International Journal of Anatomy and Applied Physiology (IJAAP)
ISSN: 2572-7451

Physiology of the Helical Heart

Research Article

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Abstract

The aim of this study was to investigate: a) beginning and ends of the myocardium; b) the slippage between the myocardial segments implies that there should be an antifriction mechanism; c) the electrical activation of the endocardial and epicardial segments; d) the mechanism of active suction during the diastolic isovolumic phase. Ten young-bovine hearts and eight human hearts were used for anatomical- histological analysis and five patients with normal QRS complexes underwent three-dimensional endoepicardial electroanatomic mapping. A nucleus (cardiac fulcrum) underlying the right trigone was found in all the hearts. Hyaluronic acid was found in the cleavage planes between the myocardial bundles. Endo-epicardial mapping demonstrated an electrical activation sequence consistent with the mechanism of ventricular twist. The finding of the fulcrum provides support to the spiral myocardial muscle. The hyaluronic acid would act as a lubricant. Electrophysiological studies explain the ventricular twist and the active suction mechanism.

Keywords: Cardiac Anatomy; Myocardium; Cardiac Fulcrum; Friction; Cardiac Electrophysiology.

Introduction

The anatomy of the heart was traditionally thought to be formed by spiraling muscle bundles, but these were never described in association with their physiology [1]. Was Torrent Guasp [2] who started the description and interpretation of the myocardial muscle, starting point to understand its motions. This was demonstrated in multiple dissections showing that the ventricular myocardium is made up of a group of muscle fibers coiled unto themselves, resembling a rope, flattened laterally, which by giving two spiral twists describes a myocardial helix that limits the two ventricles and defines their performance (Figure 1). Later, Maclvear[3]considered that the ventricular walls we are made up of an intrincate three-dimensional (3D) network of aggregated cardiomyocites.

The anatomical evolutionary state of the heart agreed with the ventricular mechanics but lacked the understanding of an electrical propagation that could accurately explain the physiology. The studies on this topic showed the integrity of an essential cardiac structure-function [4-8]. The left ventricular endocardial and epicardial electrical activation performed in patients with 3D electroanatomical mapping (TEM) allowed considering the analysis of this fundamental topic.

This pathway leading from structure to function induced gave rise to the following research studies:

1) Anatomical and histological investigation of the segmental sequence of the structure of the heart muscle.

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Received: June 22, 2021
Accepted: July 19, 2021
Published: July 26, 2021

Citation: Trainini Jorge, Beraudo Mario, Wernicke Mario, Lowenstein Jorge, Carreras-Costa Francesc, et al., Physiology of the Helical Heart. *Int J Anat Appl Physiol.* 2021;7(5):195-204. doi: http://dx.doi.org/10.19070/2572-7451-2100038

2) The myocardial muscle cannot be anatomically suspended and free in the thoracic cavity. The inevitable emerging question is that, in order for the myocardial segments surrounding the ventricles to twist, they requires a supporting point (fulcrum), similarly to a muscle in a rigid insertion. Does this exist in the heart? If this support is real, how does the myocardial muscle band insert on this structure?

3) Myocardial torsion represents the functional solution to eject the ventricular blood content with the necessary energy to supply the whole organism, overcoming the resistance of the systemic circulation. Is there a anatomical explanation supporting this fact? How is ventricular torsion produced?

4) The sliding motion between the myocardial segments during ventricular twisting-untwisting assumes that there must be an anti-friction mechanism avoiding the dissipation of the energy produced by the heart. Is there an organic lubricating element?

5) Finally, a phase of passive ventricular filling would be impossible due to the small difference between the end systolic atrial and ventricular pressures. Ventricular filling was studied as an active phenomenon with energy consumption generated by a myocardial contraction that tends to lengthen the left ventricular base-apex distance after the ejective phase producing a suction effect by an action similar to a "suction cup". Could this mechanism be explained by the persistent contraction of the ascending segment during the isovolumic diastolic phase? How is the active proto-diastolic ventricular suction produced? Is it possible to consider in the heart a coupling phase between systole and diastole where cardiac suction takes place? Which is the energy mechanism in the active suction phase?

Material And Methods

1) Cardiac dissection in ten young (two years old)-bovine hearts (800-1000 g).

2) Cardiac disection in eight human hearts: one embryo, 4 g; one 10 years old, 250 g; and six adult, mean weight 300 g.

The hearts examined correspond to material from morgue (human) and slaughterhouses (bovine). Before dissection the hearts were boiled in water with acetic acid (15 mL per liter) for approximately two hours. Prior work, before unfolding the muscle band, consisted in separating the atria from the ventricles with a very simple maneuver demonstrating the different evolutionary origins between the two types of chambers. Then, the aorta and the pulmonary artery were divided three centimeters distally to their origin, separating the attachment between them extending transversally along the anterior wall of the ventricles (Figure 2).[1] The prior boiling of the anatomical piece allowed the easy execution of all these steps. Between the atrial and the ventricular walls there was only connective tissue, allowing the easy separation of these chambers due to the denaturation produced by heat.

The key maneuver to unfold the myocardial band consisted in entering the anterior interventricular sulcus with a blunt instrument, leaving on the left side of the operator the end of the band corresponding to the pulmonary artery and its continuity with the right ventricular free wall (right segment). Next, traction was applied towards the same left side, completely releasing the pulmonary artery from the rest of the myocardial band. Below the aorta we found the cardiac fulcrum, where the origin and end of the ventricular myocardial band are attached.

As the myocardial band was unfolded, separating the pulmonary artery and the pulmo-tricuspid cord (anterior) from the ascending segment (posterior), the vision of the homogeneous anatomical reality was lost. This concurrence of the beginning and end of the ventricular myocardial band in the cardiac fulcrum constitutes a meeting point between the right segment and the ascending segment, origin and end of the ventricular myocardial band. Thus, both band ends are attach to the same point, with the origin placed anteriorly to the end of the band. Once the initial end of the band was separated from this meeting point, of great stiffness and certain resistance to the maneuver, the heart lost its functional anatomy.

The progression of the myocardial dissection implied finding the whole extent of the right segment, the beginning of the left segment and, at the posterior margin of the right ventricular chamber, the dihedral angle formed by the interventricular septum and the right ventricular free wall (right segment).

The next step (the most delicate one) consisted in entering the dihedral angle between the right ventricular and intraseptal fibers. This separation from the right ventricle allowed entering a cleavage between the anterior septal band and the intraseptal band (final segment of the myocardial muscle band), at the ventral part of the septum. Then, the dorsal part of the septum was dissected between the posterior septal band and the left descending segment to remove and separate the aorta.

Finally, the trajectories of the muscle planes belonging to the descending segment were separated in blunt fashion from those of the ascending segment leading to the cardiac fulcrum, contiguously with the aorta at the opposite end of the muscle band, to the right of the operator, allowing the band to be unfolded in all its length.

3) Histological and histochemical analysis of anatomical samples. All samples underwent histological and histochemical analysis with Alcian blue staining, a reliable marker to identify the presence of hyaluronic acid, as an antifriction mechanism and even provide a semiquantitative assessment.

4) The left ventricular endo and epicardial electrical activation sequence has been studied using TEM with a navigation system and Carto mapping, enabling three-dimensional anatomical representation, with activation maps and electrical propagation. Isochronic and activation sequence maps were performed, correlating them with surface ECG. An average of 50±8 endocardial and epicardial points were acquired for ventricular activation maps. The study included patients who had signed an informed consent previously approved by the Institutional Ethics Committee. The work described has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki).

Electroanatomic mapping was performed during the course of radiofrequency ablation for arrhythmias owing to probable abnormal occult epicardial pathways. Mapping was carried out at the

onset of studies, followed by ablation maneuvers. The presence of abnormal pathways did not interfere with mapping, as during the whole procedure baseline sinus rhythm was maintained. All patients were in sinus rhythm, with normal QRS and had no demonstrable cardiac disease by Doppler echocardiography and resting and stress gamma camera studies (Table 1).

As the descending segment is endocardial and the ascending segment is epicardial (Figure 3), two approaches were used to perform mapping. The endocardial access was achieved by conventional atrial transeptal puncture and the epicardial access by percutaneous approach in the pericardial cavity with an ablation catheter. They were then superimposed, synchronizing them with electrocardiographic timing.

The Carto system was used for 3D mapping, performing voltage, activation and propagation maps. The technique for epicardial recording was performed through the left paraxyphoid space. A decapolar catheter in the coronary sinus and a 4-polar catheter in the bundle of His were placed as fluoroscopic reference.

Results

Segmentation Of The Myocardium

Being able to unfold the myocardium with a similar thickness in all its extension proved that the helix spatial arrangement is real and not a "heuristic" or biased construction. In its course, the myocardial muscle adopted a helical configuration defining the two ventricular chambers.

The myocardial muscle describes two spiral turned with the insertion of its initial end along the line extending from the pulmonary artery to the orifice of the tricuspid valve, called the pulmo-tricuspid cord, in front of the aorta, while its final end attached below the aortic root. Both ends are fixed by an osseous, chondroid or tendinous nucleus, depending on the different species (animal or human) used in the studies. This nucleus, which we have called cardiac fulcrum, was the only perceptible edge where the ventricular muscle fibers originate and end (Figures 4 and 5). These insertions are considered the supporting point of the myocardium to fulfill its hemodynamic function. In analogy, as with skeletal muscle, we found in the myocardial muscle that its contraction takes place between a fixed point of support (insertion of the ascending segment in the fulcrum) and a more mobile one (insertion of the right segment in the anterior face of the fulcrum). This last point was shown in the dissection of a fragile character, totally opposite to the solidity of the opposite end of the myocardium in its attachment to the fulcrum. In our investigations we have not found insertion of cardiomyocytes in the collagen matrix of the trigones.

In bovines, its consistency, osseus at palpation, has been confirmed by histological studies, and its size, according to our studies, was of approximately 45 mm × 15 mm, with triangular shape. The microscopic analysis of the bovine cardiac fulcrum showed a trabecular osteochondral matrix with segmental lines[9]. Its general structure resembled the metaphyseal growth of the long bones (Figure 4). The same findings have been found in chimpanzees[10].

The analysis of a 10-year-old human heart showed in the same place a myxoid-cartilaginous formation with approximately 2 cm diameter. A similar finding, both in structure and location, occurred in the heart of a 23-week gestation human fetus (Figure 6).[9].

A fact defying logic is having found in the adult human heart a formation presenting consistent characteristics, both to observation and palpation, in the same location and with similar triangular morphology with 20 mm of length. However, the histological analysis revealed a matrix similar to that of a tendon. In principle, there is constancy in the detection, site and morphology of the fulcrum in all the hearts analyzed. This means that from a functional point of view, its presence is akin to myocardium insertion, as established in the histological analysis, becoming a solid point of interpretation to achieve its biomechanical function (Figure 6).

Histological Analysis Of The Myocardium

In the myocardial muscle we can distinguish the basal and apical loops. The basal loop extends from the base of the pulmonary artery to the central muscle twist. On the other hand, the apical loop courses from this point of inflexion to the base of the aorta. In turn, each loop consists of two segments. The basal loop consists of the right and left segments and the apical loop of the descending and ascending segments. In the general loop configuration, the basal loop envelops the apical loop, so that the right ventricular chamber presents as an open slit in the muscle mass thickness forming both ventricles. The fundamental point for cardiac mechanics is that the base and apical muscle fibers course in different directions. This disparity finds correlation with the fiber trajectories and the helical pattern of the muscle band limiting the ventricles (Figure 1).

The myocardium is a syncytial muscle with lateral bridges between its muscle fibers and a laminar histological arrangement of the muscle bundles. Due to its histological arrangement, the myocardium (syncytium) behaves like a auxetic biological material; in other words, it contracts simultaneously in two directions (longitudinal and circunferential) and thickens in a third direction (radial, towards the ventricular centroid). The histological analysis sequence of the unfolded myocardium (Figure 7) demonstrates its linear orientation according to the segmental continuity of its spatial organization when the myocardium is coiled, both in its internal and external surfaces of each segment. These orientations are identical in both surfaces (internal and external).

The lattice concept used was developed due to the band folding resulting in overlapping segments, which are functionally independent and with friction between their surfaces[11]. This arrangement is essential to achieve myocardial torsion, the fundamental action of cardiac mechanics. As the external surface of the distal descending segment (Figure 7, lower panel) twists to become the ascending segment, the cardiomyocytes generate in the planimetric histological sections a different architecture in their orientation from that of the internal surface, only site (cardiac apex) where this situations occurs. The rest of the orientation is always parallel. In the apex, the spiral course of the myocardial fibers, which shift from the periphery towards the center, determine a torsion where the subepicardial fibers become subendocardial, overlapping like the tiles of a roof, as evidenced in this image.

Table 1. Patients characteristics

Patient	Age (years)	Gender	Study indication	Other diseases
1	42	F	Isolated atrial fibrillation	No
2	19	M	Abnormal left epicardial pathway	No
3	23	M	Abnormal left epicardial pathway	No
4	29	M	Abnormal left epicardial pathway	No
5	32	M	Abnormal left epicardial pathway	No

Table 2. Activation times (ms)

Site	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	X	SD
Figure 3A	10	12	13	15	12	12.4	1.816
Figure 3B	35	38	37	41	40	38.2	2.135
Figure 4A	45	47	49	52	49	48.4	2.332
Figure 4B	55	59	57	61	58	58.0	2.000
Figure 5A	94	98	98	99	95	96.8	1.939
Figure 5B	115	118	114	120	116	116.6	2.154

Table 3. Radial propagation time (ms)

ms: milliseconds, Pat: patient, SD: Standard deviation.

Time	Pat.1	Pat.2	Pat. 3	Pat. 4	Pat. 5	Mean	SD
Radial time from descending to ascending band	25	26	24	26	28	25.8	1.483

Tabla 4. Segments

Segment	Motion
Right	Narrowing (systole)
Left	Narrowing (systole)
Descending	Shortening-twisting (systole)
Ascending	Shortening-twisting (systole)
Ascending (final part)	Lengthening-untwisting (suction phase)

Figure 1. Rope model of the ventricular myocardial band. It illustrates the different segments that form the band. In blue: Basal loop. In red: Apical loop. The insert shows the myocardial helix.

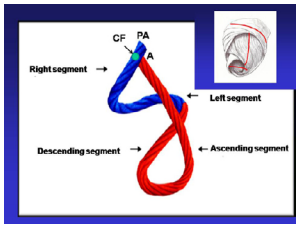


Figure 2. Unfolding of the myocardial band (bovine heart).

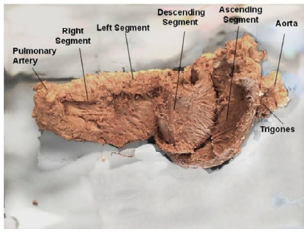


Figure 3. Spiral fibers. The ascending and descending segments can be seen in a cross section near the apex. They are correctly visualized as the fibers are spiraling along their path (bovine heart). RV: Right ventricle. LV: Left ventricle.

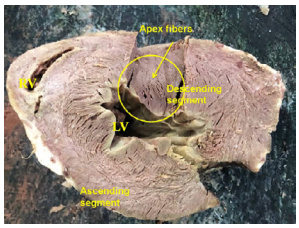


Figure 4. Cardiac fulcrum (bovine heart). A: resected piece; B: mature trabecular bone forming the cardiac fulcrum tissue. Hematoxylin-eosina stain at low magnification (10x); C: cardiac fulcrum in other view

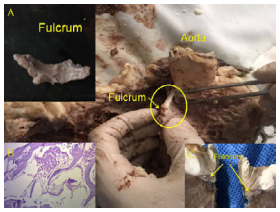


Figure 5. Cardiac fulcrum (human heart). A: 10-year-old; B: 23 week gestation human embryo heart; C: fulcrum resected from an adult human heart.

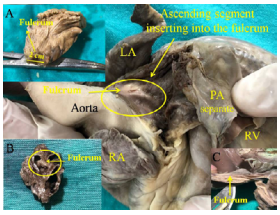


Figure 6. A: Ten year old human heart. Central area of the fulcrum formed by chondroid tissue. Hematoxylin-eosin stain (15x). B: Cardiac fulcrum in a 23-week gestation fetus showing prechondroid bluish areas in a myxoid stroma. Masson´s trichrome staining technique (15x). C: Cardiomyocytes penetrating in the the fulcrum (adult human heart). The circle details the insertion site. 1: cardiomyocytes; 2: fibrocolagenus matrix. Hematoxylin-eosin stain (15x).

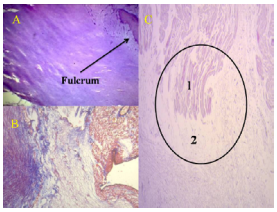


Figure 7. Segment sequence from the myocardial band histological analysis (bovine heart). The orientation of the internal (endocardic) and external (epicardic) surfaces of each segment is shown with the myocardial helix unrolled. RS: Right segment; LS: Left segment; DS: Descending segment; AS: Ascending segment.

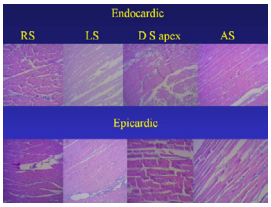


Figure 8. Longitudinal section of the left ventricle. It shows the descending segment adjacent to the ascending segment. The circle indicates the end of the ascending segment, which runs alone to attach to the cardiac fulcrum. This area is activated in the cardiac suction phase.The histology shows the different orientation of the longitudinal fibers of the ascending segment (AS) in relation to the transverse fibers of the descending segment (DS) (bovine heart).

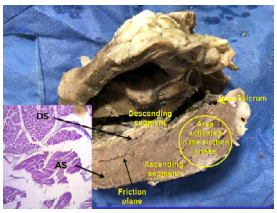


Figure 9. Interstitial space between cardiomyocytes showing hyaluronic acid (HA), stained in pale blue with Alcian blue stain (15x) (human adult heart).

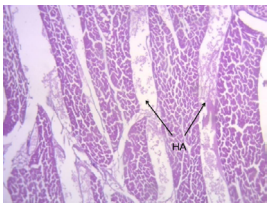


Figure 10. A: Onset of left ventricular activation. The depolarization of the interventricular septum, corresponding to the descending band is seen in the left panel. In the right panel, the ventricular epicardium (ascending band), has not been activated yet. B: Simultaneous band activation. Activation progresses in the left ventricular septum through the descending band (axial activation) and simultaneously propagates into the epicardium (radial activation) activating the ascending band.

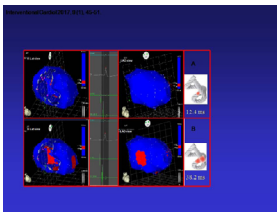


Figure 11. A: Bidirectional activation of the apex and the ascending band. The final activation of the septum is observed, progressing towards the apex, synchronously with the epicardial activation in the same direction. At the same time the epicardial activation is directed towards the base of the left ventricle. B: Progression of the activation. Activation progresses in the senses of the previous figure.

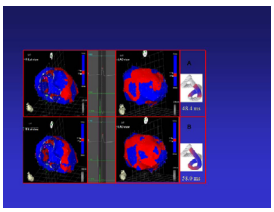


Figure 12. A: Late activation of the ascending band. At this moment, which corresponds to approximately 60% of QRS duration, the intraventricular activation (descending band) has already been completed. The distal portion of the ascending (epicardial) band is depolarized later. This phenomenon correlates with the persistence of its contraction in the initial phase of diastole. B: Final Activation In the right panel, the left anterior oblique projection was modified to a left lateral posterior projection, evidencing the very late activation of the distal portion of the ascending band.

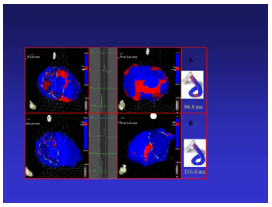
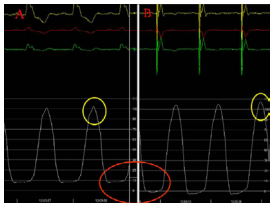


Figure 13. A: Left intraventricular pressure with the resynchronizer turned off. B: In the same patient the drop in left ventricular diastolic pressure is observed after resynchronization is restarted. Yellow circles show the increase in blood pressure with resynchronization established.



Muscle Friction

The opposing sliding motion of the left ventricular internal segments in relation to the external segments to achieve the mechanism of ventricular torsion, generates an inevitable friction between them (Figure 8).

It is natural to assume that this opposing motion of the ascending and descending segments, and also of the latter against the septal region of the myocardial band, would generate friction between their sliding surfaces in their motions of twisting (systole) and untwisting (suction).This was also observed in the modeling achieved in our study. Sliding between the internal and external myocardial segments takes opposite directions during the ejective

and suction phases of the heart, generating friction. The model of the myocardial network is not compatible with the opposing motions of these segments producing ventricular torsion, as evidenced by the speckle tracking technique developed by echocardiography. Therefore, to fulfill this dynamics, it is crucial to assume that the myocardial band has a supporting point (cardiac fulcrum), as corroborated in this chapter through anatomical and histological studies.

We know that the helical structure of the ventricular muscle allows fulfilling the dynamics of ventricular twisting-untwisting. Anatomical studies, the progress in the knowledge of cardiac mechanics through the analysis of electrical impulse pathways and echocardiographic studies of ventricular rotation, confirm the structure-function relationship of the heart. Ventricular torsion represents the functional need that makes the heart fibers adopt an 8-shape configuration. This is manifested as strain in the three myocardial axes: longitudinal, circumferential and radial, depending on the helical fiber orientation. The study of this rotation is a potential marker of myocardial disease severity when its twists depart from normal values. Ventricular torsion may become a more adequate marker of the heart's condition superior to functional class and ejection fraction in the understanding of cardiac failure. The ejection fraction only considers changes in ventricular volume, being highly dependent on preload and afterload.

During systole, the three cardiac axes contract, twisting the muscle mass, which is wrung as a towel to achieve its ejective effect. To analyze this action and explain the myocardial motions, it has been essential to understand the pathway followed by the stimulus in the myocardium. In the suction phase, first 100 ms of diastole, the longitudinal axis lengthens and then with ventricular filling it expands and untwists, increasing its circumferential and radial axes. The anatomy of the ventricular muscle, ventricular torsion, friction and the intraventricular ejective blood vortex correlate in the explanation of cardiac dynamics.

In this regard, the presence of hyaluronic acid in the cleavage planes between myocardial bundles in bovine and human hearts (Figure 9), together with Thebesian and Langer venous conduits as branches for the necessary hydration of this element, could explain this lubricating effect, counteracting the abrasion between surfaces as an antifriction mechanism[12-14].

Cardiac Electric Activation

As electroanatomical mapping corresponded to the left ventricle, the activation wave previously generated in the right ventricle was not obtained. Electroanatomical mapping took an average of 20 minutes. There were no complications related to the procedure itself or any of the approaches. Figures 10 to 12 show the projection of the endocardial an epicardial electric activation. In all the Figures the right projection was observed in the left panel and the left anterior oblique projection was simultaneously observed in the right panel. The zones activated at each moment are detailed in red. On the lateral part, the activation of the descending and ascending muscle bands that make up the muscle structure of the ventricular band in the rope model is represented. In it, the area depolarized at that moment is represented in red and those that were previously activated and are in the refractory period are represented in blue. Below the rope model, the average electrical propagation time along the muscle band can be seen measured in ms at the analyzed site (Tables 2 and 3).

Activation of the left ventricle occurs 12.4 ± 1.816 ms after its onset in the interventricular septum (Figure 10 A). At that moment it also spreads to an epicardial area - ascending bands- evidencing a radial activation at a point we call "band crossover" that occurs on average 25.8 ± 1.483 ms after septal stimulation (Figure 10 B) and at 38.2 ± 2.135 ms from cardiac activation onset. Synchronously, following the anatomical arrangement of the descending band, the activation moves axially towards the ventricular apex reaching it at an average of 58 ms ± 2.0 ms (Figures 11 A and B). At the band "crossover" the activation loses its unidirectional character and becomes slightly more complex. Three simultaneous wave fronts are generated: 1) the distal activation of the descending band towards the apical loop; 2) the depolarization of the ascending band from the crossover towards the apex and 3) the activation of this band from the crossover towards the end of the muscle band in the aorta. Figures 11 B, 12 A and 12 B show the continuation and completion of this process. Intraventricular activation ends long before the termination of the QRS. The rest of the QRS corresponds to the late activation of the distal portion of the ascending band, which justifies the persistence of its contraction during the diastolic isovolumic phase, constituting the basis of the ventricular suction mechanism (Figure 12 B).

Discussion

Myocardic Architecture

Regarding the argued difficulty to dissect the myocardium, more apparent than real, we should consider that the evolutionary goal was to develop a sufficiently solid hemodynamic structure with the strength to generate the suction and pumping of the blood volume that supplied the whole organism. Thus, every attempt to dissect an anatomical segment from the rest of the myocardium, avoiding the real cardiac arrangement, always turned into an obstacle due to the structural plan of the axes where the orientation of the myocardial band courses.

The myocardial fibers forming the myocardium cannot be considered as absolutely independent entities within a defined space. Despite the intricacy of fiber bundles with polygonal shape, which in addition receive and give off collateral fibers, a predominant course of central fibers is defined with sliding planes, which together form the myocardial muscle. It should be recalled that the myocardium constitutes a spiraling continuum in its fibers responding to the helical pattern in its muscle bundles. This arrangement indicates the need of generating a mechanical work dissipating little amount of energy. Therefore, the fiber layers very gradually shift their orientation, with more or less acute angles, to avoid that abrupt changes in the spatial organization dissipate the necessary work for cardiac function. The fan of fibers that is formed reduces the stress among them.

This situation generates a tangle of fibers that allows the band to behave as a continuous transmission chain with the epicardial fibers taking an oblique direction, the intermediate fibers a transverse course and the endocardial fibers also an oblique direction, but contrary to that of the epicardial plane. The endocardial and epicardial plane access angle is approximately 60 degrees in relation to the transverse fibers. Fiber orientation defines function and thus the ejection fraction is 60% when the normal helical

fibers contract and falls to nearly 30% if only the transverse fibers shorten. This occurs when the left ventricle dilates in cardiac remodeling and the fibers miss their oblique orientation, loosing muscular and mechanical efficiency.

It should therefore be acknowledged that a gradual change in orientation is generated from the superficial to the deep fibers that form the different segments of the muscle band. In the progression from the ventricular base to the apex, the number of horizontal fibers decreases in relation to the oblique fibers, showing that the heart is organized as a continuous muscle helix. The ventricular mechanical activity must be heterogeneous during diastole with subendocardial-subepicardial relaxation gradients. Echocardiography evidence in the apex greater curvature radius or torque. In the middle region, the descending and ascending segments become balanced, while the base of the heart shows the continuity of the right and left segments.

During systole, the muscle layers of the myocardium evidence pronounced and opposite torsion in the subendocardium in relation to the subepicardium, whereas in the apex the subepicardial fiber rotation acquires more relevance. The model is more consistent with the helix ventricular muscle concept, visualized through a longitudinal axis as long as this term takes into account interconnections between the tracts, beyond the works that detail the myocardium as a mesh based on the existence of crossed fibers. The concept of the helix adequately interprets cardiac the movements, moving away from a purely morphological definition.

The myocardial muscle cannot be anatomically suspended and free in the thoracic cavity because it would be impossible to eject blood with a speed of 200 cm/s. Therefore, there must be a point of attachment, which was identified as the cardiac fulcrum (supporting point of leverage). In this supporting site, the muscle fibers are inevitably forced to "intertwine" with the connective, chondroid or osseous fulcrum, and our anatomical and histological investigations have shown that this insertion attaches both the origin and end of the myocardium. This finding of the cardiac fulcrum in our research responds to the words of MacIver (3) by denying the existence of the muscle band: "None of the histological studies of the myocardium that we are aware, in contrast, have provided any evidence for an origen and insertion as described for the alleged unique myocardial band".

Muscle Friction

This functional association between the Thebesian and Langer venous conduits and the substantial amount of hyaluronic acid found in our research in bovine and human hearts, added to the knowledge of its lubricating role in the rest of the organism, could be crucial to understand the cardiac dynamics. In this way, ventricular torsion is correlated with a mechanism that facilitates the myocardial segment sliding to reduce the loss of energy. These venous conduits and the helical contraction would therefore continuously drive the plasmatic fluid with hyaluronic acid through a rich capillary network. As confirmation of our hypothesis, between the cardiomyocytes, we have found spaces with a capillary network and plasmatic fluid rich in hyaluronic acid.

Stimulus Propagation And Left Ventricular Twisting

The hypothesis of a continuous ventricular myocardial band in cardiac mechanics implies a series of associated muscular movements. These occur in the band forming four phases: narrowing, shortening-twisting, lengthening-untwisting and left ventricular expansion phases during the cardiac cycle allowing it to perform its functions of systole, suction and diastole. The ventricle expels its contents through torsion and not the approximation of its walls. The fundamental motions of which the different segments are shown in Table 4.

According to Torrent Guasp, longitudinal diffusion of stimuli along the ventricular myocardial band explained the performance of the heart. However, this sequential "peristaltic" activation does not correlate with some currently well-known fundamental phenomena, as clockwise and counterclockwise twisting at the left ventricular apex and base, which are mainly responsible for its mechanical efficiency[15].

In the narrowing phase there is a consecutive contraction of the right (free wall of the right ventricle) and left (edge of the mitral valve orifice) segments which represent the basal loop. According to Armour and Randall [16], this contraction constitutes an outer shell within which the apical loop is to contract. In this shell the stimulation goes from the subepicardium to the subendocardium. Then it runs through the descending segment and, according to our studies, the ascending segment is stimulated at an average of 25.8 ms. This probably happens at the level where the subendocardial fibers of the descending segment, in the anterior surface of the left ventricle, pass through the mesocardium crossing obliquely with those of the ascending segment, thus establishing a radial stimulation. Finally, the stimulation ends in the ascending epicardial segment to achieve the active isovolumic phase that generates a suction mechanism ("suction cup" action). The echocardiographic work of Mora Llabata et al. [17] finds a difference in the development of systolic strain of 88±7.1 ms between the systolic and postsystolic phases, a value that is coincident with duration values of ascending segment activation in the suction phase found in our studies.

As noted, left ventricular activation begins in the endocardial descending segment, which is almost simultaneously depolarized axially and radially. At the crossover point of descending and ascending segments, the activation spreads from the endocardium to the epicardium through radial propagation, progressing from the descending to the ascending segments[1].

From this point onwards, the ascending band depolarizes in two directions: towards the apex and towards the base, at the same time that the descending band completes its activation towards the apex.

Thus, two essential phenomena occur:

1. As the apical loop depolarizes from the segments crossover in two simultaneous wave fronts (from the descending and from the ascending segments) it generates their synchronized contraction.

2. The activation of the ascending segment propagates from the muscle crossover in two opposing directions: towards the apex and towards the base. The resulting mechanical contraction will also have a divergent direction, giving origin to the apical and basal clockwise and counterclockwise rotations, respectively.

According to Lewis [18] stimuli were transmitted from the endocardium to the epicardium through the muscle walls. Contrary to this concept, Robb and Robb [19] published in 1936 that stimuli propagation occurred longitudinally, and in 1942 inquired "How is it possible that impulse transmission occurs from the endocardial to the epicardial surface… given that the ventricular wall is composed of well differentiated bundles, separated by sheaths of connective tissue?"[20]. Surprisingly, according to their experimental studies, Armour and Randall [16] concluded that stimuli propagation in the left ventricular anterior wall was generated from the endocardium to the epicardium. This local event going on in the left ventricular anterior wall contrasts with previous concepts and with the remaining muscle mass where the electrical activity of subepicardial muscle bundles takes place before those of the subendocardium. However, this discrepancy of the impulse transmission theory through the ventricular myocardial muscle was not resolved until our research clarified its understanding, with patent relevant considerations for cardiac mechanics.

The findings of our research modify these concepts, since the stimulus propagation is simultaneously axial and radial. The ventricular narrowing phase (isovolumic systole) at the beginning of systole is shaped by the contraction of the basal loop right and left segments. The overlapping shortening phase due to the descent of the base while twisting occurs, is produced longitudinally, as the ring contracts before the apex. The fact that the apex remains fixed is due to the movement of the base, descending in systole and ascending in diastole. This is better explained because the ascending band, rigid at the beginning of diastole, acts as a tight tutor keeping the apex immobile. The pressure generated to eject the highest amount of blood at the onset of ejection during an interval lasting 20% of the systolic phase is feasible due to the twisting motion. This action is achieved because the electrical stimulation propagates towards the descending segment (axial propagation) and simultaneously to the ascending segment (radial propagation). Although the electrical conduction progresses along the myocardial muscle, radial propagation towards the ascending band plays an essential role in ventricular twisting by allowing opposing forces on its longitudinal axis to generate the necessary intraventricular pressure to achieve abrupt blood ejection. Thus, a twisting mechanism similar to "wringing a wet towel" would be produced. This concept had already been issued by Richard Lower in 1669, being the same in mice and humans[21].

This bidirectional activation does explain the generation of a force capable of ejecting the ventricular blood content at a speed of 200 cm/s at low energy expenditure. This is understood by the simultaneous axial and radial activation found in our studies.
Active suction in the diastolic isovolumic phase.

We have found that the endocardium is completely depolarized during the first part of the QRS. If according to our studies the depolarization of the ascending segment starts 25.8 ms on average after that of the descending segment and its contraction persists for the same period of time, the condition of ventricular contraction will last approximately 400 ms. On the other hand, as ventricular systole lasts about 300 ms, the remaining 100 ms correspond to the diastolic isovolumic phase (erroneously called isovolumic relaxation, because as we proved there is an active ventricular contraction). Briefly, during the initial part of this phase the ascending segment remains contracted as a result of the depolarization that occurred during the QRS.

The final part of the QRS corresponds in our investigation to the activation of the ascending segment. In this way, during the diastolic isovolumic phase, the contraction necessary to generate suction occurs. With the onset of untwisting during the diastolic isovolumic phase the ascending segment progressively lengthens, generating negative intraventricular pressure with this segment still contracted (active process) as an energy residue of the twisting process (Figure 13) [22-25] .

The suction phase of the heart is not feasible due to the small difference between the left atrial and ventricular pressures. Neither can it be passive. The untwisting of the heart in the first 100 ms of diastole (isovolumic diastolic phase) generates the negative intraventricular force to draw blood into the left ventricle, even in the absence of the right ventricle, as shown in experimental animals[26]. This suction phase is active with energy expenditure, and implies that the heart cycle consists of three stages: systole, suction and diastole. Left ventricular energetic suction is the nexus of continuity between the pulmonary and systemic circulations.

Potential Limitations

This research was conducted in a relative small number of hearts and patients, and therefore is advisable to replicate our findings in a greater number of hearts and patients.

Conclusions

1.There is sufficient evidence that the orientation of the fibers and the opposited base –apex rotacional movement of the heart, justifies the myocardic architecture.
2.The finding of the cardiac fulcrum gives support to the spiral ventricular muscle being the point of fixation that allows the helicoidal torsion.
3 .This structural composition corresponds with the electrical activation of the myocardium. The stimulus runs by its muscle pathways, but in order to fulfill the function proposed by its helical arrangement, it is essential for it to simultaneously activate the left ventricular descending and ascending segments. The transmission of the stimulus between them generates the necessary ventricular torsion (a situation similar to "wringing a wet towel") that enables the ejection of the blood content in a limited time span with the necessary force to adequately supply the whole body.
4 .The hyaluronic acid would act as a lubricant.
5. This study explains the ventricular twist and the active suction mechanism during the isovolumic diastolic and early ventricular filling phases, in contrast with the traditional concept of passive relaxation during the diastolic isovolumic phase.

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