Helical Structure of the Ventricular Myocardium. A Narrative Review of Cardiac Mechanics.

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Abstract

Introduction. The concept of ventricular anatomy as a single muscular band was initially described by Francisco Torrent-Guasp in 1972 [1] and since then, there have been multiple novel contributions to the understanding of the morphology and function of the heart. To date, this concept has been supported and denied by multiple groups of professionals reflecting both the enthusiasm and resistance to the new concept of the single muscle band model. Currently, the single muscular band is the model that meets all the requirements to explain the performance of cardiac mechanics, and its distinctive feature to determine the characteristics of the structure.

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Key Words

Myocardial band; Echocardiography; Myocardial mechanics ; Cardiac anatomy; Ventricular function; Cardiac strain; Cardiac physiology.

Introduction.

The concept of ventricular anatomy as a single muscular band was initially described by Francisco Torrent-Guasp in 1972[1] and since then, there have been multiple novel contributions to the understanding of the morphology and function of the heart. To date, this concept has been supported and denied by multiple groups of professionals reflecting both the enthusiasm and resistance to the new concept of the single muscle band model. Currently, the single muscular band is the model that meets all the requirements to explain the performance of cardiac mechanics, and its distinctive feature to determine the characteristics of the structure.

The Helical Myocardial Band Model.

The ventricular muscle has been the object of study for years and has presented multiple difficulties in defining its definitive structure. The practically indefinite global and local structural anisotropy of its fibers and other constituents of the ventricular wall creates non-linear electrical and mechanical properties, anisotropic, time-varying, and spatially inhomogeneous. This has created great difficulties in research trying to integrate the structure and function of the heart at different scales, from the molecular level to the level of tissues and organs[2,3]. The definitive understanding of the structure of the myocardial fibers aims to translate the global behavior of the uniaxial shortening of the myocytes into the harmonious and efficient three-dimensional deformation of the ventricles of the heart, which until now can be considered as a phenomenon of a stochastic nature.

Julien Hoffman[4] cites *"A model is a representation of reality but should never be confused with that reality, models do not have to be correct to be useful, but generally, they work within certain limits. If those limits are exceeded, the model ceases to be useful"*. Given this, the helical model fully explains many of the normal and abnormal functions as discussed in the following lines.

Helical Anatomy of the Ventricular Myocardium.

The longitudinal and continuous nature of the myocardium divides it into two loops forming the base (basal loop) [BL] and the apex of the heart (apical loop) [AL]. Both loops are separated by a 180° central fold, (Figure 1) determining macroscopically identifiable helical directions (spirals within spirals), recalling the principle of self-similarity and fractal dimension described by Benoit Mandelbrot[5]

Basal loop.

It predominantly comprises the transverse muscle fibers, subdivided into two segments, right (RS) and left (LS). The RS forms the free wall of the right ventricle (RV) from the anterior to the posterior interventricular sulcus, including the infundibulum and the root of the pulmonary artery. The LS forms part of the free wall of the left ventricle (LV) and begins in continuity with the posterior interventricular sulcus, accompanied during its course from the left marginal artery to the base of the LV and the left fibrous trigone.

(Figure 1: LS-RS)

Apical Loop and Interventricular Septum

In the continuity of the RS of the BL there is a 180° turn, forming the central fold that divides both loops, being the point of change of direction of the myocardial fibers in a descending direction. The subendocardial fibers in the segment reaching the apex overlap helically to form the "vortex cordis" described by Richard lower in the 17th-century[6]. Then they change direction and the subepicardial forms the AS ending in the insertion of the left fibrous trigone at the base of the left ventricle. (Figure 1: Asterik, 5)

The previous configuration leaves two lateral segments (LS, RS) with predominantly transverse fibers covering or "embracing" the (AL) containing two muscular segments with subendocardial (DS) and subepicardial (AS) direction, interposing each other at an angle of 90°, called septal segments. The 180° central fold dividing both loops preserves the continuity of endo-epicardial directions and the longitudinal axial continuity of the adjacent segments in the unfolded band.

The two large muscle segments are distinguishable by macroscopical anatomical dissection[7], can be observed by tractography[8], defined as an interseptal hyperechoic line in echocardiography[9], and identified as the path of dissection of some hematomas, participating in the formation of septal intramyocardial dissecting agents[10].

The BL helix embracing the base of the heart, the 180° central fold, and the helical twist that superimposes AL subendocardium on subepicardial that generates the vortex cordis and the interventricular septum, form the fractal principle of self-similarity and its global three-dimensional structure a non-orientable geometric surface of triple torsion like a Moebius strip.

Myocardial Dissection and Percentage Segmental Contribution of Ventricular Mass.

The standardization and protocolization of its procedure have been widely reported previously by various authors[3,7]. However, questions cited by a current of thought that is parallel and discordant to the single muscle model of the myocardium are taken up again[11]. The "predominance principle"[12] was discussed as one where muscle fibers reveal their unique functional anatomic vector planes, rather than specific eclectic ones within the ventricular mass, demonstrating that myocardial fiber fields follow a consistent and comparable organizational pattern. within normal four-chamber mammalian hearts.

Cardiac muscle is also embedded in a specific connective tissue scaffold, so the term "myocardial fiber" refers to cardiac myocytes surrounded and interconnected by a perimysium, this being the scaffolding for the hierarchy of myocardial fibers, and the predominant local direction of its longitudinal axes defines the macroscopic architecture becoming evident on blunt dissection. In Editorial Letter[11] it is argued that Professor Streeter refers to the following as *"the wall of the heart" with a three-dimensional continuum formed basically by the one-dimensional cylindrical element, the cardiac muscle cell"* [13]. However, in another article, given the reproducibility of the dissection technique, he comments as follows *"all myocardial dissection is an artifact but said artifact is reproducible and significant when the main pathway of the fiber is the only arbiter of the dissection protocol"* [14], Torrent-Guasp never questioned the existence and importance of structural and functional interconnections at different length scales, allowing the myocardium to be considered as a "functional syncytium", identifying pattern driving its behavior, respecting the predominance and fiber direction.

Antúnez-Montes and Sosa-Olavarría quantified the mass contribution provided by each segment in which the myocardium is divided, according to the single muscle band model (unpublished data), finding that on average the RS contributes 25%, the LS 12%, the DS 37%, and the AS 26%.In this way, the BL contributes approximately 37% and the AL 63% to the ventricular economy. (Table 1) This does not differ from what was recently reported by Trainini, analyzing the contribution of different segments of the helicoidal heart in the free walls of the right and left ventricle and the septum, they have reported histologically a wide area of osteochondroid tissue in a vicinity of the right fibrous trigone which they have decided to call (cardiac fulcrum), something that Torrent-Guasp had attributed to the residual volume of blood calling this fulcrum as (Hemoskeleton), supposed to facilitate the ventricular torsion movements[15, 16, 17]. The osteochondroid structure had already been identified as similar to "bone tissue" attributing it to the nature of the fibrous trigone by Antunez-Montes in 2014, with comments in a video class found on the virtual platform https://www.youtube.com/watch?v= HZmaPA837Q8&t=727s at 10:22.

Cardiac Mechanics and Helical Structure.

The sequential and continuous contraction of the different muscle segments and, thanks to the global three-dimensional orientation as a non-orientable triple torsion geometric surface, explain the mechanical phenomena that occur during the cardiac cycle, as we will review below. Therefore, the traditional concept of

electromechanical apex-base activation[18] contradicts what has been shown by experimental studies[17,19,20] as well as data from echocardiography strain and strain rate quantify an initial movement of the base before shortening ventricular[21,22] MRI analyses also support the evidence of contradirectional base-apex rotations in the different phases of the cardiac cycle[23, 24].

Isovolumetric Contraction.

It has been documented that during this phase there is a mechanical shortening of the transverse fibers belonging to the free wall of the RV in continuity with the LS, conditioning the narrowing of the cavity. This forms an "external armor" keeping the base of the ventricles fixed, and compressing the AL (the cross-sectional diameters of both ventricles decrease) causing a brief temporary longitudinal lengthening of the LV apex during the pre-ejection interval (increase in the longitudinal diameter of the LV)[19,20,23]. The global translation of these movements of BL is the counterclockwise rotation of the entire heart as seen from the apex as reported by MRI studies[23,24]. LV elongation during the pre-ejection phase and compression secondary to BL contraction can generate a "loading" effect on the AL to produce a Starling effect through the titin mechanism for posterior ejection[26]. Until this moment, the contraction of the subendocardial DS is prepared, which is continuous to the LS but directionally opposite, which during the isovolumetric contraction phase does not produce enough force to cause the longitudinal shortening movement as it happens in the next phase (ejection), the lack of chamber shortening during the isovolumetric phase implies that the counterclockwise rotation of the heart is due to circumferential muscle shortening of the BL.

Ejection Phase.

During this phase, the circumferential fibers of the BL accompany the AL during its shortening, producing a force perpetuating the continuity of the narrowing during ejection. This will keep the base narrow, counteracting the vectorial forces generated in opposite directions by the DS and AS in movements. of torsion and untorsion of the LV[27] (Figure 3: 1A, B, C) at the moment of ejecting and sucking the blood. (Figure 2)

At this moment, the key piece is the entry into contraction of the DS, its shortening moving the base towards the apex of the LV, turning the base clockwise while the apex rotates counterclockwise due to the torque of torsion of the longest lever radius of the AS. (Figure 3: 1B) The mechanical phenomenon of shortening the LV and consequently ejecting blood is provided by the shortening of the DS, although the subepicardial AS is adjacent to it, reports of longitudinal strain at the level of the AS interventricular septum located in both segments of the AL show a positive wave deflection showing its elongation not it's shortening, the longitudinal strain of the subendocardial DS shows a negative wave deflection denoting its shortening during the ejection phase[28]. This superimposition of segments in the interventricular septum was documented as an interseptal hyperechoic line delimiting both segments[21] and is demonstrated in great detail in the anatomical correlations to the echocardiogram[7] (Figure 2 panel 4-3: 3) and tractography[8]. This difference in directions in the septum can capture opposite shortenings in the longitudinal strain. This activation and deformation of the sequential fiber also coincides with the findings of experimental studies by sonomicrometry reported by various authors[19,20,27](Figure 3) where it is evident how there is a shortening of the subendocardium (DS) moments before the ejection phase with a maximum shortening before the traditional isovolumetric relaxation phase, the maximum shortening of the subepicardial AS is recorded once the rapid filling has begun, demonstrating that there is a sequential mechanical activity which follows the segments of the helical pattern (Figure 3: 1-2) , itself the apical short-axis strain velocity vectors demonstrate a radial inward direction of the cavity throughout the endocardium formed by the DS (Figure 2: 1A-B) , in an apical window the strain velocity vectors show an inward motion of the LV cavity of right, left, and apex during early ejection, as ejection period temporally progresses downward motion persists (displacement of base toward apex) during late ejection and now shows leftward directional shift in basal portion of the septum by rotation clockwise by the start of shortening of the AS that will give way to the first diastolic phase (Figure 4: 1A, B, C).

The increased tension in the DS explains its ability to cause shortening and clockwise twisting of the base

of the heart, but the simultaneous counterclockwise apical twisting is due to AS torsion (Figure 4: 1 A-C transition of apical vectors in opposite directions) , (Figure 2 B) . Contracting together with a greater radius of curvature. This interaction is responsible for systolic torsion, and recent electrophysiological studies denote a radial transmural activation in the middle third of the interventricular septum,[17] explaining this phenomenon by coactivation of the DS and AS simultaneously (Figure 3) . The AS shortens during co-contraction to compress the cavity, but its effect by raising the base of the ventricles is counteracted by the dominance of the SD contraction, which, as we have seen, already contributes a greater percentage to the ventricular mass[17, 29].

Reinterpreting Isovolumetric Relaxation: Decompression Phase.

As a consequence of the shortening of the DS during ejection, lengthening of the AS occurs, beginning its intervention in this phase with its contraction, it shortening during the criticized phase of isovolumetric relaxation[30]. At this moment the overall result of its shortening causes a clockwise reversal after ejection since it reverses its ejection-conditioned movement which is counterclockwise, (Figure 2: 2A-B) as a result, the LV elongates from the straightening of the AS just when the DS has stopped contracting but maintains rigidity and tension[33](Figure 4: 2A-C) together with the recently reported osteochondroid tissue, the fulcrum or support point of the myocardium is obtained[17] for the AS straightening, base elevation as it continues to contract unopposed for approximately 90 ms more. The AS according to experimental studies begins to contract approximately 60 ms later than the DS, followed by a time interval of 90 ms that marks the difference between the end of the DS contraction, followed by the shortening of the AS[31, 32]. (Figure 3: Transition from color yellow to green)

With an apical 5-chamber echocardiographic window, during the closure of the aortic valve a longitudinal elongation of the ventricles is observed, as well as a subsequent widening of the cavities. This simultaneous elevation and straightening are due to the shortening of the AS, its activity as already mentioned, is delayed until the interval of the isovolumic relaxation when its current contraction is not accompanied by that of the DS, observed as a positive deflection of the longitudinal strain of the AS ([28]) and a velocity vector directed upwards (the base rises and separates from the apex.)[7] (Figure 4: 2 A-C observe the change of direction of the subepicardial vectors undertakes a direction towards the base which is manifested at the end of the isovolumetric relaxation.) also occurs the widening of both cavities without flowometric exchange related to the retreat of the circumferential muscle that has stopped contracting, particularly in the RV, different characteristics have been experimentally reported, defining a phase of longer ejection divided into early and late and the absence of a phase of isovolumic relaxation[34]. This detorsion of the LV generated by the contraction of the AS produces the elongation that creates a negative pressure and a vacuum generating the suction of blood during rapid filling[35]. (Figure 4: 2C)

Fast Filling: Suction Phase.

It begins after the isovolumetric relaxation or decompression phase when the decrease in pressure falls below the atrial pressure and is associated with a rapid additional accentuation of the twisting of the apex in a clockwise direction. (Figure 4: 3A-C) , (Figure 3: 4) At a basal level, it produces an elongation due to the movement of the AS, which simultaneously produces the final clockwise rotation of the ventricular base, in turn leading to the detorsion of the entire ventricular mass[24,30](Figure 2: 3A-B). During this phase, an active suction of blood from the atria is produced. It has been argued that the continuous unwinding process is caused by elastic recoils of compressed titin within the AS, however the active and unopposed shortening due to subepicardial contraction is responsible for the suction during the rapid filling, the straightening of the LV during the "relaxation" isovolumetric, is a very important component which, if altered, would trigger phenomena such as diastolic dysfunction[7].(Figure 4: Note the prevalence of the subepicardium over the subendocardium, 3A-C)

Divergences and Implications of Cardiac Mechanics.

Until now, the mechanical path through the myocardial fibers to perform their arduous work during the discussed cardiac cycle has been exposed. Now certain divergences and implications that these movements

have with the opening and closing phenomena of the mitral valve, isovolumetric relaxation, torsion and detorsion, longitudinal and circumferential tension, right ventricular function, and diastolic dysfunction will be discussed.

The mitral valve opening phenomenon

Since the late 1970s, irregularities have been described in the descriptions of the opening and closing times of the mitral valve by various echocardiography modalities, and findings of the start of transmitral flow by Doppler have been described before showing a loss of coaptation of the mitral valves leaflets during diastolic suction[36], an early mitral valve opening suggests a shorter duration of isovolumetric contraction[37]. More recently, MRI has documented, as previously discussed, that the start of elongation and detorsion by AS determines the opening of the mitral valve approximately 27 ms before the coaptations of the aortic valve[24], this muscular action potentially triggering diastolic dysfunction, coinciding in experimental studies with the LV negative dP/dt pressure curve due to (Figure 4: 3A) AS contraction, which is the key point to identify the unwinding and elongation process[19,20,27].

As we have seen, the BL causes clockwise recoil by rotating the apex with its subendocardial fibers and papillary muscles that connect the leaflets to the mitral annulus, (Figure 2: 4A) when it rotates counterclockwise due to DS contraction during ejection, it allows closure the mitral annulus, (Figure 2: 1A-B) (Figure 4: 3B-3C) to subsequently open again with the contraction of the AS that elongates and untwists the LV[38](Figure 2: 3A-B) (Figure 4: 2C-3A) . Therefore, the mechanical explanation of mitral valve opening with LV elongation as a consequence of AS contraction poses a mechanical explanation for understanding diastolic dysfunction.

Isovolumetric Relaxation.

It has been previously explained that during this badly named period, the whole heart does not relax but rather the AS contracts., There are records where the opening of the mitral valve begins with the unwinding and elongation of the LV (even with the aortic valve open) during this period, 50% to 60% of recoil and detorsion occur, promoting a drop in LV pressure below left atrial pressure, conditioning explosive LV suction, the interdependence of twisting and recoil is an essential interaction[39], because twisting must stop before unwinding can begin. When an increase in DS contraction torsion occurs and isovolumetric relaxation time is prolonged, as in aortic stenosis, hypertension, and ischemia, diastolic dysfunction can develop[40].

Implications of the Mechanics of Torsion and Detorsion.

There is morphological agreement that the architecture of the LV muscle fibers produces systolic twists during ejection, with differential rotation of the LV base (clockwise) and apex (counterclockwise). The torsion phenomenon refers to the change in its angle of rotation, along the longitudinal axis of the LV. The intrinsic muscular mechanisms responsible for this phenomenon have only been deduced.

The helical architecture generates LV torsion when the DS contracts turning the base clockwise and when the AS co-contracts the apex counterclockwise, causing the torsion to be generated *"between the segments of the helicoid"* [29,41]. The interactions between torsion and recoil are independent of the maximum value of torsion because prolonged torsion delays unwinding, a process necessary to develop suction, affecting ventricular performance. The torsion ends when the DS stops contracting, since the unwinding by the AS cannot start until that happens (the isovolumetric relaxation phase), this process of prolonged torsion will eventually become a precursor of diastolic dysfunction. This interdependence between torsion and recoil emphasizes why diastolic dysfunction cannot be thought to exist in a patient whose ejection fraction is considered normal and healthy.

The evidence of septal deformation confirms the displacement in different longitudinal directions and the transition of the functionally overlapping fibers that pass along its septal hyperechoic line, where the longitudinal tension of the AS during pre-ejection and ejection shows the positive deformation (elongation) continuing during recoil, as shown by sonomicrometric and magnetic resonance imaging[20,21]. During cardiac resynchronization therapies, therapeutics should approach returning to the myocardium the mechanisms of

myocardial torsion and detorsion, seeking better anatomical sites for LV stimulation, as we have discussed in this review, the movement of the heart is sequential, during an experimental study Liakopoulos etal[43] evaluated ventricular torsion after isolated ventricular or biventricular stimulation and the torsion was inconsistent, finding that the interruption of the DS and AS contraction sequence explains the mechanisms of asynchronous ventricular shortening. On the other hand, the same study group showed that when the His-Purkinje conduction system is stimulated, the torsion phenomenon is restored[44]. This can be explained because the speed of electrical propagation is 10 times faster through the natural His-Purkinje fiber conduction system (3 m/s) than through direct stimulation of the ventricular muscle (0.3 m/s)[45]therefore the helical arrangement must be taken into account.

Longitudinal and Circumferential Strain.

Concerning the shortening, and thickening in the circumferential, longitudinal, or radial dimensions, it is not addressed what it means or why this deformation occurs, which is linked to its helical morphology. Longitudinal strain reflects downward displacement of the AL fibers to generate the normal ejection fraction on average of 60%. (Figure 1: 3 DS-AS) In contrast, short-axis shortening arises from predominantly circumferential fiber deformation, but only a 30% ejection fraction occurs[46] (Figure 1: 3 RS-LS) These changes in fiber angulation have implications because systolic contractile resistance Impaired AL develops when the ventricle becomes spherical and the AL muscle fibers lose their obliquity to a more transverse orientation. Longitudinal strain quantifies ventricular shortening and reflects the action of the DS and its sequential shortening in the base-apex direction, so "pull-down" inferences contradict the helical dynamics of motion[42].(Figure 4: 1A-C, predominance of vectors towards apical direction and endocardial direction.)

The short-axis shortening of the transverse fibers of the BL and the transverse shortening produced by subendocardial DS are two factors explaining why circumferential deformation produces ventricular compression or cardiac narrowing their contributions cannot be determined by the overlap within the free wall of the LV, the interventricular septum does not contain a circumferential envelope, so its measurement of longitudinal deformation is possible, this can be measured by recording the excursion of the mitral annulus towards the apex (MAPSE). Altered longitudinal strain is an early finding in ventricular dilatation and develops when the conical ventricular shape becomes spherical, causing the natural oblique fibers to develop a transverse configuration that disrupts their torsion capacity[47]. The functional validity of this concept is confirmed by the consistent return of cardiac torsion when the spherical shape of the failing heart is restored to its natural elliptical shape, as seen after properly performed ventricular restorative surgical procedures[48, 49].

Functional Relations to the Right Ventricle.

The RV is formed in its free wall by the circumferential sheath of transverse fibers that make up the RS, however, the septum of this cavity is formed by the continuity of the AS of the AL, (Figure 1: 5) until its insertion in the fibrous trigone and the recently described cardiac fulcrum[17,50] the failure of this ventricle is related to septal lesions such as interdependence related to pulmonary hypertension, pulmonary thromboembolism, or occlusion of posterior septal branches of the right coronary artery[51,52] so it is must take into account the difference between transverse fibers that form an envelope and helical ones.

The dynamics behind TAPSE (tricuspid annular plane systolic excursion) is the longitudinal translation of the shortening that causes the contraction of the subendocardial DS, this extension and speed of base-apex shortening so that both cavities are interconnected by a muscular unit the only one that during its torsion explains TAPSE in the RV and MAPSE in the LV. In cases of increased afterload, as in pulmonary hypertension, the force to be overcome would be increasing the torsion phenomenon of the DS. Saleh et al. describe that in the RV the helical fibers of the septum determine the highest ejection fraction; in situations where there is septal damage, therefore, a deterioration in torsion, the ejection fraction is compensated by the circumferential constriction caused by the free wall of the RV with fibers of BL, recognizing how septal dysfunction can be the cause of RV failure, this phenomenon does not cause catastrophic LV dysfunction due to the presence of compensation to a greater area of subendocardial fibers, however, in the RV its free wall is predominantly transversal generating low pressure before a high resistance circuit. (Figure 4: Observe the

participation of the septal subepicardial velocity vectors, which belong to the AS and therefore to the RV septum).

Torsion-Elongation-Detorsion Interdependence as the Genesis of Diastolic Dysfunction.

During the evaluation of diastolic dysfunction, emphasis should be placed on the moment of detorsion, which occurs with the elevation of the BL, which stopped contracting during ejection; however, in the period that coincides with isovolumetric relaxation, the AS is contracting to perpetuate a turn counterclockwise against the base clockwise, likewise in experimental studies, it has been documented that at the end of the isovolumetric relaxation phase the AS has stopped to give step to its reconstitution by the release of accumulated kinetic energy during torsion, it is clear that its contribution is important to untwist, elongate and reconstitute the myocardium. Recoil does not start until torque ends. (Figure 3) The recoil process causes the predominance of ventricular filling due to the genesis of negative pressure during the so-called isovolumic relaxation phase due to contraction of the AS, and elevation of the base, the recoil ends during rapid ventricular filling and is due to the elastic recoil of the AS. An increase in torque will delay the onset of recoil and de-torque, therefore a late and short recoil will restrict suction.

The time interval between the end of DS contraction (end of ejection) and the end of AS contraction (end of isovolumetric relaxation) is the central time interval for understanding the mechanical actions responsible for diastolic dysfunction (Figure 3: yellow and green shade) . Buckberg et al. have shown in multiple studies and reviews the importance of the interdependence between DS and AS. In an experimental study, he shows[25] the correlation between the start of DS contraction, which corresponds to the QRS, and when the dP/dt curve is positive, however once the QRS has finished and when the maximum peak of the dP/dt reaches the AS starts its contraction, the ejection is finished and when the dP/dt starts its negative change the DS ends its contraction, the records show that the subepicardial AS still continues to contract, and it is at this point where the controversial isovolumetric relaxation phase begins, the AS ends its contraction at the end of the isovolumetric phase and when dP/dt returns to baseline, something very interesting to analyze is that AS contraction coincides with the T wave, because it does not reflect repolarization and another meaning must be depicted, as Manel Ballester does, attributing it to the electromagnetic field associated with the mechanical activity of the myocardium and the movement of blood, an effect called magnetohydrodynamic[53]. Considering diastolic dysfunction as impaired ventricular relaxation, the concept should be reviewed: Aortic stenosis, hypertrophic cardiomyopathy, transmural ischemia can prolong torsion compromising unwinding, aortic valve replacement in stenosis allows hypertrophic LV regression, improving torsion and recoil, post-ischemic diastolic dysfunction is reversed when sodium and hydrogen ion inhibitors limit calcium accumulation within the internal helix[54, 55].

Conclusions.

Cardiac mechanics have been scrutinized in detail, with the available knowledge of the spatial distribution of myocardial fibers to understand their functional repercussion, for this, the dissection technique, or the use of didactic models, contributes to its understanding, the helical myocardial band model is the hallmark to explaining its function. The process of isovolumetric contraction shortens the BL and forms a rigid shell that allows the first clockwise rotation and initial elongation of the LV. During ejection, torque is generated by the initial activation of the DS, and shortly after the AS, the DS ends its contraction at the end of the ejection, the isovolumetric relaxation period is a contractile phenomenon where the AS is contracting. Diastolic dysfunction implies understanding the mechanical phenomena of prolonged torsion and altered detorsion, the period between the end of the contraction of the DS and the AS are key to understanding the mechanical genesis of diastolic dysfunction. In the absence of any better and more reliable model, Francisco Torrent-Guasp's ventricular myocardial band remains the best anatomical model explaining the relationships between heart structure and function.

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Most used abbreviations

BL	Basal loop
AL	Apical loop
LS	Left segment
RS	Right segment
DS	Descending segment
AS	Ascending segment

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