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Ventricular synchrony in artificial cardiac pacing: The role of the helical myocardium and fulcrum in the electromechanical coupling of the heart

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ABSTRACT

About 30% of patients with Heart Failure (HF) have wide QRS complexes. In left bundle branch block (LBBB), the electromechanical activation sequence in the Left Ventricle (LV) is asynchronous with deterioration of pump function, higher frequency of hospitalizations and mortality. Definitive artificial cardiac stimulation, especially when from the apex of the Right Ventricle (RV), entails ventricular activation identical and as antiphysiological as LBBB, and therefore, in a percentage of cases, a worsening of the degree of HF and a higher incidence of arrhythmias occurs. headphones, hospitalizations, and mortality.
In search of "physiological" stimulation, alternative RV electrode placement sites (non-apical sites) have been evaluated. We highlight the stimulation of the bundle of His, the area of the left branch, or biventricular stimulation for cardiac resynchronization, strategies included in a generic term: Cardiac Physiologic Pacing – CPP (1) and with promising results in preserving and/or restoring synchrony. cardiac electromechanics. However, due to the complexity of the procedures, especially technical and resources, the first two have not had the consensus or the expected growth. On the other hand, biventricular Cardiac Resynchronization Therapy (CRT) was also shown to be ineffective in about 30% of patients (non-responders).
Dr. Francisco Torrent Guasp, demonstrated that the ventricular myocardium is made up of a continuous muscular band with a helical shape, which explains the great efficiency of cardiac systole, where blood is expelled through torsion-detorsion contraction mechanisms, with a phase active suction in protodiastole. The cardiac fulcrum functions as a fulcrum and support for the helical myocardium. According to this author, the propagation of the electrical stimulus and the contraction of the myocardium (electromechanical coupling) begin in the region of the right ventricular outflow tract (RVOT) anatomically related to the cardiac fulcrum, advancing towards the rest of the helical myocardial segments and continuing the longitudinal direction of the muscle bundles, which could explain the results of definitive cardiac pacing in the RVOT septum with electrical synchrony in some cases.
Synchromax® (Exo S.A., Buenos Aires, Argentina) is a software which records non-invasively during lead implantation: spatial coincidence, area and direction of QRS in leads D2 and V6 from the ECG. Intraoperatively, after analysis and in real-time, generates a mathematical index called cross-correlation cardiac synchrony index (CSI) which at this time generates a couple of synchrony curves. A CSI value of 0.0 corresponds to maximum coincidence between both leads (perfect ventricular synchrony), 0.0-0.39 demonstrate adequate synchrony, 0.4-0.7 equal poor synchrony, and values > 0.71 correspond to cardiac dyssynchrony.
In this chapter we present a new perspective. An anatomical-mechano-physiological vision in which the coordination of cardiac activity is based on the interdependence between structure, activation and function: cardiac synchrony. In this context, physiological cardiac stimulation seeks to artificially reproduce that the result is, as in nature, maximum electromechanical coordination with the goal of the best cardiac systolic function. As will be explained, it is difficult to explain efficient and coordinated (synchronous) cardiac performance without an adequate structure, or to accept a non-complex anatomy for the competent development of the function.

Keywords: Ventricular synchrony, Artificial heart stimulation, The role of the helical myocardium.

1 INTRODUCTION

1.1 ASYNCHRONY IN VENTRICULAR CONTRACTION AS A DETERMINING FACTOR IN THE PATHOPHYSIOLOGY OF HEART FAILURE (HF)

About 30% of patients with HF have wide QRS complexes (>120 ms) (2). In left bundle branch block (LBBB), the activation sequence of the different LV walls is asynchronous, with a delay of the lateral in relation to the septum (3). This results in a decrease in effectiveness in ventricular systole, with deterioration of pump function, pre-systolic mitral insufficiency and increase in LV end-diastole pressure (4). Comparative studies in patients with HF showed a higher frequency of hospitalizations and higher mortality in those with wide QRS (figure 1) (5). These data are more evident in patients with genuine or true LBBB and QRS duration greater than 150 ms, while the results are not as notable or significant in patients with narrow QRS, right bundle branch block (RBBB) or nonspecific intraventricular conduction disorders (6, 7). However, these characteristics and the absence of eventual response to cardiac resynchronization therapy (CRT) do not mean that there is no cardiac asynchrony in these patients, but perhaps they are translating into a lack of scope of the implemented strategies.

Figure 1: Graph of year mortality in patients with heart failure with and without left bundle branch block.

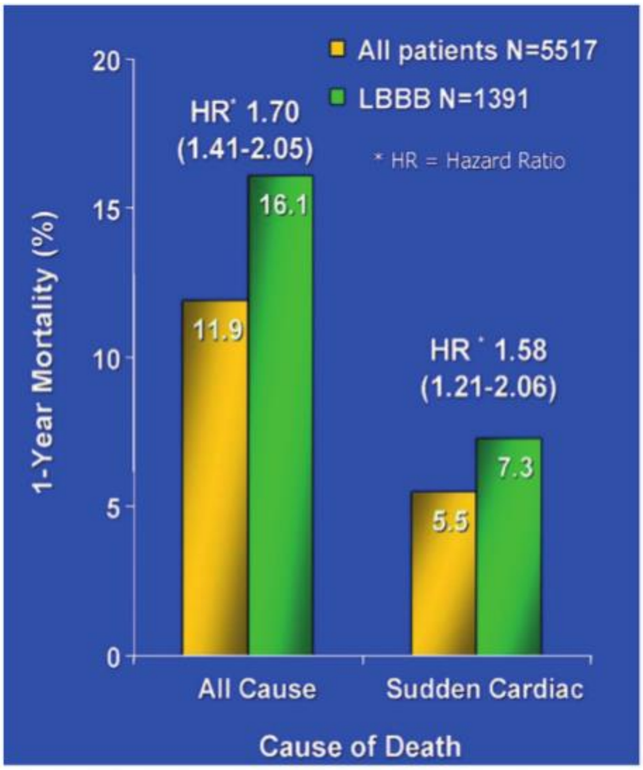

Figura 1
Gráfico de mortalidad al año en pacientes con Insuficiencia Cardíaca con y sin bloqueo de rama izquierda.

2 DEFINITIVE APEX STIMULATION AS A CAUSE OF LEFT VENTRICULAR DYSSYNCHRONY

The first pacemaker implantation was performed in 1958 by Dr. Ake Senning at Karolinska Hospital, (Stockholm, Sweden). Those first pacemakers were implanted with electrodes in the epicardium, later evolving over time to endocardial implantation, transvenously, traditionally with definitive positioning of the electrode in the apex of the right ventricle (RV). The results of definitive cardiac stimulation managed to improve the survival of patients with severe symptomatic bradyarrhythmias and today it is the undisputed treatment when any pathology in this scenario is irreversible. However, a percentage of them (difficult to predict in advance) will present deterioration in LV function, worsening of the degree of HF and a higher incidence of atrial arrhythmias (figure 2)(8). The explanation for such an undesirable effect is that by stimulating the RV apex we would be producing an “iatrogenic” LBBB, since ventricular activation is identical and as antiphysiological as in said conduction disorder. In this way, a new entity arises that we can call cardiomyopathy induced by artificial dyssynchrony (MIDA). (9, 10)

Figure 2: This review paper shows the deleterious effect of definitive pacing in the RV apex, which may lead to deterioration of LV function, worsening of HF and increased incidence of atrial fibrillation.

The Deleterious Consequences of Right Venricular Apical Pacing

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Trial	No. of Patients	Mean Age (y)	Mean FU (y)	LA Diameter	LV Function	CHF	AF
Tantengco et al.[28]	24	19.5	9.5	NA	↓	2 pts	NA
Karpawich et al.[29]	14	15.5	5.5	NA	Altered Histology	NA	NA
Thambo et al.[30]	23	24	10	NA	↓/DS	NA	NA
Tse et al.[31]	12	72	1.5	NA	↓/MPD	NA	NA
Hamdan et al.[32]	13	66	NA*	NA	↓/↑SNA	NA	NA
DAVID[36]	506	64	1	NA	NA	↑	NA
MADIT II[37,38] Substudy	567	64	1.7	NA	NA	↑	NA
Wonisch et al.[39]	17	59	0.25	NA	NA	**	NA
Thackray et al.[40]	307	72	5.2	NA	NA	↑	↑
MOST[41]	1,339	74	6	NA	NA	↑	↑
Nielsen et al.[43]	177	74	2.9	↑	↓	NA	↑
O'Keefe et al.[44]	59	69	1.5	NA	↓	NA	NA

Figura 2
En este trabajo de revisión se evidencia el efecto deletéreo de la estimulación definitiva en el ápex del VD, la cual puede producir deterioro de la función VI, empeoramiento de la IC y aumentar la incidencia de fibrilación auricular.

Given the demonstration with multiple scientific evidence of the existence of MIDA, algorithms were implemented to minimize ventricular stimulation, being in a way a contradiction, since a pacemaker is implanted with the purpose of artificially activating the heart for the shortest possible time. , especially in those patients who do not depend on artificial ventricular stimulation. On the other hand, paradoxically, the action of these algorithms triggers atrio-ventricular intervals (PR interval in the surface ECG), often very long, also anti-physiological and capable of triggering dyssynchrony in the same way, only this time between atria and ventricles. (atrioventricular - AV dyssynchrony) (11, 12). That scenario: avoiding wide QRS – artificial LBBB and its consequences vs. Maintaining narrow QRS causing AV dyssynchrony due to long PRs that are also antiphysiological, poses the challenge of finding alternative forms and sites of stimulation in order to avoid this dyssynchrony and MIDA. It also confronts us with the alternative need to have a real-time diagnosis of cardiac electromechanical coordination (synchrony), which helps us during the intraoperative period to have a better notion of the effect generated by the artificial stimulation by the electrode from that specific site.

3 CARDIAC RESYNCHRONIZATION THERAPY (CRT) AS A TREATMENT FOR HF

In order to correct intra- and interventricular dyssynchrony, the use of CRT by biventricular artificial stimulation began to be implemented. Since the first publication of CRT implantation by Serge Cazeau in 1994, several multicenter studies demonstrated the effectiveness of CRT in improving the morbidity and mortality of dilated patients, with grade III-IV HF, systolic failure with EF <35%. and BCRI (Figure 3A) (13, 14). However, CRT is ineffective in about 30% of patients, classified as "Non-Responders" (Figure 3B) (15, 16). Among the reasons for this lack of adequate response we can mention the technical difficulties in catheterizing the coronary sinus, many of them due to unpredictable anatomy, failure to stimulate or position the cable - electrode in a vein on the lateral aspect of the LV (the most delayed due to LBBB), diaphragmatic stimulation, high thresholds, etc. (17–19).

Figure 3: A) Effect of CRT on morbidity and mortality in patients with HF (CARE-HF Trial Cardiac Resynchronization-Heart Failure). B) Percentage of patients not responding to CRT and its causes.

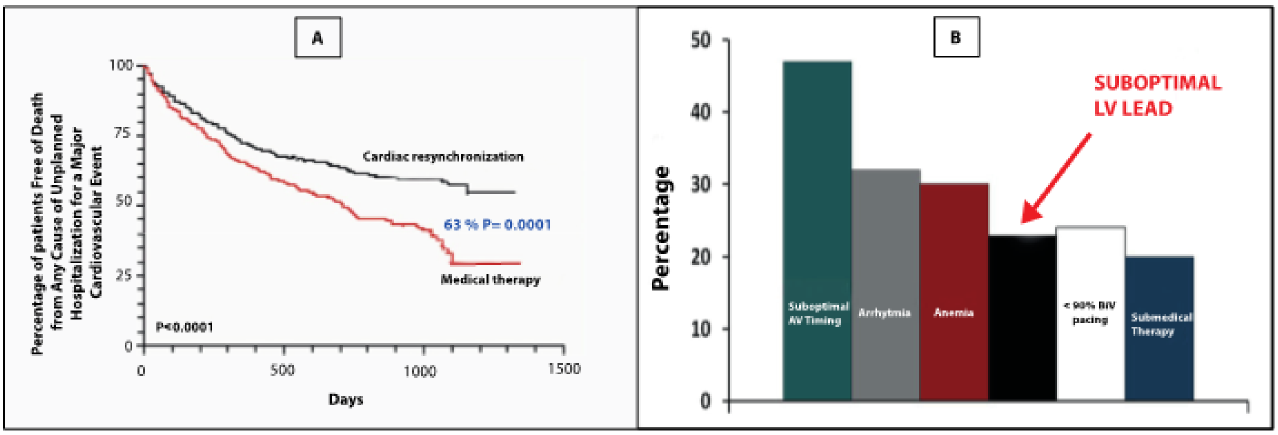

Figura 3: A) Efecto de la TRC sobre la morbi-mortalidad en pacientes con IC (CARE-HF Trial Cardiac Resyncronization-Heart Failure). B) Porcentaje de pacientes no respondedores a la TRC y sus causas.

4 THE ROLE OF CONDUCTION SYSTEM PACING AND ALTERNATIVE VENTRICULAR PACING SITES IN PREVENTING DYSSYNCHRONY

The alternative non-apical RV ECA sites have the purpose of achieving a more coordinated and close ventricular electromechanical sequence than the natural one due to the intrinsic conduction system. It is understood that this will result, therefore, in obtaining better, almost physiological activation (cardiac synchrony) (20). Non-selective or selective capture of the His bundle proved to be superior to RCT of the RV apex, especially in those patients with a pacing load greater than 20-40% and a LV ejection fraction less than 50% (Figure 4) (21, 22). However, these procedures require a long learning curve due to their technical difficulties, are complex and high cost due to the use of special materials, have a higher risk of complications and reinterventions, higher capture thresholds, shorter battery life, and doubts about their long-term safety, as a consequence they have not had the expected consensus or growth (23). Furthermore, to carry out these procedures it is necessary to have a polygraph and the presence of a specialist in invasive electrophysiology trained in intracavitary mapping, which is presented as a limitation, since we must keep in mind that, in most countries, Cardiac pacing devices are generally implanted by cardiovascular surgeons.

Figure 4: Mortality and HF hospitalizations are higher in patients with LBBB-when LV ejection fraction is less than 50%.

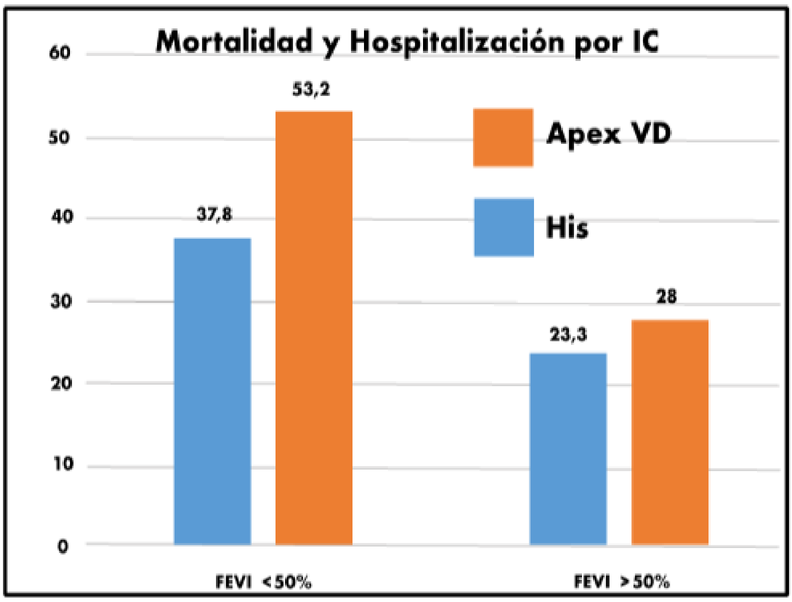

Figura 4: La mortalidad y hospitalizaciones por IC es mayor en pacientes con BCRI cuando la fracción de eyección del VI es menor de 50%.

On the other hand, the stimulation of the tissues of the His area (parahisian or His Area Pacing - HAP) and the left branch (Left Bundle Branch Area Pacing - LBBAP) were shown to be non-inferior to that of the non-selective His, and are gaining place progressively as more experience is acquired and technical resources improve (24, 25). The concept of physiological cardiac stimulation must always be considered before implanting a device, even in patients with a normal heart and a narrow QRS, because it is important to avoid the damage that we can generate in a patient without dyssynchrony, solving the electrical problem at the cost of a mechanical problem for the heart (cardiomyopathy induced by artificial dyssynchrony – MIDA).

5 FULCRUM AND TORSION OF THE HELICAL MYOCARDIUM. NEW PARADIGMS IN THE CONCEPTION OF THE ANATOMICAL-FUNCTIONAL PHYSIOLOGY OF VENTRICULAR CONTRACTION

The Danish anatomist Nicolaus Steno, in 1663, established for the first time the muscular nature of the heart ("Cor vero musculus est"). Since then, future generations of researchers strove to understand how the architectural structure of the ventricular myocardium explained the relationship between ventricular form and function. The overall arrangement of the ventricular muscle was 'easy to observe and difficult to understand'. The muscle fibers of the ventricular walls presented a pronounced global and local anisotropy, with electrical and mechanical properties that varied in time and were spatially inhomogeneous, constituting a true "Gordian knot" of the functional anatomy of the heart, which persisted for almost five centuries.

The Spanish doctor and scientist Dr. Francisco Torrent Guasp, through multiple dissections in human hearts and other species, demonstrated that the ventricular myocardium is made up of a set of muscle fibers twisted on themselves resembling a laterally flattened rope like a double-turned band, like a figure-eight helicoid (figure 5) (26). Torrent Guasp's theory of heart function is based on the grouping of the muscle fibers that make up the ventricular myocardium in a continuous band that is wound into a double helix. In this way, physiologically, the organization of the myocardium in a band with two helicoids greatly facilitates the understanding of cardiac mechanics and its effectiveness in generating the cardiac cycle: torsion – detorsion, pressure and ejection (27).

The Torrent Guasp Helical Ventricular Myocardial Band (BMVH) is a revolutionary concept in the understanding of the global three-dimensional functional architecture of the ventricular myocardium, and defines the integration between tissue form and function, where the ventricles are located both anatomically and functionally. well coupled, so that 'two hearts can beat as one'.

The Argentine heart surgeon Dr. Jorge Trainini continued and advanced the studies of this mechanistic and biophysical theory, and through deep, thorough and exhaustive research work, he managed to reinforce these concepts and expand them until achieving an understanding of the functional anatomy of the heart, conceiving revolutionary contributions, such as: Protodiastolic suction as an active process, the cardiac fulcrum as support for the contractile muscle and hyaluronic acid as a lubricant in the antifriction mechanism (28).

Figure 5: Location of the cardiac fibers in the continuous myocardium (chordal model). The right angle shows the three-dimensional arrangement of the continuous myocardium).

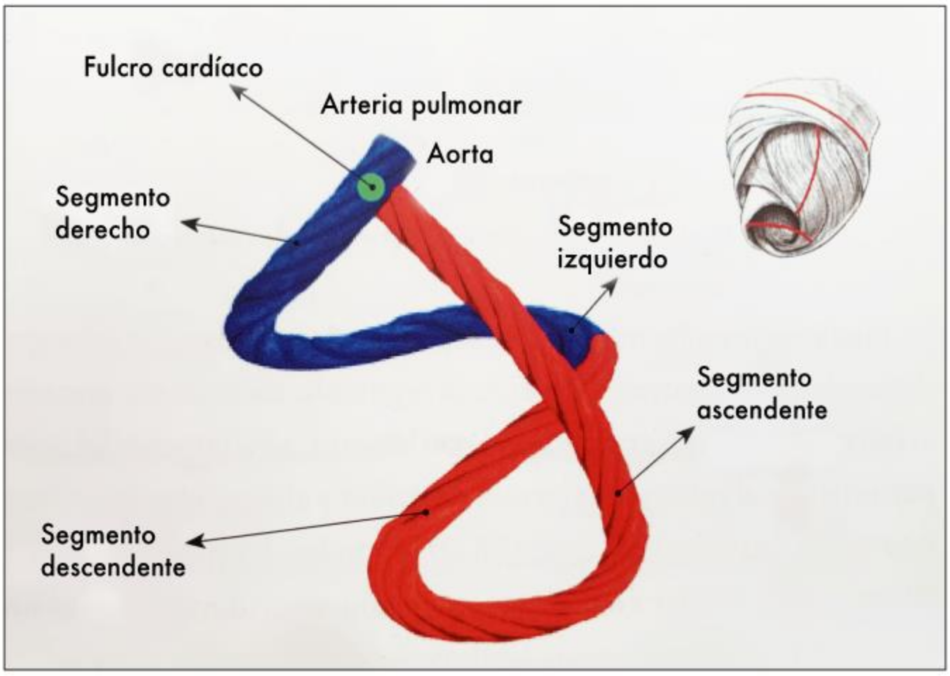

Figura 5: Ubicación del fulcro cardíaco en el miocardio continuo (modelo de la cuerda). En el ángulo derecho se muestra la disposición tridimensional del miocardio continuo).

5.1 DISSECTION METHOD AND PARTS THAT MAKE UP THE HELICAL MYOCARDIUM

The anatomical dissection is carried out with a standardized methodology, progressively advancing on defined muscular planes, with the purpose of unwinding the helical structure, which favors the successive and concatenated physiological movements of contraction and relaxation (torsion-detorsion), dependent on the propagation of the electrical stimulation through its muscular pathways. It is not the purpose of this chapter to make a detailed description of the dissection method; there are multiple publications that describe it, as well as videos on the internet and books published by Dr. Trainini (28).

Meanwhile, briefly and for educational purposes, the heart is previously prepared by boiling a mixture of water and acetic acid. The dissection maneuver carried out with the hands, without the need for any instruments, begins by applying pressure with the fingers on the anterior interventricular groove, through which it is possible to detach or separate the free wall of the RV, which is on the left side of the operator (figure 6A).

Next, the cardiac muscle is uncoiled through digital traction and pressure maneuvers that follow the direction of the muscle fibers, until it becomes a continuous band rolled up in a figure of eight, where two loops can be identified:

The Basal Loop has two segments: The Right Segment begins in the pulmonary artery including the outflow tract and the free wall of the right ventricle, contours on the outside of the orifice of the tricuspid valve, to continue behind with the Left Segment located in the wall. free of the left ventricle outside the orifice of the mitral valve.

Apexian Loop: It in turn has two parts, the Descending Segment in continuity with the left segment of the basal loop, descends obliquely, from the lateral wall of the VI towards the septum, and then retracts and rotates at the apex forward and above. From there, it takes the name of Ascending Segment, which runs in a more external position (subepicardial) and in a vertical direction, ascending in front and superimposed on the descending segment, to end below the base of the aorta in a structure in the that bind the fibers, the cardiac fulcrum.

5.2 CARDIAC FULCRUM

Todo músculo, por naturaleza, requiere un punto de apoyo o palanca para cumplir su función de contraction and relaxation, as evident in skeletal muscle. It is difficult to explain how, physiologically, the muscular segments that make up the ventricular cavities, in the thoracic cavity, free of all support, can develop a pressure of 120 mmHg in a few thousandths of a second and push 4 to 6 liters/minute of blood at a speed of 300 cm/s.

In his multiple dissections and investigations in human hearts and other species, Dr. Jorge Trainini discovered and described an osteo-chondroid-tendinous nucleus which he called Cardiac Fulcrum (29), describing its anatomical location and its histological and functional. This structure is located below and in front of the aortic root near the origin of the right coronary artery, in front and to the left of the tricuspid annulus and behind and to the right of the pulmonary valve (figure 6B). The cardiac fulcrum represents a meeting point where both ends of the helical myocardium are inserted "like ivy to a stone": Its initial end (the right segment of the basal loop) is tied to the anterior face of the fulcrum; Its final section (the ascending segment of the apex loop) is inserted into the fulcrum at the bottom and in a deeper plane than the initial end.

The fulcrum as an anatomical structure had been previously described in animals, especially in dissections of bovine, ovine and porcine hearts, identified as having a bony consistency and called "os cordis", however it was never assigned any function (30). Dr. Trainini's studies identified it in human hearts, confirming its existence not only in dissections, but also in computed tomography and nuclear magnetic resonance studies, and also defining its function as a fulcrum and support of the helical myocardium, explaining and resolving the physiological relationship between structure and function.

Figure 6: A) Dissection of a bovine heart. The Helicoidal Muscular Band can be observed unfolded (own experience). B) Image where the cardiac fulcrum and the different segments of the helical muscular band can be appreciated.

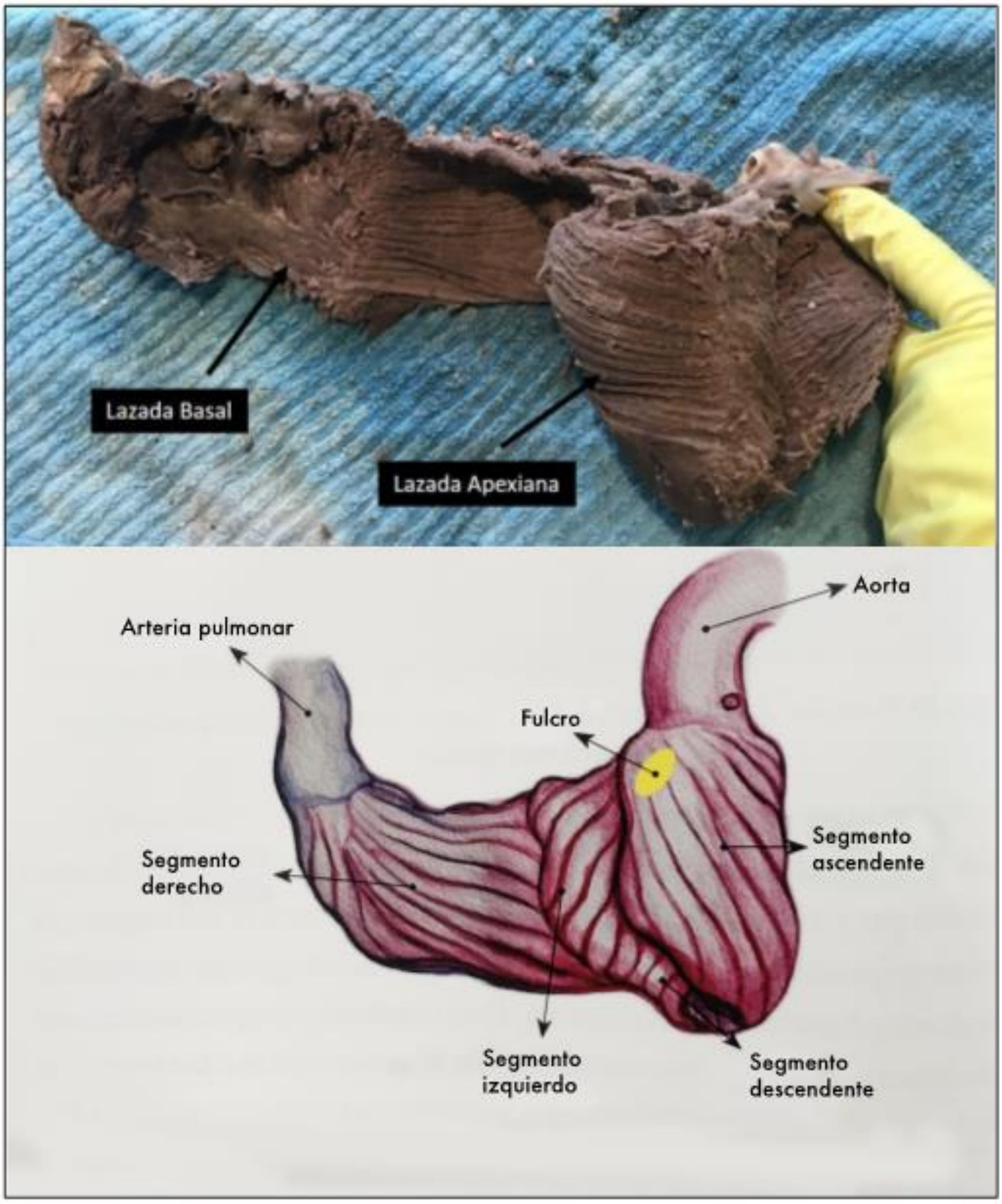

Figura 6: A) Disección de un corazón bovino. Se puede observar la Banda Muscular Helicoidal desplegada (experiencia propia). B) Imagen esquemática donde se puede apreciar el Fulcro Cardíaco y los diferentes segmentos de la banda muscular helicoidal.

5.3 CONTRACTION, TORSION-DETORSION, SUCTION PUMP FUNCTIONS (PROTODIASTOLE)

The heart is a pump that expels 70% of the left ventricular content with only 12% shortening in its contractile unit (the sarcomere), with great mechanical efficiency, that is, the work/energy ratio, superior to most of the pumps. machines (50% vs 30%). This function could not be justified with only the radial shortening during systole, described in classical physiology texts. The myocardial continuity in the form of a helix supported at both ends on the fulcrum allows us to explain a structure-function relationship. Through torsion-detorsion mechanisms, during contraction, the muscle mass is “squeezed like a towel” shortening in its three radii, longitudinal, circumferential and radial. The tip of the heart is fixed in its position, while the base descends like a piston, shortening the longitudinal diameter, while an opposite rotation movement of the muscle fibers occurs, clockwise in the apex part and in opposite direction at the base.

In the most initial phase of diastole (protodiastole), contrary to the traditional concept, there would not be isovolumetric relaxation, but rather an active suction phase. During the first 100 ms of diastole, due to the contraction of the vertical ascending segment of the apex loop, the longitudinal diameter of the LV increases, with the aortic and mitral valves closed, generating negative intraventricular pressure with energy expenditure (figure 7). This sudden lengthening of the base-apex distance of the left ventricle, after the ejection phase, produces a suction effect through an action similar to that of a "suction cup", configuring a dynamic in three stages (systole, suction and diastole) (28).

Figure 7: Diagram showing the phases of the cardiac cycle, with particular attention to the protodiastolic active suction phase, where there is widening of the LV longitudinal diameter with closed valves, with consequent drop in intraventricular pressure, which generates an aspiration mechanism.

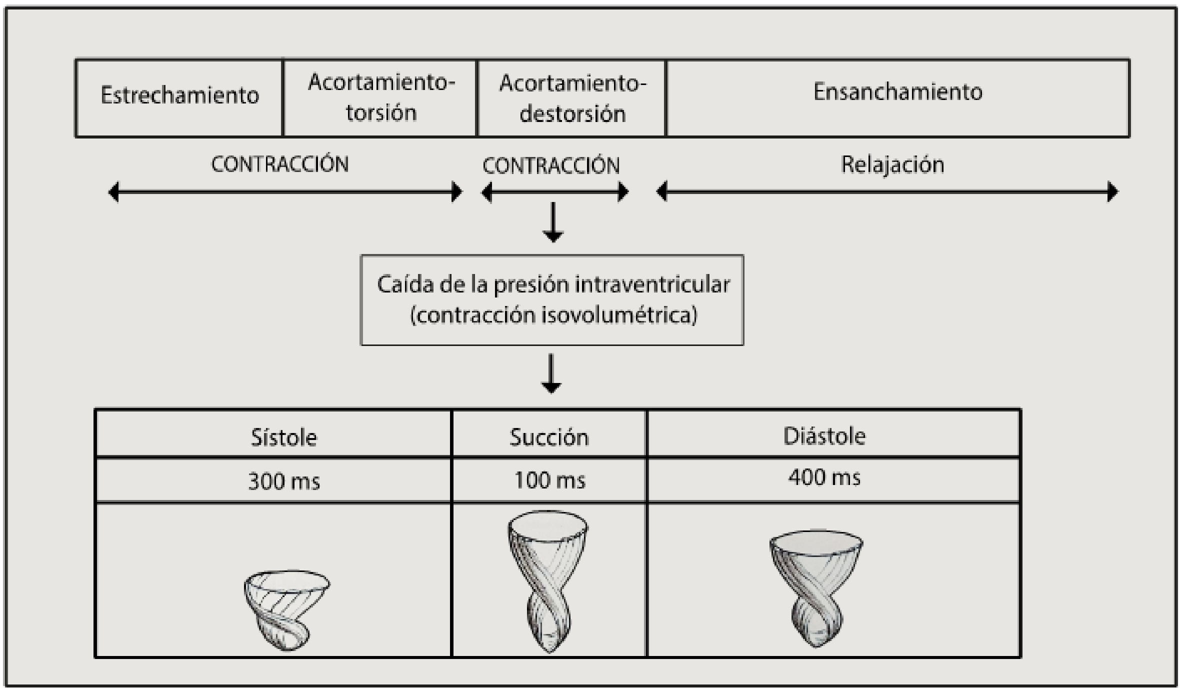

Figura 7: Esquema que muestra las fases del ciclo cardíaco, prestando especial atención a la fase de succión activa protodiastólica, donde hay alargamiento del diámetro longitudinal del VI con válvulas cerradas, con la consiguiente caída de la presión intraventricular, lo cual genera un mecanismo aspirativo.

5.4 FRICTION BETWEEN ASCENDING AND DESCENDING SEGMENTS, HYALURONIC ACID

During ventricular torsion-detorsion movements, sliding and friction in the opposite direction occurs between the descending and ascending segments of the apex loop, which are superimposed and perform an opposite movement during contraction, due to the direction of their fibers. . This implies that there should be an anti-friction mechanism that facilitates the sliding of the muscle bundles. Small venous conduits that originate from the intramyocardial veins (Thebesius) or the tributary veins of the greater coronary vein (Langer), cross the myocardium and drain into the cardiac chambers, and provide hyaluronic acid, which would function as "lubricating oil." organic (31).

5.5 ELECTRICAL ACTIVATION AND MECHANICAL FUNCTION

Using three-dimensional electro-anatomical mapping techniques with a 3D navigation system, it was evident that both the propagation of the electrical stimulus and the contraction of the myocardium begins in the RVOT region anatomically related to the cardiac fulcrum, advancing towards the basal loop, and to its time activating the muscle bundles of the descending and ascending segments of the apex loop, following the longitudinal direction of the muscle bundles towards the apex and finally ending in the ascending segment near the aortic annulus (figure 8). This electrical activation sequence is what allows and enables the torsion-detorsion mechanisms during myocardial contraction (32).

Figure 8: Schematic image of the helical myocardium represented by the asplaned chordae. The activation/contraction sequence is observed. In red the earliest and in blue the latest.

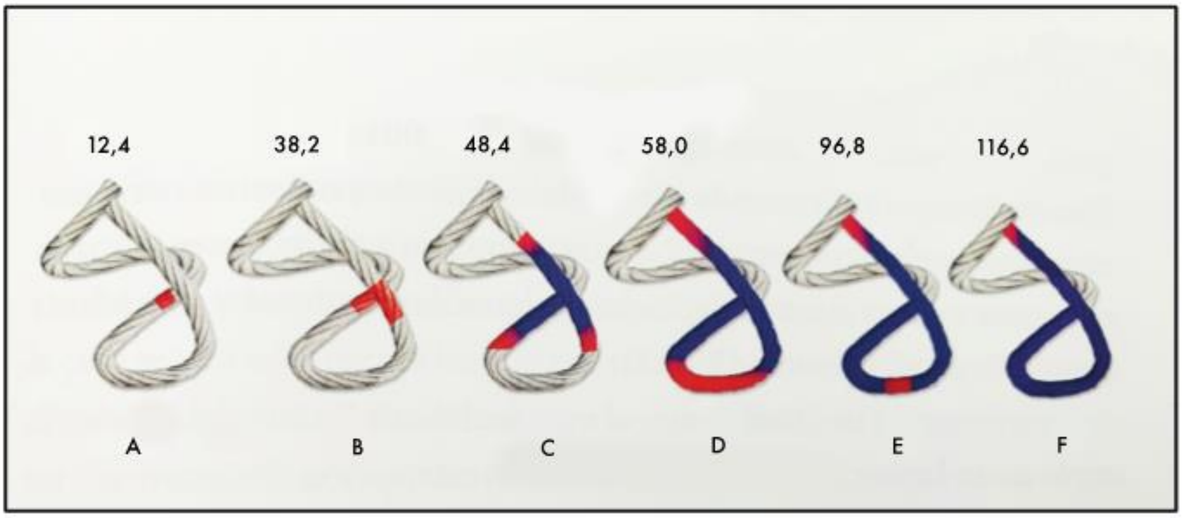

Figura 8: Imagen esquemática del miocardio helicoidal representado por la cuerda aplanada. Se observa la secuencia de activación/contracción. En rojo lo más precoz y en azul lo más tardío.

The direction and spatial sense of the contraction motivated by electrical activation from a physiological site and based on the architecture of the band itself, determine the effectiveness of the work (synchronous coordination) (27). Affecting the propagation of the electrical stimulus alters the contraction synchrony, and therefore the torsion-detorsion mechanisms, as can be observed in patients with LBBB and some with definitive stimulation in the RV apex. Studies are underway to analyze the hypothesis that heart failure with preserved ejection fraction could be caused by failures in the protodiastolic suction mechanism.

5.6 RESEARCH METHODS AND CLINICAL STUDIES ON HELICAL MYOCARDIUM

Images obtained with cardiac magnetic resonance by diffusion tensor (27), echocardiographic analysis with the speckle tracking technique, electrophysiological studies with three-dimensional electro-anatomical mapping techniques and laboratory investigations were procedures used in the extensive studies carried out by the Dr. Trainini's team and categorically support his claims. The dizzying changes in the advancement of cardiac imaging techniques will surely enable a better understanding of the three-dimensional myocardial structure and its function.

6 DEFINITIVE STIMULATION OF THE RVOT GUIDED BY ELECTRICAL SYNCHRONY IN REAL TIME

Physiological artificial cardiac stimulation (ACE) means and translates the capture of the patient's intrinsic conduction system to improve cardiac electromechanical activation. Prevents damage due to antiphysiological contraction of the “conventional” ACE (apical of the right ventricle or the purely septal muscle). In search of this “physiological” alternative, different electrode placement sites in the right ventricle have been evaluated. We highlight the stimulation of the bundle of His, the area of the left branch, or biventricular stimulation for cardiac resynchronization, strategies included in a generic term: Cardiac Physiologic Pacing – CPP (1) and with promising results in preserving and/or restoring synchrony. cardiac electromechanics. To achieve this, in any case, it is of fundamental importance to have a tool that allows us to evaluate the result in terms of synchrony, ideally in real time during the ventricular lead implantation procedure.

The Synchromax® (Sy) is a device developed by a group of Argentine researchers led by Dr. Daniel Ortega (33). This device is capable of non-invasively identifying electrical dyssynchrony through a spectral and averaged analysis of many QRS of leads DII and V6 as expressions of the activation of the septum and the lateral wall of the LV respectively. As a result, two curves are obtained, one blue (DII) and the other red (V6), which, when superimposed and compared, report the direction of activation (from base to tip or vice versa), simultaneity of activation, and delays in spread (wide QRS). In addition to the curves, a synchrony value is obtained that is classified as Synchronous (0-0.4), Intermediate (0.4-0.7) and Asynchronous (>0.7) (figure 9).

Figure 9: Synchromax curves and values.

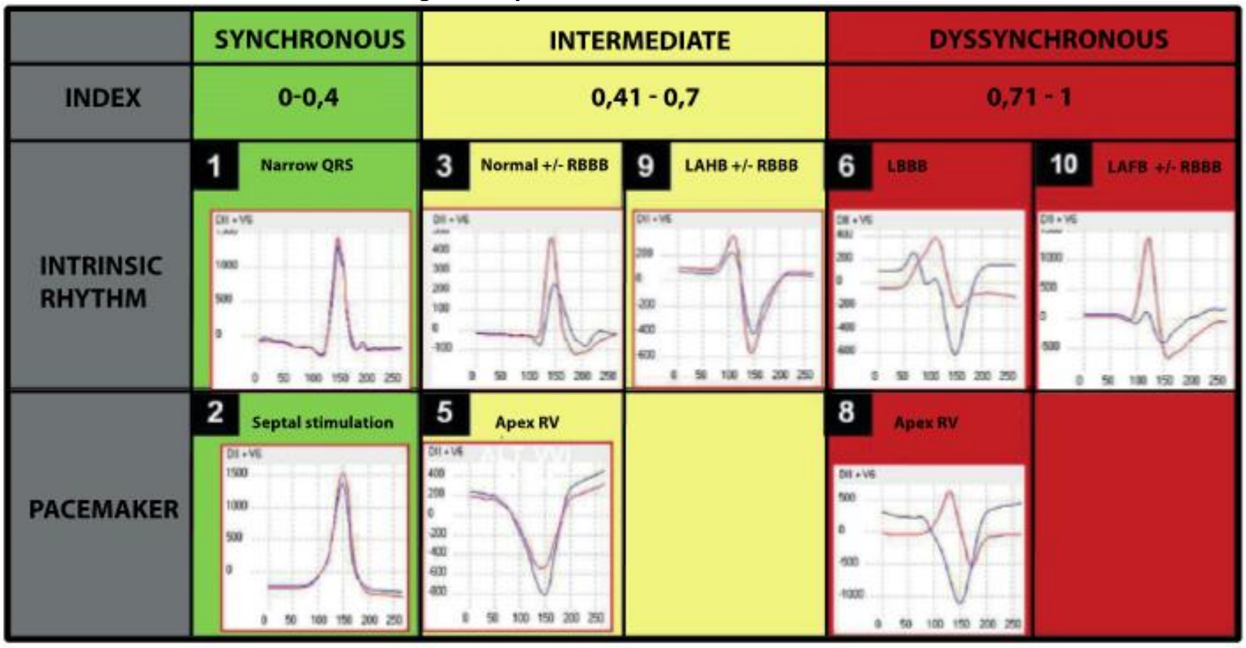

Figura 9: Curvas y valores del Synchromax.

By stimulating the septal aspect of the RVOT guided by Sy non-invasively and in real time, it is possible to obtain curves and adequate marker values of electrical synchrony. This could be explained by the coincidence of the site of activation by the artificial stimulus with the starting point of the electrical and mechanical activity described above in relation to the fulcrum and the helical myocardium.

7 OUR EXPERIENCE IN THE EVOLUTION OF CARDIAC STIMULATION

We evaluated at Cordis-Instituto del Corazón (city of Resistencia, Chaco, Argentina), 132 patients with bradyarrhythmias in whom pacemakers were implanted in the asp septal ecto of the RVOT (table 1). The majority were female (43%) and the average age was 62 years (44-81). In all cases, conventional active fixation catheters were used and real-time Sy was used during the procedure as a guide to define the appropriate implantation site of the stimulation catheter, looking for synchronous curves and indices (<0.4).

In the implants, curves and synchrony values were achieved, with adequate parameters at 129p (98%). The synchrony indices achieved were 0.20 ± 0.11 and the stimulation thresholds were 0.8 ± 0.7. No complications related to the procedures were observed. A follow-up period was carried out between 6 and 36 months, with no catheter displacements observed, the thresholds were stable and an upgrade of the PM stimulation site was not required in any case.

In conclusion, it is understood that the functional properties of myocardial fibers could be summarized in: a) their longitudinal contraction that gives rise to an increase in thickness perpendicular to the shortening process of the myocytes; b) the transmission of the electrical activation of one fiber to the neighboring ones following the length of the myocardial band, and c) the fibers of the specific conduction tissue transmit the stimuli more quickly, and as they are distributed throughout the length and width of the subendocardium , linked directly to the atrioventricular conduction system, allow the action – coordinated and efficient electromechanical function of the heart (cardiac synchrony).

Based on our experience, we highlight that the technique and strategy used allows us to simplify and standardize the implantation of definitive electrodes in the RVOT, being an effective, safe and reliable procedure. Probably this form of stimulation may be ideal to avoid asynchrony in pacemaker-dependent patients. The advent of Sy as a tool to non-invasively define synchrony in real time during implants may be promising for standardizing the ideal site for definitive artificial ventricular pacing.

Table 1: Demographic characteristics and results of the population studied in our center.

A) CARACTERÍSTICAS DEMOGRÁFICAS DE LA POBLACIÓN	
Edad	62 (48-81)
Sexo femenino	56 (43%)
Tipo de marcapasos:	
- Unicamerales	31 (23%)
- Bicamerales	101 (77%)
B) RESULTADOS	
Implante TSVD	129 (98%)
Indices de Sincronía	0,20 ± 0,11 (0,07-0,38)
Detección de R (mV)	3,2±5
Impedancia (Ohms)	536±90
Umbral de estimulación (V)	0,8±0,7(0,4-1,1)
Tiempo de radioscopía (min)	7 (5,2-21)
Ancho QRS post implante (ms)	132±5

Tabla 1: Características demográficas y resultados de la población estudiada en nuestro centro.

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