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Research Article

Therapeutic Procedures in The Helical Heart

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Abstract

This research reveals coherence between the structure and organizational function of the heart. The description of the continuous myocardium, from its support (cardiac fulcrum) to the intraventricular vortex, explains its high mechanical efficiency and also the therapeutic procedures that have been used in practice according to the progress of the research, which was both experimental and clinical in nature. In this respect, given the anatomy and physiology of the helical heart, it is now essential to incorporate clinical and surgical therapeutic procedures that benefit this true structure of the "center of the pulse" in its pathologies. A misunderstanding of the physiological biological mechanisms, divorced from the morpho functional structure of the helical heart, can wreak havoc on the understanding of cardiac morbidities as well as their treatment.

Keywords: Helical Heart, Cardiac Fulcrum, Therapeutics

Introduction

We believe that to introduce the therapeutic procedures offered by the helical heart, a brief review of our research findings is necessary [1-3]. The heart is a network composed of structures that were integrated as evolution necessitated this development to adapt to different needs. From the circulatory tube of annelids to the four-chambered heart of mammals, constituents were added to the organization to construct a functional pattern that fulfilled a circulation adapted to aerial life. Individually, these components had distinct structures and functions, but they became complementary in the cardiac organizational function as a system. Only in the physiological pattern is the meaning of each structure found. First of all, it is necessary to understand that the helical spatial arrangement of the four cardiac chambers requires a nomenclature that corresponds to their three-dimensional reality, which is why the names right atrium, antero-right ventricle, postero-left ventricle and posterior atrium are proposed (Figure 1).

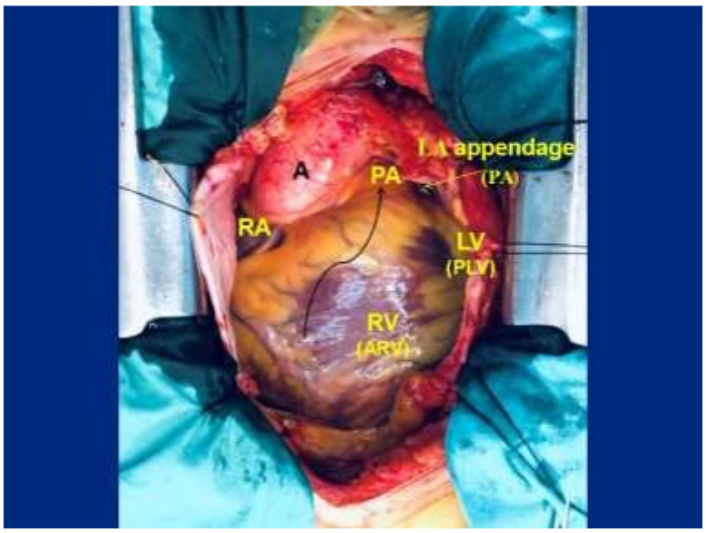

Figure 1: Photograph of a human heart "in situ" in a patient prior to cardiac surgery. The proposed nomenclature is shown in parentheses. References. A: aorta; PA: pulmonary artery, RA: right atrium; LA (PA): left atrium (posterior atrium); LV (PLV): left ventricle (postero-left ventricle); RV (ARV): right ventricle (antero-right ventricle). The black arrow indicates the direction of counterclockwise helical torsion of the venous circuit around the aorta.

The anatomical and physiological study conducted in this research begins with the description of the continuous myocardium. When it folds and assumes a functional helical shape, it corresponds to the mechanical torsion evidenced by the different segments that comprise its structure (Figure 2). The sequence in the histological analysis of the unfolded myocardium demonstrates a linear orientation according to the continuity of the segments, which, when folded, determine its three-dimensional spatial configuration. In the natural state of the folded myocardium, the segments are superimposed in the left ventricle and the septum. This folding demonstrates the different orientation of the fibers in each segment, which allows for their functional anisotropy, a determining factor in myocardial torsion. The crescent-shaped free wall of the right ventricle, formed by a single segment, is constituted by the right segment of the continuous myocardium. Hence, the thickness of the left ventricle is twice that of the right ventricle, as it is composed of overlapping segments. No segment of the sequential histology of the continuous myocardium, explored in our research, presented a mesh-like arrangement. The anatomical and histological studies of the myocardium in its helical arrangement were corroborated through mechanical investigation using echocardiography, magnetic resonance imaging (diffusion tensor imaging), and cardiac modeling studies.

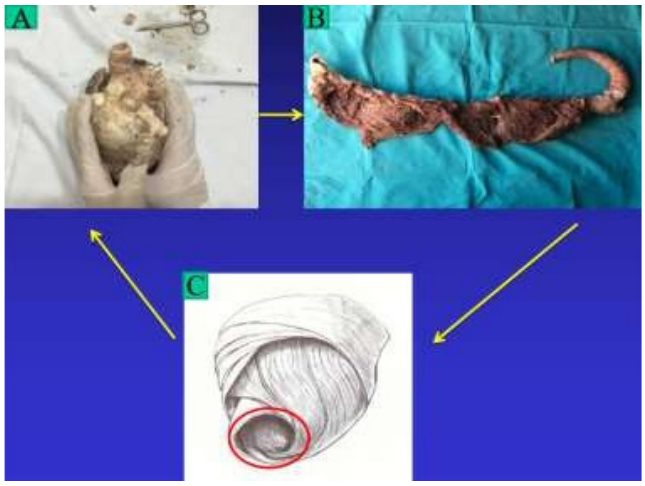

Figure 2: A: Folded heart. B: Cardiac unfolding. C: The figure shows the helical arrangement of the myocardium. The red ring details the apex, clearly demonstrating the change in direction of the fibers, constituting a conclusive sign of the helical nature of the heart.

The helical arrangement is achieved through a situation not classically studied. The three twists that occur in the continuous myocardium constitute a hallmark of the helical heart, since in the classic quasi-spherical system of a cavity surrounded by homogeneous muscle, these twists would lack the necessary function for the heart's torsion/detorsion. This arrangement allows the ventricles to be aligned adjacently by forming the septum. This is achieved by directing the descending segment (continuity of the left segment) parallel and contiguous with the ascending segment, which originates from the continuation of the descending segment as it changes direction at the apex, orienting itself towards the base of the heart. In this way, the myocardium, by achieving contiguity between the descending and ascending segments, determines with the anisotropic properties, understood as the different orientation of its fibers, that the stimulation, when passing between these segments, can produce a helical movement with opposing forces, which leads to myocardial torsion-detorsion (Figure 3).

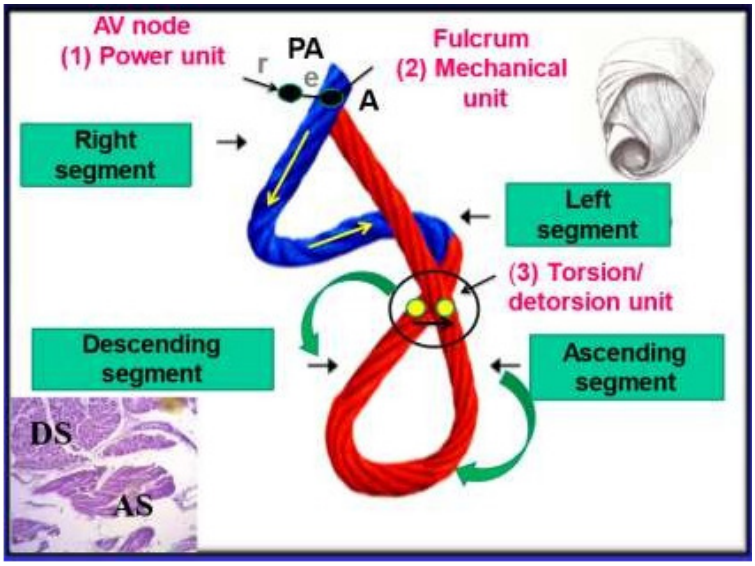

Figure 3: Helical myocardium in the cord model that simplifies the spatial structure. It shows the different segments that comprise it. In blue: basal loop. In red: apical loop. PA: pulmonary artery; A: Aorta; r: stimulus reception; e: stimulus emission. The three anatomical and functional units that allow integration between the AV node, the cardiac fulcrum, and myocardial torsion are detailed. The black circle details the site we have called band crossover. Given the different anisotropic orientations of the fibers, this area corresponds to the beginning of the helical opposite movement that produces myocardial torsion. The spatial arrangement of the continuous myocardium can be seen in the upper corner. Histology (left inset) details the different orientation of the longitudinal fibers (ascending segment, AS) in relation to the descending fibers (transverse fibers, DS).

The apex, the space created when the descending segment rotates toward the base of the heart and becomes the ascending segment, normally possesses the ability of annular narrowing (a sphincter mechanism) to withstand the retrograde intracavitary pressure produced by the ejection of blood. It undergoes almost no measurable displacement. It remains practically immobile throughout the cardiac cycle, exerting only a slight pressure on the rib cage. It is the base of the heart that performs the displacements, descending (systole) and ascending (suction). The helical arrangement of the myocardium is fully evident at the apex, as its direction abruptly changes from descending to ascending at this site. This forms the apical recess, which is lined internally by the endocardium and externally by the epicardium with few intervening muscle fibers, as evidenced by positive transillumination. A simple cardiac image can provide insight into cardiac efficiency, since the apex of the normal human heart has a "gothic" morphology, and the tendency toward sphericity with a "romanesque" appearance is always a pathological conformation, with high myocardial resistance but low efficiency, due to compromise of the descending subendocardial spiral longitudinal fibers.

A crucial point in the research was the discovery of a support for the myocardium, which we have termed the cardiac fulcrum. The heart cannot be anatomically suspended and free in the thoracic cavity, because it would be impossible for it to eject its contents at a speed of 200 cm/s. It undoubtedly had to have an attachment point, which, once found, we called the cardiac fulcrum (the lever's pivot point). At this pivot point, the muscle fibers insert into its structure, which has a connective, chondroid, or osseous nature, depending on the specimens analyzed. In our research, all the hearts studied (bovine, porcine, and human) using anatomical and histological techniques confirmed this fulcrum. The fibers that give rise to the myocardium, as well as those at its termination, are attached to the fulcrum, leaving the rest of the muscular structure free in the mediastinum. Furthermore, the cardiac fulcrum is

visualized in humans using ultrasound scans during pregnancy and in adults, computed tomography, and magnetic resonance imaging. This finding was subsequently corroborated by other researchers. Without this support, the heart could not expel 70% of its ventricular contents with only 12% of the sarcomere shortening.

The contraction and relaxation of myocardial muscle fibers, whose ends must rest on a fixed point to have a mechanical effect, would not be effective without the cardiac fulcrum. The fulcrum is located at the center of gravity of the lever, which ensures the functionality of the cardiac system by allowing forces to be distributed evenly. For this mechanism to be subjected to tensile forces one hundred thousand times daily, it must meet certain conditions: a) stability; b) resistance; c) heterogeneity; d) anisotropy; e) elasticity; and f) plasticity. These properties allow it to reach a certain level of stress when subjected to loads and then recover its shape when these loads are removed.

The anatomical proximity of the cardiac fulcrum to the AV node, surrounded by a rich plexus of neurofilaments, leads us to consider the anatomical structure of an electromechanical unit in which stimulation energy and muscle mechanics participate simultaneously. The effectiveness achieved with the placement of the pacemaker catheter in the vicinity of the right ventricular outflow tract validates the research findings. During the investigation, in both human and bovine hearts, histological analysis revealed that the cardiac fulcrum (the beginning and end of the continuous myocardium) is adjacent to the AV node, defining a space rich in neurofilament plexuses. A crucial finding was that the neurofilaments also occupy the fulcrum to stimulate the myocardial fibers that insert into it.

We mapped left ventricular activation on its endocavitary and epicardial surfaces. The mapping was performed simultaneously with the surface electrocardiogram. This study, the first using the Carto system in humans, provided a unified temporal reference frame, allowing us to correlate both recordings and obtain a synchronized view of the simultaneous activation observed in various electro anatomical scenarios. This research found a relationship between myocardial stimulation and its mechanical product. The mechanical consequence of the cardiac structure is the beginning of stimulation in the anatomical-functional unit between the AV node and the cardiac fulcrum, and its continuity in myocardial activation to the activation zone with a slight delay between the descending and ascending segments, which generates torsion of the left ventricular myocardium, by opposite rotation between the base and the apex with simultaneous shortening of both ventricles.

The three-dimensional anatomical composition of the heart corresponds to the direction of activation by the different segments of the continuous myocardium, in which their overlapping in the left ventricle is fundamental. The stimulus travels along its muscular pathways, but to fulfill the function dictated by the helical arrangement, it must primarily activate the descending and ascending segments of the left ventricle simultaneously and in opposite directions. The transmission of the stimulus between these segments generates the necessary ventricular torsion, which prompted the expression of similarity to "wringing out a towel," described by Giovanni Borelli and Richard Lower (17th century) to facilitate its physiological understanding. This function facilitates the expulsion of ventricular contents with the necessary force in a limited time to adequately irrigate the entire body. Ventricular torsion is almost exclusively the domain of the left ventricle in relation to its power.

The concept of anisotropic myocardial structure is a simple yet compelling argument for explaining its function. Cardiac mechanics play a key role in the physiological function and pathological dysfunction of the heart. The helical organization of myocardial fibers within the ventricular wall is responsible for torsional movement and the resulting functional efficiency of the heart, since myocardial fiber is fundamentally functional, not morphological. It depends on the orientation of the fibers in space. Thus, anisotropy should be considered a directionally dependent property. It is important for inducing torsion. For proper function, there must be cooperation between cardiac mechanics and the anisotropy of its fibers, since altering the latter will impair the ability to generate torsion/detorsion. This action is possible due to changes in the contractility of cardiomyocytes. Aging and hypertrophy lead to dysfunction. In this regard, the different orientations of endocardial and epicardial fibers, if modified, alter the functional balance. This organization is fundamental to cardiac efficiency. Myocardial wall deformation, degree of rotation, and angular velocity are sensitive indices of ventricular performance.

Following systole, the subsequent suction phase of the heart is not feasible through such a small pressure difference with the periphery. Nor can it be passive. The detorsion of the heart in the first 100 ms of diastole, a phase we have termed the Protodiastolic Phase of Myocardial Contraction (PPMC), generates the negative intraventricular force to draw blood primarily into the left ventricle, even in the absence of the right ventricle, as we have demonstrated in our research. This suction phase is active, requiring energy expenditure, and implies that the cardiac cycle consists of three phases: ejection, PPMC, and diastole. Our research supports the existence of an active suction coupling phase between systole and diastole, involving muscle contraction, energy expenditure, and a significant drop in intraventricular pressure. This effect draws blood into the left ventricular cavity through a pressure difference, relative to the periphery, generated by this active suction.

There is only one period in the circulatory cycle in which negative pressure can occur in the ventricular chambers at a specific moment. This phenomenon occurs during the PPMC, when the pressure drops to -3 mmHg, according to our measurements. Between the closure of the aortic valve and the opening of the mitral valve, and therefore between the closure of the pulmonic valve and the opening of the tricuspid valve, there is a sharp drop in intraventricular pressure with energy expenditure that can reach

negative values. It is during this phase that the muscular contraction of the terminal ascending limb at its insertion on the cardiac fulcrum-myocardial support-produces myocardial lengthening and detorsion with the ventricular chambers closed. This contraction of the septum, due to its ventricular interdependence, determines, as we will see, the PPMC in both ventricles, generating a suction process.

The opposing sliding of the inner and outer segments of the left ventricle, necessary to achieve ventricular torsion, generates unavoidable friction between them. It is reasonable to understand that this friction, from a physics perspective, also implies resistance to movement. As Newton's first law states, friction limits the continuity of movement. There would be a high expenditure of energy without the spongy system in the myocardium, which includes the Thebesian and Langer venous canaliculi, with their antifriction lubrication system. The opposing contractions of the descending and ascending loops facilitate the release of hyaluronic acid-rich plasma from the Thebesian vessels into the interstitium, ensuring continuous lubrication by compressing and dilating the vessels that traverse the myocardium. Histological studies of this spongy network and its canaliculi revealed the antifriction effect of hyaluronic acid, which flows throughout the thickness of the myocardium (Figure 4).

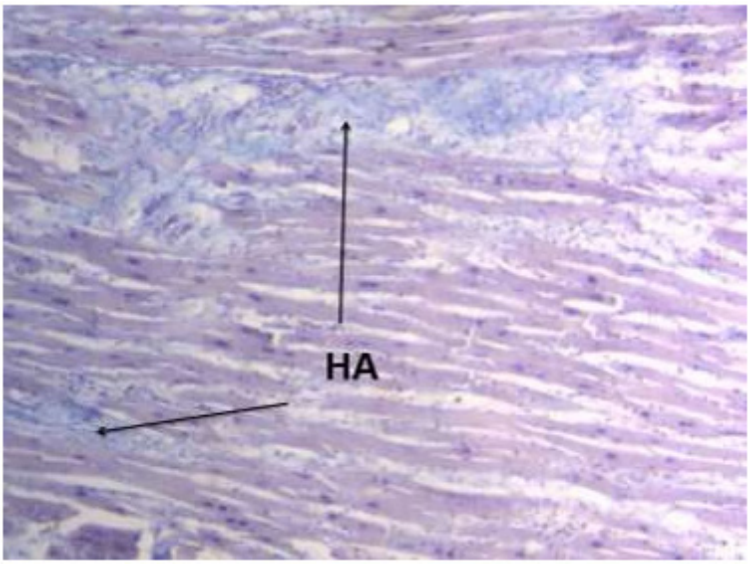

Figure 4: Interstitium between cardiomyocytes showing hyaluronic acid (HA) stained light blue with Alcian blue (15x) (adult human heart).

The continuous helical organization of the myocardium (structure) and ventricular torsion (function) lead to the intraventricular blood content becoming a small tornado, explained by the physical laws of dissipative structures. This vortex, resulting from ventricular torsion, allows blood to be ejected with the necessary force to meet the circulatory needs of the various organs. The initial asynchrony between the right and left ventricles in the opening of the semilunar valves explains the necessary functional complementarity of circulatory movement and provides the basis for the anatomical and physiological structure of the continuous helical myocardium that constitutes the heart. In this respect, the pulmonic valve opens approximately 40 ms before the aortic valve opens. The systemic and pulmonary vascular beds are connected in series to form a continuous circuit through the active, ejection, and suction phases of each ventricle, since while the former generates positive pressures, the latter tends toward negative pressures.

Since each ventricle has only one chamber to perform two active phases with opposing functions- suction and ejection- and a passive diastolic filling phase, we must analyze how, to complete their unidirectional circulatory system, they require an a synchronicity between these phases so that blood can circulate in one direction and with an effective sequence. In this respect, our research also focused on the study of anurans. Although the systemic and pulmonary circulations appear to be independent circuits, the synchronized activation of ejection and suction in both ventricles is consistent with the accepted concept of active suction, supported by recent research. In this way, the circulatory system comprises two subsystems in which the ejection and suction of the different ventricles act in alternating complementarity, with both phases actively involving energy expenditure and muscle contraction:

a) systemic subsystem: ejection by the left ventricle + suction by the right ventricle.

b) pulmonary subsystem: ejection by the right ventricle + suction by the left ventricle.

As a fundamental phylogenetic process, we have studied anurans, since the initial asynchrony in the mammalian heart between the right and left ventricles in the opening of the

pulmonary and aortic semilunar valves explains the necessary functional complementarity of circulatory movement and provides the basis for the anatomical and physiological structure of the continuous helical myocardium that constitutes the heart. In the investigation of specimens such as anurans (Rhinella arenarum) and the mammalian heart, we find incipient functional analogies such as the complementarity between the ventricles and the asynchrony within them. We might ask whether the physiological characteristic of ventricular complementarity in mammals was already functionally represented in anurans, which, with two atria (the left being smaller) and a single ventricle, make the atria act asynchronously to avoid mixing oxygenated and deoxygenated blood in the single ventricle. The single ventricle of anurans is connected to an outflow tract from which the pulmonary artery and the aorta emerge at its distal end.

Regarding cardiac mechanics, the use of equations based on the concept of energy (integrated variables) in a ventricular chamber is more evident than simply expressing the function in terms of a pressure variable. The concept of blood pressure is determined by the properties of the vascular system and the heart's capacity. While this concept is correct, the notion that stroke volume is synonymous with the heart's functional capacity is misleading. For example, the right and left ventricles eject similar volumes, but their energies differ. Hypothetically, if the aortic diameter is reduced, stroke volume decreases. This implies that the volume changes according to resistance; therefore, stroke volume is not an independent value. Stroke volume reflects the heart's capacity as a function of two independent variables: stroke energy and systemic vascular resistance.

Based on these considerations, it would be more logical to speak of cardiac stroke energy as a parameter that summarizes the heart's potential and to which non-independent variables contribute. In this model, energy expenditure extends from systole to the period known as PPMC, while energy consumption is absent only during the diastolic relaxation phase. This concept is fundamental for understanding the suction energy that occurs during PPMC. This phase can be a promising clinical indicator, as the energy suction of the left ventricle is the link between the pulmonary and systemic circulations.

Based on the results obtained in our clinical research presented in this text, it can be interpreted that dysfunction in Heart Failure with Preserved Ejection Fraction (HFpEF) is primarily due to an alteration in ventricular suction, excessively prolonged during PPMC of the left ventricle, compared to control groups. This would lead to an increase in filling pressures of the cardiac chambers with the consequent dyspneic symptoms that characterize these patients.

This research reveals coherence between the structure and organizational function of the heart. The description of the continuous myocardium, from its support (cardiac fulcrum) to the intraventricular vortex, explains its high mechanical efficiency and also the therapeutic procedures that have been used in practice according to the progress of the research, which was both experimental and clinical in nature. In this respect, given the anatomy and physiology of the helical heart, it is now necessary to incorporate clinical and surgical therapeutic procedures that benefit this true structure of the "center of the pulse" in its pathologies. A misunderstanding of the physiological biological mechanisms that are far removed from the morpho functionality of the helical heart can wreak havoc on the understanding of cardiac morbidities as well as their treatment.

Magnitude of Heart Failure

Heart failure is one of the most significant public health problems due to its incidence and its social, economic, and, above all, human impact. It currently affects 64 million people worldwide. Its prevalence in the adult population is 1.3%, with an increase of over 10% in those over 70 years of age. The mortality rate is 50% within 5 years. The prevalence in the US is 5 million patients, with 300,000 deaths per year and 500,000 new cases per year [4]. The impact on healthcare systems is 15 million outpatient visits per year, 6.5 million hospital days, and $38 billion in healthcare expenditure. In Europe, the incidence is 1.3 cases per 1,000 habitants per year in those over 25 years of age, reaching 11.6 per 1,000 habitants per year in those over 85 years of age [5], while 5% of the European population has problems related to heart failure [6,7]. It is the leading cause of cardiovascular hospitalization, with high rates of hospital readmission and high healthcare costs.

The leading cause of heart failure is ischemic heart disease, responsible for 50% of cases in the US. Within ischemic heart disease, acute myocardial infarction is the most common single cause, with a risk of heart failure ten times higher than that of the general population during the first year after the infarction and up to twenty times higher in subsequent years. Following an acute myocardial infarction, there is a loss of cardiomyocytes which, combined with ventricular remodeling, triggers heart failure. This remodeling is a complex phenomenon involving molecular, neurohormonal, and genetic processes, resulting in left ventricular dilation, morphological abnormalities, and dysfunction. Furthermore, HFpEF currently accounts for 50% of all heart failure cases.

Early revascularization of acute myocardial infarction using angioplasty and stenting has not reduced the incidence of left ventricular dysfunction and remodeling. Thus, while in older series with conventional treatment, 20% of patients with transmural infarction developed ventricular dilation and dysfunction, in recent series of infarctions treated with angioplasty and stenting during the acute phase, 30% develop alterations in shape or function within six months. The evolution in these patients, in relation to mortality and complications, is directly related to ventricular dilation [8-10].

Five percent of patients present with Stage IV heart failure, are highly symptomatic, require frequent hospitalizations, and

have a one-year survival rate of less than 30% [11]. The increase in ventricular volume and the acquisition of a spherical shape is responsible for the progression of heart failure. The classic surgical treatment for these patients is heart transplantation, with a five-year survival rate exceeding 70% and a 20-year survival rate exceeding 25% [12]. However, the mismatch between the number of recipients and donors means that less than 20% of patients with Stage IV heart failure can benefit from transplantation. Hence the need to reserve heart transplantation for those patients who have no other treatment options and, concurrently, to develop other alternatives. Recent therapeutic strategies are designed to integrate biology and new medical technologies, generating alternatives that can improve the prognosis and functional status of these patients. Treatments include ventricular restoration surgery, passive ventricular containment, permanent ventricular assist devices or those used as a bridge to recovery, immunoadsorption, and tissue engineering [13-15].

Neurohormonal models do not explain the progression of heart failure, and pharmacological treatments that act on neurohormonal activation slow, but do not stop, disease progression, or are otherwise ineffective [16,17]. Size and geometric changes are responsible for the structural abnormalities of myocytes and the extracellular matrix that worsen cardiac function, increase neurohormonal activity, and reduce cardiovascular response. Studying the anatomical basis is necessary for the application of new techniques aimed at restoration, returning the ventricle's geometry to its native volume and conical configuration. The prognosis of patients with heart failure is directly related to ventricular dilation. Myocardial capacity is conditioned by the orientation of the cardiac fibers. In this respect, the conical heart has helical and circumferential myocardial fibers. The orientation of the fibers determines function; thus, the ejection fraction is 60% when normal helical fibers contract and falls to 30% if only the transverse fibers shorten. The development of a spherical configuration modifies the orientation of the helical fibers toward transverse dilation and decreases contractile force. In this way, the development of surgical techniques for ventricular restoration has revived the work of Torrent-Guasp in his myocardial muscle band hypothesis, as well as the cardiac mechanics based on research into cardiac electrical activation that we have developed. This understanding of the helical myocardium also improves the potential for efficient pacemaker stimulation.

Clinical Procedures

When beginning an investigation, it is necessary to have a certain orientation regarding what should be analyzed. A shift in the research concept also occurs when the problem is examined from a different angle, from a new perspective on the reality of the problem. Peter Medawar stated: "Scientific research begins with a possible world or a fragment of it" [18]. Therefore, imagination is only a narrow margin in science, where there must be a confrontation between what could be and what is, between the possible and the real.

In the concept of describing the human body, there have been arbitrary and sometimes fanciful interpretations, since myths and science often intertwine. Scientific answers tend to be partial and provisional. Myths attempt to explain everything. In science, general questions tend to have limited answers, while limited questions tend to have general answers. A clear advance has been the understanding that biology needs current science to explain itself. Culture is historical and imbued with narratives that are often seized upon by those in power, thus exonerating scientific rationality. As a result, the sciences are caught between reductionist unification and self-sufficient fragmentation.

Imagination is always lurking in the construction of the possible, which is why we must accept that our approach to science can occur through unsettling and unexpected concepts. It is its way of developing. Furthermore, humans have not only a need to explain reality, but also a need to enter the realm of possibility in dreams. The conclusions of this research regarding myocardial stimulation can be summarized in the following points:

a) Three-dimensional endoepicardial mapping demonstrates an electrical activation sequence of the apical loop zone consistent with the synchronous contraction of the descending and ascending bands.

b) The simultaneous and opposing activation of the ascending band, originating from its radial activation by the descending band, is consistent with the simultaneous clockwise and counterclockwise rotation of the apical and basal zones (ventricular torsion mechanism).

c) The late activation of the ascending band, compatible with the persistence of its contraction during the initial phase of the isovolumetric diastolic period, more accurately termed, given its energy expenditure, the Protodiastolic Phase of Myocardial Contraction (PPMC), occurs without the need to postulate electrical activations following the QRS complex.

The activation sequence of the helical heart found in this study, consistent with the functional torsion/detorsion it exhibits, explains the preceding process that triggers ventricular torsion and the suction mechanism. Furthermore, it confirms that activation of the ascending band completes the QRS complex. This finding demonstrates the persistence of contraction of this muscle segment during the first part of diastole, refuting the traditional concept of passive relaxation. At the moment, it is necessary to incorporate the advances of current quantum physics into the study of living beings. This step should not be seen as esoteric or mystical, but rather as the use of information held by other sciences in order to achieve negentropy, thus advancing biological knowledge. The path of depolarization energy in our research leads to the understanding that the stimulation circuit is completed at its origin, in the electromechanical unit. We believe that these contributions, based on the electromechanical unit, design it as an attractor of the helical convolution that establishes the path of the myocardium.

The myocardium, being the only muscle that originates and terminates at the same point (the cardiac fulcrum), analogous to a Möbius strip, combined with the understanding of the QRS complex as a continuous information pattern for achieving sequential cardiac mechanics through the negentropic effect, and the evidence of the time factor as a fundamental element in a four-dimensional myocardium, are essential for understanding cardiac physiology. Without this adherence to the timing factor in each cardiac cycle, life would not be possible, this characteristic being unique in its brevity within the organs of the human body. Undoubtedly, the heart is subject to the rigor of time as a fundamental element of its continuous function, since any delay in its cycle carries the possibility of imminent death. Our work proposes the existence of a "three-phase heart": systole, suction, and diastole. We consider the data obtained to be of particular importance because they were recorded in human beings with structurally normal hearts under physiological (non-experimental) conditions. Further investigation is needed into what occurs in various pathologies.

In recent years, the importance of ventricular filling pathology and diastolic insufficiency has become evident [3,19]. In this regard, almost all studies have focused on alterations in the passive properties of the myocardium. It is possible, however, that these pathologies are due to dysfunctions of ventricular contraction during the PPMC. Similarly, systolic alterations could, in some cases, be due to modifications in the activation of the apical loop. These phenomena have very significant clinical and therapeutic consequences, making it feasible to develop classifications of heart failure based on the suction phase, as well as pharmacological, surgical, or device-based therapies that take into account the regulation of the persistence of contraction during PPMC of the ascending limb, the timing and/or synchronization of the contraction of both limbs, etc. In fact, the pathophysiological basis of cardiac resynchronization therapy could correspond to this phenomenon.

We have also highlighted the importance of the apex in resolving ventricular reduction techniques, as well as anatomical speculations regarding the great band and the different functionalities of the basal and apical loops, the former involved in the ejection phase and the latter also in the suction phase. The path remains open, especially given the understanding that a dilated heart does not have an adequate suction phase and, therefore, an efficient subsequent contraction. This active mechanism of the myocardial band on the protodiastolic effect opens up a broad panorama for surgical techniques to restore both the shape and volume and consequent function of the left ventricle [20]. One clinical aspect, derived from the anatomical and physiological studies we have conducted and presented in publications, shows promising clinical prospects for its resolution. This is heart failure with preserved ejection fraction, which we will discuss in the following section.

Contributions of the Helical Heart Organization to Heart Failure with Preserved Ejection Fraction

According to what has been developed, the suction produced by negative pressure in PPMC cannot be explained by a passive mechanism, given the low gradients reached at the entrance to the atria, and should be considered the fundamental element facilitating venous return in complementarity with the systolic impulse of the opposite ventricle. Since an alteration in the ventricular suction mechanism could be an initial, even subclinical, stage of ventricular dysfunction, the objective of this analysis has been to identify whether there is a relationship between the parameters that determine the impairment of the PPMC phase of the left ventricle and Heart Failure with Preserved Ejection Fraction (HFpEF). This should be classified as having an ejection fraction greater than 50%, the mechanisms underlying the onset and development of this heart failure being not well understood [8,17,21-23]. In this regard, the National Heart, Lung, and Blood Institute has stated that HFpEF is the greatest unmet need in the cardiovascular setting [22,24-30].

Is poor LV suction the cause of this characteristic of heart failure?

Material and Methods

A retrospective study was conducted on echocardiographic studies performed in the last six months. The study population consisted of three groups:

a) Group I: Ten (10) young patients (5 male, 5 female) without heart disease, with mean age 30.3 ± 9.2 years and body surface area of 1.81 ± 0.16 m^2.

b) Group II: Ten (10) adult patients (5 male, 5 female) without heart disease, with mean age 66.2 ± 4.1 years and body surface area of 1.73 ± 0.16 m^2

c) Group III: Ten (10) patients (6 female, 4 male) with HFpEF, mean age 81.1 ± 11.3 years and body surface area of 1.76 ± 0.20 m^2.

In this study, patients provided their informed consent. The research was previously approved by the Ethics Committee. All patients were in sinus rhythm with no abnormalities in the electrocardiogram. The variables analyzed were: Cardiac cycle (ms); Left ventricular systole (ms); Left ventricular PPMC (ms) (LVPPMC); Left ventricular diastole (ms); Relative wall thickness (RWT) (%); Left ventricular mass (LVM) (g/m2); E/E´ ratio; Left ventricular ejection fraction (%); Left atrial volume (LAV) (ml/m2); Pulmonary artery pressure (mmHg); and End-systolic volume (ml).

Statistics

We defined cohorts C1, C2, and C3 according to whether they belonged to Group I, II, or III, respectively. For each variable, the values were plotted for each cohort, and the mean, standard deviation, minimum, maximum, median, and confidence interval for the mean was calculated. The difference between means was

studied using Student's t-test for the difference of means in paired samples, with a confidence level of 95%. A p-value <0.05 indicated a positive test result, meaning that the means differed. Finally, confidence intervals were established for the difference between means of the indicators when comparing C1-C2 and C2-C3, in order to quantify their variation.

Results

(Table 1) shows the results in the three groups with the echocardiographic variables studied. In this study, it was observed that in all patients with HFpEF (Group III) there was a longer LVPPMC time: 134 ± 18.97 ms, compared with the groups without heart disease (Groups I and II), which had a significantly shorter duration: 83 ± 16.36 ms and 83.10 ± 18.45 ms, (p < 0.01) and (Tables 2, 3), respectively.

Note: *The p-values between GI and GII are those obtained by Student's t-test for the difference of means in paired samples, with a confidence level of 95%, comparing Group I with Group II. The p-values between GI and GIII are those obtained by the test comparing Group I with Group III. Values with p < 0.05 indicate a positive test result and are interpreted as meaning that the compared means differ. Values with p ≥ 0.05 indicate a negative test result and are interpreted as meaning that the compared means coincide. The lower the p-value, the greater the probability that the compared means differ.*

Table 1: Echocardiographic Values. References. ms: milliseconds; LV: left ventricular; LVPPMC: left ventricular protodiastolic phase myocardial contraction; RWT: relative wall thickness; LVM: left ventricular mass; LVEF: left ventricular ejection fraction; LAV: left atrial volume; PAP: pulmonary artery pressure.

Variable	Group I	GI-GII	Group II	GI-GIII	Group III
		p – valor		p – valor	
Cardiac cycle (ms)	783 ± 129	0,1900	855 ± 97	0,8290	865 ± 148
LV Systole VI (ms)	346 ± 44	0,0100	300 ± 27	0,0030	423 ± 99
LVPPMC (ms)	83 ± 16	0,9870	83 ± 18	0,0009	134 ± 18
LV diastole (ms)	354 ± 111	0,0290	471 ± 78	0,0010	333 ± 82
RWT (%)	0,33 ± 0,03	0,1110	0,36 ± 0,03	0,0001	0,50 ± 0,05
LVM (gr/m^2)	67,3 ± 13,33	0,2831	72,5 ± 15	0,0040	106,3 ± 25
E/E' Ratio	6,34 ± 1,46	0,1454	7,5 ± 1,53	0,0040	16,13 ± 6,47
LVEF (%)	61,80 ± 3,46	0,8827	61,5 ± 4,9	0,3328	63,6 ± 4,88
LAV (ml/m^2)	19,2 ± 4,08	0,0320	25,2 ± 5,79	0,0020	43,9 ± 14,6
PAP (mmHG)	22,7 ± 6,77	0,0060	22,5 ± 5,64	0,0450	32,9 ± 15,89
LV End Systolic Volume (ml)	32,8 ± 7,84	0,0940	27,20 ± 8,48	0,7460	25,5 ± 12,65

Table 2: Ratio of percent left ventricular cardiac cycle, systole and diastole duration with the protodiastolic phase of myocardial contraction.

Phase	Group I	Group II	Group III
Systole / LVPPMC ratio (%)	23	27	31
Diastole /LVPPMC ratio (%)	23	17	40
Cardiac Cycle / LVPPMC ratio (%)	10	9	15

Table 3: Protodiastolic phase of myocardial contraction duration in each patient with heart failure with preserved ejection fraction for a normal investigated value of 83 ms.

Patient	1	2	3	4	5	6	7	8	9	10
Duration (ms)	130	140	160	120	110	160	150	110	140	120

Concomitantly, an increase of LVM was also found in Group III with an average of 106 g/m2 compared with the other groups (67 and 72 g/m^2, respectively). Also, RWT increased from 0.33% and 0.36% in groups I and II to 0.49% in group III, and LAV reached 43 ml/m^2 for a value in the control groups of 19 and 25 ml/m^2. In this analysis (Table 1), it is clear that in Group III, LVM, E/E', PAP, and LAV are significantly increased. In terms of total cardiac cycle duration, systole, and diastole, the cohorts are basically the same (p >0.05 for all comparisons) except for left ventricular suction time, which is longer in patients with HFpEF (p <0.01).

The mean values obtained when comparing C1-C2 and C2-C3 to study whether they differ or coincide are summarized in (Table 4).

Table 4: Comparison of C1-C2 and C2-C3 cohorts.

Comparison	Result		Interpretation of the indicator/Impact on Diagnosis
C1-C2	Coincide	CC	Neither age nor disease alter it Scarcely significant.
C2-C3	Coincide		
C1-C2	Coincide	CD	Age does not alter it, but disease does Very significant.
C2-C3	Differ		
C1-C2	Differ	DC	Age alter it, but not disease Scarcely significant.
C2-C3	Coincide		
C1-C2	Differ	DD	Age alter it and also disease. Significant.
C2-C3	Differ		

Discussion

There is a significant increase in the time to LVPPMC in Group III (patients with HFpEF) compared with the group without heart disease (Groups I and II). Furthermore, the tissue deformation curve in these cases loses its steep slope and becomes irregular, requiring a longer time to generate the pressure difference necessary to open the mitral valve (Figures 5, 6). This variable correlates with the E/E′ ratio, as in Group I this value was 6.34 ± 1.46 and in Group II 7.50 ± 1.53, compared with Group III, which reached a value of 16.13 ± 6.47 ($p < 0.01$) (Table 1). An abnormal effect on the negative pressure generated in this phase can be interpreted, as the process is slowed down with increased time to open the mitral valve.

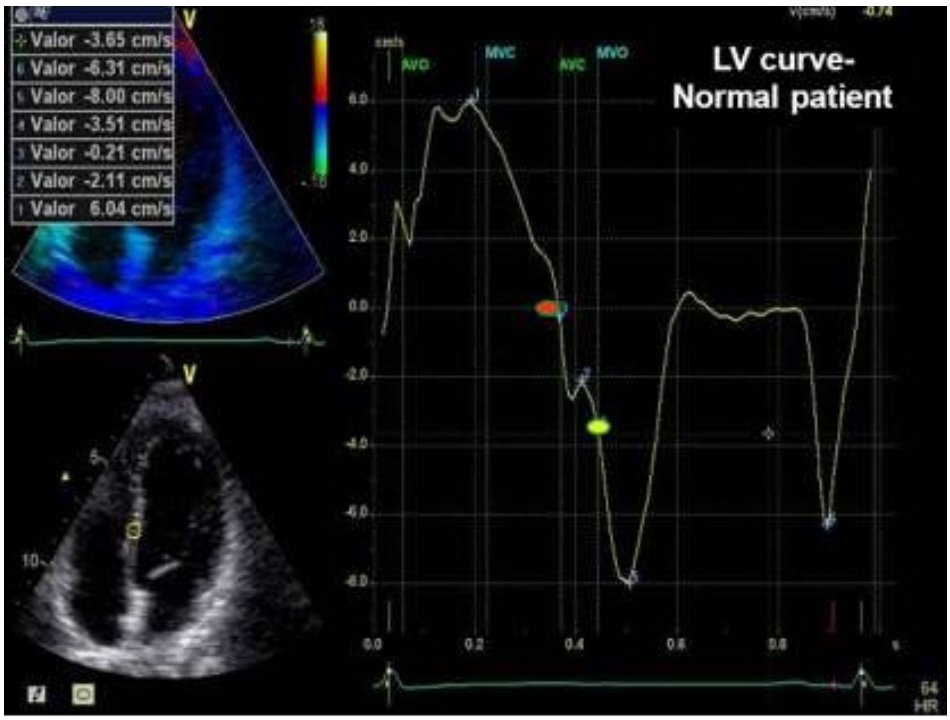

Figure 5: LVPPMC curve in a normal patient. The red dot indicates the beginning of the phaseand the yellow dot its completion. Duration 80 ms; left ventricular mass: 67 gr/m2; relative wall thickness: 0.33%.

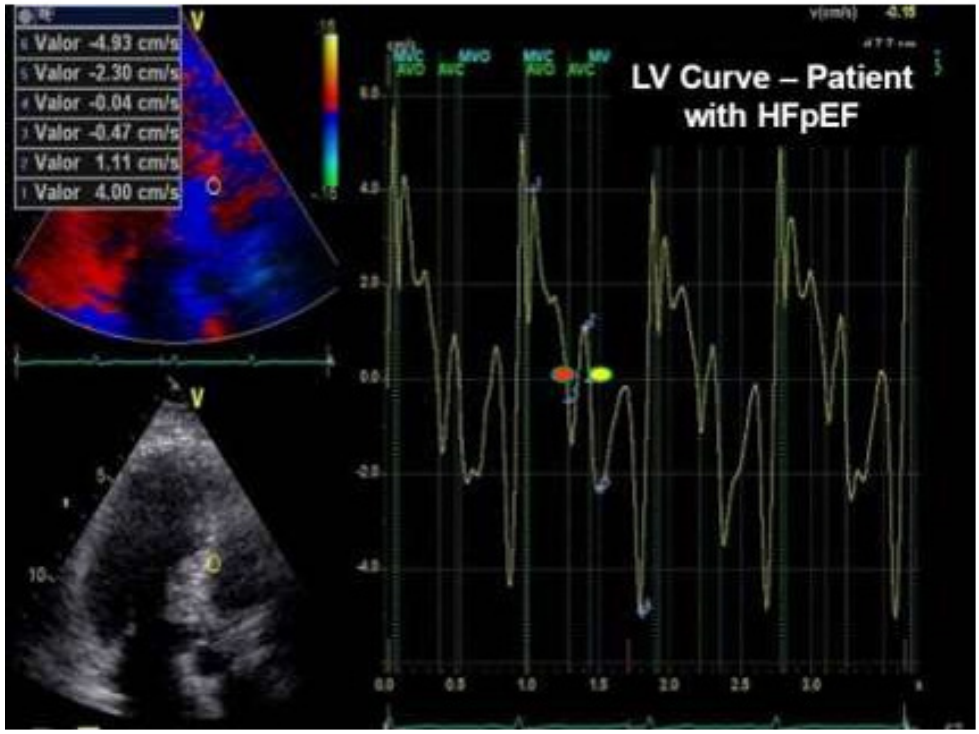

Figure 6: LVPPMC curve in a patient with HFpEF . The red dot indicates the beginning of the phase and the yellow dot its completion. Duration 160 ms; left ventricular mass: 106 gr/m^2; relative wall thickness: 0.49%.

We also observed that the duration of diastole itself (passive filling phase without energy expenditure) remained largely unchanged in all the groups (354 ms in Group I; 471 ms in Group II and 333 ms in Group III), which confirms that the alteration of the suction mechanism that occurs in LVPPMC is primarily involved in the dysfunctional process. The increase in LVM, RWT, and LAV in Group III, all of which are significant, are measurements that correspond to an increased PAP to 32 mmHg in Group III compared with 22 mmHg in Groups I and II. These concepts would explain why pulmonary wedge pressure ≥15 mmHg or left ventricular end-diastolic pressure ≥16 mmHg is often found in HFpEF.

The possible interpretation is that as its mass increases, the left ventricle does not achieve adequate detorsion in a normal time to generate a pressure drop with a suitable slope to allow mitral valve opening. In terms of flow, when the inflow to the left ventricle decreases by 1 cc per cardiac cycle due to suction deficit and the right ventricle continues to pump blood into the pulmonary system, dyspnea appears. This is understandable, since at 1 cc per beat, every hundred beats represent 100 cc, which are held in the lungs.

Consensus statements on heart failure highlight concentric left ventricular hypertrophy as a characteristic of this disease [31]. Other characteristics include reduced ventricular wall distensibility, and ventricular and aortic valve stiffness. In addition, excessive myocardial fibrosis is also mentioned, due to an increase in type 1 collagen in the extracellular matrix and an inflammatory process with increased fibroblasts and cytokines. Regarding the increase in LAV, we must understand that the atria are compensatory volume chambers that prevent the ventricles from becoming overloaded. This increase in LAV in patients with HFpEF should be considered a consequence of LV suction deficit, probably as a mechanism to reduce the increase in wall tension and prevent a significant increase in atrial pressure. This observation, present in all patients with this pathology, is clinically accompanied by both exertion and rest dyspnea.

In the LV, EF and end-systolic volume are normal in all groups, implying that the altered values corresponding to LVPPMC indicate the moment in the cardiac cycle where the pathophysiological alteration is located. The duration of the total cardiac cycle, LV systole, PPMC, and diastole were measured in the three groups. The results (Table 2) are consistent with the contributions of research. It shows that in patients with HFpEF, the duration of LVPPMC is prolonged in relation to the duration of the total cardiac cycle, systole, and diastole. This would demonstrate the possibility that patients with HFpEF may experience their problem in LVPPMC, as it requires a longer time to achieve adequate intraventricular pressure to open the mitral valve (Table 3).

Conclusions

Based on the results obtained, it can be interpreted that the mechanism of HFpEF is primarily due to ventricular suction dysfunction, which is excessively prolonged during LVPPMC compared with control groups. This would result in increased filling

pressures in the cardiac chambers, with the resulting dyspneic symptoms that characterize these patients.

Procedures in Electrophysiological Therapy

Throughout history, daily clinical practice has always encountered situations where classical pathophysiology proved insufficient for accurate interpretation. Often, only after the emergence-and acceptance-of diametrically opposed and resisted alternatives to the prevailing theories were adequate and effective interpretations and therapeutic approaches achieved. From Harvey's foundational description to the implantable cardioverter-defibrillator, including cardiac surgery and the use of beta-blockers in heart failure, new concepts marked milestones in cardiology. Sometimes these ideas were immediately applicable, but other times they had been described years earlier and required subsequent studies to demonstrate their validity. This is probably the case with the Torrent-Guasp model. In the research presented in this text, his concept of the helical heart has been revisited, expanded upon, and reinterpreted in light of new evidence.

The clinical implications of this model remain to be discussed. It will likely take years to verify them in the various cardiac syndromes and pathologies. However, one situation in which its application may be immediate is in the loss of ventricular synchrony and its counterpart, biventricular resynchronization therapy. In patients with advanced heart failure of various etiologies, biventricular dyssynchrony significantly exacerbates the condition. When a left bundle branch block occurs in an already severely damaged heart, the contraction of the left ventricle is delayed. Consequently, cardiac contraction ceases to be simultaneous or synchronous, becoming asynchronous and further worsening cardiac mechanics.

Central Resynchronization Therapy (CRT) is a procedure that restores biventricular synchrony. This resynchronization is achieved using a cardiac stimulator similar to a sequential pacemaker, but which, in addition to the right ventricle, also stimulates the left ventricle. However, to achieve adequate resynchronization, stimulation of this chamber is not enough; it must also be done from specific sites or "effective zones," typically the medial lateral or posterolateral zone of the left ventricle. The standard procedure for implanting this catheter involves cannulating the coronary sinus and then a tributary vein leading to the "effective" area. However, this procedure presents numerous problems and drawbacks, resulting in a failure rate of 20 to 30% in patients undergoing Cardiac Resynchronization Therapy (CRT). These patients are referred to as "non-responders."

Before proceeding, it is crucial to emphasize the significance of this situation. CRT is a procedure of critical importance. It is performed in patients with advanced heart failure for whom all other available pharmacological and surgical treatments have proven ineffective. If successful, it leads to a very significant improvement in the patient's quality of life and survival; failure results in the patient remaining in Functional Class III-IV. While heart transplantation could be considered as an option, in practice, very few patients have the opportunity to undergo it.

There are some striking elements in this pathology and its treatment:

1) Despite tens of thousands of patients treated with this approach, current cardiac mechanics has not clearly elucidated the concept of biventricular dyssynchrony. Even in cases of very severe cardiac mechanical dysfunction, the most advanced diagnostic methods, including sophisticated echocardiography techniques, gamma camera imaging, and MRI, do not allow for the diagnosis of this pathology, at least not with useful and reliable sensitivity and specificity. The diagnosis continues to be based on the presence of left bundle branch block, logically within the context of dilated cardiomyopathy in NYHA class III or IV. Even this seemingly classic and unchangeable criterion is under discussion, with debate about whether the important factor is its morphology ("typical" or "atypical"), its critical duration, or some other as-yet-unresolved criterion.

2) The same applies to the evaluation of the effectiveness of Cardiac Resynchronization Therapy (CRT). None of the techniques mentioned manage to document variations of any parameter in a sufficiently significant and early measure that reliably correlates with the almost surprising improvements in the clinical evolution of patients in whom CRT is successful.

In our opinion, these "failures" are due to the evaluation of classical cardiac mechanics parameters that are not responsible for either the pathology or the response to therapy. On the contrary, it is very appealing to reconsider these phenomena in light of the new findings on stimulus propagation and subsequent cardiac mechanics discovered in this research.

The principal parameter of "dyssynchrony," as we have pointed out, is left bundle branch block. This phenomenon, which, we reiterate, hardly explains the severity of the mechanical disturbances, would, moreover, severely alter the mechanics of the ascending bundle. In fact, it would seem quite coherent to suggest that a failure in activation at the level of the "crossing of bundles" mentioned above could generate the image of left bundle branch block (and its "atypical" forms) and, of course, severely alter the activation sequence of said bundle, but not in the way proposed in the "classical" conception. There would now be an alteration in the "radial" activation of the descending endocardial band to the ascending epicardial band and in the "bidirectional longitudinal" activation of the epicardial band (Figure 3). We emphasize that standard methods for assessing ventricular mechanics, particularly left ventricular mechanics, generally do not take these essential factors into account. Therefore, it would not be surprising if these methods were unreliable in evaluating both ventricular dysfunction and its improvement with CRT.

The critical importance of the left ventricular pacing catheter's location for CRT effectiveness is also striking, as is the failure of currently used methods to explain this fact. It has been suggested that the best catheter location is that corresponding to the area of

latest ventricular activation; while this is true in many cases, it is not always the case. However, there is agreement that anatomical placement in the medial area of the posterolateral or lateral wall of the left ventricle is effective, regardless of whether it coincides with the late potentials. This could be coherently interpreted using the Torrent-Guasp model and our findings, where the aforementioned location corresponds to the crossing of the ventricular bands. The left ventricular catheter would activate it at the precise point required to "restore" the normal activation of the ascending segment in its double-front progression towards the apical loop and ventricular base [32,33].

Finally, there is another fact that strongly supports this interpretation: we have already pointed out that the usual route for implanting a left ventricular catheter is through the coronary sinus, which provides access to the epicardial veins of the left ventricle, thus diffusing activation from the epicardium to the endocardium. Even when the catheter is correctly positioned in the aforementioned "effective zone," a percentage of patients are "non-responders." Studies such as the Alsync trial [34] found that 20% of patients were non-responders despite the catheter being perfectly positioned when implanted via the coronary sinus. Fifty percent of these patients became responders when the catheter was implanted endocavitary. The explanation for this, based on the classical view, is a rather vague notion that endo-epicardial activation is "more physiological" than the reverse.

Viewing this phenomenon through the lens of our research, the explanation becomes much more coherent: activation of the ascending band at the point of intersecting the bands restores its longitudinal activation, with the beneficial consequences already mentioned. However, radial activation from the descending to the ascending band is lost, as is the persistence of normal distal activation of the descending segment. Endocavitary stimulation (descending segment) would almost completely restore normal electrical activation and, consequently, its mechanical function. Based on this principle, endocavitary CRT has been under investigation for several years at the Presidente Perón Hospital in Avellaneda, Argentina. It has already been used in numerous patients, with excellent surgical and clinical results. In summary, Torrent Guasp's hypothesis and fundamentally anatomical model, expanded and modified in our research from an electromechanical perspective, offers countless possibilities for development, both theoretically and in terms of clinical and therapeutic application. Much of cardiology will likely need to be re-examined in light of this new paradigm, with perhaps unpredictable results.

Surgical Procedures

Ventricular dilation is an adaptive state in response to various cardiovascular diseases. This condition results in cardiac remodeling, leading to increased wall tension and progressive dilation. In the myocyte, this process leads to irreversible slippage and a consequent rightward shift of the pressure/volume curve, increased ventricular volumes, and added mitral regurgitation. This continuous dilation process is the primary cause of increased mortality [35]. This situation has made the study of altered ventricular geometry in heart failure of paramount importance for the application of new surgical therapies aimed at restoring this state and improving prognosis.

Leonardo da Vinci (1452-1519), when studying the left ventricle and the aortic root, made geometric drawings for his analyses [36]. William Harvey, in his work "Exercitatio anatomica de motu cordis et sanguinis in animalibus" (1628), described the left ventricle as narrow and elongated during the ejection phase, tending towards a sphere during diastole [36]. Interest in these studies dates back to Woods in 1892, but it was in the mid-20th century that Burton found that the increase in cardiac volumes, coupled with a larger internal radius of the ventricle, implies greater tension or stress on its wall [20].

In the evolution of various surgical techniques, different approaches have been considered clinically to address both ventricular dilation and non-contractile post-infarction areas. This analysis indicates the current need for a strategy that restores the left ventricle to its native configuration through ellipsoidal reconstruction. The fundamental principles for restoring the geometry of the left ventricle to an ellipsoidal shape are: a) geometric; b) anatomical; c) functional; and d) volumetric.

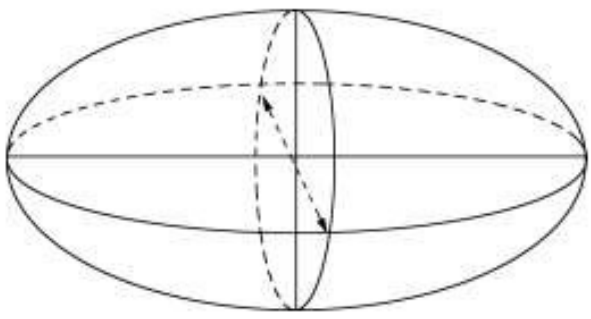

Figure 7

Figure 7

a) Geometric: The ellipsoidal reconstruction, which is the geometric shape assumed by the normal left ventricle, is related to its functional efficiency. Structure and function are inextricably linked to achieve maximum mechanical performance. The ellipsoidal shape of the normal left ventricle is characterized by the presence of one major diameter and two minor diameters of equal dimensions (Figure 7).

b) Anatomical: The studies carried out by Torrent Guasp [37] have been fundamental in linking the anatomical relationship of the heart to cardiac mechanics. In his description, the ventricular chambers are defined by the continuous myocardium that describes two spiral turns extending from the root of the pulmonary artery to the root of the aorta. Within this helical configuration, a descending and an ascending band must be distinguished. The ventricular myocardial band thus describes two spirals, which implies that the ventricles are the chambers of a circular muscle. This contraction is exerted on a cardiac fulcrum located where the continuous myocardium begins and ends. We have termed this finding from our research the cardiac fulcrum. At this site, the myocardial fibers attach to exert the myocardium's extraordinary power.

c) Functional: Physically, intraventricular pressure exerts a tension on the wall that contains it, which is explained by Laplace's law (1749-1827). This equation indicates that wall Tension (T) is directly proportional to transmural Pressure (P) and the Radius of the vessel (r), and inversely proportional to the thickness of the vascular wall (w): T = Pr/w. This principle explains the value of the general concept developed with ventricular reduction techniques, which is consistent with the aforementioned physical postulates [38,39].

d) Volumetric: It becomes essential to manage not only the shape (ellipsoid) but also the cardiac volume. Greater volume results in greater sphericity, and vice versa. When 20% of the ventricular mass is complicated, remodeling begins, triggering volume overload (Figures 8, 9). When the left ventricular end-systolic volume is greater than 100 ml/m2, the probability of being free from heart failure is only 31.4%. Conversely, if the volume is less than 100 ml/m2, this probability increases to 85% [20].

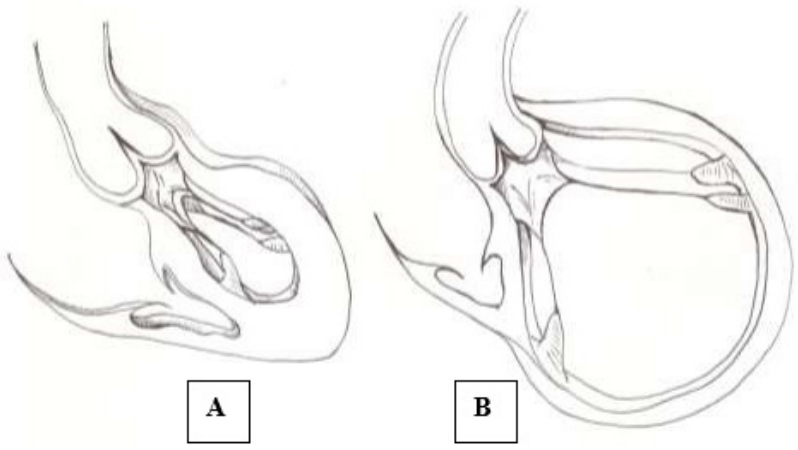

Figure 8: A: normal (ellipsoidal); B: dilated (spherical).

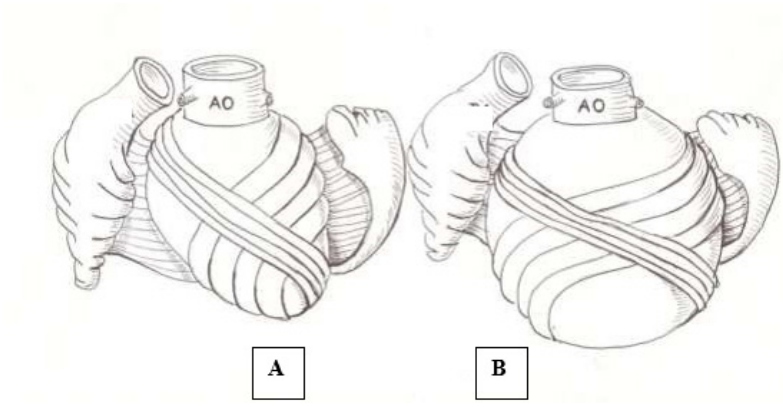

Figure 9: A: normal (ellipsoidal); B: dilated (spherical). The dilated heart shows separation between the descending and ascending segments.

It is essential to understand that ventricular sphericity leads to increased wall stress. Wall stress acts in three directions: meridional or longitudinal, circumferential, and radial. When ventricular dysfunction is present, the greatest increase in stress is longitudinal. These findings are not only observed in patients with idiopathic dilated cardiomyopathy but also in those with ischemic heart disease with depressed ventricular function, aortic insufficiency, mitral insufficiency, ventricular septal defects, and aortic stenosis with depressed ventricular function. Furthermore, these alterations in left ventricular geometry are key determinants of the development of functional mitral regurgitation in both ischemic and idiopathic heart disease.

Given this situation, we have implemented an ellipsoidal reconstruction using a technique that combines the geometric, anatomical, and functional principles presented here [20,40]. The ellipsoidal reconstruction technique of the left ventricle investigated and carried out during this research assumes the possibility of restoring the geometry to the form required for its mechanical function. It consists of the following steps (Figures 10-13):

1) A longitudinal incision is made along the left anterior descending artery in the avascular wall of the left ventricle (Figures 11, 12).

2) The left edge of the incision is brought to the level of the preserved interventricular septum with a continuous suture (Figure 13).

3) The remaining marginal edge, i.e., the right edge, is sutured to the free wall of the left ventricle (Figure 14).

4) Both the original incision and the size of the flaps should be adjusted to the cavity that is to be preserved, in order to reduce ventricular volume (Figure 15).

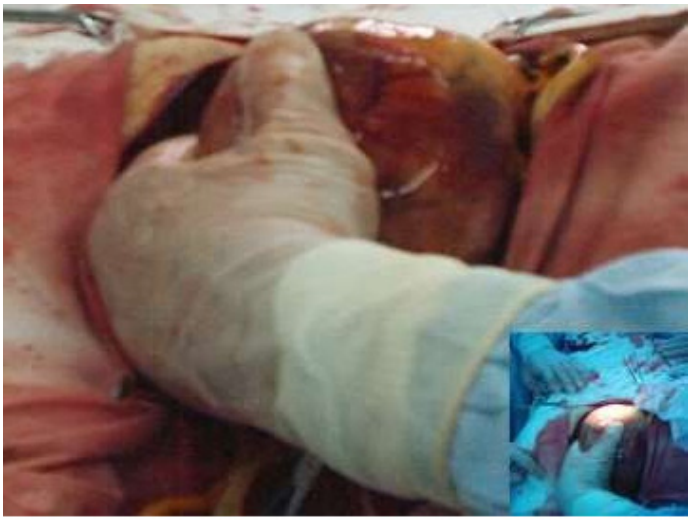

Figure 10: Dilation of the left ventricle is observed at the start of ellipsoidal reconstruction surgery.

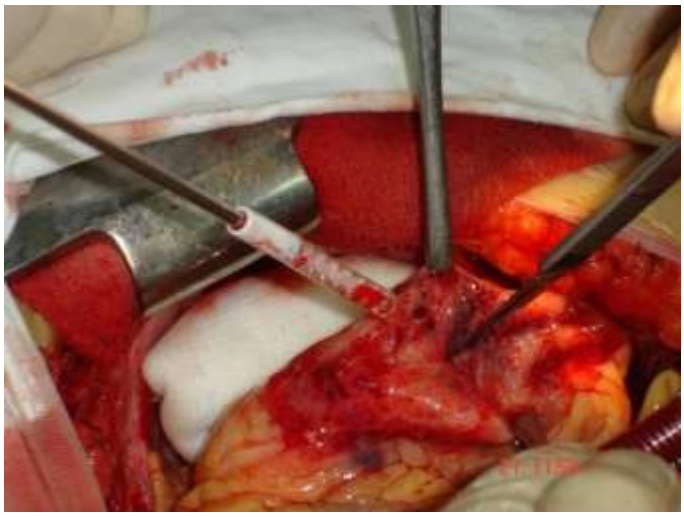

Figure 11: With the patient on extracorporeal circulation and the aorta clamped, the wall of the dilation is observed to be flaccid and ready to be excised.

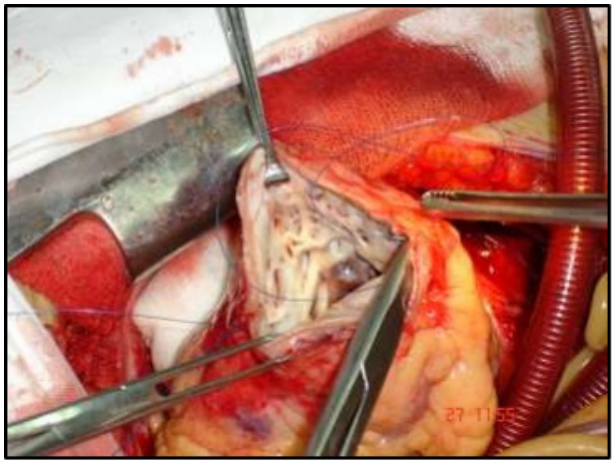

Figure 12: Longitudinal incision in the avascular left ventricular wall showing the aneurysmal area to be excluded when the flap with myocardial margins is formed.

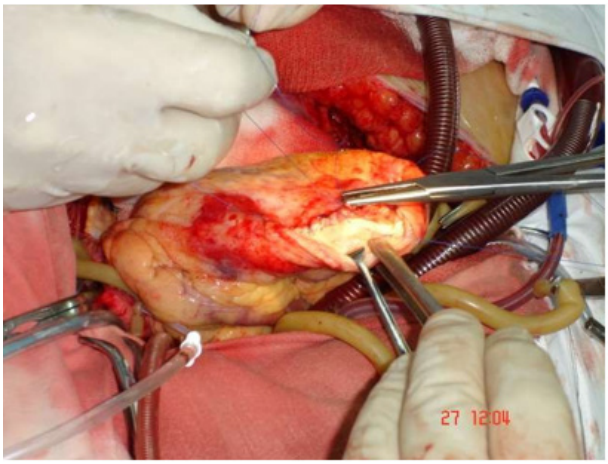

Figure 13: Internal suture procedure. The left incision margin is brought to the Interventricular septum. The unsutured margin will be conveyed to the left ventricular free wall.

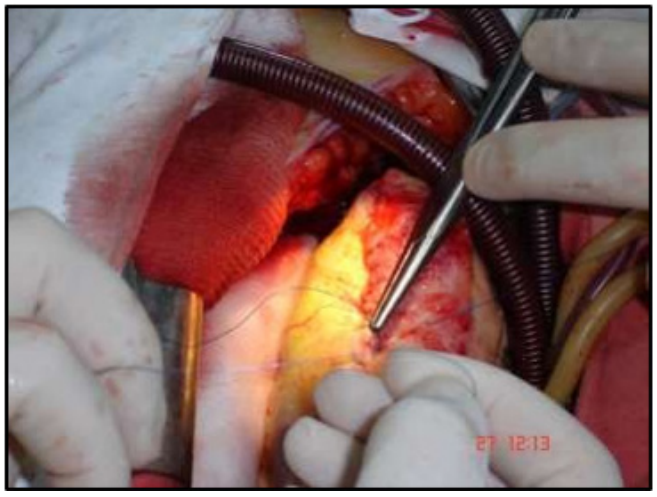

Figure 14: External suture procedure. The right margin with the part of the excluded septum is sutured to the left ventricular free wall.

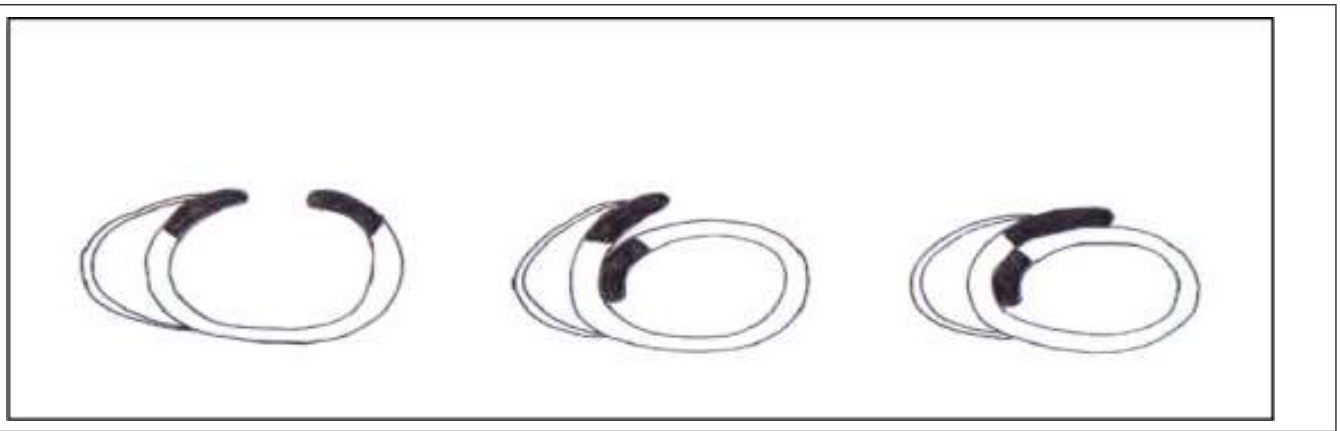

Figure 15: Diagram of the successive steps in ellipsoidal reconstruction.

This technique offers several advantages when compared to traditional techniques (Jatene, Dor, Batista) (13,15,35) and aligns with the strategy presented by Matsui [41] and Menicanti [42,43]. These are:

a) Preservation of the cardiac muscle by operating on the area limited by the descending and ascending segments of the ventricular myocardial band, according to the work of Torrent Guasp [44]. At this geographic point of the heart, called the apex, a virtual conduit is defined in which the endocardium adheres to the epicardium, constituting a weak zone prone to dyskinesia [45,46].

b) This apical topographic zone of the heart is avascular, which prevents the arterial system from being pulled during resection.

c) It preserves the circumflex artery by choosing the avascular left ventricular wall along the left anterior descending artery as the incision site. In other techniques, with a lateral approach to the left ventricle, circumflex branches are sacrificed.

d) The overlapping geometric effect achieved with this technique results in a ventricular girdle effect.

e) The flap constructed with the incision edges excludes the remodeled area (anterior and tip of the septum), leaving the new cardiac apex viable to withstand cardiac pressure, as it is reconstructed with healthy tissue.

f) Given the distortion of the apical septum that occurs during remodeling, which appears thinned, this technique eliminates dyskinesia in this region by providing rigidity to the septum.

g) This technique does not involve the placement of synthetic patches, thus avoiding leaving non-contractile areas on the left ventricular contraction surface.

h) The aforementioned technique brings the ascending and descending segments of the apical loop closer together, which are separated in heart failure, contributing to improved function.

i) We believe this technique can treat both the septal distortion and improve diastolic function.

j) Restoring the heart's ellipsoidal shape could address the structural alteration of the mitral apparatus. We have experienced that after ventricle reduction; insufficient mitral valves did not require surgical repair [46].

Material and Methods

Twenty-nine patients (27 men) with a mean age of 65 ± 8.4 years underwent surgery. The mean NYHA functional class was 3.25, with a mean ejection fraction of 19.8%. The mean end-systolic volume was 106 ml/m^2. The study was conducted at the Presidente Perón Hospital (Argentina). As concomitant treatment, one patient underwent a bone marrow-derived cardiomyoimplantation; seven patients underwent coronary revascularization (three in conjunction with cell therapy); and one patient underwent mitral valve replacement followed by a cell-derived cardiomyoimplantation. All patients underwent ellipsoidal ventricular reduction using the described technique. Thirty-day mortality occurred in only one patient (due to infection). At an average follow-up of 27.8 months, the functional class was reduced to an average of 1.37, with the average end-systolic volume index decreasing from 108 ml/m2 to 56 ml/m2 ($p<0.05$) and an increase in ejection fraction from 19.8% preoperatively to 37.1% ($p<0.05$) post-surgery.

Conclusion

Restoring ventricular geometry has stimulated the use of techniques for its evaluation and has also allowed for the elimination of spatial distortion in heart failure. Undoubtedly, the spherical shape that the left ventricle assumes during heart failure has an ominous prognostic value. Ventricular geometry is a sensitive marker of function and prognosis. Whether a cause or

a consequence, its appearance determines an increase in oxygen consumption in the patient through increased wall stress. It is certainly a consequence, but it also carries the responsibility for perpetuating the alteration.

Pacemaker Placement: Anatomical and Physiological Relationship between the Fulcrum and the AV Node

This research analyzed the anatomical and histological relationship between the cardiac fulcrum and the atrioventricular node in human and bovine hearts, as well as the potential functionality between these two structures. Samples were taken from the Aschoff-Tawara AV node and the bundle of His in Koch's triangle (Figure 16). The helical conformation of the heart, combined with the myocardial extension to and retraction of an attractor-the cardiac fulcrum-and the configuration of the electromechanical unit (fulcrum + AV node), has led to advancements in the knowledge of catheter placement for cardiac pacing, resulting in improved physiological performance compared to traditional approaches.

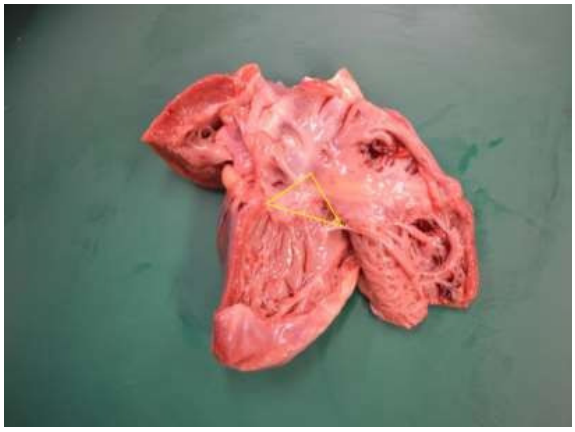

Figure 16: 4-year-old human heart: Macroscopy of Koch's triangle, delimited by the septal leaflet of the tricuspid valve, the tendon of Todaro and the coronary sinus. This triangle is a reference to find the AV node.

The fulcrum, located at the atrioventricular junction at the insertion of the interventricular septum, below the aorta and pulmonary artery, is adjacent to the AV node of Aschoff-Tawara, which lies to its right (Figures 17, 18). The AV node is located at the atrioventricular junction, at the base of the muscular septum, below the origin of the great vessels. It is adjacent to the cardiac fulcrum, situated between it and the insertion of the septal leaflet of the tricuspid valve. It constitutes a cluster of cells (specialized myocytes) that Rushmer defines as a spherical or bulbous end composed of bundles of fibers [47] for the purpose of transmitting electrical impulses to the myocardial mass. Continuing along its length, it transforms faintly into the bundle of His, which is short in length, sometimes even nonexistent. In humans it measures approximately 5 mm in length, 3 mm in width and 1 mm in thickness and is irrigated by a branch of the coronary arteries, usually from the right in 90% of cases and from the left circumflex in the remaining minority [48-50].

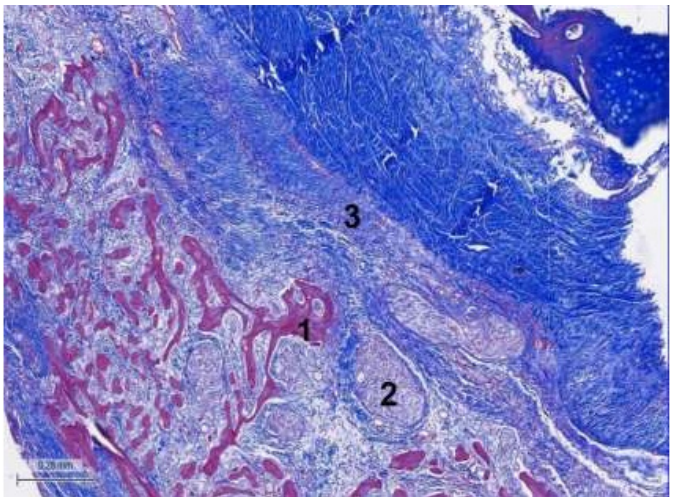

Figure 17: (Bovine heart). Masson's trichrome technique, 25x. Tangential section of the cardiac fulcrum.

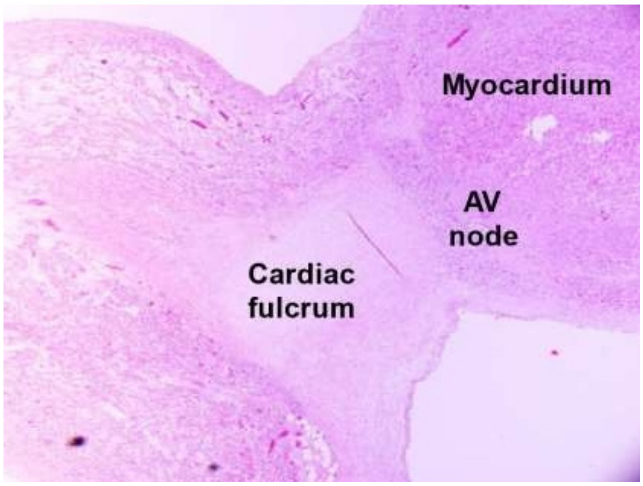

Figure 18: 36-day-old newborn human heart. Magnification 20x. The cardiac fulcrum of cartilaginous matrix is seen with the myocardium and adjacent AV node. AV: Aschoff-Tawara atrioventricular node.

This analysis reveals, in both human and bovine hearts, a topic of importance for cardiac stimulation therapy. The histological study found that the fulcrum is adjacent to the AV node, forming a cellular cluster rich in neurofilament plexuses. This contiguity between the two structures was found in all specimens studied, both bovine and human hearts. The key finding of this research is that neurofilaments (Figure 19) are also located within the cardiac fulcrum. Fibroblasts and connective tissue are situated within the conduction system and between it and the working myocardium, acting as an insulating layer. Connective tissue also forms the fibrous ring and the central fibrocartilaginous portion of the fulcrum, thus electrically isolating the atrial and ventricular chambers.

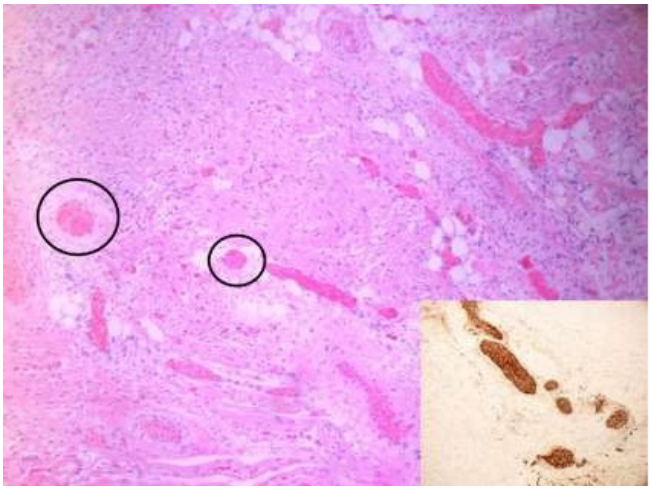

Figure 19: 27-week-old infant heart. Thick nerve trunks are seen in the cardiac fulcrum (arrows) adjacent to the AV node. HEx200. Inset shows thickened nerve trunks in the cardiac fulcrum confirmed by immunohistochemistry for S-100.

The AV node has a compact portion and a marginal portion of transitional cells. The transitional zone is located between the wall of the right atrium and the AV node, forming its outer layer. The compact component has a semi-oval shape and is continuous with the cardiac fibrous tissue. The cells of the compact portion of the node are smaller and more spindle-shaped than the transitional cells. The transitional cells are arranged parallel to each other and have intermediate dimensions between the conduction and working myocardial cells. They are surrounded by a greater amount of connective tissue and are responsible for transmitting electrical signals.

The helical spatial arrangement of the myocardium forces the muscle to overlap segments in its spatial configuration. This anatomical situation is closely related to myocardial movements and the stimulation that travels through its segments, according to the electrophysiological studies we have conducted. The interpretation of the anatomical relationships between the cardiac fulcrum and the AV node implies the complementarity of anatomy

with the physiology of the continuous helical myocardium, since their contiguity lies at the site where stimulation begins and ends, producing the mechanical action of torsion and detorsion during the systolic and suction phases of the ventricles. The cardiac fulcrum, the support and insertion point of the myocardium that acts as a lever during its movements, is located adjacent to the AV node of Aschoff-Tawara. This constitutes an electromechanical unit situated at the beginning and end of the continuous, helical myocardium. This anatomical and functional arrangement of the myocardium is supported by a rich plexus of specialized filaments that interact with the mechanically active cardiomyocytes [49].

This interpretation of the findings in the research conducted with human and bovine hearts inevitably leads to therapeutic action. What explains why, in our experience, we observed better synchronization of the pacemakers with the catheter placed near this electromechanical unit? The AV node is located at the base of the muscular septum at the base of the tricuspid valve's septal leaflet, at the insertion point of the interventricular septum with the aorta and pulmonary artery. In this respect, the proximity between the cardiac fulcrum and the beginning of the continuous myocardium along its helical course, in relation to the AV node, demonstrated that stimulation of the right ventricular outflow tract was more effective. In this experience with pacemakers implanted at different points in the right ventricle (apex, parahisian, outflow tract), using standard active fixation catheters, the right ventricular outflow tract achieved better electrical synchrony in the left ventricle [3,48]. The ideal region for the pacemaker pacing catheter would be located high in the right ventricular outflow tract, below the pulmonic valve, and preferably over the septum, not on the free wall.

The function of the myocardium gives it a fulcrum, like any skeletal muscle, both at its origin and its end. If it did not have this helical anatomical configuration, but rather an insertion at its ends located at the base of the heart, instead remaining free at the apex, or hanging like a pendulum in the thorax; and if it did not receive stimulation that allows for its torsion and detorsion, it could not fulfill its extraordinary muscular power. The proximity of the cardiac fulcrum to the AV node, surrounded by a rich plexus of neurofilaments, leads us to consider the anatomical structure of an electromechanical unit in which stimulation energy and muscle mechanics participate. The effectiveness achieved with the placement of the pacemaker catheter in the vicinity of the right ventricular outflow tract validates the findings of this research.

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None.

Conflict of Interest

None.

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