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Basis of the New Cardiac Mechanics

The Suction Pump

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Dedication To Francisco Torrent Guasp (1931-2005)

There is need to grasp the past. To retrace in its steps towards the bifurcations that determine the mesh of history. Precision fades with time and circumstances. It becomes uncertain and misses the crossroads. Progress is made over the incomprehension of oblivion, treading over those brown and crispy leaves that return to the anonymous dust in spinning crosswinds. Which are destroyed shedding the pollen that had constituted their purpose of "existence". The incomprehension of what occurred with the knowledge developed by Francisco Torrent Guasp is stirring. We arrive at the site of memory. Then, from the street that borders his house, we perceive the man who loved the stoicism of effort, the observation of nature and the skepticism in ephemeral, futile conquests. He did not desire success nor took refuge in inconsequential interests; he barely explored the imagined human existence. He became an artist able to paint realities in the dark, those that testify figures similar to the torn sound of a string, slashing the air to produce the cry.

Francisco loved the sublime of the infinite task. He launched in frenzied days of research, in opposition to the established dogma and to the disbelief distilled by the fear of change. Today life seems to soothe him with the achieved explanation of his original theory. In the face of the rigidity of paradigms, this idea defied the inviolability determined by history, in an attempt to undermine an unbeaten foundation, never defeated by the evolution of knowledge; only defied by time as the slave of his evolution, which in turn dominated him in an enduring monotony. Torrent Guasp realized that as long as there is movement there is time and that with the construction of his own historicity revision was possible, creating the coordinates to which man belongs.

And in this need for comprehension Torrent Guasp's chronic becomes similar to our daily experience until we fuse with his own prominence, that which sought the reality of things without being blinded by

the uncertainty of success, equipped with a self-criticism that prevented him from the celebrity settled in the temporary and the trivial. We are guided by the reflective glow emanating from his wisdom. We perceive him entering our consciousness from above, similar to a bird that observes the knowledge he generated, diluting the darkness.

The authors, 2015

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Foreword

When the authors of this original and splendid book asked me to write some introductory lines, I was greatly honored by their request and also felt a great pleasure to perform the task at hand, as I consider its contents crucial for the progress of knowledge on cardiac contraction and subsequent relaxation, and thus of ventricular mechanics.

I have been a cardiologist for over five decades and we have always considered the heart as a pump which is active during systole and passive during diastole, but when my brilliant colleague Dr. Jorge Trainini put me in touch with the ideas and work of Dr. Francisco Torrent Guasp, and of the recent contributions of those who wrote this book, I was captivated reflecting on them. I quickly realized that cardiac diastole plays an active phase in the heart's pumping function since the effectiveness of manual or external cardiac massage would be impossible, if a previous mechanism of blood withdrawal or suction from the ventricles were not present, to then prompt an acceptable blood ejection by wringing them. An isolated first pumping cannot possibly generate "vis a tergo".

This work, with the historical contributions of Dr. Torrent Guasp and the subsequent work of the authors, discusses in detail the anatomical structures that make up Torrent Guasp's ventricular myocardial band, its functional bases, electrical propagation, cardiac apex composition, electrophysiological and mechanical effects generated by muscle contraction and twisting, those that cause the ventricular suction effect during diastole (the authors' "plunger" mechanism).

The originality of this physiological concept is that the first phase of ventricular diastole (protodiastole), the isovolumic phase, has an active character due to the late myocardial contraction that produces left ventricular lengthening by separating the base from the apex (cardiac suction). This phase of ventricular mechanics becomes an intermediate phase followed by the real diastole (mesodiastole) which produces ventricular expansion and filling by decompressing the elastic elements of the heart, completing the cycle with ventricular presystolic atrial contraction and the subsequent ventricular systole. All this establishes the three-stage heart mechanics described in the text.

Faced with these facts there arises for the future a thorough study and analysis that will bring a wealth of knowledge to the prognosis and solutions for the treatment of heart failure and ventricular dysfunction, perspectives well discussed in this book.

Dr. Torrent Guasp's work is stimulating, and the deep study on the furtherance of the research performed by the authors of this book allows me to say without doubt that it is a great contribution to the better understanding of cardiac mechanics and future management of heart failure.

Pedro Ramón Cossio

Preface of the authors

Any knowledge is a sequence of the previous, but it is not excerpted of being considered a turmoil. It happens when the bases that are intended to be reformed are very anchored in the time and the praxis. The history of the circulation of the blood evolved through three fundamental jumps. The initial one, through Claudius Galeno (second century A.D.) that persisted until 1628, when William Harvey published his book *"Exercitatio anatomica de motu cordis et sanguinis in animalibus"*, establishing the modern physiology of circulation. This movement of the blood lacked the description of the pathway for returning of the blood to the heart. The appearance of the microscope allowed Marcelo Malpighi in 1661 to close he breach between arteries and veins with the discovery of the capillary vessels. His writing *"De pulmonibus observations anatomicae"*, that appeared four years after Harvey´s death, shows excellent pictures, taking as an investigation model the lungs of the frog.

The first visual demonstration of the circulation of the blood corresponded to the cleric Lazarus Spallanzini in 1771, as he observed in a chicken embryo the blood circulating from the arteries to the veins. His relation describes the event with these words*: "The room where I was didn´t have enough light, and willing anyway to satisfy my need, I decided to examine the egg under the direct light of the sun. Once placed the egg in Lyonnet device, I directed the lens, and, in spite of the great clarity that surrounded it, I was able, by sharpening my sight, to observe the blood running through the complete circuit of umbilical vessels, arterial and venous. Overwhelmed then by an unexpected happiness I exclaimed eureka! Eureka!"*.

In spite of the righteous of this idea, it was not simple Harvey´s discovery to supplant the fourteen centuries of the hegemony imposed by Galeno´s system. The blood returned, it did not remained in the perifery as thouth by the Galeno. In relation to cardiac movement, through his observations Harvey explains the function of this organ in two phases: systole and diastole, considering the later one as a simple cardiac dilatation. With this position, he opposed to Galeno´s doctrine, who believed

that the activity of the heart was manifested by the dilation, through the *"vis pulsifica"*. By 1980 Francisco Torrent Guasp originates fissures in the understanding of the dominant cardiac mechanics. He conceives an anatomic explanation adapted to the real facts of its function, which will advance in the theoretical understanding of its mechanics, in works together with Gerard Buckberg (2001).

The investigation in human beings in relation to the electrophysiological aspects of the heart that we expose in this text modifies the conception of the cardiac function. It allows to understand the anatomical-functional unity, the propagation of the electric impulse through the myocardium not visualized until now and to sustain the concept of a three phased heart.

Cardiac electrical activation is a consequence of the propagation of the stimuli through the muscular structure of the heart, both of Torrent Guasp´s myocardial band and the rest of the fibers that constitute its structure. The cardiac mechanism of *aspiration* and *expulsion* requires an integration of the structure/function that takes into account several mechanisms of its dynamic due to the propagation of the excitation. This implies that the diastolic isovolumetric phase is an active process of contraction with a growing aspiration action that we called by its similarity as a plunger mechanism, which at a certain level of intraventricular pressure level produces the opening of atrioventricular valves, originating the rapid ventricular filling.

In our experience, we found a pathway of spreading of the electric impulse different from the one described by Torrent Guasp that explains the phase of torsion of the heart, defined as a rotation movement opposed of the base and the apex.

This activation conceives, between the systole and the diastole, a third phase of active coupling of suction, with muscular contraction, energetic spending and remarkable fall in the intraventricular pressure, defining a three phase heart.

The basically anatomic model of Torrent Guasp, amplified and modified by our investigation from an electromechanically point of view, offers countless possibilities of development both at a theoretical level as well as from tis clinical a therapeutic application. It is probable that a great part of the cardiology should be reexamined in light of this new paradigm, with potential unpredictable results.

Buenos Aires, 2015

Index

Intertext 21
Genesis of the idea. Torrent Guasp's "key doubt" 21

Chapter I. Torrent Guasp´s Ventricular Myocardial Band In Ventricular Anatomy 27
1. Ventricular myocardial band anatomy 27
2. Phylogenetic development of the circulatory system 35
3. The cardiac apex 37

Chapter II. Electrophysiological Interpretation Of The Ventricular Myocardial Band 39
1. Historical concepts on myocardial electrical activation 39
2. Stimuli propagation, muscle torsion and cardiac suction effect through electrophysiological research . . . 40
3. Electrocardiographic correlation 56
4. Functional aspects of the ventricular myocardial band 58

Chapter III. The Three Stage Heart 63
1. Chronology of the suction mechanism concept 63
2. Cardiac mechanics 64
3. How is diastolic suction produced? 70
4. Structural basis of diastolic left ventricular limitation in the suction mechanism 73
5. Echocardiographic concepts 77

Chapter IV. Clinical, Surgical And Electrophysiological Perspectives Derived From This Research 81
1. Extent of heart failure 81
2. Clinical perspectives 83
3. Surgical perspectives 84
4. Electrophysiological therapy perspectives 90

References 95

The heart has the gift of oblivion. The grace that allows each dawn to restart the utopia of remaining in force. Despite memory and boredom. Of the personal history that reflects in its own mirror the circular flow of blood and destinies.

Jorge Carlos Trainini

"Heart mechanics is homologous to that of the circular fibers of blood vessels, which carry out their function without fixed fulcrums"

***Francisco Torrent Guasp* (68)**

Intertext

Genesis of the idea. Torrent Guasp's "key doubt"

> *"Francisco [Torrent Guasp] never believed that blood could enter the left ventricle other than by a suction device"*
>
> Juan Cosín-Aguilar
> (personal interview, Valencia, June 2010)

The disciple slightly shrugged his shoulders recalling the question Francisco Torrent Guasp asked himself. -*You know*…- he said, facing us with his light-colored eyes while slowly stirring the coffee: -… *Paco's thesis began with a key doubt.*

Juan Cosín-Aguilar, the speaker and faithful friend of the person we were trying to unravel held upon us a remarkable fascination in that warm night of Valencia. No one had been so close to the master's "doubt" in those essential years when different hypotheses on heart functioning were being developed.

-*What was the doubt*- we asked in unison, impertinently driven by the force of our anxiety.

An external uproar filtered through all the cracks in the tavern, ornamented with red and yellow headscarves on that festive day. Even the most frivolous had been caught by the merriment. At a moment when the excitement took a break he began outlining the answer stored in the memory.

- *"Man,* [said Paco] *blood does not return to the heart vis a tergo or by a peripheral pressure difference with the heart. This is small. The gradient that returns it is ventricular suction".* To this statement I retorted with another: *"So the heart is a suction pump?"* At that point his shyness turned into a candle. He ignited. *"Look Juan, all dissections made in animals and humans*

clearly explain this possibility. The heart is formed by a large muscle band that begins at the insertion of the pulmonary artery and ends at the level of the aorta, forming a double helical structure which limits the ventricles. The two ventricular chambers wrapped by the large muscle band are the left chamber with ellipsoidal shape and the right chamber of a semilunar structure. Well, the contraction of this band not only explains heart's systole but also blood suction".(72)

After a pause owing to the outside din and with his undisguised sensitive condition of a Spanish native he proceeded. -'I sometimes became the devil's advocate. "*Paco, to allow the bands surrounding the ventricles to contract, they should need a rigid support just as a tendon uses the bone insertion as lever Are there any in the heart?"* In those moments he would make a quiet patient pause and from his working place in the attic would pensively gaze for a while at the long alley of his house in Denia, his adoptive village in Alicante, which appeared and disappeared at every turn of its curves. *"You do not need that support. When the heart fills with blood it behaves as a bone insertion. The large muscle band is a double helix suspended between the pulmonary artery and the aorta using the hemoskeleton i.e. cardiac filling as its fulcrum. At this point the left ventricle turns counter-clockwise and the right ventricle turns clockwise. Exactly like wringing out a towel".*(68, 87)

"Free, master of his time and his ideas"

Jesús Herreros, Eduardo Otero Coto, Salvador Mercé and his father Mercé Vives, the other participants of the meeting, must have looked astonished as Jorge Trainini lashed in the memory of the disciple trying to exhume the magic that the figure of Francisco Torrent Guasp stirred upon him.(82)

-Cosín … Torrent must have been a special man! I picture him working in anonymity, far from the media noise which often appears with the advances in our profession. What was hisinner-self?

-Paco was a free man, master of his time and his ideas.(19) To know him you had tolet go of prejudices. He lacked the need to enter a career of honors.

-If you are free you'll die alone- managed to interrupt Trainini.

-Something of that emerged in his recognition. He was cultivated, lucid, curious, imaginative, spontaneous, nonconformist, rebellious, enthusiastic, committed.(19) Since 1954, while still a student, he devoted himself to the physio- structural study of cardiac mechanics. He was born to change these

things of the heart. I think the rest of his life was an addition to that passion. He was an anonymous and colossal worker. He used more than a thousand bovine, horse, dog, pig, sheep, cat, hen, turtle, fish and obviously human hearts.

-He reminds me of Galen. The pergamum dissected all kinds of animals, even an elephant. However, since autopsies were forbidden in Rome [second century A.D.] he could only do so in a corpse found floating after a flood had swept his tomb.(79)

-He had an artistic mind. He liked to paint and even exhibited his paintings in Paris. Perhaps this explains his way of being. He had ideas and action. He lacked the dialectic.

-I picture him with the characteristics of a rebel. Dialectic, essential to be recognized is not usually well valued by men who are pioneers.

The interviewee agreed, nodding with a slight tilt of his head. Then he added, almost resignedly: *-especially with today's computing progress the space you do not occupy is quickly usurped.*

Trainini reiterated the words of the disciple *-Juan, I have always believed that personality has a lot to do with success. Extreme self-criticism conspires against it. I have the sensitivity to understand that Torrent Guasp's almost silent work still needs a posthumous tribute.*

-But in the last years he received some honors and acknowledgements. His idea of the ventricular myocardial band still requires certain explorations in other fields such as electrophysiology.

-Incidentally, concerning this multidisciplinary approach that you confer to it, did Torrent explore into microscopy?- Now everyone asked in unison resembling a circumstantial choir.

-At first he worked on it but quitted without clear results.

-However, the helical physio-structure he proposes would have correspondence with the microscopic universe known today. Sarcomeres are bundled by collagen fibers in the shape of double helical bands whose function would be limiting expansion and setting the mechanical recoil, in addition to storing the restoring force of energy for relaxation- explained Trainini with a parsimony not devoid of passion. The evening continued further on with other conversations, but Trainini would return to the subject… outside the joy persisted without shapes or boundaries.

Denia

… –Cosín, perhaps this invitation for a conference in the field of electrophysiology that Torrent desired so much has been yet another scorn of fate in its insult to men?

One could feel in the air that everyone remained stationed in their own impressions. Time-detained silence waited for the affectionate reminiscence of the disciple. They all directed their eyes towards Cosín. The disciple relaxed slightly allowing time for the word not to become an emotional fraud. The glasses and coffee cups seemed suspended in the air waiting for a decision to break the spell. He coughed softly to clear his knotted voice. Perhaps to divert some tears.

-Paco was happy with the invitation to the electrophysiology meeting. He had always longed to talk with cardiologists to explain that the contraction of the heart began in the right ventricular outflow tract and ended at the left apex. According to him the "cardiac piston" worked in that way. So he went to Madrid despite having been for weeks in a wheelchair. His lecture was bold, characteristic of a leader, exultant. He was ebullient with the reception he had been offered. It was the 25th of February 2005. He died just after his last conference.

We walked away from the meeting at midnight. The last celebrations were fading in the streets of a bright city. Golden-walled buildings seemed mirrors in a maze we were trying to solve thinking of that village, of that attic, of those projects developed by Francisco Torrent Guasp, indifferent and detached from the clamor that usually blesses the medical community. We would arrive to his home in Denia, even if only for a silent exercise of admiration.

So we did the next day. Denia was born Roman, was then Arabic, but definitely remained Spanish. An Alicant village, at a very short distance, it runs behind a rock against another gem, Javea, belonging to the Valencian community. Located in the last rugged steps of the appeased mountains in the jagged coastline of the Mediterranean, it stands between uneven streets and white houses that seem to lean against each other. Its past shows the offering of each conqueror. Its figure outlined by an infinite light is drawn in the rolling emerald waters that conferred it glory and destiny. We meandered the street where Torrent Guasp had lived. We imagined that from his working attic a watchful eye was still peering to where the future of silent men reached. One of us remembered François Jacob [Nobel Prize in Medicine, 1965] who always repeated that *"humility does not suit the wise or the ideas he has to defend"*.(32) In our return the sun spilled fully without casting any shadow. It had stationed at the exact point where emotion meets absence.

Buenos Aires

When Jorge Trainini returned to Buenos Aires he carried in his memory the words of Juan Cosín in relation to Torrent Guasp's hypothesis: *"His idea of a myocardial band still needs certain explorations in other fields such as electrophysiology"*. In Buenos Aires, he met with Benjamin Elencwajg, Nestor López Cabanillas and Noemí Lago and together with Jesús Herreros in Spain they formed a working group. They articulated a human research project that did not infringe the ethics. The literature was meticulously examined. Several proposals were considered for electrophysiological testing. The recent advent of navigation systems and three-dimensional endocardial and epicardial mapping (CARTO, Biosense Webster, California, USA) provided the ideal instrument. Then the steps were accomplished in a continuous and tenacious research. The first steps led to others in the required paradigm of science. The point reached and presented in the text opens a perspective for the physiological understanding of the heart and its resulting clinical, surgical and electrophysiological implications. Future research will help to improve all the aspects developed so far.

The authors, Buenos Aires, 2015

Chapter I

Torrent Guasp´s Ventricular Myocardial Band In Heart Anatomy

1. Ventricular myocardial band anatomy

The fibers forming the cardiac muscle cannot be considered as separate entities contained in a defined space; however, independently of the intricate polygonal fiber bundles, which also receive and give off collaterals, a main trajectory of central fibers is defined forming the ventricular myocardial band (Figure 1) described by Torrent Guasp.(68) It is then necessary to establish the concept of linear and laminar trajectories. Essentially, myocardial muscle bands and bundles constitute an indispensable dynamic master axis, derived from phylogenetic evolution allowing the passage of fishes to amphibians and then to birds and mammals. The muscle structure forming the ventricular myocardial band has a dual function: a) to define the boundaries of ventricular chambers and b) to perform the ejection and suction actions as cardiac pump. From an evolutionary point of view this organization of the myocardial mass was a necessary change for aerial life (Figures 1 and 2).

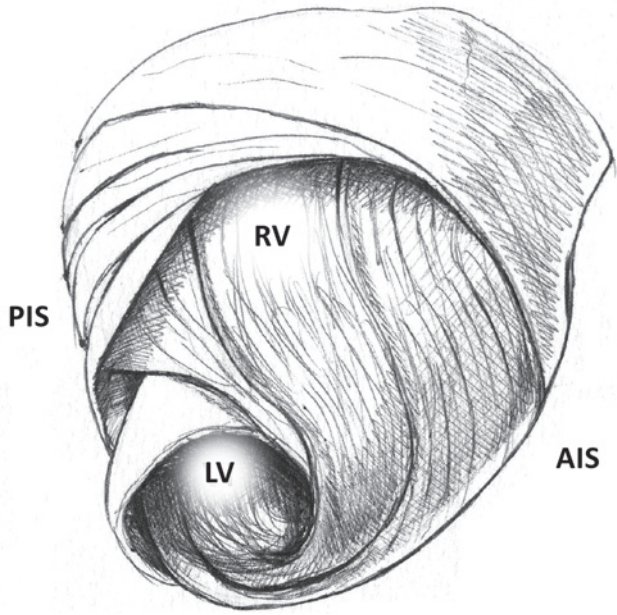

Figure 1. Torrent Guasp´s ventricular myocardial band. **RV:** Right ventricle; **LV:** Left ventricle; **PIS:** Posterior interventricular sulcus; **AIS:** Anterior interventricular sulcus.

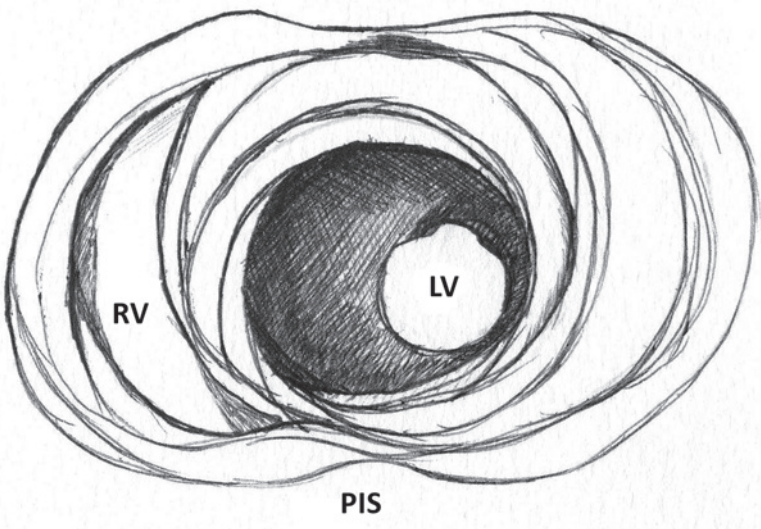

Figure 2. Apical view of the left and right ventricles. **RV:** Right ventricle; **LV:** Left ventricle; **PIS:** Posterior interventricular sulcus.

Left ventricle. The entire apex belongs to the left ventricle. In its distal part, called **apical**, a muscle layer with spiral trajectory extends from the surface to the center, undergoing a rotation that turns subepicardial into subendocardial fibers, overlapped like the tiles of a roof. Consequently, the left ventricular distal end, the apex, surrounds a virtual tube with no muscular plane, lined at its two ends by the endocardium and epicardium. It is essential to consider that in the apex the fibers undergo a helical spinning movement with sphincter-like arrangement as they transform from subepicardial to subendocardial fibers, following a clockwise trajectory (apical view of the diaphragmatic surface of the heart in anatomical position) (Figure 3).(66)

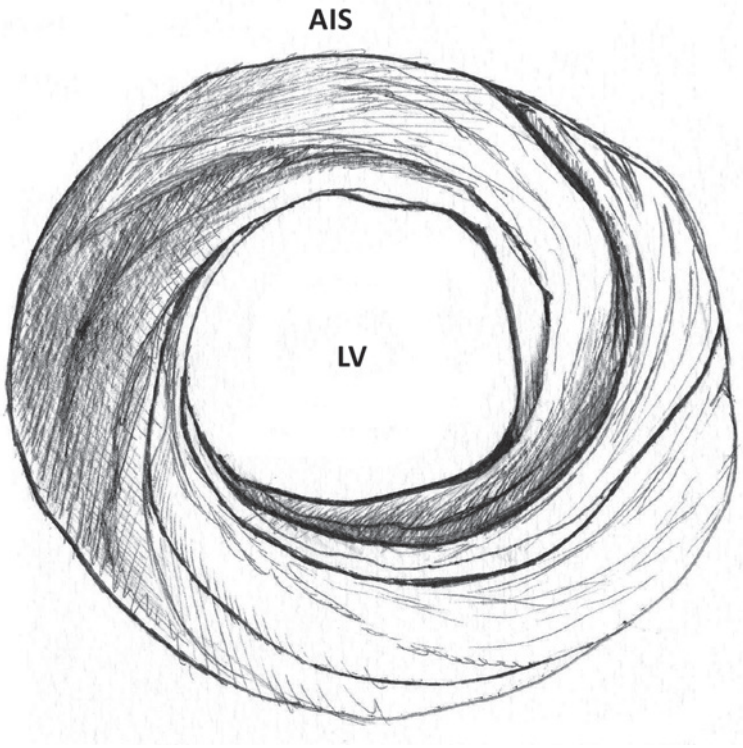

Figure 3. Spiral arrangement of apical muscle layers. **AIS:** Anterior interventricular sulcus; **LV:** Left ventricle.

In the left ventricular **basal** half, at the level of its free wall (Figure 4), the fibers are ordered similarly to the apical half. A muscle layer with spiral trajectory extends from the surface to the center arranging paraepicardial and paraendocardial regions from the outside to the inside. At this level their orientation is opposite to that of the apex, following a counter-clockwise trajectory (apical view of the diaphragmatic surface of the heart in anatomical position). This arrangement of the spiraling muscle layer limits a cavity which at the base of the heart is real and not virtual as in the apex.

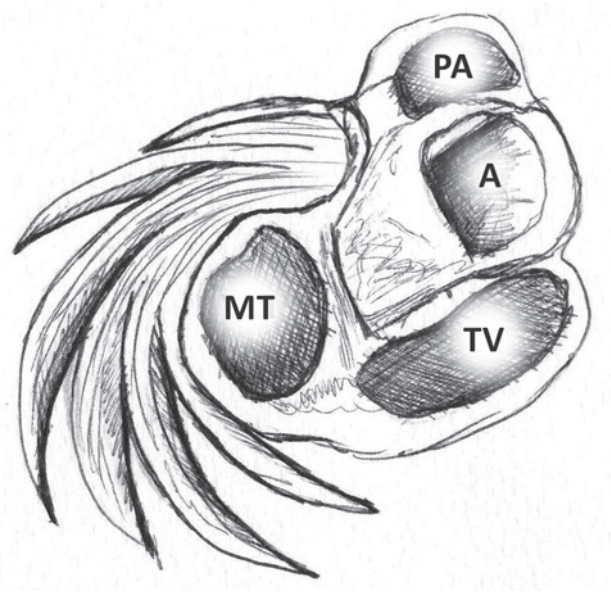

Figure 4. Basal third of the left ventricle, showing the free wall muscle layers. **PA:** Pulmonary artery; **A:** Aorta; **TV:** Tricuspid valve; **MT:** Mitral valve.

The apex should be considered as a tunnel with a rim in its entire ring, while at the ventricular base this ring has two parts. One part corresponds to the left ventricular free wall and the other to the interventricular septum. In addition, the superficial basal fibers make contact with the fibrous mitral annulus, absent at the apical level. However, the essential functional difference between basal and apical segments is the opposite trajectory of their fibers. This characteristic determines muscle torsion to achieve blood flow.

Right ventricle. According to their orientation, two types of fibers can be identified in its **distal** half: paraendocardial and paraepicardial

fibers. The former extend from the pulmonary root backwards and downwards to the ventricular tip, whereas the others go from the anterior interventricular sulcus towards the back near the base of the heart. This X cross layout allows the fibers in the distal end of the right ventricle to adopt a helical arrangement, turning from subepicardial to subendocardial fibers (Figure 5).

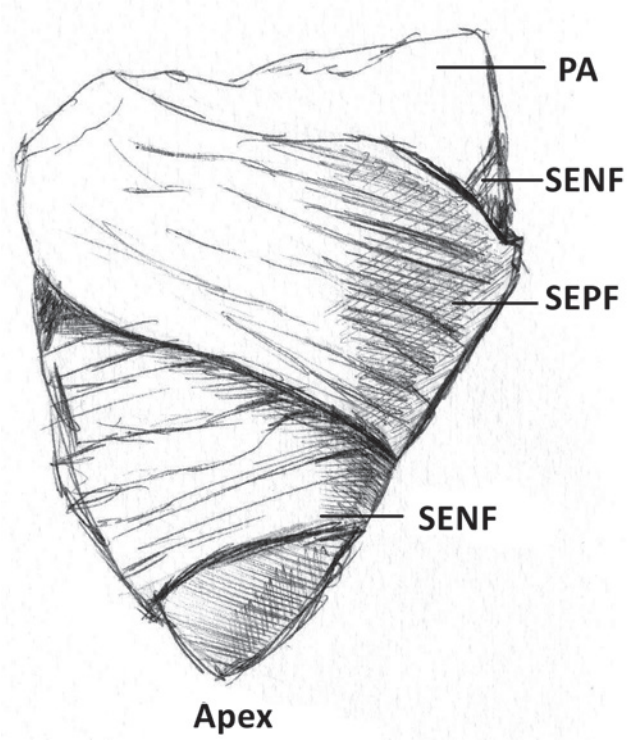

Figure 5. Right ventricular free wall. **PA:** Pulmonary artery; **SENF:** Subendocardial fibers; **SEPF:** Subepicardial fibers.

Three segments can be identified at the **basal** half of the right ventricle (tricuspid orifice perimeter): free wall, supraventricular crest and interventricular septum. The free wall presents the same general configuration of spiraling fibers that go from subepicardial to subendocardial positions. Similarly to the left ventricle, there is a difference in the rotating sense of the basal fibers with respect to the distal ones. They follow a counterclockwise trajectory at the basal end and a clockwise trajectory at the distal end (apical view of the diaphragmatic surface of the heart in anatomical position).

Interpretation. It can be seen that the spatial configuration and rotating movement of the fibers at the basal and distal levels of both ventricles correspond to Torrent Guasp's ventricular myocardial band. The author considered that the ventricular myocardium is formed by the assembly of muscle fibers twisted like a rope (*rope theory*) (Figure 6), flattened laterally as a band, which presents two spiral turns defining a helical structure that limits the two ventricles and defines their function.(70)

A classical interpretation of blood circulation through the different cardiac chambers has been made bearing no correlation with their muscle dynamics. However, this dynamic behavior is essentially the circulatory motor established by the muscle mass, which also defines the chamber boundaries through which blood flows. This arrangement of the ventricular myocardial band confers ventricular chambers a leading role in cardiac function.

Cardiac muscle lacks fixed points of attachment as those of the skeletal system to develop force. In this sense, the ventricular myocardial band would act as the circular muscle of the arteries, supporting itself in its own cavity content (hemoskeleton). Moreover, attachment to the origin of the great vessels could also be considered an insertion point to drive the mechanics of the ventricular myocardial band.

Segmentation of the ventricular myocardial band. Ventricular chambers are defined by Torrent Guasps´s ventricular myocardial band, which describes two spiral turns with one end inserted in the pulmonary artery and the other in the aortic root. In its trajectory it adopts a helical configuration forming the two ventricular chambers. The figure in 8 defined by its course outlines two loops: a basal and an apical loop. The basal loop extends from the root of the pulmonary artery to the central twist of the band. On the other hand, the apical loop courses from this twist to the aortic root. Moreover, each loop is formed by two segments. The basal loop consists of the right and left basal segments and the apical loop by the descending and ascending apical segments (Figure 6). In the general configuration, the basal loop embraces the apical loop, so that the right ventricular chamber is more like an open slit in the muscle mass thickness forming both ventricles (Figure 1). The segments are defined by anatomical structures.

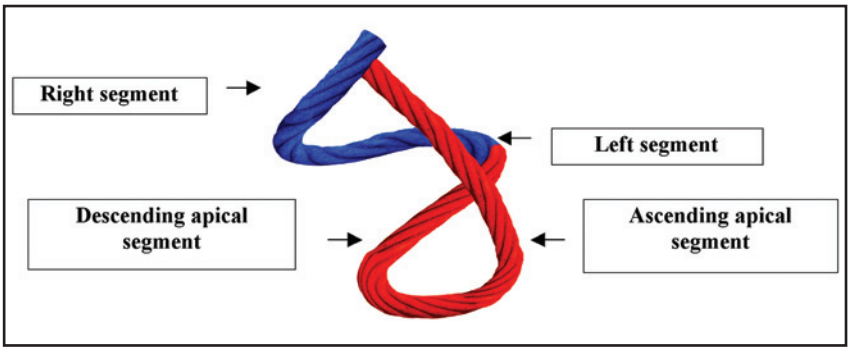

Figure 6. Torrent Guasp´s ventricular myocardial band showing its different constitutive segments. Basal loop (blue). Apical loop (red).

Basal loop. The posterior interventricular sulcus presents a trough that determines the limit between the right and left basal loop segments. The right segment constitutes the right ventricular free wall and surrounds the tricuspid valve orifice externally. The left segment situated in the left ventricular free wall externally defines the mitral valve orifice.

Apical loop. The descending apical segment extends from the twist of the band to the apex, where it becomes the ascending apical segment to end at the aortic root. Both segments mainly form the interventricular septum, and are separated by the anterior papillary muscle.

These concepts indicate that the right ventricular free wall is formed by one loop (basal) and the left ventricular free wall by both loops (basal and apical).

Interband fibers. When the ascending apical segment reaches the anterior interventricular sulcus, some fibers instead of continuing their intraseptal course towards the end of the aortic root band, coat the right ventricular free wall and reach the anterior left ventricular surface after crossing the posterior interventricular sulcus. They are inserted along the whole extension of the ventricular base comprising the pulmonary, tricuspid and mitro-aortic valve annuli. These muscle bundles were called "*aberrant fibers*" by Torrest Guasp, who pointed out: "*Owing to their spatial arrangement they surround the basal loop in all its extension, thus enveloping both ventricles*".(70) Torrent Guasp believed that the function of these fibers was to separate the right and left ventricular walls to expand both chambers. He ended his report expressing with certain skepticism: "*This expansion, due to its relatively low magnitude, has less importance than was normally assigned*".(70)

In the light of our findings on the trajectory of cardiac stimuli and the anatomical size of the fibers between the two ventricles, our interpretation (Chapter II) is that they fit perfectly well in the direction of radial impulse transmission which courses from the descending to the ascending band, so that the heart can fulfill its twisting, untwisting and suction mechanical function. Therefore, we decided to name them **interband fibers** (Figure 7).

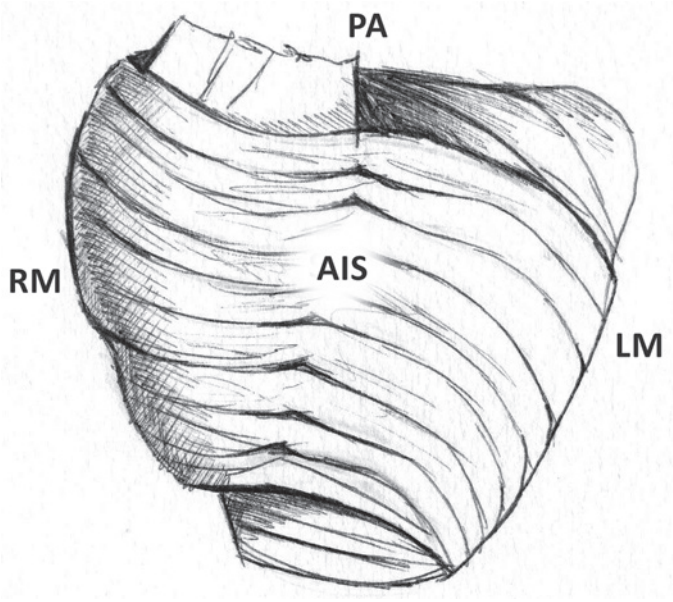

Figure 7. Interband fibers. **PA:** Pulmonary artery; **RM**: Right margin; **LM:** Left margin; **AIS:** Anterior interventricular sulcus.

Origin and end of the ventricular myocardial band. Embriological studies to elucidate the origin and end of the ventricular myocardial band in bovine hearts could not find a solution. Phylogenetic studies filled this gap across 600 million years of circulatory system evolution, arriving to the conclusion that these ends are located at the root of the pulmonary artery and the aorta.

The musculature forming the right ventricle corresponds to the origin of Torrent Guasp's ventricular myocardial band which originates in the pulmonary artery annulus and its related structures with the tricuspid annulus (pulmonary-tricuspid cord and tricuspid annulus). Regarding the autochthonous muscle bundles forming the left ventricle, they end at the root of the aorta and in the right and left trigones

(constituting the aortic annulus) (Figure 8). This arrangement allowed Torrent Guasp to deduce that the aorta, at its root, represents the end of the heart's ventricular myocardial band.(69)

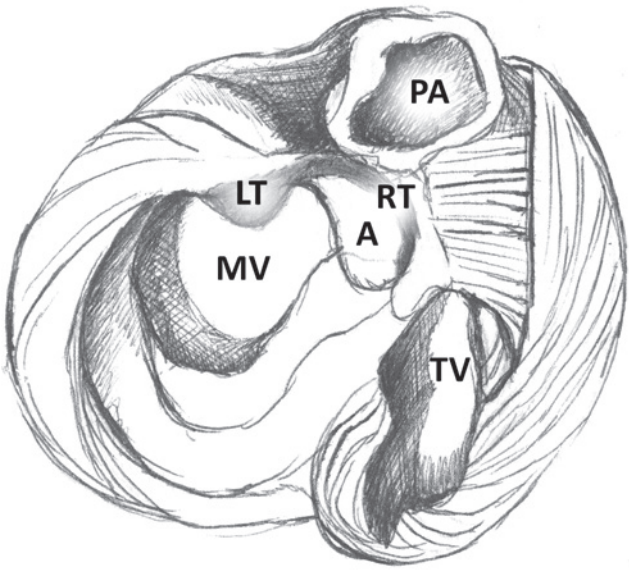

Figure 8. Septal portion of the aortic annulus between the right and left trigone. **PA:** Pulmonary artery; **LT:** Left trigone; **RT:** Right trigone; **A:** Aorta; **MT:** Mitral valve; **TV:** Tricuspid valve.

2. Phylogenetic development of the circulatory system

The circulatory apparatus of worms (Annelida, Nemerthea) consists of a closed system with two capillary beds (respiratory and systemic) with semicircular arterial and venous segments. In this unique circulatory system blood is pumped by peristalsis (expression and suction) as it lacks a heart impulse (Figure 9 A).

In the evolution to fishes three linear dilatations appear in the venous semicircle (venous sinus, atrium and ventricle) forming a primitive heart. Although it is still a single circuit a pumping organ was inserted increasing the possibility of intravascular pressure transmission (Figure 9 B).

In the following evolutionary stage of amphibians and reptiles more prominent modifications are produced. At this stage of evolutionary development two circuits are identified: the systemic and respiratory circuits, with the heart presenting two atria and one ventricle, this last generated by the incipient twist of the arterial circuit.(74) This self-rotation in a segment of the arterial semicircle (systemic circuit) is a crucial step in the evolutionary development of species (Figure 9 C). This rotation forms the future ventricular chambers in their definitive configuration, a fact well understood when the ventricular myocardial band forming the ventricles is unfolded.

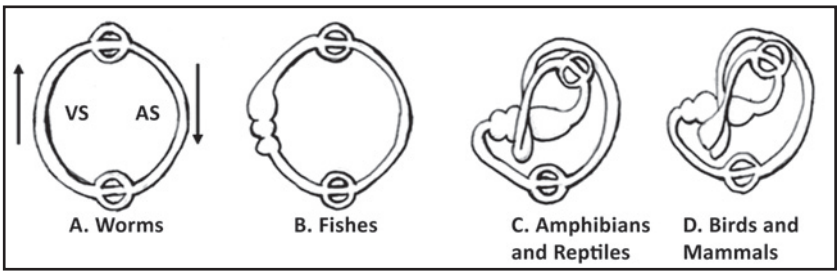

Figure 9. Phylogenetic development of the circulatory system. VS: venous segment; **AS:** arterial segment. Arrows indicate the direction of circulation.

The development of the circulatory system of birds and mammals shows two atria and two ventricles. The twist of the tube which started in amphibians and reptiles is completed in the arterial semicircle forming a helical system. In this segment, the longitudinal incision, as a knife slit in the primitive arterial tube, gives rise to canals that make up the band separating the two ventricles (Figure 9 D). The pulmonary artery and aortic roots present today evidence of this evolutionary incision in the arterial circuit. This process succeeds in pumping blood from the loop exit into the systemic bed with low energy cost and high speed (in humans its reaches 300 cm/s) via the left ventricle and into the pulmonary artery via the right ventricle at pressures which are 20% those in the systemic circuit. This stratagem allowed the development of intravascular pressure in the arterial circuit segment that propels blood at a speed that manages to irrigate the whole organism.

If we step back to the primitive stage in the evolution of the circulatory system we can appreciate the phylogenetic hallmarks of different species. The atria belong to the venous segment and the ventricles to the arterial segment. A subsequent more pronounced twist of the arterial segment puts into contact the atria with their corresponding ventricles. In summary, the comparison of fish and mammals shows that the single

atrium and ventricle of the former become the right and left atria in the latter. In turn, the mammalian ventricles originate from the budding arterial segment twist corresponding to the arterial circuit of amphibians and reptiles.(68,74)

The concept of circulatory trajectory prevented a clear understanding of the muscle anatomical unity. This muscular mass forms the chambers where blood circulates, since its dynamic movement is determined by the ventricular myocardial band. The arrangement of both atria in a horizontal plane (chambers originated in the venous semicircle) attaches to the ventricular component plane (arterial semicircle) where the chambers shaped by the myocardial band receive the motor impulse for ventricular contraction and suction.

In conclusion, according to Torrent Guasp's concept, the unification of the arterial and venous semicircles originating the circulatory system of birds and mammals determines the emergence of the ventricular myocardial band from an arterial loop and the differentiation of the right and left ventricular chambers when that tube segment is split into two.(68)

3. The cardiac apex

The apex -formed exclusively by the left ventricle- is a region situated in a twist of the descending band in its ascending continuation. This helical rotation of its fibers that from a subepicardial position become subendocardial, forms a coil of circularly interdigitating muscle layers that create a virtual rather than a real tunnel, as systolic contraction narrows it similarly to the mitral orifice. The apical "cul de sac" is lined in the inside by the endocardium and outside is covered by the epicardium.

The spatial configuration of the double consecutive passage of the descending band situated posteriorly to the ascending band (Figure 6) would allow the apex to turn first to the left during systole (seen from the apex) and then to the right, at the onset of the isovolumic diastolic phase with persisting contraction of the ascending segment. The continuation of descending and ascending bands is a continuum that in this vertex allows the apical loop to act as a bellows that shortens during systole and lengthens during the isovolumic diastolic phase.

As a result of this anatomo-functional process the apex enables the approximation of the base to the tip of the heart during systole (shortening) and its separation during diastole achieving ventricular elongation.

This mainly longitudinal functional interplay favors the residual systolic volume (30% of total diastolic volume) in the apical cul-de-sac.(81) At this point, we consider that the spatial arrangement of the double consecutive passage of the descending band situated posteriorly to the ascending band allows the non-ejection of part of the cardiac volume at end systole, remaining as residual volume. This remaining fluid acts as a limiting layer for correct suction during the isovolumic diastolic phase (Chapter II).

The apex does not make any measurable movement. It remains practically immobile during the whole cardiac cycle producing only a certain pressure on the chest wall (apex beat). It is the base of the heart which shifts as it descends (reducing ventricular volume) and ascends (increasing ventricular volume).

During systole, the heart undergoes a jet propulsion motion (principle of action and reaction). The apex is the main subordinate region of the retrograde force affecting the ventricular chamber when blood is ejected during systole. Similarly to other body regions with stress overload (J.L. Petit triangle)[1] it lacks muscle. In addition, it presents precarious irrigation and is submitted to a final pressure in its cul-de-sac when the aortic valve closes. This relatively immobile avascular apical area, without interposing muscle, submitted to the maximum effect of left ventricular residual pressure, becomes the place where ventricular wall aneurysms originate in 90% of cases.

1. Weak area of the abdominal antero-lateral wall. It has a triangular shape whose sides are composed of the medial margin of the external abdominal oblique muscle, the lateral margin of the latissimus dorsi muscle and the iliac crest as inferior boundary. (A.N.)

Chapter II

Electrophysiological Interpretation Of The Ventricular Myocardial Band

1. Historical concepts of myocardial electrical activation

In 1998 Torrent Guasp wrote: "*It would therefore be convenient, in order to validate the statement of a new concept of cardiac mechanics, to first perform an experimental study exclusively directed towards demonstrating the reality of the new interpretation on the propagation of stimuli*".(70)

The propagation of stimuli throughout the ventricles has to follow a pattern that matches the topography of muscle bundles. This interpretation undoubtedly concerns the heart function in its contraction, suction and dilatation phases. Structure and function are thus intimately linked and this concept has been the fundamental basis of our electrophysiological research to understand muscle twisting and untwisting and its cardiac suction effect.

In 1915, Thomas Lewis (39) had established that the stimuli arriving along the bundle of His are transmitted through the ventricular walls in an endocardial-epicardial direction, making the papillary muscles the first electrically activated structures. This position was confirmed by Parker in 1930.(52)

However, J. Robb and R. Robb posed in 1942 a fundamental question: "*How is it possible that the transmission of electrical impulses occurs, as all electrical data indicate, from the endocardial to the epicardial surface, given that the ventricular wall is composed of well differentiated bundles separated by sheaths of collagen tissue?*"(55) These same authors in dissection studies performed in 1936 had already supported that propagation runs lon-

gitudinally (axially) and not transversally, as defined by the classical view of Lewis with these statement: *"These data indicate that the excitatory process is conducted axially in the muscles studied along a pathway parallel to fiber direction"*.(54)

Later, J. Armour and W. Randall (1) in 1970 demonstrated experimentally that subepicardial contraction precedes subendocardial contraction. "*Subepicardial muscle contraction rather forms a rigid shell within which the subsequent contraction of the remaining myocardial mass takes place, generating intraventricular pressure*". They also declared "*the electrical activity propagates from the endocardium to the epicardium in the anterior surface of the left ventricular wall*". These concepts are in accordance with the anatomical arrangement of both ventricles within the myocardial mass, as in the integral loop configuration, the basal loop embraces the apical loop, determining a right ventricular chamber that resembles an open slit in the muscle thickness forming both ventricles (Chapter I). C. Roy and J. Adami (57) confirmed these conclusions in 1980 when they established the chronology between subepicardial contraction and mitral valve motion.

In 1987, García Civera, Cavadés and J. Cosin posed an open question abandoning the subtle discussion that "*the sequence of epicardial activation does not bear exact correspondence with endocardial activation, the former being earlier and variable, even from individual to individual*".(25) Francisco Torrent Guasp confirmed in 1998: "*the succession of functional difficulties of cardiac mechanics represents unequivocal proof of the longitudinal diffusion of stimuli along the ventricular myocardial band*".(70) This concept remained unchanged until the studies performed in the present investigation, based on the hypothesis that the axial transmission of the stimulus along the ventricular myocardial band supported by Torrent Guasp did not explain satisfactorily the twisting and untwisting movement indispensable to understand cardiac mechanics.

2. Stimuli propagation, muscle torsion and cardiac suction effect through electrophysiological research

Introduction

The hypothesis proposed by Torrent Guasp considers that the myocardium consists of a continuous single muscle band originating in the pulmonary valve and extending to the aortic root, thus limiting the two

ventricular chambers. In its spatial trajectory two muscle bands can be identified: the descending endocardial band and the ascending epicardial band, twisted in a double helical coil forming a basal loop (left and right basal segments) and an apical loop (descending and ascending apical segments). In this spatial arrangement, the descending and ascending bands cross each other at a point we will call "band intersection".(9, 68) This anatomical peculiarity forming a figure of 8 and its stimulation sequence explains fundamental aspects of left ventricular dynamics: 1) the mechanism of ventricular torsion; 2) the physiology of rapid diastolic filling by the suction effect and 3) the residual systolic volume. Despite anatomical and then functional studies performed so far, it was necessary to establish a correlation with the endo-epicardial stimulation circuit, since there were no studies in humans.

Ventricular filling is generally assumed as a passive ventricular function, resulting from ventricular relaxation, elastic fiber action and circulatory vis-a-tergo. Some authors (8,20,72) questioned that these mechanisms were sufficient to explain rapid left ventricular filling. A mechanism was postulated by which at the onset of diastole (isovolumic diastolic phase) the ventricle actively aspirates blood by persistent muscle contraction of the "ascending band". This action would produce apex-base lengthening with concomitant sudden decrease of intraventricular pressure until atrioventricular valve opening and subsequent rapid ventricular filling. (3-5,87) Various aspects of this theory have been challenged, mainly due to lack of an electrophysiological basis to support it through the study of cardiac activation.(18) We investigated the sequence of endo and epicardial band electrical activation to explain ventricular torsion, the active suction effects in the isovolumic diastolic phase and the meaning of residual systolic volume.

Methods

The left ventricular endo and epicardial electrical activation sequence has been studied using three-dimensional electroanatomic mapping (EAM) with a navigation system and Carto (Biosense Webster, California, USA) mapping, enabling three-dimensional anatomical representation, with activation maps and electrical propagation. Isochronic and activation sequence maps were performed, correlating them with surface ECG. An average of 50±8 endocardial and epicardial points were acquired for ventricular activation maps achieving detailed high density recordings. Apical, lateral and basal views were analyzed.

The study was performed at Hospital Presidente Perón (Buenos Aires, Argentina) and included patients who had signed an informed consent previously approved by the Institutional Ethics Committee. All patients were in sinus rhythm, with normal QRS and had no demonstrable cardiac disease by Doppler echocardiography and resting and stress gamma camera studies (Table 1).

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

Table 1. Patients characteristics

Electroanatomic mapping was performed during the course of radiofrequency ablation for arrhythmias owing to probable abnormal occult epicardial pathways. Mapping was carried out at the onset of studies, followed by ablation maneuvers. No complications developed. The presence of abnormal pathways did not interfere with mapping, as during the whole procedure baseline sinus rhythm was preserved.

As the descending band is endocardial and the ascending band epicardial, two approaches were used to perform mapping. The endocardial access was achieved by conventional atrial transeptal puncture and the epicardial access by percutaneous approach in the pericardial cavity (62) with an ablation catheter (Navistar™ curve F, Biosense Webster). Endocardial and epicardial mapping was immediately and consecutively performed. They were then superimposed, synchronizing them with electrocardiographic timing. Thus, simultaneous mapping of both ventricles was obtained.

The anatomo-functional Torrent Guasp theory lacked an essential research: the clinical documentation of the electrophysiological mechanism supporting the mechanical activation of the model. At the time Torrent Guasp performed his research, ECG was the only available study method. Theoretically, the ECG consists in the moment-to-moment recording of the potential resulting from the vector summation

of multiple simultaneous action potentials generated from the local activation of each area of the heart. Thus, the information provided by this method -invaluable in multiple aspects of cardiology- is limited to assess the discriminated spatio-temporal activation sequence for each ventricular region. The advent of clinical EAM overcame that limitation, as it not only allows the independent recording of different ventricular areas but also exclusive or integrated endocardial and epicardial areas.

We performed a high density and very detailed mapping of left ventricular endocardial and epicardial activation, according to the methodology described above. Mapping was carried out simultaneously with surface ECG, providing a unified temporal framework that enabled the correlation of both recordings and the synchronized view of the simultaneous activation observed in different electroanatomic conditions.

Percutaneous access technique. The Carto system was used for 3D mapping, performing voltage, activation and propagation maps, and the Sosa E and d´Avila (62) technique for epicardial recording through the left paraxyphoid space. A decapolar catheter in the coronary sinus and a 4-polar catheter in the bundle of His were placed as fluoroscopic reference. Mapping was performed in the pericardial cavity with a Navistar catheter (Navistar™ curve F, Biosense Webster, California, USA) (Figure 1). After both ventricles were externally recorded, left ventricular endocardial mapping was performed. The left ventricle was accessed by transeptal puncture through the right femoral artery using standard technique and then its inner surface was mapped with a catheter similar to that used for the epicardium.

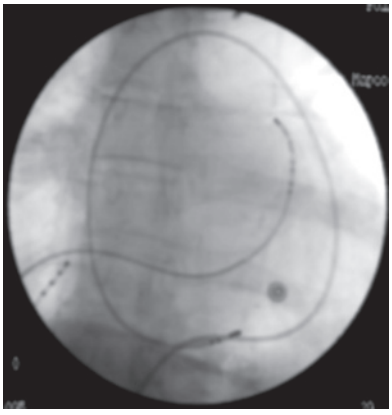

Figure 1. Epicardial mapping showing the epicardial catheter in the pericardial sac and the catheters in the bundle of His and coronary sinus.

Results

Isochronic mapping. Mapping enabled detailed activation recordings (Figure 2). Although the activation sequence varied in its details, in each case it was similar in its general aspects, forming an endo-epicardial depolarization matrix. As EAM corresponded to the left ventricle, the previously generated activation wave in the right ventricle was not obtained.

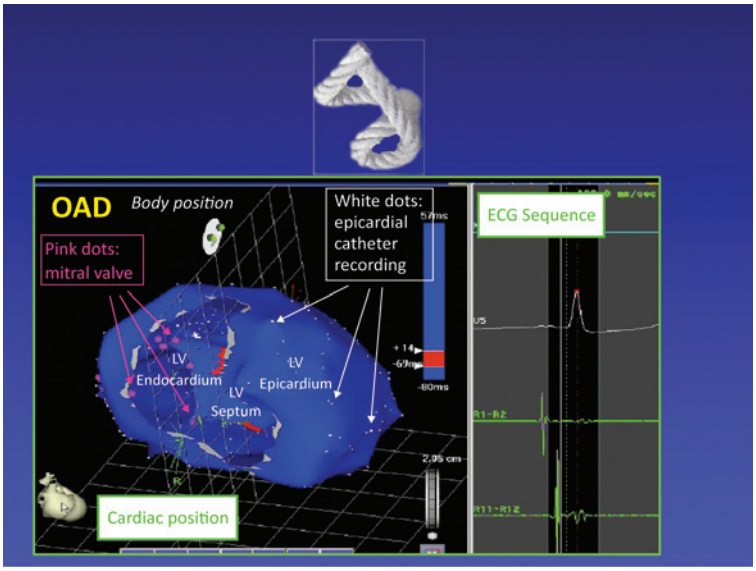

Figure 2. ***Integrated endo-epicardial mapping***. The left panel shows: the mitral valve (limited by pink dots), the left ventricular (LV) endocardium, the LV septal endocardium and the LV epicardium. The blue bar at the right shows total cycle duration and the red zone inside, the activation moment corresponding to the activation graph on the left. The right panel depicts surface ECG. The red dot at peak QRS indicates gated stimulus. Green channels correspond to reference electrograms. The dotted vertical line shows QRS onset and the full line the present recording moment. The upper panel represents Torrent Guasp's rope model.

Activation sequence. Figures 3 to 5 illustrate endo and epicardial propagation of electrical activation. In all figures, the left panel shows the right lateral projection and the right panel the simultaneous left anterior oblique projection. The activated areas at each moment are seen in red. The lateral part represents the activation of the bands in Torrent Guasp's rope model, where the depolarized zones are shown in red and previously activated zones in refractory period in blue (for more details see Figure 2).

Left ventricular activation starts at the interventricular septum (Figure 3 A). Next, following the anatomical arrangement of the descending band it spreads axially towards the ventricular apex. At that moment, the epicardial zone is also activated -ascending band- evidencing radial activation in a region that can be termed as "segment intersection" (Figure 3 B). This finding, as later discussed, modifies Torrent Guasp's model and constitutes the electrical foundation for the mechanical phenomenon of ventricular twisting. From "segment intersection" the activation loses its unidirectional character and becomes more complex. Figure 4 A shows 3 simultaneous wavefronts: 1) The distal activation of the descending band towards the apical loop; 2) the depolarization of the ascending band from the intersection towards the apex and 3) the activation of the ascending band from the intersection towards the end of the muscle band in the aorta. Figures 4 B, 5 A and 5 B show the progress and end of this process. In figure 5 A it can be seen that endocardial activation finishes much earlier than the end of the QRS period; the rest of the QRS period corresponds to the late activation of the distal portion of the ascending band, explaining its persistent contraction during isovolumic diastole, which is the basis of the ventricular suction mechanism (Figure 5 B). Figure 6 summarizes the stimulation of this rope model.

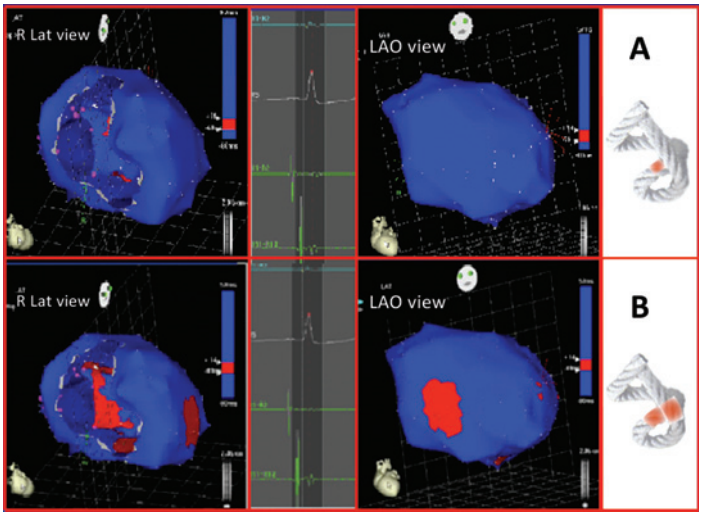

Figure 3. A: ***Onset of left ventricular activation***. The left panel illustrates the depolarization of the ventricular septum, corresponding to the descending band. In the right panel, the ventricular epicardium (ascending band) has not been activated yet. ***B. Simultaneous band activation.*** The activation progresses in the left ventricular septum along the descending band (axial activation) and at the same time propagates to the epicardium (radial activation), activating the ascending band.

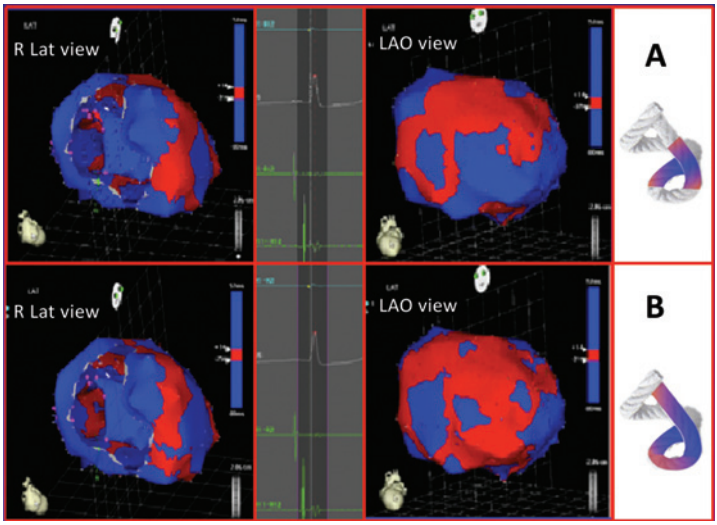

Figure 4. ***A: Bidirectional activation of the apex and the ascending band***. The final septal activation is seen propagating towards the apex, synchronously with the epicardial activation in the same direction. At the same time, the epicardial activation propagates towards the base of the left ventricle. ***B: Propagation progress***. The activation progresses in the directions of the previous figure.

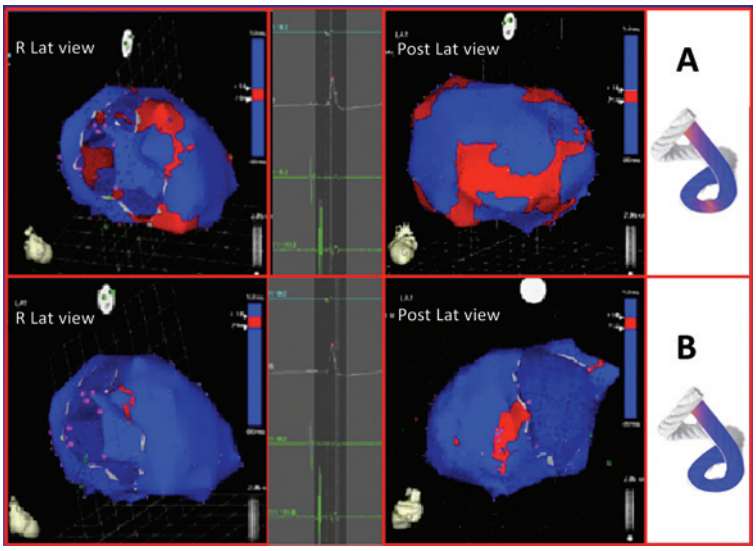

Figure 5. ***A: Late activation of the ascending band***. At this moment, corresponding to approximately 60% of QRS duration, endocardial activation (descending band) has already been completed. The distal portion of the ascending band (epicardial) depolarizes lately. This phenomenon correlates with its persistent contraction at the initial phase of diastole. ***B: Final activation.*** In the right panel, the projection was modified from left anterior oblique to left postero-lateral, showing the very late activation of the distal portion of the ascending band.

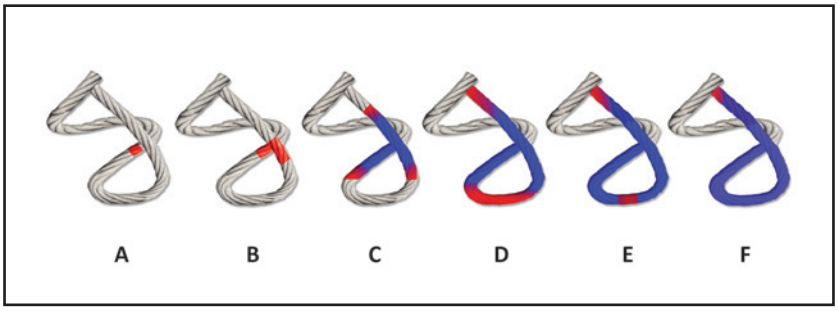

Figure 6. ***Rope model.*** Activation sequence of Torrent Guasp's ventricular myocardial band (A-F) according to our findings. Depolarization (red); repolarization (blue).

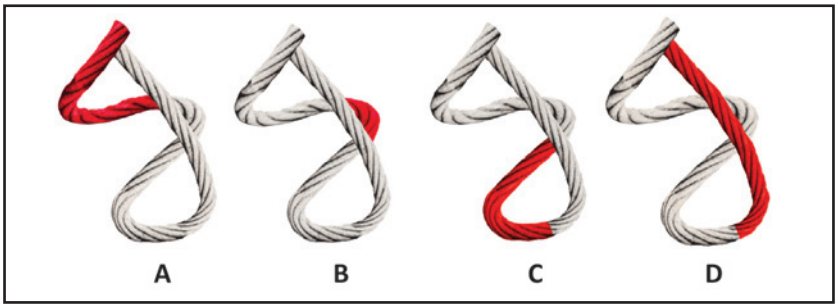

Figure 7. ***Rope model.*** Unidirectional propagation of excitation (red) in the ventricular myocardial band according to Torrent Guasp (A-D). Notice the difference with Figure 6.

Discussion

Torrent Guasp's continuous ventricular myocardial band hypothesis in cardiac mechanics implies a series of associated muscular movements. These occur in the band eliciting left ventricular narrowing, shortening, twisting, lengthening-untwisting and expansion phases during the cardiac cycle.

According to Torrent Guasp, longitudinal diffusion of stimuli along the ventricular myocardial band explained heart performance (Figure 7).(68) However, this sequential "peristaltic" activation did not correlate with some currently well-known fundamental phenomena, as clockwise and counter-clockwise twisting at the left ventricular apex and base, which are mainly responsible for its mechanical efficiency (Figure 6). In an attempt to explain the mechanism of muscle twisting, we studied the sequence of ventricular electrical activation by means of simultaneous three-dimensional endo-epicardial segment mapping.

Electrical activation is the consequence of the propagation of stimuli through the muscular structure of the heart, both of Torrent Guasp's ventricular myocardial band as the rest of the fibers involved in its structural framework. The cardiac mechanism of suction and ejection requires structural-functional integration capable of unraveling the different dynamic aspects arising from the propagation of excitation. This indicates that the diastolic phase is an active process of contraction producing increasing suction (due to its similarity we have called it "plunger mechanism"), (83) which at a certain level of intraventricular pressure opens the atrioventricular valves producing rapid filling.

Stimulus propagation and left ventricular torsion

The integrated endo-epicardial three-dimensional mapping performed in this research supports the activation model that propagates along the descending and ascending bands. Figures 3 to 5 elucidate the activation sequence of the contractile areas and their entry into cardiac dynamics associated to the course of the excitation wave with a coordinated pattern according to muscle structure.

In this experience we found a stimulus trajectory different from that described by Torrent Guasp, but which explains the twisting phase of the heart, defined as the opposing rotational movement of the base and apex. At the point of band intersection the activation propagates from the endocardium to the epicardium (radial propagation), that is, from the descending to the ascending band. From the anatomical point of view, this passage could be mediated by "interband fibers" (Torrent Guasp's "aberrant fibers") (71) (Chapter I).

From this point onwards, the ascending band depolarizes in two senses: towards the apex and towards the base, at the same time that the descending band completes its activation towards the apex (Figure 5). Thus, two essential phenomena occur:

1. As the apical loop depolarizes from band intersection in two simultaneous wavefronts (from the descending and from the ascending bands) it generates their synchronized contraction.
2. The activation of the ascending band propagates from band intersection in two opposing directions: towards the apex and towards the base (Figure 5). The resulting mechanical contraction will also have a divergent direction, giving origin to the apical and basal clockwise and counter-clockwise rotations, respectively.

According to Lewis (39), stimuli diffused from the endocardium to the epicardium through the muscle walls. Contrary to this concept, Robb (54) published in 1936 that stimuli propagation occurred longitudinally, and in 1942 inquired: "*How is it possible that impulse transmission occurs from the endocardial to the epicardial surface… given that the ventricular wall is composed of well differentiated bundles, separated by sheaths of connective tissue?"*.(55) Surprisingly, according to their experimental studies, Armour and Randall (1970) concluded that stimuli diffusion in the left ventricular anterior wall was generated from the endocardium to the epicardium.(1) This local event in the left ventricular anterior

wall contrasts with previous concepts and with the remaining muscle mass where the electrical activity of subepicardial muscle bundles takes place before those in the subendocardium. However, this discrepancy of the impulse transmission theory through the ventricular myocardial band was not resolved until our research shed light on its understanding, with patent relevant considerations for cardiac mechanics. In 1960, Torrent Guasp expressed: "*The subendocardial layers contracted by the descending segment come into activity before the subepicardial ones, which are components of the ascending segment*" (66) and in 1988 he reaffirmed "*the descending segment and then the ascending segment successively enter into activity*".(70) Towards 2001, Buckberg and Torrent Guasp ratified the hypothesis that excitation spreads unidirectionally along the ventricular myocardial band (Figure 7).(8)

Our research modifies these concepts since stimulus propagation is simultaneously axial and radial. This activation sequence has an important anatomical component in cardiac architecture not evidenced by Torrent Guasp's description (66), to the point of calling them "aberrant fibers" and that we will name "interband fibers" (Chapter I), since they constitute the anatomical possibility of radial impulse propagation from the descending to the ascending band.

The ventricular narrowing phase (isovolumic systole) at the beginning of systole is produced by the contraction of the basal loop right and left segments. The overlapping shortening phase is due to the descent of the base, at the same time as twisting occurs, which is produced longitudinally, as the ring contracts before the apex. The fact that the apex remains fixed, is due to the movement of the base, descending in systole and ascending in diastole. This is explained better because the ascending band, rigid in systole and at the beginning of diastole, acts as a tight tutor keeping the apex immobile. The pressure generated to eject the highest amount of blood at the onset of ejection during an interval lasting 20% of the systolic phase is feasible due to the twisting movement. This action is achieved because the electrical stimulation propagates towards the descending band (axial propagation) and simultaneously to the ascending band (radial propagation). Although the electrical conduction progresses along the ventricular myocardial band, radial propagation towards the ascending band plays an essential role in ventricular twisting by allowing opposing forces on its longitudinal axis, generating the necessary intraventricular pressure to achieve abrupt blood ejection. The interband fibers that cross from the descending band to the ascending band would be responsible of impulse transmission between the bands (Chapter I). Thus, a twisting mechanism similar to "wringing a towel" would be produced.(80)

The historical term of systole and diastole (79) did not take into account the meaning of electrical activation and contraction, but only the hemodynamic concept of ventricular ejection and filling. It is therefore necessary to find a relationship between activation and the mechanical outcome. The explanation is provided by the simultaneous axial and radial electrical conduction when it reaches band intersection, also confirmed by the spatial arrangement of fibers, with subendocardial fibers on the right side and subepicardial fibers on the left.(58) This layout also agrees with the evolutionary loop of the circulatory system forming the two developed ventricles in birds and mammals. Torsion -different rotation between the apex and base of the heart- generates: a) high pressures, b) reduces ventricular stress, and c) homogenizes its distribution in the ventricular wall thickness.

Torrent Guasp declared that "*the sequence of ventricular muscle entry into activity in the different ventricular regions takes place along the band*", (70) similarly to a peristaltic movement (Figure 7). Then, how could the ventricle achieve its twisting movement, since this action requires two opposing forces at the same time? Unidirectional activation does not explain twisting or the evolutionary-structural development designed to apply a force capable of ejecting the ventricular content at a speed of 300 cm/s at low energetic cost. This is understood by the simultaneous axial and radial activation we have found, whose sequence can be illustrated as (Diagram 1):

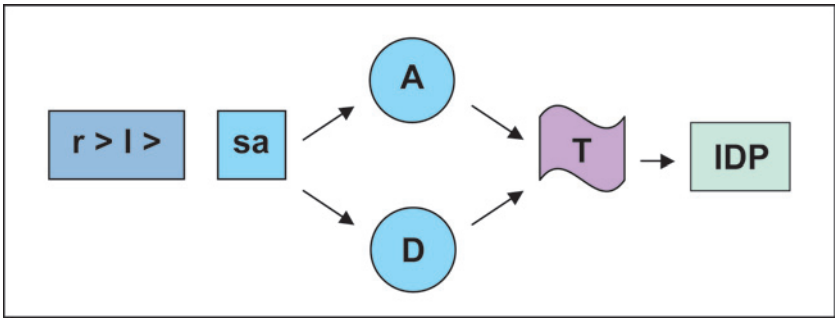

Diagram 1. References: r, right segment; **l**, left segment; **sa**, simultaneous activation of the ascending and descending segments (interband fibers); **A**, ascending segment; **D**, descending segment; **T**, torsion; **IDP**, isovolumic diastolic phase (active).

Active suction in the isovolumic diastolic phase

The investigation of another previously unexplained point was to consider ventricular filling as an active phenomenon generated by myocardial contraction tending to increase left ventricular apex-base distance after ejection, thus producing a suction effect through a "plunger" mechanism. This mechanism is explained by the persistence of the ascending band contraction during isovolumic diastole.

We have found that the endocardium depolarizes completely during the first part of the QRS. In turn, Buckberg found that the mechanical contraction triggered by this electrical mechanism is initiated 50 ms after its occurrence and persists for approximately 350 ms. If the depolarization of the ascending band starts 50 ms after that of the descending band and its contraction persists for the same length of time, the contractile state will last approximately 400 ms. If ventricular systole lasts about 300 ms, the remaining 100 ms correspond to the isovolumic diastolic phase (usually called isovolumic relaxation, though it can be seen that there is ventricular contraction). To summarize, during the initial part of this phase, the ascending band remains contracted as a consequence of the depolarization that took place during the QRS. The explanation of this late contraction does not require depolarizations after the QRS, as postulated by Pedro Zarco.(88)

In our investigation, the final part of the QRS corresponds to the activation of the ascending band (Figure 5), resulting in the necessary contraction to generate suction ("plunger effect") during the isovolumic diastolic phase.

With the initiation of untwisting during isovolumic diastole, the ascending band progressively lengthens, generating negative intraventricular pressure while it is still contracted (active process), as an energetic residue of the twisting process. On this point, Zarco expressed in 1998: "*there is a point about which we cannot agree: that the straightening of the ascending segment is due to an active contraction of the cardiac muscle in full diastole*".(88) This is precisely what we have found in our research, establishing the mechanism that achieves rapid ventricular filling during a short interval in diastole.(61)

Interpretation of the active suction phase

Diastolic activation explains the insertion of a suction active coupling phase between systole and diastole, with muscle contraction, energy consumption and marked fall of intraventricular pressure (Figure 8). This effect draws blood into the ventricular chamber through a pressure difference with respect to the external pressure which is responsible for 70% of the total filling volume in only 20% of the filling period.

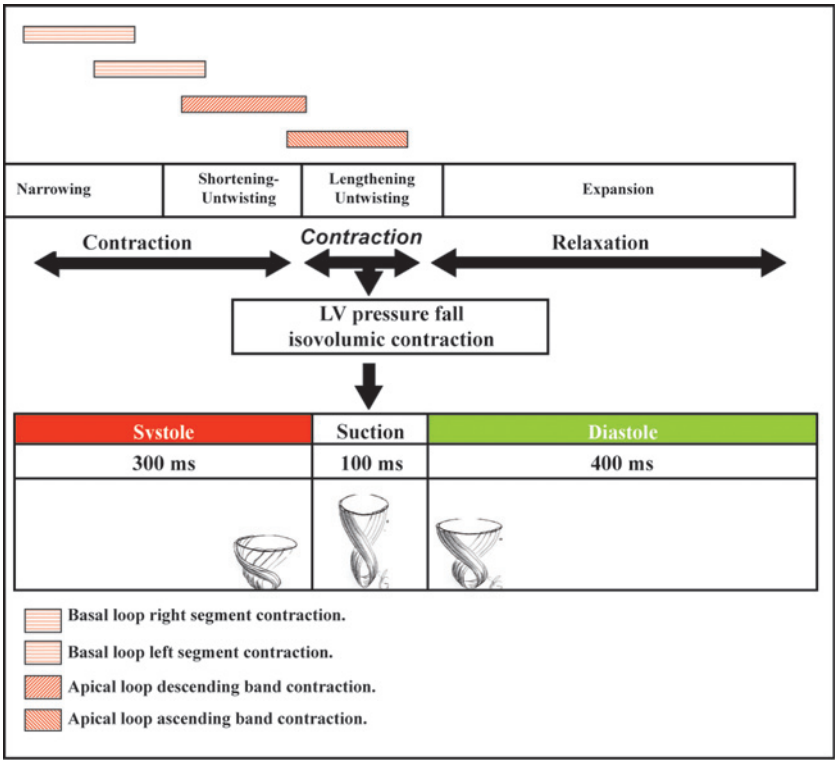

Figure 8. Effects of the cardiac activation-contraction mechanism

This suction phase between systole and diastole lasts 100 to 200 ms and is active during muscle contraction with intraventricular pressure fall below zero (Figures 9 and 10). Tyberg, (85) using mitral valve balloon occlusion in the dog, showed that during diastole the left ventricle decreases pressure below zero. As a result, the heart is a dynamic suction pump, but to be effective the elastic recoil must be limited to allow

an effective subsequent systole. The efficacy in surgeries that bypass the right ventricle (Fontan-Kreutzer) have shown this left ventricular suction effect, (73) similarly to left mechanical support with univentricular devices, where blood is delivered from the left ventricle into the aorta.

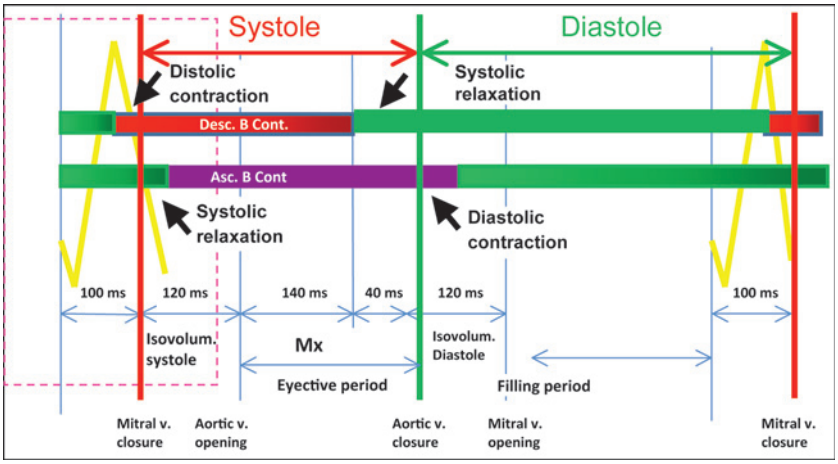

Figure 9. Diagram of the electromechanical activity

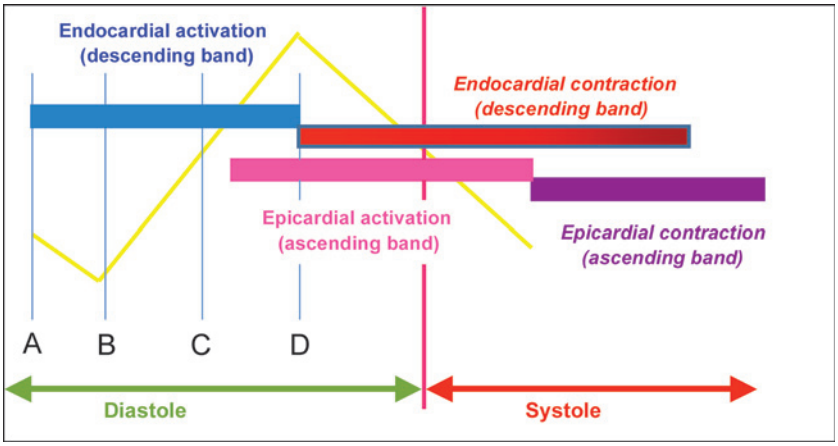

Figure 10. Electrical activation and mechanical contraction of the ascending and descending bands (enlarged Figure 9 box).

In the traditional model, cardiac filling is only determined by venous pressure. Actually, atrial pressure is too low to explain this situation. Torrent Guasp developed the concept of active suction to explain this "key doubt", supported by the physio-muscular structure he described.(82)

The last contracting areas of the ascending band produce a suction effect to draw blood towards the left ventricle. The high filling velocity at low pressures establishes that this is an active phenomenon.

Systolic residual volume

Systolic residual volume represents 30% of total diastolic volume. From the point of view of fluid mechanics, between systole and diastole we find a closed chamber that only contains blood, which is an incompressible fluid, and hence, at any level of muscle contraction, this chamber volume cannot change. Therefore, this phase is isovolumic, and as it cannot change the volume, muscle contraction produces a depression (pressure fall) in the chamber favoring subsequent diastolic filling. In these conditions, the depression generated will depend on chamber capacity of muscle contraction and geometry affecting the internal pressure distribution. It is physically necessary to have an incompressible fluid to produce this pressure fall, and that the chamber geometry and blood volume content allow an adequate depression ("plunger" mechanism).

There is a range of optimal residual systolic volume for suction. If it is higher it will require an important muscle contraction to develop the necessary depression, whereas if is lower, the interaction between walls will impair suction and hence the diastolic boundary layer phenomena will alter filling. From a physical standpoint, an excessive end-systolic volume will affect the suction capacity, which will thus be related with the volume pumped during systole.(22)

Conclusions

Our study shows a "three-stage heart": systole, suction and diastole. The data obtained are especially important, as they were recorded in humans, with structurally normal hearts and in physiological conditions. According to these findings, we may conclude:

1. Endo-epicardial 3-dimensional mapping shows a sequence of electrical activation in the region of the apical loop in agreement with the synchronous contraction of the descending and ascending bands.

2. The simultaneous and opposing activation of the ascending band to the starting point of its radial activation from the descending band, agrees with synchronous and opposite rotation of the apical and basal areas (mechanism of ventricular twisting).
3. The late activation of the ascending band, compatible with its persistent contraction during the initial phase of isovolumic diastole (untwisting and suction period), occurs without need of postulating electrical activation after the QRS.

3. Electrocardiographic correlation

The onset of the QRS correlates with early endocardial depolarization of the left ventricular anteroseptal wall. The depolarization of the epicardial segment starts about 50-60 ms after endocardial depolarization (corresponding approximately to the final 40% of QRS duration, coincident with peak positive dP/dt), generating the contraction of the apical loop ascending segment as shown in electrophysiological endo-epicardial mapping recordings. Certainly, small differences between QRS onset and end must be accounted for, as well as those of endocardial and epicardial potentials, similar to the ones recorded in electrophysiological studies.

Since Harvey and then Einthoven, contraction and electrical activation of the heart have been considered as almost linear and homogeneous processes. According to the classical concept, normal ventricular activation would have to be initiated in the septum, to continue in the apex and end in the basal region. In this way, contraction would occur "en bloc" during systole and relaxation would ensue almost homogeneously during diastole. Systole is synonym of cardiac contraction and diastole of relaxation. These have been the classical concepts, and at this stage of knowledge they must be considered more complex.

Although several aspects of ventricular electrical stimulus propagation have long been known, the advent of three-dimensional navigators and electroanatomic mapping have enabled a more detailed study of electrical propagation in the human heart in completely physiological clinical situations, revealing various interesting characteristics:

1. Normal electrical activation, though basically ordered and homogeneous, is very complex especially in lately depolarized areas.

2. Intraventricular activation "occupies" approximately the initial 60% of surface QRS duration. The "rest" of the QRS corresponds to myocardial and epicardial activation. The onset of ventricular activation evidenced by the QRS complex is "exclusively" endocardial; during the intermediate phase both activations coexist and at the end, during suction, the activation is "exclusively" myo-epicardial.
3. The end of the apical loop ascending segment contraction is diastolic, indicating that the classically termed isovolumic relaxation phase is an active process.

In conclusion, ventricular electrical activation is a very complex process generating intricate patterns in the ventricular contraction it elicits. This complexity is further complicated by the addition of electromechanical coupling factors, intrinsic contractility of different cardiac regions and mechanical coupling, among others, which substantially modify basic concepts of cardiac mechanics evidenced so far.

It is possible to hypothesize about some aspects of this research:

a. Although there is no problem in defining the onset of systole as the moment in which the mitral valve closes and that of diastole as closure of the aortic valve, this does not imply that the process of contraction and relaxation start at those precise moments. On the contrary, it is obvious that the ventricular contraction generating the increase in pressure required for mitral valve closure occurred before this point (that is, during diastole) and the relaxation which reduced intraventricular pressure allowing aortic valve closure also started before this took place, that is, during systole. Likewise, it is reasonable to infer that not all myocardial fibers contract or relax simultaneously, but that at certain periods (impossible to quantify for each individual fiber) the fibers coexist at different stages of contraction and relaxation, both during systole and diastole.
b. The final state of contraction achieved (and hence the general ventricular geometrical configuration and specially that of its chamber) must depend to a certain degree, and perhaps very significantly, on the state of contraction or relaxation of adjacent fibers or structures. It seems logical to assume that the fibers that contract in the first place, surrounded by relaxed fibers, may reach their maximum degree of potential contraction; conversely, if the

adjacent fibers are already contracted and "pulling" in an opposite direction, the degree of effective contraction will be lower.

c. Something similar might happen if we consider a structure such as the ventricle in its entirety and in the opposite situation of finding a region of the heart relaxed while another is contracted. In this case, muscle shortening is completed producing chamber dilatation.

d. Electromechanical coupling. Even though electrical depolarization triggers mechanical contraction, from this moment two different and autonomous processes take place. Membrane activation may occur without mechanical contraction, as evidenced by neuronal electrical activation eliciting the release of neurotransmitters with no contractile activity. This would be similar to having a selective inhibitor of actin-myosin activation producing a normal depolarization without muscle contraction.

On the other hand, the temporal relationship between electrical activity and mechanical contraction is probably not linear. It is known that above all, there is latency between electrical and mechanical activity. Moreover, the time between electrical activation and contraction (at least, maximal contraction) probably varies depending on numerous factors: the type of fiber, its metabolic condition, its place in myocardial wall thickness, the level of intracellular calcium, the contraction or relaxation of neighboring fibers, etc.

Let us consider typical situations: the QRS lasts approximately 100 ms, and systole 300 ms. Therefore, the QRS effects on contraction "persist" at least for those 300 ms after its end. Furthermore, fibers activated at the end of the QRS contract or remain contracted at the beginning of diastole. Therefore, depolarizing potentials in the T wave would not be necessary to explain muscle contraction during diastole (which, as already mentioned, is not synonym for relaxation of all cardiac fibers).

4. Functional aspects of the ventricular myocardial band

The analysis of the anatomical description of Torrent Guasp's ventricular myocardial band and the ensuing electrophysiological research in patients performed by us indicates the need of providing a clear interpretation of some fundamental aspects. Of the three descending

band turns relative to the ascending band, the first two successively pass in front and behind the ascending band forming the basal loop. The last step, after the twist of the large band that turns it in the descending segment, it becomes again posterior to the ascending band, with a failed helicoidal arrangement in this space of the apical loop. This spatial anatomical situation of the muscle bands establishes a significant correlation with cardiac function.

The ventricular narrowing movement takes place at the base of the heart, conditioned by the consecutive basal loop right and left segment activation that produces the narrowing phase (systolic contraction). The progression of this phase, in the stimulation process of the descending band (axial), together with the concomitant propagation (radial) towards the ascending band found in our investigations, determines a helical rotating motion with the ensuing shortening of the left ventricular vertical axis (ejection). The cardiac apex, composed of the subepicardial arrangement of fibers that in their twist become subendocardial, constitute a free apex with a "cul de sac" to support the intraventricular pressure produced by the heart impulse during ventricular shortening. The subsequent movement as a result of the ascending band contraction, its straightening and ventricular lengthening therefore determine an active mechanism during isovolumic diastole that maintains the chamber in an isovolumic condition but with intraventricular pressure fall (untwisting and active suction phase) to give place to ventricular filling with atrioventricular opening (expansion phase). These left ventricular shortening (base descent) and lengthening (base ascent) movements correlate with the principle of Newton´s action and reaction principle.

As a result of this anatomo-functional process, the apex (space between the two bands) has in normal conditions the power of annular narrowing (sphincter-like mechanism) to bear the retrograde intraventricular pressure produced by blood ejection.

At the beginning of the isovolumic (active) phase, erroneously considered as belonging to diastole, which instead should be named suction phase, the persistent contraction of the ascending band with left ventricular lengthening generates the fall of intraventricular pressure, sufficient to achieve ventricular suction. This pressure fall produced by the persistent contraction of the ascending band during the isovolumic phase with every left ventricular orifice closed, works as a "plunger" mechanism (suction phase) at the moment it starts lengthening. When this pressure is sufficiently low (less than 10 mmHg) and with the ventricle expanded and "unscrewed", the mitral valve opens and the abrupt flow of blood from the atrium takes place (filling phase).

The contractile impulse of the circulatory tube of the annelids worms through a peristalsis mechanism. The propulsion along its length preserves the pattern of axial transmission, but after the twist suffered by the cardiac tube in mammals and birds it adds radial transmission allowing both bands to have a helical motion indispensable to produce the subsequent concatenated twisting and untwisting-suction movements.

It is an arrangement of nature that the descending band passes posterior to the ascending band twice consecutively without wrapping around the latter. In this way it creates a distal cardiac region (apex) favoring the installation of the cardiac residual volume by becoming a zone with less amplitude of helical movement in this game of twisting first to the left during systole (seen from the apex) and then to the right at the beginning of the suction phase with the contraction of the ascending segment. Moreover, it makes the apical loop act as a bellows that shortens during systole and lengthens during the isovolumic phase. This longitudinal motion of apex-base shortening in systole and lengthening during the isovolumic phase is responsible for 75% of left ventricular ejection or suction power. The transverse interplay of basal loop narrowing (systole) and expansion (diastole) contributes to only 25% of cardiac output (Figure 11).

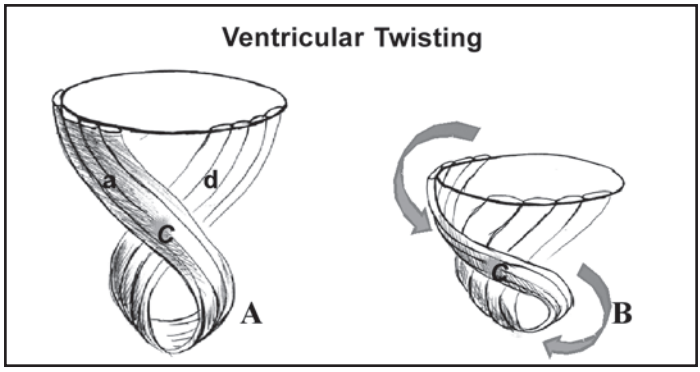

Figure 11. Ventricular twisting. A. Resting ventricular state. **B.** The subsequent mechanical contraction produces opposed rotation between the ventricular base and apex. **C:** band intersection; **d:** descending band; **a:** ascending band.

The fact that diastole only employs 15-20% of its period to attain its maximum pressure fall indisputably leads to assume an active mechanical process and not simply passive relaxation. The time employed by diastole to attain its maximum negative pressure (120 ms) is com-

parable to that used by systole to achieve its maximum ejective pressure (140 ms). Sarcomere architecture in the spatial integration and its contractile framework, as well as its biochemical structure (elastin, titin) implicates elastic properties that add to the active phase. The force generating negative intraventricular pressure through the ascending band activation acts on myocardial elastic properties to achieve the optimal effective sarcomere recoil intime and limit for adequate relaxation. The violation of this limit imposed by the architecture of cardiac muscle will have great impact on heart failure.

Neither is systole synonym for contraction nor diastole for relaxation. This denomination is a dialectic that keeps no relation with the real movements that originate ventricular systole, suction and diastole, the three heart stages.

Chapter III
The Three Stage Heart

1. Chronology of the suction mechanism concept

Erasistratus of Keos, a Greek anatomist and physician in Alexandria (300-250 B.C.), detailed the anatomy of the heart, described its valves, giving its current name to the tricuspid valve, and referred to the intermediate system between arteries and veins, which he called "*synanastomoseis*", as a first approach to the capillaries. The latter would have led Erasistratus, had he not been influenced by the pneumatic theory -according to which the pneuma had to be transported by the arteries along the body to the veins by means of *"synanastomoseis"*- to a more precise knowledge of circulation.(79) Erasistratus considered that arteries did not carry blood, but pneuma, and assumed that the fundamental movement of the heart was dilatation and was produced by a contracting muscle.(70) This concept persisted in history by the work of Galen of Pergamus (130-200 A.D.). In 1542, Andrea Vesalius in his work "*De Humani Corporis Fabrica"* stated "*when the left ventricle expands again, breath and blood are attracted once more towards the ventricle".* (79)

In 1628, William Harvey in the fundamental work of human physiology "*De Motu Cordis*" regarded systole as the main fact of cardiac function. With this perspective he opposes Galen, who believed that the activity of the heart was manifested in its expansion, by means of the *vis pulsifica*.(79) It was necessary to wait until 1954 when Francisco Torrent Guasp (67) using a theoretical approach describes diastolic ventricular suction as an active contractile process, leading G. Brecher (4) (1956) to assume that *"a demonstrative experimental evidence of diastolic ventricular suction is very difficult".* However, this same author elaborated on this concept and in 1958 established the certain possibility of ventricular suction.(5) The efforts, mainly of anatomical research, together with an intense reflective analysis on the subject, led to the acknowledge-

ment of the ventricular myocardial band postulated by Torrent Guasp (67-70) though without a clear physiological interpretation. This situation would progress in the physiological understanding of ventricular pressure in the different phases of the cardiac cycle through the work performed by Francisco Torrent Guasp (68), D. Streeter (63), P. Lunkenheimer (40) and Gerard Buckberg.(7,8)

The experimental study in pigs by Juan Cosín Aguilar et al. in 2009, using piezoelectric crystals on the ventricular wall, considered the possibility of myocardial contraction in the isovolumic diastolic phase.(20)

2. Cardiac mechanics

Background. The mechanical activity of the heart is complex because it results from integrating its ejection, suction and filling properties, in different short successive phases, correlated in short periods. Anatomy texts describe the heart but pay little attention to the position adopted by myocardial fibers. Physiologists do not even mention it, as if the mechanical heart had no bearing on the scaffolding design developed by muscle fibers in the wall of the ventricles. It is impossible to interpret cardiac function measurements with diagnostic purposes without taking into account its morphological structure. Cardiologists and clinical researchers focus on the molecular aspects to explain heart failure following neurohormonal models. However, drugs that act upon neurohormonal activation alleviate but do not cure heart failure. Size and geometrical changes are responsible for the abnormal myocytes and myocardial structure that worsen cardiac function, enhance neurohormonal activity and reduce cardiovascular system response. The morbidity and mortality prognosis of patients with heart failure is directly related to ventricular dilatation.(65)

Myocardial capacity is conditioned by cardiac fiber orientation. In that regard, the conical heart has helical and circumferential myocardial fibers. Fiber orientation determines heart function. Thus, the ejection fraction is 60% when the normal helical fibers contract and drops to 30% if the transverse fibers shorten.(7) The development of a spherical configuration modifies the orientation of the helical fibers until a transverse dilatation is achieved with consequent decrease in contractile force.

Regarding the mechanism of ventricular filling two hypotheses have been generated:

a) Classical model. According to Harvey (79), ventricular filling is achieved by atrial contraction or by pressure gradient, the latter being the hypothesis proposed by Wiggers in the 1920s.(86)

The atria have two main functions: one as transportation or pumping and another as reservoir for ventricular filling. Like the ventricles, the atria respond to an increase in fiber length with an increase in contractile force and at rest atrial contraction contributes 20% to ventricular filling. Increase in atrial contractility and deviation to the left of atrial function curves or deviation to the right of velocity-force curves may result from sympathetic stimulation, vagal inhibition or inotropic agents. In atrial fibrillation, circulatory reserve mechanisms may sustain cardiac output at rest, or otherwise, loss of atrial contraction may result in serious consequences for ventricular filling and heart function.

At the onset of the isovolumic phase, left ventricular pressure falls below left atrial pressure shortly after peak left atrial *V* wave. Moreover, the ventricular rapid filling phase at the end of the isovolumic phase coincides with the continuous atrial pressure decline that began during mitral opening, while the end of rapid ventricular filling and the beginning of slow ventricular filling is characterized by a change in the rise of the ventricular volume curve. During slow ventricular filling, left atrial and ventricular pressures increase slowly until the next atrial systole. Atrial contraction and the concomitant increase of ventricular filling are manifested by increased pressure and ventricular volume.

b) Suction mechanism model. The ventricle is an active suction pump during diastole. This hypothesis was proposed by Erasistratus in the third century B.C. and later by Galen (second century A.D.) and Vesalius (1542).(70,79) Diastolic filling by an active suction mechanism is supported by Torrent Guasp's hypothesis (71) and by experimental and clinical studies.(4,20) The three components of the heart's three-dimensional reduction interact synergistically through the ventricular myocardial band rotating around itself to achieve the ventricular twisting and then untwisting-suction mechanism. In systole there is a three-dimensional reorientation of cardiac architecture with helical shortening in a longitudinal and transverse sense, producing a twisting movement. The atrioventricular annulus contracts, the aortic annulus slightly expands, the mitral and aortic planes descend and the left ventricular outflow tract remains open.

Suction occurs in early diastole. During this period (isovolumic phase) presence of negative intraventricular pressure has been documented both in human clinical studies as in animals with balloon mitral valve occlusion.(23,56,61,85)

Mechanical energy is stored during systole and the suction phase and is then released in favor of a rapid expansion during the filling phase, (56,61) thus explaining the high filling velocity with very low pressure gradient. In Torrent Guasp's hypothesis, diastolic suction is activated by the contraction of the ascending band, a fact which was verified in our electrophysiological research. Then, relaxation (expansion) is almost explosive and the sarcomere jumps like a spring to regain its original length. If end-systolic volume is reduced, the expansive restorative forces are greater; while in pressure or volume overload (hypertrophic cardiomyopathy, heart failure) relaxation occurs more slowly and suction is reduced.

Based on the law of Laplace, after mitral valve opening the ventricular wall stress rapidly increases due to increased intraventricular radius and reduced wall thickness. This suction force (similar to the "plunger "mechanism) acts at the end of the isovolumic phase lengthening the fibers and allowing fast ventricular loading despite low filling pressure. Changes in wall thickness, i.e. thinning, determines ventricular filling.

Diastolic filling of the coronary arteries can participate in active myocardial lengthening. The plethora of coronary reservoir during diastole distends the myocardial mass as if the ventricle was a cavernous body and an erectile mechanism was generated prolonging the ventricular cavity. Coronary circulation becomes a protection for the ischemic ventricular muscle during systole and expands in diastole by filling the coronary bed. Based on this principle, a cardiac model has been developed composed of a double elastic bag whose hydraulic increase of intramural pressure generates ventricular diastolic expansion, and which has been applied in paracorporeal circulatory assist devices.

The right ventricle is designed for ejecting large volumes of blood against low resistance, owing to its large semilunar surface. Unlike the left ventricle where blood is transferred from the mitral orifice to the apex before being directed into the aorta, in the right ventricle the blood is pumped directly from the tricuspid orifice to the pulmonary artery following a central washing line that forms a 90° angle.

Suction pump. Torrent Guasp's theory has generated all kinds of objections and controversies. One of the most debated aspects of this conception is the electrophysiological rationale, namely the alleged lack of correlation between the sequences of the proposed mechanical activity and the observed electrical activation. The advent in recent years of electrophysiological three-dimensional navigators allow the acquisition of highly accurate and detailed information on the activation sequence of the various cardiac structures we have established in our research.

One of the central points of Torrent Guasp's hypothesis consists in postulating isovolumic diastole as an active phenomenon, generated by late myocardial contraction producing ventricular lengthening and separating the apex from the base. This concept has two apparent "inconsistencies" from the point of view of classical mechanics pathophysiology.

1) *Muscle contraction is always associated with reduction of the ventricular cavity.*
2) *Ventricular contraction and relaxation are considered to have "en bloc" simultaneous performance during their entire course and not in successive phases.*

In order to investigate and clarify these controversial points, we have studied the sequence of left ventricular activation using high-resolution three-dimensional mapping. The electroanatomic mapping was performe during a Carto system in patients during radiofrequency ablation procedures for different types of arrhythmias, in structurally normal hearts. Mapping was established in sinus rhythm and patients had normal QRS.

A very important finding of this study was that endocardial activation is fully completed when surface QRS duration has barely reached 60%. The rest of it therefore corresponds to epicardial activation. This late stimulation (about 100 ms) at the epicardial level (corresponding to the ascending segment of the apical loop) results in its contraction during the isovolumic diastolic phase, thus constituting an active process.

According to recordings obtained by three-dimensional mapping, the electrical impulse travels along the helical irregular arrangement of the ventricular myocardial band. Transmission is axial, longitudinal along the ventricular myocardial band, and also radial transverse, from endocardium to epicardium, through the fibers we have called interbands (TorrentGuasp's aberrant fibers) (Figures 1 and 2).

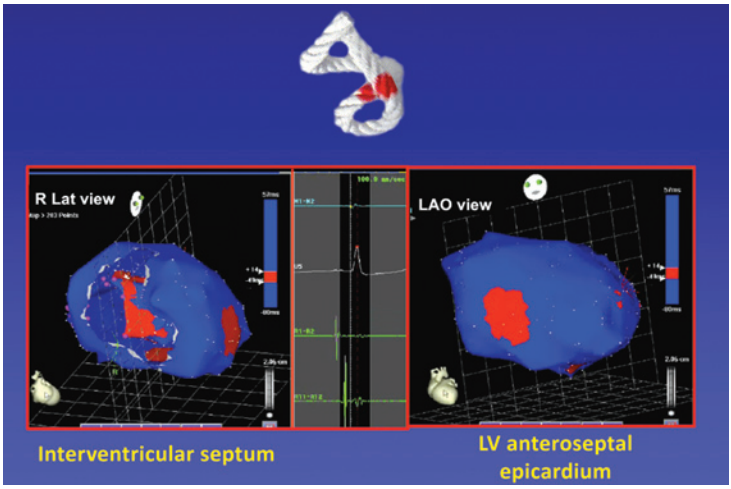

Figure 1. Transverse impulse transmission through the interband fibers.

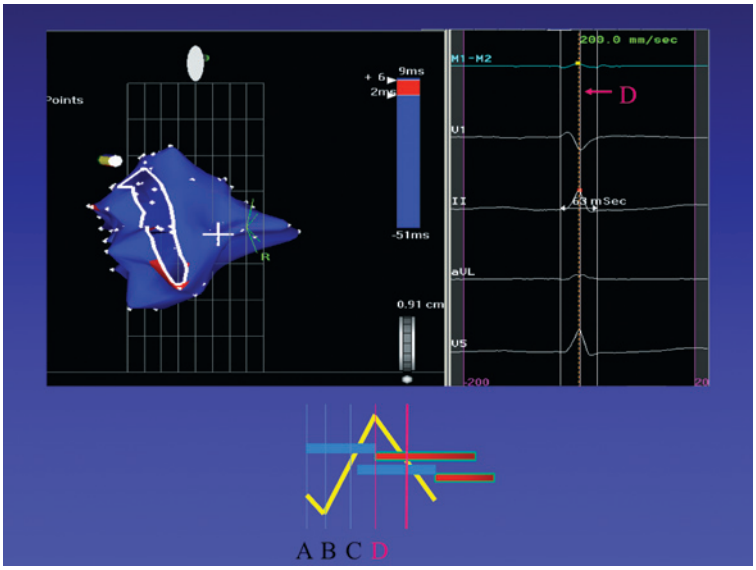

Figure 2. Continuation of the previous figure. It completes the endocardial activation in the area corresponding to the mitral annulus. Note that all the endocardial activation "occupies" about 60% of QRS duration (D line in the right panel). Epicardial activation has initiated earlier, but its completion occurs during the end of the QRS. This whole sequence is consistent with the succession of mechanical activation events set forth above (Chapter II, Figure 9) and provides its electrophysiological substrate.

In the anterior left ventricular surface, at the intersection of the ascending (subepicardial) over the descending (subendocardial) band, just at the start of isovolumic diastolic contraction, the ascending segment is contracting and the descending segment is repolarizing. This late contraction of the ascending segment during the isovolumic diastolic phase supports the mechanical process to achieve ventricular untwisting and suction, generating the pressure fall necessary to aspirate blood. (Chapter II).

The basal loop (contraction of the right and left segments) determines ventricular narrowing, while contraction of the descending and ascending segments by radial impulse transmission causes the shortening and twisting movements of systole. All these physiological processes are required for the ejection phase. As cardiac activity continues, the persistent contraction of the apical loop ascending segment causing ventricular lengthening, determines the isovolumic diastolic intermediate phase, generating intraventricular negative pressure (suction phase) through a "plunger"mechanism. Ventricular expansion, last cardiac movement, occurs during diastolic relaxation (diastolic filling).

The result of this research leads us to consider that cardiac function consists of three movements. Between systole (300 ms) and diastole (400 ms) it inserts a third movement of contraction (100 ms) which represents coupling between both and produces the intermediate cardiac suction phase, now called isovolumic relaxation phase.

In the classical model, the heart remains passive after each systole, filling with blood as a result of venous pressure. However, in the current model a *quantum* of the energy of each systole is stored in the heart, activating the following initial phase of classical diastolic relaxation (isovolumic) which we call isovolumic contraction. The physiological effect of the old model is validated by the Frank-Starling law, but actually it: a) does not reflect the interaction between systole and diastole and b) that law was enunciated in isolated hearts.(56,61)

In the current model, the relationship between the two cardiac phases is critical for the proper functioning of the heart. Now, how is the energy accumulated in systole spent in diastole?

1) By the movement achieved in systole, the base of the heart is pushed downwards and the blood in the reverse direction (Newton's principle of action and reaction). In diastole, however, the cardiac base projects upwards against the blood flow entering the heart. This increases blood velocity and helps to propel filling.

2) Systole contracts both the elastic elements of the heart and its muscle fibers to the point of inducing the natural tendency of the ventricles to expand even without any external filling. Thus, an intraventricular negative pressure or suction is generated.

Let us remember that in the traditional model the output is solely determined by the venous filling pressure of the right heart. Actually, the difference between peripheral and atrial pressure is too low to explain heart filling. Upon this "key doubt", related to the classical explanation, Francisco Torrent Guasp developed the concept of active suction pump supported by the physiologic-muscular structure he described. The last contracted areas of the apical loop ascending segment produce blood suction from the atrium into the left ventricle. With variations to Torrent Guasp's interpretation, the research we conducted allows to set solid bases on cardiac mechanics produced by the ventricular myocardial band.

Cardiac movements during systole and diastole correspond to phenomena of inertia and elastic recoil and not to static filling pressures. Mammalian hearts persist in their emptying and filling after being excised and placed in buffered solution. The length reached by cardiac muscle fibers is determined not only by the position of cardiac filling but also by the intervening suction mechanism.

Conversely, if there is failure of systolic contraction, as occurs in systolic heart failure, it is reasonable to think that energy release will not be sufficient in the suction phase. The role of venous filling pressure in these hearts is here more relevant according to the Frank-Starling law. (56,61, 81)

3. How is diastolic suction produced?

There is evidence from current functional anatomy to microscopy. The irregular helical anatomical description of Torrent Guasp's ventricular myocardial band mimics in its torsion the supporting microscopic connective structure of the heart, a condition that reinforces his investigation. Lengthening resides both in the sarcomere structure as in the muscle cell components that make up the cytoskeleton. The sarcomere expands laterally and increases its diameter. This lateral enlargement extends the Z discs. Thus it constitutes the storage mechanism of part of the energy of contraction that could be used as expansion energy.

Connective tissue is here involved. The outer surface of the muscle cells is covered by collagen and elastin connective fibers which have stress-strain properties arranged in a network. This network structure returns muscle cells to their original configuration by preventing excessive stretching of the sarcomeres in order to achieve suitable suction during the relaxation phase.

The fibrous cytoskeleton should be understood as the sum of the fibrous interweaved constituents essential for the preservation of ventricular geometry. The collagenous scaffolding coordinates the muscle fibers by collecting them in bundles of increasing structures, to keep an optimal stretching with the aim of achieving an effective subsequent contraction. This cytoskeleton is formed by a grid-shaped mesh that individually enfolds the sarcomeres, which are gathered in bundles of connective structures called straps or ropes, twisting helically around their own axis.(2)

In normal conditions, myocardial sarcomeres undergo length changes ranging from 1.85 micrometers during contraction to 2.05 micrometers in the resting period. During systole, sarcomere shortening is only of 12%, but with this small percentage the left ventricle empties by 70%. Due to this efficacy ejection is achieved at a speed of 300 cm/s at a pressure of 120 mm Hg, allowing a left ventricular three-dimensional reduction of 15% in its longitudinal, transverse and anteroposterior axes. The helical disposition of muscle fibers (macro and microscopic) enables this efficiency resembling, in the words of Torrent Guasp a "heart piston". In this three-dimensional reduction there are three components: a) a transverse component of the basal loop resulting in myocardial constriction in the mentioned plane; b) a longitudinal movement of ventricular shortening due to the contraction of the descending apical loop segment that acts as a bellows approximating the base to the apex; and c) a twisting motion of the helical component, counter-clockwise in the left ventricle and clockwise in the right ventricle.

These straps of connective tissue, with have a similar disposition to that of helically twisted guy cables of suspension bridges, have suggested the idea of systolic energy storage, which when released in the last stages of the process allow the effect of a diastolic suction pump.

Mammalian hearts placed in buffered solution are self-propelled due to the strap structure. In contrast, there is no such impulse in the frog heart because it lacks these moorings that act as inter fiber fixing elements. Paradoxically, an invertebrate such as the squid, draws water through a hollow chamber wrapped in a muscular structure achieving with its ejection the effect of jet propulsion. This can be done due to the straps present in the muscles.(56)

Left ventricular recoil lifts the right ventricle favoring its rapid and accelerated filling. The whole heart motion contributes to its filling. As a result of this mechanism the increased contractility of the left side enhances the efficiency of the right side.(37)

In short, as a result of the helical ventricular elastic recoil, suction is an active ventricular process. During the misnamed left ventricular isovolumic relaxation the ascending apical loop segment contracts. Suction in this phase is explained by a "plunger-like" mechanism. As the ventricular walls that exert suction give in and dilate, the "plunger mechanism" becomes unsteady, thus establishing, through this concept, a different assessment of heart failure and its clinical severity.

Likewise, mitral valve opening which increases wall stress and decreases its thickening, lengthens the fibers allowing the ventricle to fill up quickly. The high filling velocity with low pressures would be explained by the suction phenomenon. This active mechanism of Torrent Guasp's ventricular myocardial band on the diastolic effect opens a wide perspective for surgical repair techniques both in shape and volume and on the consequent left ventricular function.

Francisco Torrent Guasp never believed that blood could enter the left ventricle other than by a suction device. He considered that it cannot be achieved "vis a tergo" since the difference between peripheral blood pressure and heart pressure is small. The gradient that returns it is ventricular aspiration by a suction mechanism. The heart is a structure formed by a large muscular band that begins at the insertion of the pulmonary artery and ends at the level of the aorta, forming a double helix which limits the ventricles. The two chambers wrapped by the ventricular myocardial band are the left ventricle with ellipsoidal shape and the right ventricle of semilunar structure. Hence, this band's contraction explains not only heart's systole but also blood suction.

The inevitable question that arises is whether the bands surrounding the ventricles should do so on a rigid fulcrum, as does a tendon using a bone insertion as lever. Are there any in the heart? The heart does not need that support. When it fills with blood it behaves as a bone insertion. The ventricular myocardial band is a large double helix suspended from the aorta and pulmonary artery that uses the hemoskeleton, i.e. cardiac filling, as fulcrum. On this point, by rotating the base and the apex of the left ventricle in opposite directions, muscular twisting is generated. This counter-clockwise movement of the apex can generate high pressures reducing deformation. Exactly as if a towel was wrung out.(80,81,83,87) This mechanical activity is now clearly explained by the simultaneous axial and radial stimulation trajectory found in this investigation.

4. Structural basis of diastolic left ventricular limitation in the suction mechanism

As Henderson said in the distant 1923:*"In the heart, diastolic relaxation is a vital factor and not simply the stretching of a rubber bag"*.(27) In this theoretical framework, biological assistance with a conditioned skeletal muscle appeared in 1985 with the first surgery in humans. Performed by Carpentier and Chachques (12) in Paris, it stood in the clinical therapeutic horizon with surprising and enigmatic edges (Figure 3). But this demonstration had its limit of success when in 1959 Kantrovich affirmed: *"I believe that a muscle is a much more sensitive engine to be used in this type of situation. It gets its power from the eggs you take in the morning, and the system to transform this into energy is already present and working".*(35)

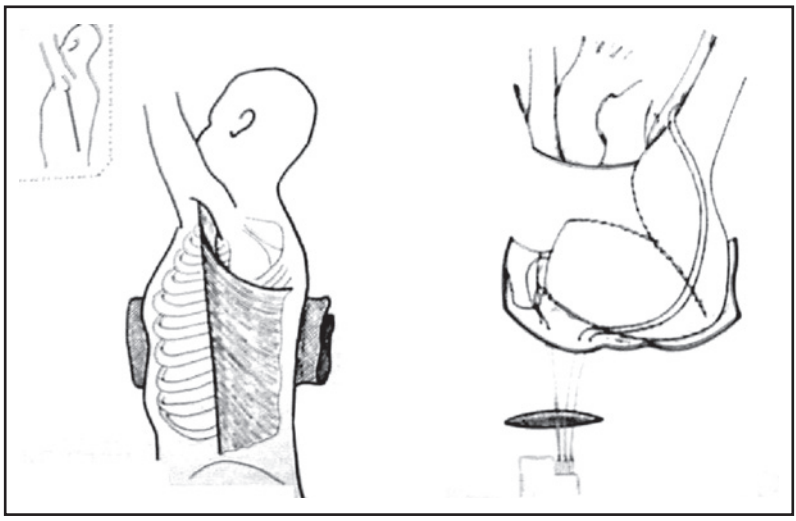

Figure 3. Dynamic cardiomyoplasty with latissimus dorsi muscle

Since then, and until being used in man, it was necessary to elucidate a number of physiological advances in the understanding of the transformation of type II muscle fibers (fast, glycolytic and fatigable) into type I (slow, oxidative and fatigue resistant).(59) In addition, both technological developments in the search of muscle stimulation generators, as progressively adapted surgical techniques, have allowed the performance of a significant number of dynamic cardiomyoplasties (DCMP) worldwide, enabling the clarification of various aspects from the results obtained.

Since heart failure poses different problems derived from its own complexity, and has led to various analyses based on the segmentation of the problem, we believe that the same cartesian reasoning should be followed in interpreting the results of DCMP. Mechanical assistance during systole would be the most logical effect to accept, but this event has no explanation in the small increase observed in left ventricular ejection fraction. Similarly, the lack of difference between assisted and unassisted beats at postoperative mid-term, casts doubt on this function. Neovascularization, i.e. the possibility of establishing irrigation channels between the latissimus dorsi muscle and the myocardium has not been demonstrated in humans so far.

If something has been demonstrated by DCMP, is its effectiveness in stopping the process of expansion of the ventricular chambers which occurs in dilated cardiomyopathy pathophysiology. This process of continuous dilatation carries in itself the greatest risk of death in these patients, as confirmed by the SOLVD group of researchers.(65)

Capouya, (11) as well as Mott (48) have suggested that the procedure serves as an interruption of progressive dilatation. Carpentier (13) reports stability in cardiothoracic index values at three-year follow-up. Our experience has been similar in this respect.(76) Moreira, (46) on the other hand, reports clinical evidence of reduced wall stress, decreased left ventricular chamber diameters and increased maximum left ventricular elastance. Kass (36) also found beneficial effects associated with "reverse remodeling"in cases studied analyzing pressure-volume relationships, reporting left ventricular decreased end-diastolic and end-systolic volume and improved ventricular function. Wall stress reduction, a central determining factor of oxygen consumption, would be explained by DCMP prevention of progressive ventricular dilatation.(34)

We have seen in our own statistics these favorable cardiomyoplasty effects regarding the progression of ventricular dilatation.(76,78) Among the 15 patients who underwent this technique, 12 patients exceeded the absolute threshold of two years (follow-up average 31.3 months per patient). In these patients the value of left ventricular diastolic diameter (echocardiography) was: 72.7 mm ± 3 mm preoperatively, 73.6 mm ± 7 mm at one-year follow-up and 72.3 mm ± 8 mm at two years.(83) The difference between these values was not significant, i.e., the left ventricle preserved its initial values, revealing no progression of dilatation. In the heart structure proper and in the pathological consequence implied by progressive and permanent dilatation we must acknowledge the beneficial effects of dynamic cardiomyoplasty in dilated cardiomyopathy.

The force generated by systolic contraction exerts a compression on the elastic-heart muscle elements of such a magnitude that even without internal diastolic filling the ventricular tendency is to expand. This negative pressure determines a suction pump mechanism. The negative intraventricular pressures determining this effect were first described in 1930. For this dynamic suction pump to be effective, the elastic recoil process must have a limit that allows a subsequent effective systole. Therefore, the possibility of sarcomere lengthening is engraved both in the muscle structure proper as in the fibrous cytoskeleton. The arrangement of the muscle straps, similar to guy cables on a suspension bridge, have suggested the idea of systolic energy storage, which once released in the diastolic process would allow the effect of a ventricular suction pump (Figure 4).(55,61,75)

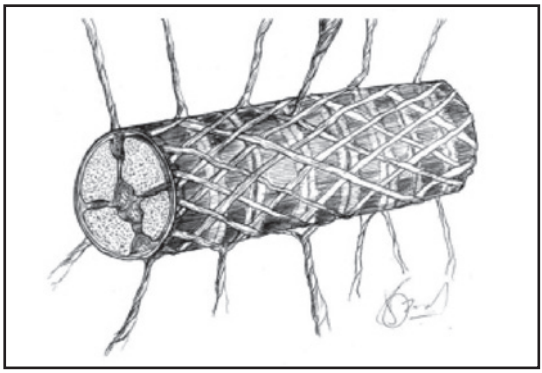

Figure 4. Collagen cytoskeleton consisting of a grid-shaped mesh surrounding the sarcomere.

The muscle girth obtained with DCMP provides an elastic constriction limiting cardiac dilatation. This brings a benefit to diastolic function, producing a decrease in end-diastolic pressure, which is enhanced when the cardiomyostimulator is switched off. Similarly, once the cardiomyostimulator has been discontinued, there tends to be a more delayed ventricular filling, indicating that the contraction of the latissimus dorsi muscle improves filling.(14,78) This situation of improved diastolic function performance would determine a decrease of mitral annulus and left atrial diameters, as was the tendency in patients of the different series published.(13.76)

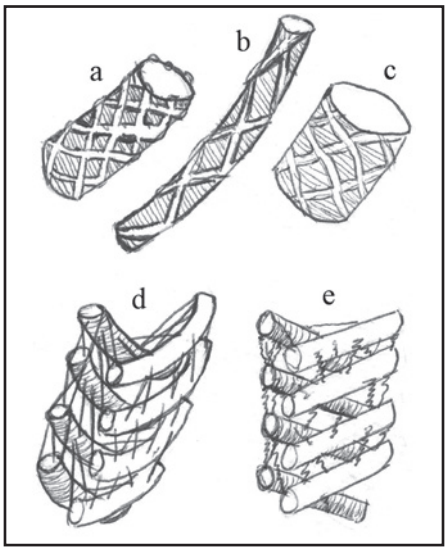

Figure 5. Collagen cytoskeleton surrounding the sarcomere; **a, b** and **c:** different expansion phases; **d** and **e:** collagen fibers that constitute the ropes that bind sarcomere bundles and achieve an integral unit.

Dynamic cardiomyoplasty is an operative situation that contributes to restore the dilated heart muscle strength in order to obtain the required internal gradient for filling through the suction pump mechanism, and has irrefutably demonstrated a significant improvement in these patients' functional class. In our experience, at two-year follow-up, it had passed from 3.06 ± 0.2 to 1.7 ± 0.6, with a survival rate of 75% during this period, similar to other reports.(77) To date, functional class III should be considered as the only one capable of being subjected to this technique. In some of this functional class series, long-term follow-up has shown (13.48) a survival rate of 66% at 7 years. Other publications have reported less optimistic results: about 56% at two years.(24) In order to achieve the diastolic effects of DCMP, tissue network shave been developed which are inserted around the ventricles through a ministernotomy. It has been considered that they control the deterioration of cardiac function by stabilizing the dimensions of the heart.(53)

It is understood that the straps between sarcomeres coordinate the action of a large number of muscle cells (Figure 5). The fact of providing solidary architecture for heart deformation allows better suction and a faster recovery of its initial length. Given the heart's anatomo-physiological composition and the application of Laplace's law to the progressive dilatation mechanism, the beneficial diastolic effect observed

in DCMP monitoring is explained through the basis of left ventricular structural control in the active suction mechanism. The simplest interpretation of these findings is that the grid structure of the collagen fibers prevents excessive sarcomere stretching. Cardiac muscle damage determines poor suction. With no limit to recoil, the mechanical activity of suction affects ventricular filling with the consequent failure of the suction pump, as there is no available intraventricular pressure drop in an adequate degree and suitable time.

5. Echocardiographic concepts

Echocardiography is currently able to provide noninvasive information on the complex mechanism of myocardial contraction. By analyzing 2D deformation with speckle tracking technique the opposite rotation between the apex and the base of the heart (Figure 6) is evidenced, thus achieving ventricular twisting (systolic contraction) and subsequent untwisting (suction mechanism, "plunger" effect) (Figure 7). Apical rotation (counter-clockwise, viewed from the apex) (Figure 8) is considered positive while basal rotation (clockwise, viewed from the apex) is considered negative (Figure 9). To calculate the degree of twisting, the software performs an algebraic subtraction (it adds the value of the apical positive twisting to the negative value of the base). In normal subjects it is about +11 degrees, always with prevalent tip rotation.

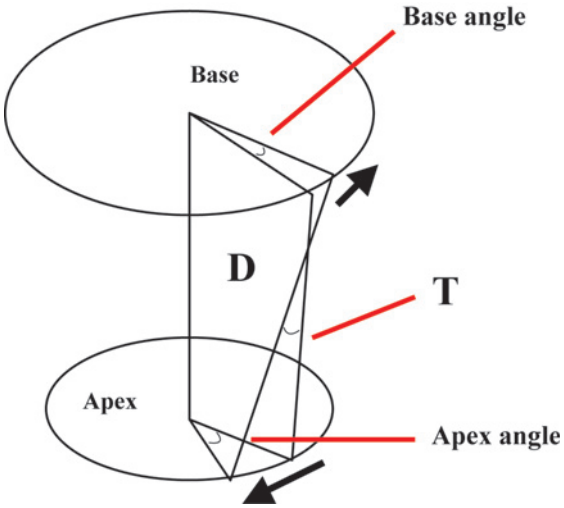

Figure 6. Twisting (T) considers the distance between the rotating points.

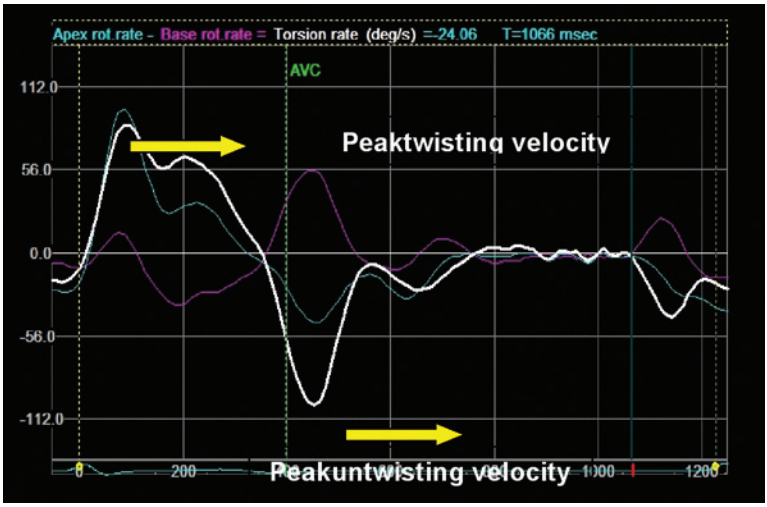

Figure 7. Peak twisting and untwisting velocity.

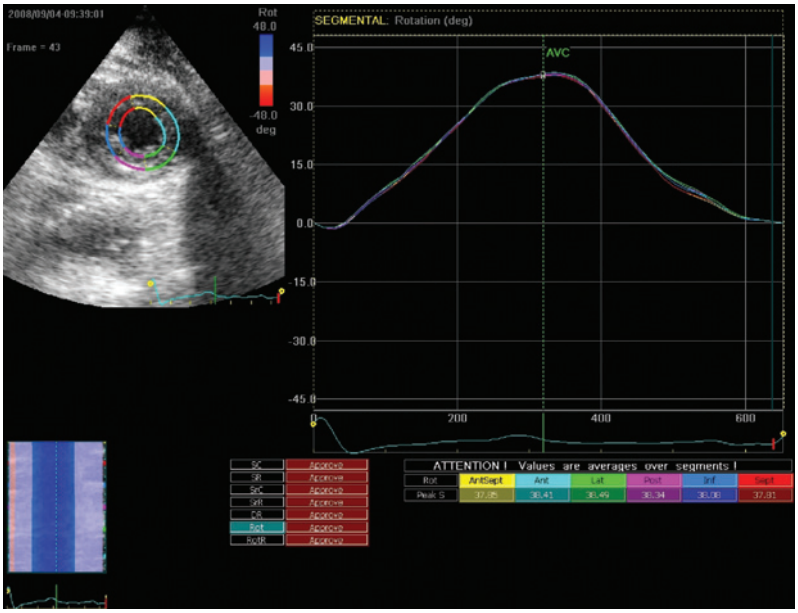

Figure 8. Apical rotation (counter-clockwise, viewed from the apex).

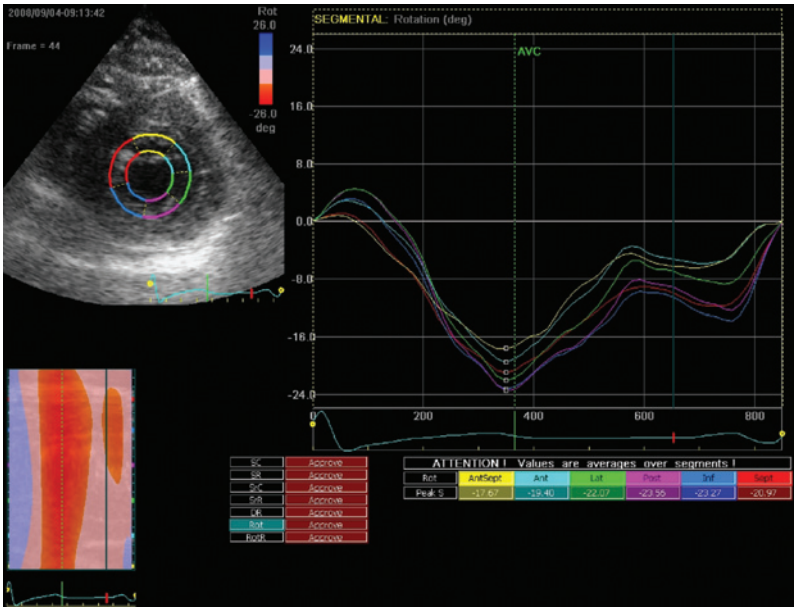

Figure 9. Basal rotation (clockwise, viewed from the apex).

The deviation from this value is generally a marker of heart disease, although it is necessary to consider that the normal values of left ventricular rotation and twisting are variable and depend on the technique used, the location of the area of interest (subendocardium, subepicardium), subject age and loading conditions.

Ventricular twisting and untwisting can be explained by the angular distribution of the ascending and descending fibers.(15) Rotation is produced by contraction of the helical fibers and the strength and direction are given by the balance of subendocardial and subepicardial fibers. If subendocardial fibers are responsible for longitudinal deformation, we must consider that the mid-fibers and especially the subepicardial fibers contribute fundamentally to rotation and twisting.(26)

The radius of subepicardial rotation is greater than that of subendocardial rotation; therefore, the subepicardium provides greater rotational force than the subendocardium, and as result subepicardial rotation expresses more significantly at the apical level.(49)

The mathematical difference between twisting and torsion is that the latter also considers the distance between the two points that rotate in opposite directions, although in practice and in the literature they are confused and are often used as synonyms.

At the papillary muscle level rotation is neutral, constituting a transition point between counter-clockwise apical rotation and clockwise basal rotation, so in general it is not considered. There is a clear relationship between twisting and age, showing that it increases with age possibly by progressive deterioration of the subendocardial fibers.(51) In coronary disease with subendocardial perfusion deficit, longitudinal strain is attenuated. Myocardial ischemia does not affect twisting unless infarctions are very extensive and transmural (affecting circumferential and subepicardial fibers).

In dilated cardiomyopathy, twisting is reduced proportionally to systolic function, and is responsible for the attenuation of apical rotation, whereas basal rotation may be normal or reduced. In some of these patients the apex and base rotate in the same clockwise direction.(60) In patients who respond to Cardiac Resynchronization Therapy a rapid normalization can predict inverse remodeling at 6 months.

In each of the different pathologies, rotation, twisting and untwisting are variably affected depending on the extent and distribution of the fibers affected, adopting characteristic patterns in ventricular hypertrophy, diabetes, amyloidosis, valvulopathies and pericardial diseases.

In conclusion rotation/twisting is fundamental for the heart contraction and suction mechanism. Its echocardiographic assessment is an accurate and feasible method to detect both systolic and diastolic myocardial dysfunction in daily practice, even in the early stages of cardiovascular disease.

Chapter IV

Clinical, Surgical And Eletrophysiological Perspectives Derived From This Research

1. Extent of heart failure

Heart failure is one of most important public health problems due to its social, economic and especially human impact with a prevalence of 5 million patients, 300,000 deaths per-year and 500,000 new cases per year (16) in the United States. The implications for health systems are 15 million visits per year; 6.5 million days of hospitalization and 38,000 million dollars of health costs. In Europe, the incidence is 1.3 cases per 1000 inhabitants per year in subjects over 25 years, reaching 11.6 cases per 1000 inhabitants per year in patients over 85 years (21), while 5% of the European population has problems associated to heart failure.(17,50)

The main cause of heart failure is coronary artery disease also responsible for 50% of cases in the United States. In ischemic heart disease, acute myocardial infarction is the first single factor, with a tenfold increased risk of heart failure than in the normal population during the first year post infarction and up to twenty times higher in the following years. After acute myocardial infarction, there is loss of cardiomyocytes which added to the process of ventricular remodeling triggers heart failure. This remodeling is a complex phenomenon involving molecular, neurohormonal and genetic processes leading to left ventricular dilatation, abnormal morphology and dysfunction.

Early revascularization of acute myocardial infarction with angioplasty and stenting has not reduced the incidence of dysfunction and left ventricular remodeling. Thus, if in old series with conventional

treatment, 20% of patients with transmural infarction developed ventricular dilatation and dysfunction; in recent series of myocardial infarction treated with angioplasty and stenting during the acute phase, 30% develop alterations in the shape or function at six months. The evolution of these patients regarding mortality and complications, is directly related to ventricular dilatation.(28,44,45)

In 5% of cases, patients present with Grade IV heart failure, are very symptomatic, require frequent hospitalizations and survival is less than 30% at one year. (33) The increase in ventricular volume and the acquisition of a spherical shape is responsible for the progression of the disease. The classic surgical treatment of these patients is heart transplantation, with a survival rate of over 70% at five years and 25% at 20 years.(64) However, the disproportion between the number of recipients and donors, only allows less than 20% of patients with Grade IV heart failure to benefit from heart transplantation.

Hence it is necessary to preserve cardiac transplantation for patients who have no other treatment options and concomitantly develop other alternatives. Recent therapeutic strategies are designed to integrate biology and new medical technologies, generating alternatives that may improve the prognosis and the functional status of these patients. Treatments include surgical ventricular repair, passive ventricular restraint, permanent ventricular assistance or as bridge to recovery, immunoadsorption and tissue engineering.(30)

Neurohormonal models do not explain heart failure progression, and drug treatments that act on neurohormonal activation delay but do not stop disease progression, or are ineffective.(41,42) Size and geometrical changes are responsible for myocyte and extracellular matrix structural abnormalities that aggravate cardiac function, increase neurohormonal activity and reduce cardiovascular response. The study of the anatomical basis is necessary for the application of new techniques aimed at restoring ventricular geometry to the native volume and ventricular conical configuration. The prognosis of patients with heart failure is directly related with ventricular dilatation.

Myocardial capacity is conditioned by the orientation of cardiac fibers. In that respect, the conical heart has helical and circumferential myocardial fibers. Fiber orientation determines function; thus, ejection fraction is 60% when the normal helical fibers contract and falls to 30% if only the transverse fibers shorten. The development of a spherical configuration modifies the helical fiber orientation towards a transverse dilatation and decreases contractile force. Thus, the development of surgical ventricular repair techniques has reenacted the work of Tor-

rent Guasp and his hypothesis of the ventricular myocardial band (7) as well as cardiac mechanics based on the research of cardiac electrical activation developed by us.

2. Clinical perspectives

The conclusions of the research presented in this manuscript can be summarized as follows:

1. Endo-epicardial three-dimensional mapping shows a sequence of apical loop electrical activation consistent with the synchronous contraction of the descending and ascending bands.
2. The simultaneous and opposed activation of the ascending band, to the starting point of its radial activation from the descending band, is consistent with the simultaneous clockwise and counter-clockwise rotation of the apical and basal regions (ventricular twisting mechanism).
3. The late activation of the ascending band, compatible with its persistent contraction during the initial phase of the diastolic isovolumic period, occurs without postulating electrical activations after the QRS.

The novel activation sequence of Torrent Guasp's ventricular myocardial band found in this work, explains the previous process that triggers the ventricular twisting and suction mechanism. Furthermore, it confirms that the activation of the ascending band completes the QRS. This finding demonstrates the contractile persistence of this muscle segment during the first part of diastole, cancelling the traditional concept of passive relaxation.

Our work suggests the existence of a "three-stage heart ": systole, suction and diastole. We believe that the data obtained are of special importance since they were recorded in humans, with structurally normal hearts and in physiological (non-experimental) conditions. Further research is needed to elucidate what happens in different diseases.

In recent years, the importance of pathological ventricular filling and diastolic failure has been put into evidence.(38) In this respect, most studies have been based on the alterations of the passive myocardial properties.

It is possible however, that at least some of these diseases are due to ventricular contractile dysfunction during diastole. Similarly, systolic alterations may be due in some cases to modifications in apical loop activation. These phenomena have very significant clinical and therapeutic implications, and probably heart failure classifications based on the suction phase can be developed, as well as pharmacological, surgical and device-implemented therapies that take into account the regulation of diastolic contractile persistence of the ascending band, the timing and/or synchronization of the two bands' contraction, etc. In fact, the pathophysiological basis of cardiac resynchronization therapy could correspond to this phenomenon. We have also rescued the value of the apex in the solution of ventricular reduction techniques, as well as the anatomical speculation of the myocardial band in relation to the different functioning of the basal and apical loops, the former committed to the ejective phase and the second one to suction and filling phases. The approach is open, mainly because we agree that an enlarged heart has no adequate suction phase and consequently a subsequent inefficient contraction. This active mechanism of the ventricular myocardial band on the diastolic effect opens a wide scenario for surgical repair techniques both of the heart shape and volume and the resultant left ventricular function.(6)

3. Surgical perspectives

Ventricular dilatation is an adaptive condition to different cardiovascular diseases. As a result, there is cardiac remodeling presenting increased wall tension and progressive dilatation. In the myocyte this process leads to irreversible stretching with subsequent deviation of the pressure/volume curve to the right, increased ventricular volumes and mitral regurgitation. This continuous dilatation determines increased risk of mortality in these patients.(10) Therefore, the study of altered ventricular geometry in heart failure is of major interest for the practice of new surgical therapies that try to restore this situation in order to improve prognosis.

Leonardo da Vinci (1452-1519) had already drawn geometrical designs for his analyses of the left ventricle and aortic root.(79) On the other hand, William Harvey, in his anatomical *"Exercitatio anatomica de motu cordis et sanguinis in animalibus"* work (1628), made a description of the left ventricle with a long narrow shape during the ejective phase, tending towards a sphere during diastole.(79) The interest in these

studies dates back to Woods in 1892, but it is in the mid-twentieth century when Burton found that the increase in cardiac volumes coupled to a larger ventricular internal radius implies a greater wall tension or stress. (81)

Various surgical techniques have been put into clinical consideration to solve the issue both of ventricular dilatation as well as of the post-myocardial non-contractile areas. Their analysis shows the need for a strategy to return the left ventricle to its native configuration through an ellipsoidal reconstruction. The rationales involved in restoring left ventricular geometry to an ellipsoidal shape are: a) geometrical; b) anatomical; c) functional and d) volumetric.

a) Geometrical. 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 obtain the highest mechanical performance. The ellipsoidal shape presented by the normal left ventricle is characterized by the presence of a major diameter and two smaller diameters of equal dimensions (Figure 1).

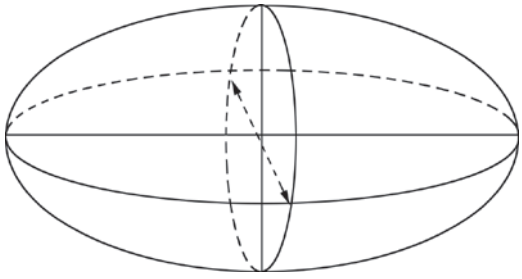

Figure 1

b) Anatomical. The studies carried out by Torrent Guasp (68) have been fundamental to couple the heart's anatomical structure to cardiac mechanics. In his description, the ventricular chambers are defined by a myocardial band that describes two spiraling turns extending from the root of the pulmonary artery to the aortic root. In this helical configuration a descending and another ascending band must be differentiated. Thus, the ventricular myocardial band describes two coils, which means that the ventricles are the cavities of a circular musculature. The contraction is exerted on a movable fulcrum which is represented by the content limited by the ventricular myocardial band proper. i.e., there are no extrinsic fulcrums (e.g. skeletal muscles) for muscle contraction which is supported by the intraventricular volume (hemoskeleton) allowing both blood ejection as storage functions. On this point, Torrent

Guasp expressed: *"Heart mechanics is homologous to that of blood vessels' circular fibers, which carry out their function without fixed fulcrums"*.(68)

c) *Functional*. Physically, intraventricular pressure exerts on the wall that contains it a tension that is described by the law of the Marquis Pierre de Laplace (1749-1827). This equation determines that wall tension (T) is directly proportional to transmural pressure (P) and to the vessel radius (r) and inversely proportional to the thickness of the vascular wall (w): T = Pr/w.

This physical principle supports the valuable general concept developed with ventricular reduction techniques.(87-88)

d) Volumetric. It becomes imperative to manage not only cardiac shape (ellipsoidal) but also volume; the higher the volume, the higher the sphericity and vice versa. When 20% of ventricular mass is injured remodeling starts triggering volume overload (Figures 2 and 3).

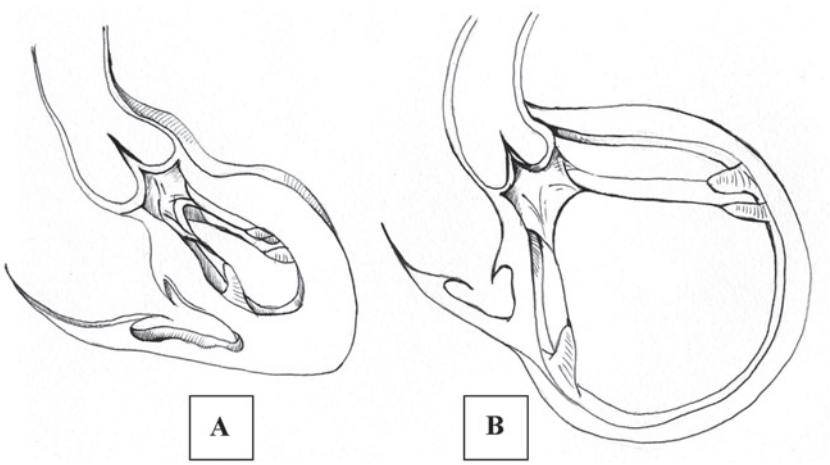

Figure 2. A: normal (ellipsoidal); **B:** dilated (spherical).

It is essential to know that ventricular sphericity leads to increased wall stress which acts in three directions: meridional or longitudinal, circumferential and radial. When there is ventricular dysfunction the highest stress increase is longitudinal. These findings not only apply to patients with idiopathic dilated cardiomyopathy but also in those with ischemic heart disease with depressed ventricular function, aortic regurgitation, mitral regurgitation, ventricular septal defects, and aortic stenosis with depressed ventricular function. Moreover, these changes in left ventricular geometry are leading determinants to the appearance of functional mitral regurgitation in both ischemic and idiopathic heart disease.

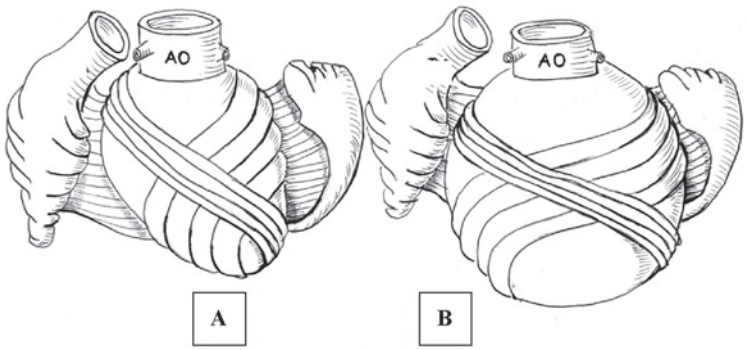

Figure 3. **A:** normal (ellipsoidal); **B:** dilated (spherical). The dilated heart shows separation between the descending and ascending bands.

In view of this situation, we have implemented ellipsoidal reconstruction according to a technique that links the anatomical and functional geometrical bases presented here.(80,81) The **left ventricular ellipsoidal repair technique** we have researched and performed (80,81) assumes the possibility of restoring ventricular geometry within the shape needed for its mechanical function. It consists of the following steps (Figure 4):

1) A longitudinal incision along the anterior descending artery in the avascular left ventricular wall (Figure 5).
2) The left incision margin is brought by means of a continuous suture to the level of the preserved interventricular septum (Figure 6).
3) The remaining right margin is sutured to the left ventricular free wall (Figure 7).
4) Both the original incision as well as the size of the flaps must observe the cavity to be preserved in order to reduce ventricular volume.

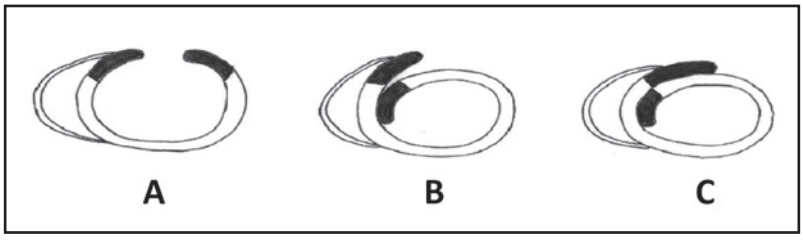

Figure 4. Diagram of the successive steps in ellipsoidal reconstruction.

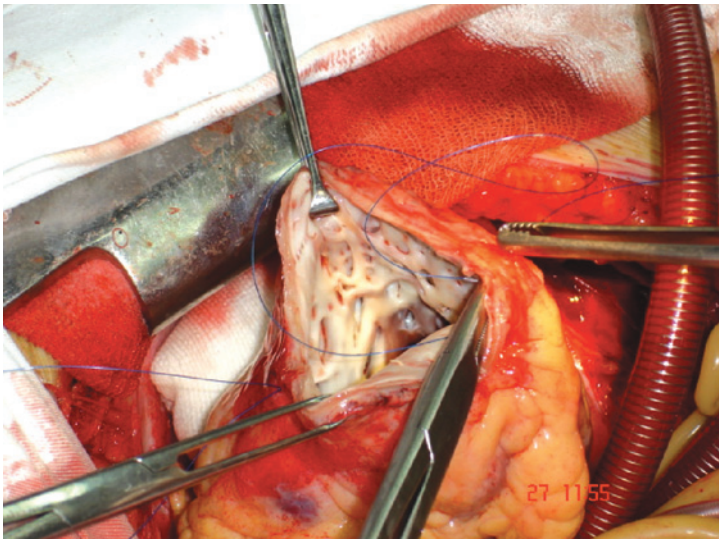

Figure 5. Longitudinal incision in the avascular left ventricular wall showing the aneurysmal area to be excluded when the flap with myocardial margins is formed.

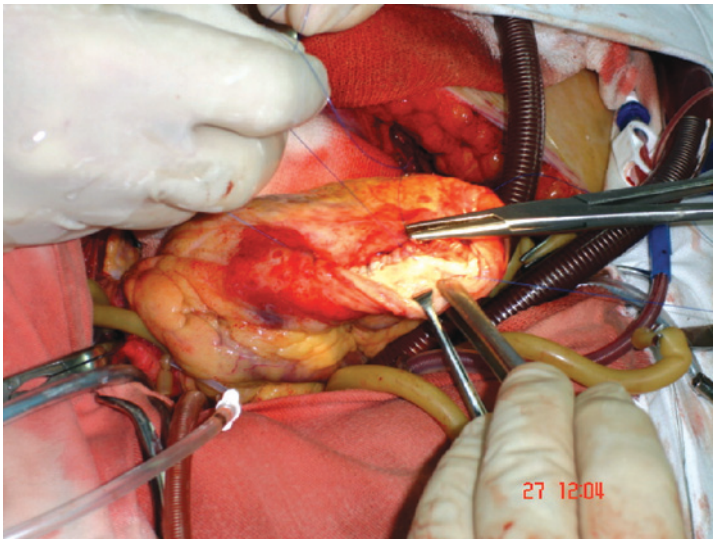

Figure 6. Internal suture procedure. The left incision margin is brought to the interventricular septum. The unsutured margin will be conveyed to the left ventricular free wall.

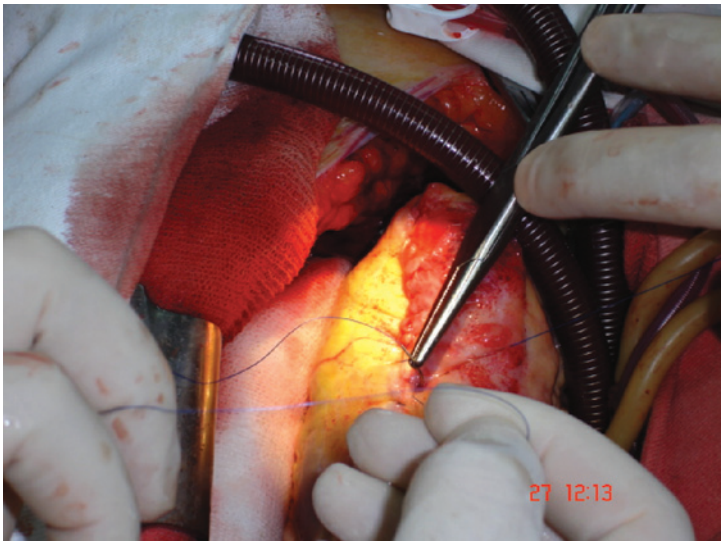

Figure 7. External suture procedure. The right margin with the part of the excluded septum is sutured to the left ventricular free wall.

This technique adds a number of benefits when compared to traditional techniques (Jatene, Dor, Batista) (10,29,31) and follows the strategy presented by Matsui (43) and Menicanti (44,45):

a) Preservation of the heart muscle by acting on the area bounded by the ascending and descending segments of the ventricular myocardial band according to the work of Torrent Guasp. (68) In this geographical tip of the heart, called the apex, a virtual conduct is defined in which the endocardium attaches to the epicardium constituting a weak zone favorable to dyskinesias.(66, 78)

b) This topographical heart area is avascular, which prevents pulling the arterial system during resection.

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

d) The geometrical flap effect made with this technique has the effect of a ventricular girdle.

e) The flap built with the overlapping incision margins excludes the remodeled area (anterior and septal tip) leaving the cardiac

apex with new vitality to bear heart pressure as it is rebuilt with healthy tissue.

f) In the thin apical septum with distortion produced by remodeling, this technique eliminates dyskinesia, providing septal rigidity.

g) This technique does not involve placing synthetic patches, thus avoiding noncontractile areas on the left ventricular contraction surface.

h) This surgical approach approximates the ascending and descending apical loop segments, which are separated during heart failure, contributing to improve ventricular function.

i) We undertook treating both septal distortion and diastolic function with this technique.

j) The recovery of the heart's ellipsoidal shape might act upon the structurally altered mitral apparatus. We have experienced that once the ventricle is reduced, the insufficient valves did not require surgical repair.(78)

Conclusion. The restitution of ventricular geometry has stimulated both the use of techniques to assess it as well as having solved spatial distortion in heart failure.(29) Certainly the sphericity to which the left ventricle turns during heart failure has an ominous prognostic value. Ventricular geometry is a sensitive marker of function and prognosis. Be it cause or consequence, the presence of spherical geometry in the patient determines increased oxygen consumption through increased wall stress. It is certainly the consequence, but it also carries the blame of perpetuating the alteration.

4. Electrophysiological therapy perspectives

Throughout time, there were always situations in daily practice where classical pathophysiology was not enough to interpret them correctly. Often, only after the appearance -and acceptance- of diametrically opposed and resisted alternatives to those currently existing, therapeutic interpretations and appropriate and effective behaviors were achieved. From Harvey's seminal description to the implantable cardioverterdefibrillator, passing through heart surgery and the use of beta blockers in heart failure, there appeared new concepts which marked milestones in cardiology. Sometimes these ideas were implemented immediately

but in other cases they were described years earlier and had to wait for further studies to prove their validity. This is probably the case of Torrent Guasp's model. In the research presented in this text his initial ideas have been summarized, completed and reinterpreted in the light of new evidence.

The clinical implications of this model remain to be discussed. It will probably take years to verify them in the various syndromes and heart diseases. However, a situation in which its application may result immediate is in the loss of ventricular synchrony and its counterpart, biventricular resynchronization therapy.

In patients with end-stage heart failure of different etiologies, this is significantly aggravated by biventricular dissynchrony. When left bundle branch block occurs in an already severely damaged heart, left ventricular contraction is delayed. Therefore, cardiac contraction ceases to be simultaneous or synchronous and becomes dissynchronous, further worsening cardiac mechanics.

Resynchronization Therapy (RCT) is a procedure that restores biventricular synchrony. This resynchronization is achieved via a heart stimulator similar to a sequential pacemaker, which stimulates not only the right ventricle but the left ventricle as well. However, to achieve proper resynchronization the stimulation of the left ventricular chamber is not enough; it must also be achieved from specific sites or "effective areas", typically the medial lateral or posterolateral area of the left ventricle.

The usual catheter implantation procedure is coronary sinus cannulation and then a tributary vein that reaches the "effective" area. However, this method has numerous problems and disadvantages failing in 20% to 30% of patients in whom RCT is performed. These patients have been referred to as "non-responders".

Before proceeding, it is important to note the significance of this circumstance. Resynchronization Therapy is a crucial procedure. It is performed in patients with end-stage heart failure in whom all other available pharmacological and surgical treatment options were ineffective. If it is successful, a significant change is achieved in the quality of life and survival of the patient; its failure means remaining in Functional Class III-IV. Although cardiac transplantation could be raised as an option, in practice only very few patients have the opportunity of accessing to it.

In this pathology and its therapy there are extraordinarily remarkable elements:

1. Despite the tens of thousands of patients treated with this method, current cardiac mechanics has failed to clearly elucidate the concept of biventricular dissynchrony. Even in cases of very severe mechanical cardiac dysfunction, the most advanced diagnostic methods, including the most sophisticated echocardiography, gamma camera or MRI techniques cannot diagnose this disease, at least with a useful and reliable sensitivity and specificity. The diagnosis is still based on the presence of left bundle branch block, logically in the context of dilated cardiomyopathy in Functional Class III or IV. Even this apparently permanent and classical criterion is under discussion, debating whether its morphology ("typical" or "atypical"), its critical duration, or some other as yet not elucidated criterion is important.
2. The same applies for the evaluation of RCT effectiveness. None of the mentioned techniques has been able to document a parameter with a sufficiently significant and early variation to consistently correlate with the almost surprising improvements in the clinical outcome of patients in whom RCT is successful.

In our opinion these "failures" are due to the fact that the assessed cardiac parameters of classical mechanics are neither responsible for the pathology nor for the therapeutic response. On the contrary, it is very attractive to reconsider these phenomena in the light of the recent findings on stimulus propagation and subsequent cardiac mechanics resulting from this investigation.

The princeps "dyssynchrony" parameter is, as we have pointed out, left bundle branch block. This phenomenon, which we again mention, hardly explains the severity of mechanical disorders, on the other hand would seriously alter the mechanics of the ascending band. In fact, it would seem very logical to say that a fault in the activation at the level of "band intersection" could generate the image of left bundle branch block (and its "atypical" forms) and of course seriously alter the activation sequence of the ascending band, but not in the way postulated by the "classical" conception. There would be an alteration in the "radial" activation of the descending endocardial band to the ascending epicardial band and of the "bidirectional longitudinal" activation of the epicardial band. The phenomena that these activations cause in cardiac twisting at the level of the apical loop and in the active protodiastolic ventricular suction mechanism have already been explained in detail.

We insist that the usual methods of ventricular mechanical assessment, especially of the left ventricle, usually do not take into account

these essential factors. Therefore, it would not be surprising that these factors lack the accuracy to evaluate ventricular function alteration as well as its improvement with RCT.

The left ventricular stimulation catheter positioning has critical importance in the remarkable RCT effectiveness and in the failure of the current methods used. It has been suggested that the best position of the catheter is the one corresponding to the late ventricular activation area; although in many cases this is true, in others it is not. There is agreement, however, that the anatomical location in the medial area of the left ventricular posterolateral or lateral surface is effective whether or not it coincides with late potentials. This fact could be interpreted consistently with Torrent Guasp's model and our findings, as this location corresponds to the point of band intersection. The catheter would activate the left ventricle at the precise point required to "restore" the normal activation of the ascending segment in its double progression towards the apical loop and the ventricular base.

Finally, there is another fact that strongly reinforces this interpretation; we have indicated that the normal pathway for left ventricular catheter implantation is through the coronary sinus, which gives access to the left ventricular epicardial veins, so that activation propagates from the endocardium to the epicardium. Although the catheter is properly located in the mentioned "effective area", there are some "non-responder" patients. In recent studies (Alsync trial) (47) 20% of patients are non-responders although the catheter implanted through the coronary sinus is perfectly positioned. When implanted through an endocavitary pathway, 50% of these patients become responders. The explanation using the classical conception, is a fairly vague consideration in the sense that endo-epicardial activation is "more physiological" than the reverse.

From the perspective of our research, the explanation is much more consistent: the activation of the ascending band at the point of band intersection restores its longitudinal activation, with the beneficial consequences mentioned above. However, the radial activation from the descending to the ascending band and the persistent normal distal activation of the descending segment is lost. Endocavitary stimulation (descending segment) would almost completely restore the normal electrical activation and therefore its mechanical result. Based on this principle, at Hospital PresidentePerón de Avellaneda we have been researching endocavitary RCT for several years. This has already been employed in 34 patients, with excellent surgical and clinical outcomes.

In conclusion, Torrent Guasp's hypotheses and fundamental anatomical model, extended and modified in our electromechanical research offers countless opportunities for the development of both theoretical and clinical and therapeutic applications. It is possible that much of cardiology should be reviewed in the light of this new paradigm, with perhaps unpredictable results.

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