects less than 0.3% of the population and is the result of a disorder in endomyocardial morphogenesis during weeks five to eight of fetal life. It is characterized by the lack of compaction of the LV myocardium, resulting in multiple trabeculae in the LV that form a layer of non-compacted myocardium (NCM) associated with a thin subepicardial layer of compacted myocardium (CM). This condition can lead to the clinical presentation of heart failure or arrhythmias. As for athletes, as mentioned in the second section, the prevalence of LV hypertrabeculation is higher than in the general population, being more common in athletes of African descent or Afro-Caribbean ancestry (Gati et al., 2013).

**Electrocardiogram/echocardiography**

The ECG in NCCM is quite non-specific and is of limited use for early diagnosis in cardiovascular screening programs for this cardiomyopathy. Nevertheless, the presence of negative T waves in

the inferolateral leads could be suggestive of NCCM (Gati et al., 2015), and this is a finding that should be considered.

There are several diagnostic criteria for NCCM; in general, all of them are based on the relationship between CM and NCM. In echocardiography, the most well-known criteria are those of Chin et al. (1990), who consider that for the diagnosis of NCCM, the ratio between CM and total wall thickness should be <0.05 in the short axis at the end of diastole, and the criteria of Jenni et al. (2001), who consider that the ratio between NCM and CM should be >2 in the short axis and at the end of systole.

**Cardiac MRI**

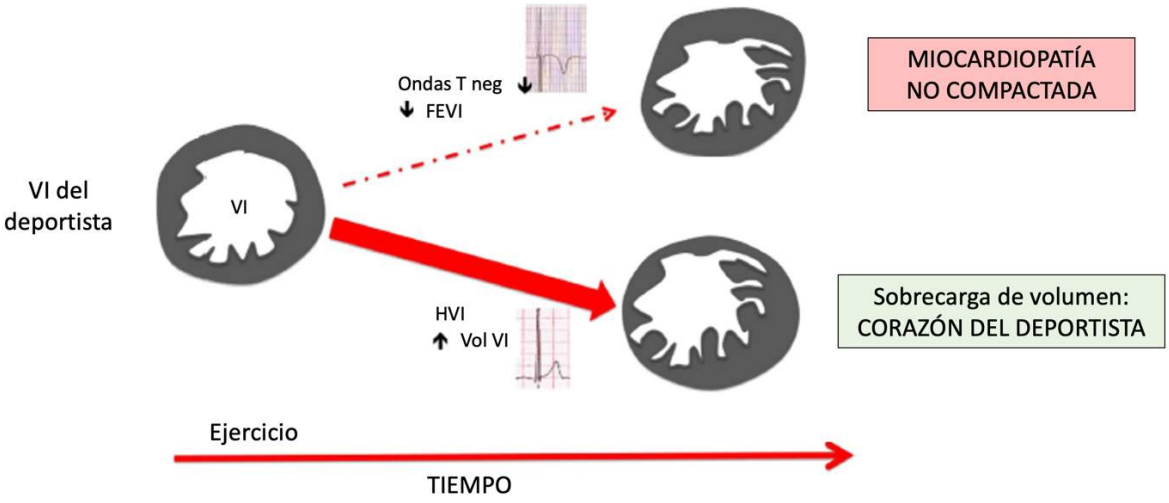
In CMR, the most commonly used criteria are those of Petersen et al. (2005), who consider that, in order to diagnose NCCM, the ratio between NCM/CM should be >2.3 in the long axis and at the end of diastole. However, it is important to note that all these diagnostic criteria are derived from cohorts of non-athletes, so caution should be exercised when applying them to athletic populations.

Furthermore, while it is known that prominent trabeculae are common in athletes, it is also known that hypertrabeculation can be seen in up to 15% of the healthy population. In prospective studies with follow-ups of up to ten years, it has been shown that LV myocardial hypertrabeculation, with normal systolic and diastolic

function and no LCE on CMR, has a benign prognosis (Andreini et al., 2016; Weir-McCall et al., 2016; Zemrak et al., 2014).

In any case, to accurately differentiate between highly trained athletes and early stages of NCCM, it is essential to consider not only the imaging diagnostic criteria mentioned earlier but also other factors such as race, LV volumes, LVEF, and the low pre-test probability of NCCM (for example, if the identification of trabeculae occurred during an echocardiogram as part of a cardiovascular screening program in a healthy, asymptomatic athlete). Although there are no specific criteria to differentiate between an athlete's physiological remodeling and a NCCM, Gati et al. (2013) suggest that if, during follow-up, an athlete with prominent LV trabeculations, these are only associated with slight hypertrophy and an increase in LV diastolic volumes, they could be considered adaptive changes secondary to volume overload from sports. On the other hand, the same authors argue that if negative T waves appear or LVEF decreases during follow-up, NCCM should be considered (Figure 6).

**Figure 6. Proposed scheme for classifying a young individual with left ventricular trabeculations**




Source: own source based on Gati et al., 2013.

| | |
|---|---|
| VI del deportista | Athlete's LV |
| VI | LV |
| Ondas T neg | Negative T waves |
| FEVI | LVEF |
| HVI | LVH |
| Vol VI | LV Vol. |

| | |
|---|---|
| MIOCARDIOPATÍA NO COMPACTADA | NON-COMPACTION CARDIOMYOPATHY |
| Sobrecarga de volumen: CORAZÓN DEL DEPORTISTA | Volume overload: ATHLETE'S HEART |
| Ejercicio | Exercise |
| TIEMPO | TIME |

**Figure 6.** Proposed scheme to classify a young individual with left ventricular trabeculations as an adaptive response to sport (athlete's heart) or as a non-compaction cardiomyopathy.

LV: Left ventricle

neg: Negative

LVEF: Left ventricular ejection fraction

LVH: Left ventricular hypertrophy

Vol: Volume

In addition, Gati and Sharma (2015) suggest several factors that may indicate pathology rather than athlete's heart. These include inferolateral T-wave inversion on the ECG, cardiac symptoms, a family history of NCCM, a first-degree relative with a similar cardiac phenotype, left bundle branch block [of the His bundle], reduced LVEF on echocardiography or stress MRI, a maximum oxygen consumption of less than 100% of the expected value in a cardiopulmonary exercise test, arrhythmias on a Holter study or the presence of myocardial fibrosis in the LCE sequence on CMR (Figure 7).

**Figure 7. Cardiac magnetic resonance images**

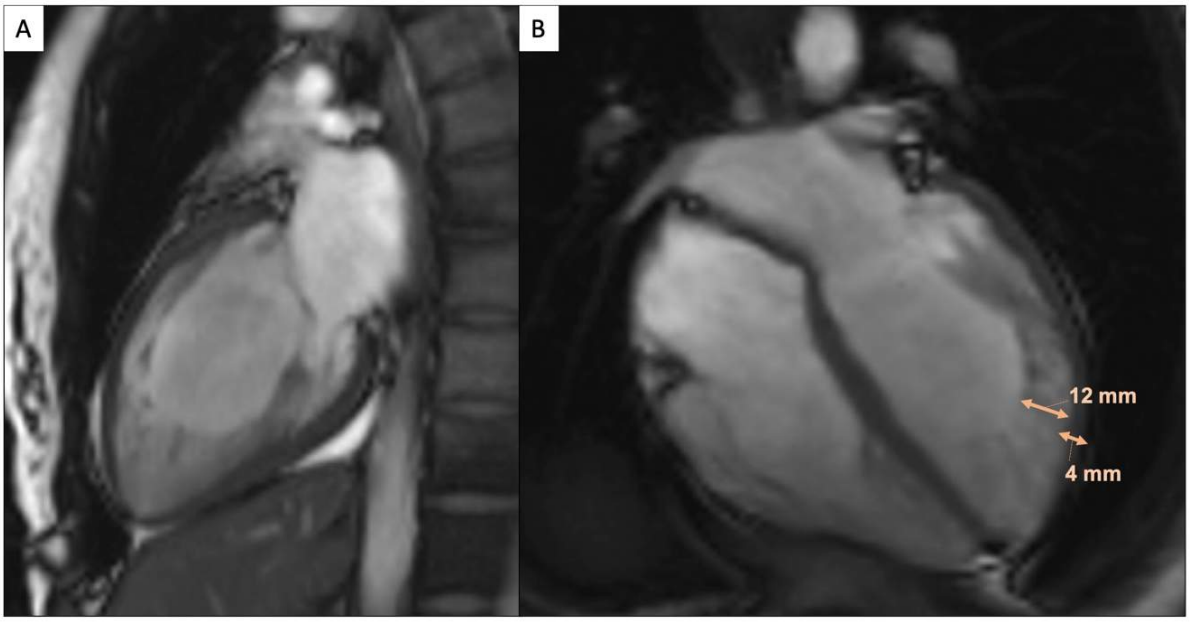



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**Figure 7.** Cardiac magnetic resonance images of a healthy athlete with marked LV hypertrabeculation at the midventricular lateral level and throughout the apical region, in long axis (A) and four-chamber (B) view, meeting NCCM criteria (NCM/CM ratio of 3).

**Genetic testing**

In the case of NCCM, genetic tests do not have specific utility for diagnosing hypertrabeculation. However, genetic testing may be considered if a patient with NCCM presents with reduced LVEF, LCE, or a family history, as some sarcomeric mutations have been associated with a worse prognosis due to the occurrence of cardiac events (heart failure or ventricular arrhythmias), and therefore may benefit from close monitoring (Casas et al., 2021).

**Table 1. Summary of the differential diagnosis between athlete's heart and dilated cardiomyopathy and non-compaction cardiomyopathy**

| | **Athlete's heart** | **DCM** | **NCCM** |
|---|---|---|---|
| | | ± | ± |

| | | | |
|---|---|---|---|
| Symptoms | No | | |
| Family history | No | Yes | Yes |
| ECG | . Normal<br>. Mild to moderate sinus bradycardia<br>. Incomplete right bundle branch block | . Complete left bundle branch block<br>. Pathological Q waves<br>. T-wave inversion | . Co<br>bundl<br>block<br>.<br>T-wav |
| Echocardiography | . Harmonic dilatation of the cavities<br>. Preserved or slightly reduced LVEF<br>. Improvement of LVEF during | .<br>Disproportionate dilatation of the LV<br>. Reduced LVEF<br>. No improvement or slight | .<br>Hyper<br>with<br>at rest |

| | | | |
|---|---|---|---|
| | exercise | improvement of LVEF during exercise | |
| CMR | . Absence of LCE or LCE at the interventricular insertion point<br><br>. Low extracellular volume values<br><br>. Improvement of LVEF during exercise | . Presence of LCE<br><br>. Increased native T1 or extracellular volume values<br><br>. No improvement or slight improvement of LVEF during exercise | . Prese |

Source: own source.

## Conclusions

Athlete's heart is characterized by adaptive cardiac remodeling secondary to sports practice which, particularly in endurance athletes, typically involves harmonious dilation of the cardiac cavities

with generally preserved LVEF and the absence of LCE or the presence of focal LCE at the inferior interventricular insertion point. In those athletes where LV dilation is more significant, with diameters that could overlap with those seen in DCM, or in those with marked LV trabeculations that could overlap with those seen in NCCM, distinguishing between athlete's heart and cardiomyopathy can be challenging, as these different entities may share phenotypic characteristics. However, it is important to consider that even if an athlete exhibits significant LV dilation or hypertrabeculation, if there are no systolic function abnormalities and no pathological fibrosis, these findings are most often adaptive and physiological cardiac changes secondary to high-intensity sports practice.

Finally, we must remember that to perform an accurate differential diagnosis, it is essential to integrate all available information about the athlete, including race, gender, the sport they practice and training load, as well as to rely on the full spectrum of data provided by multimodal imaging (echocardiography, myocardial strain, CMR, and exercise testing).

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