

Electrical Propagation in the Mechanisms of Torsion and Suction in a Three-phase Heart

Propagación eléctrica en los mecanismos de torsión y succión en un corazón de tres tiempos

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ABSTRACT

Background: The hypothesis of Torrent Guasp considers that the ventricular myocardium consists of a continuous muscular band that begins at the level of the pulmonary valve and ends at the level of the aortic root, limiting both ventricular chambers. This anatomy would provide the interpretation for two fundamental aspects of left ventricular dynamics: the mechanism of left ventricular torsion and rapid diastolic filling due to the suction effect.

Objectives: The aim of this study was to investigate the electrical activation of the endocardial and epicardial bands to understand ventricular torsion, the mechanism of active suction during the diastolic isovolumic phase and the significance of the residual volume.

Methods: Five patients underwent three-dimensional electroanatomic mapping. As the descending band is endocardial and the ascending band is epicardial, two sites of puncture were used.

Results: Three-dimensional endo-epicardial mapping demonstrates an electrical activation sequence in the area of the apical loop in agreement with the synchronic contraction of the descending and ascending band segments. The simultaneous and opposing radial activation of the ascending band segment, starting in the descending band segment, in the area in which both band segments intertwine, is consistent with the mechanism of ventricular torsion. The late activation of the ascending band segment is consistent with its persistent contraction during the initial period of the isovolumic diastolic phase (the basis of the suction mechanism), and takes place without need of postulating further electrical activation after the QRS complex.

Conclusions: This study explains the process of ventricular torsion and the suction mechanism, and demonstrates that the activation of the ascending band segment completes the QRS, ruling out the traditional concept of passive relaxation during isovolumic diastole.

Key words: Heart/physiology - Cardiac Electrophysiology - Diastole

RESUMEN

Introducción: La hipótesis de Torrent Guasp plantea que los ventrículos están conformados por una banda muscular continua que nace a nivel de la válvula pulmonar y se extiende hasta la raíz aórtica delimitando las dos cavidades ventriculares. Esta anatomía brindaría la interpretación para dos aspectos fundamentales de la dinámica ventricular izquierda: el mecanismo de torsión y el llenado diastólico rápido por efecto de succión.

Objetivos: Investigar la activación eléctrica de las bandeletas endocárdica y epicárdica para comprender la torsión ventricular, el mecanismo de succión activa en la fase isovolumétrica diastólica y el significado del volumen residual.

Material y métodos: La investigación se realizó mediante un mapeo electroanatómico tridimensional en cinco pacientes. Al ser la bandeleta descendente endocárdica y la ascendente epicárdica, se utilizaron dos vías de abordaje por punción.

Resultados: El mapeo tridimensional endoepicárdico demuestra una activación eléctrica de la zona de la lazada apexiana concordante con la contracción sincrónica de las bandeletas descendente y ascendente. La activación simultánea y contrapuesta de la bandeleta ascendente con punto de partida de su activación radial desde la bandeleta descendente, en la zona de entrecruzamiento de ambas, es coherente con la torsión ventricular. La activación tardía de la bandeleta ascendente se compatibiliza con la persistencia de su contracción durante el período inicial de la fase isovolumétrica diastólica (base del mecanismo de succión); se produce sin necesidad de postular activaciones eléctricas posteriores al QRS.

Conclusiones: Este trabajo explica el proceso de la torsión ventricular y el mecanismo de succión. Comprueba que la activación de la bandeleta ascendente completa el QRS anulando el concepto tradicional de relajación pasiva en la fase isovolumétrica diastólica.

Palabras clave: Corazón/fisiología - Electrofisiología cardíaca - Diástole

Abbreviations

EAM Electroanatomic mapping

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INTRODUCTION

The hypothesis of Torrent Guasp considers that the ventricular myocardium consists of a continuous muscular band that begins at the level of the pulmonary valve and ends at the level of the aortic root, limiting both ventricular cavities. Two band segments can be differentiated: the endocardial descending segment and the epicardial ascending segment, which describe a helix with two spiral turns forming a basal loop (right and left segments) and an apical loop (descending and ascending segments). In this spatial arrangement, the descending and the ascending band segments cross at a point that we called “crossing of band segments” (1, 2) (Figures 1 and 2). This anatomical feature has a figure of 8 shape and its sequence of stimulation explains fundamental aspects of left ventricular dynamics: 1) the mechanism of ventricular torsion; 2) the physiology of rapid diastolic filling due to the suction effect, and 3) the residual volume. Despite the anatomical and functional studies hitherto performed, a correlation with the epicardial and endocardial activation sequences in humans has not been evaluated. (3, 4)

Ventricular filling has always been considered a passive ventricular function. A mechanism has been postulated, by which during the initial phase of diastole (isovolumic diastole) the ventricle actively sucks blood due to persistent muscular contraction of the “ascending band segment”. This mechanism would produce apex-base lengthening with an abrupt decrease of intraventricular pressure (isovolumic diastole) until the atrioventricular valves open and the rapid filling phase starts. (5-7) Many aspects of this theory have been argued, mainly due to the lack of an electrophysiological basis evaluating cardiac activation. (8) The aim of this study was to investigate the sequence of electrical activation of the endocardial and epicardial bands to understand ventricular torsion, the mechanism of active suction during the diastolic isovolumic phase and the significance of the residual volume.

METHODS

The sequence of left ventricular endocardial and epi-

cardial electrical activation was studied by three-dimensional electroanatomic mapping (EAM) with the Carto navigation and mapping system (Biosense Webster, California, USA), which allows a three-dimensional anatomical representation with activation maps and electrical propagation. Isochronic and activation sequence maps were constructed and correlated with the surface electrocardiogram. Ventricular activation maps were built with 50 ± 8 endocardial and epicardial points, providing detailed high density recordings. Apical, lateral and basal views were obtained.

All patients had sinus rhythm, normal QRS and no structural cardiomyopathy by Doppler echocardiography and resting and stress myocardial perfusion scintigraphy tests.

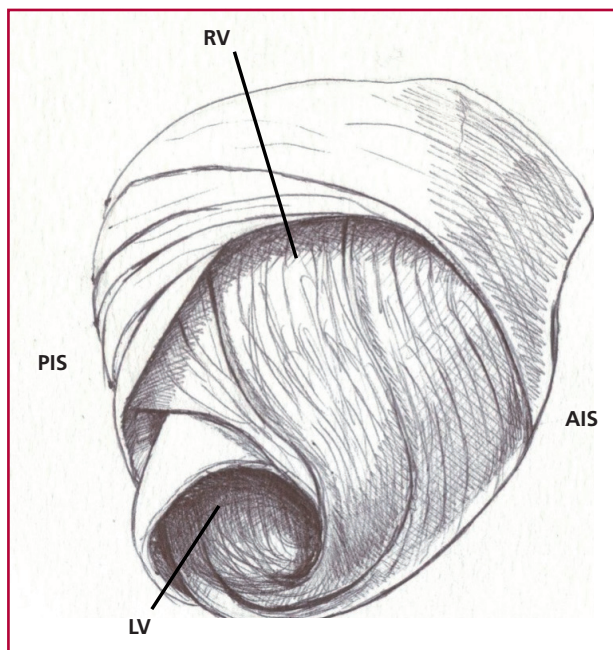


Fig. 1. Torrent Guasp's ventricular myocardial band. RV: Right ventricle. LV: Left ventricle. PIS: Posterior interventricular sulcus. AIS: Anterior interventricular sulcus.

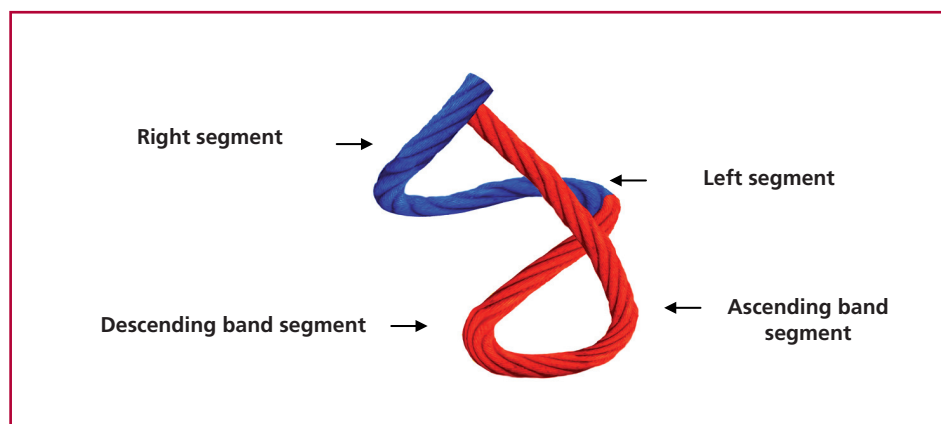


Fig. 2. Different segments of Torrent Guasp's ventricular myocardial band. In blue: basal loop. In red: apical loop.

The EAM was performed during radiofrequency ablation of arrhythmias due to probable abnormal epicardial pathways. Mapping was performed at the beginning of the studies, followed by the ablation procedures. No complications were encountered. The presence of abnormal pathways did not interfere with the mapping, since baseline sinus rhythm was maintained throughout the entire procedure.

As the descending band is endocardial and the ascending band is epicardial, two sites of puncture were used for mapping. The endocardium was accessed by conventional transeptal puncture and the epicardial access was obtained by a percutaneous pericardial approach (9) using an ablation catheter (Navistar® F curve, Biosense Webster). Endocardial and epicardial mappings were successively performed. They were then superimposed, synchronizing the results with electrocardiographic temporization to obtain the simultaneous mapping of both ventricular surfaces.

Ethical considerations

The study was performed at Hospital Presidente Perón, (Buenos Aires, Argentina), and included patients who signed an informed consent previously approved by the Institutional Ethics Committee.

RESULTS

Isochronic mapping

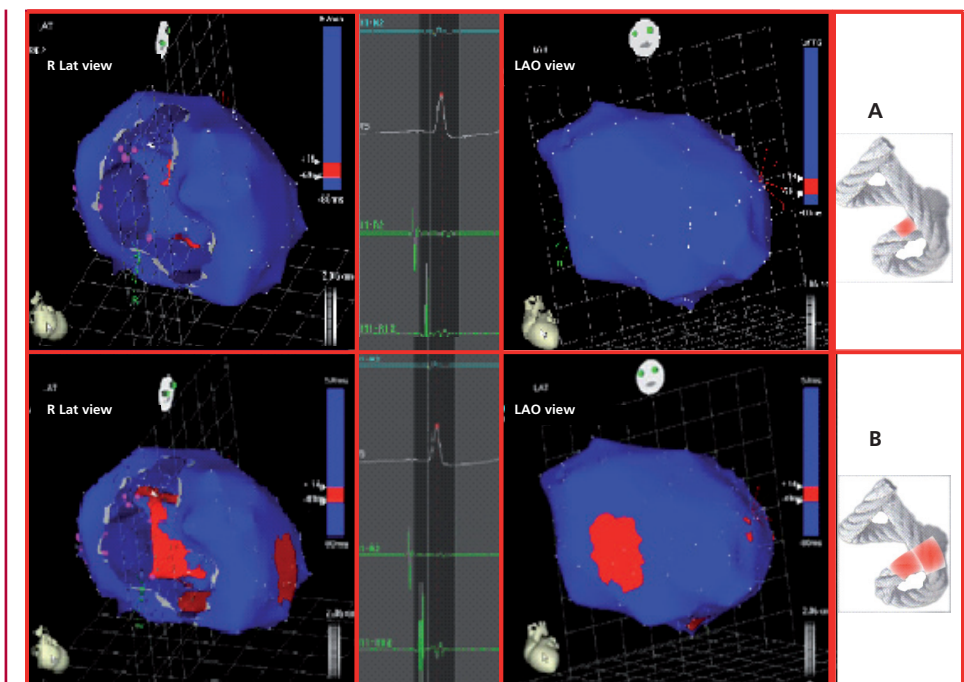
Three-dimensional mapping allowed detailed recording of the activation sequence. As the EAM corresponded to the left ventricle, the previous activation wavefront originating in the right ventricle was not recorded.

Activation sequence

Figures 3 to 5 show the propagation of electrical endocardial and epicardial activation. In all the figures, the left panel shows the right lateral projection and the right panel shows the simultaneous left anterior oblique projection. The activated zones at each moment are shown in red. The lateral part represents the activation of the band segments in Torrent Guasp’s rope model. The depolarized area at that moment is represented in red and those areas previously activated and in refractory period are represented in blue. (Figure 2)

Left ventricular activation is initiated in the interventricular septum (Figure 3 A). It then extends axially towards the ventricular apex, following the anatomical arrangement of the descending band segment. An epicardial area is also activated at that moment –the ascending band segment- evidencing radial activation at a point that we called the “crossing of band segments” (Figure 3 B). This finding, as stated in the discussion section, modifies the Torrent Guasp model and constitutes the electrical basis of the mechanical phenomenon of ventricular torsion. After the “crossing of band segments”, the activation loses its unidirectional character and becomes more complex. Figure 4 A shows three simultaneous wavefronts: 1) the distal activation of the descending band segment towards the apical loop; 2) the depolarization of the ascending band segment from the crossing point towards the apex; and 3) the activation of this band segment from the crossing point towards the final end of the muscular band in the aorta. Figures 4 B, 5 A and B show the progression and the end of this process.

Fig. 3. Onset of left ventricular activation. The left panel shows the depolarization of the interventricular septum, corresponding to the descending band segment. In the right panel, the ventricular epicardium (ascending band segment) has not been activated yet. B. Simultaneous activation of the band segments. The activation progresses in the left ventricular septum through the descending band segment (axial activation) and simultaneously propagates towards the epicardium (radial activation) activating the ascending band segment.



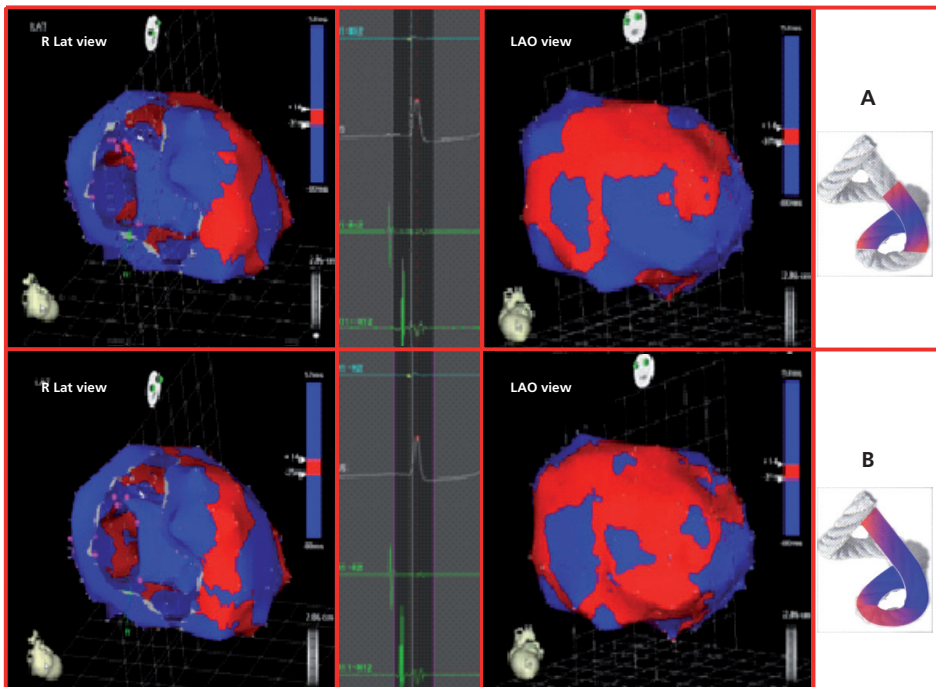


Fig. 4. A. Bidirectional activation of the apex and the ascending band segment. The panel shows the end of septal activation, extending towards the apex, synchronously with the epicardial activation in the same direction. At the same time, the epicardial activation is directed towards the base of the left ventricle. **B.** Progression of activation. The panel illustrates the progression of activation in the directions of the previous panel.

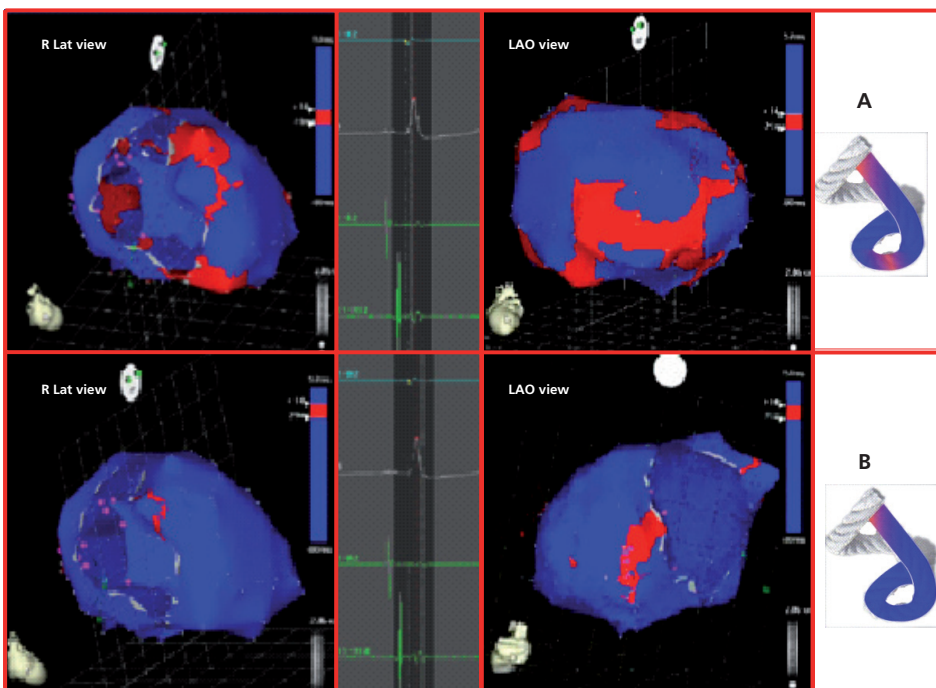


Fig. 5. A. Late activation of the ascending band segment. At this moment, corresponding to approximately 60% of QRS duration, subendocardial activation (descending band segment) is already complete. The distal portion of the ascending band segment (epicardial segment) is depolarized later. This phenomenon correlates with its persistent contraction during the initial diastolic phase. **B.** Final activation. The right panel shows the very late activation of the distal portion of the ascending band segment in a modified left anterior to left posterior-lateral oblique projection.

Figure 5 A shows that the activation of the endocardial descending segment finishes well before the end of the QRS, and the rest of this process corresponds to late activation of the distal portion of the ascending band segment, which explains its persistent contraction during the isovolumic diastolic phase, basis of the mechanism of ventricular suction (Figure 5 B). Figure 6 shows a summary of the activation with this rope model.

DISCUSSION

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

According to Torrent Guasp, the longitudinal diffusion of the stimuli along the ventricular myocardial

Fig. 6. Rope model. Upper panel: Activation sequence (A-F) of Torrent Guasp’s ventricular myocardial band according to our investigation. In red: depolarization; in blue: repolarization. Lower panel: Unidirectional propagation of the excitation wavefront (in red) in the ventricular myocardial band according to Torrent Guasp (A-D)

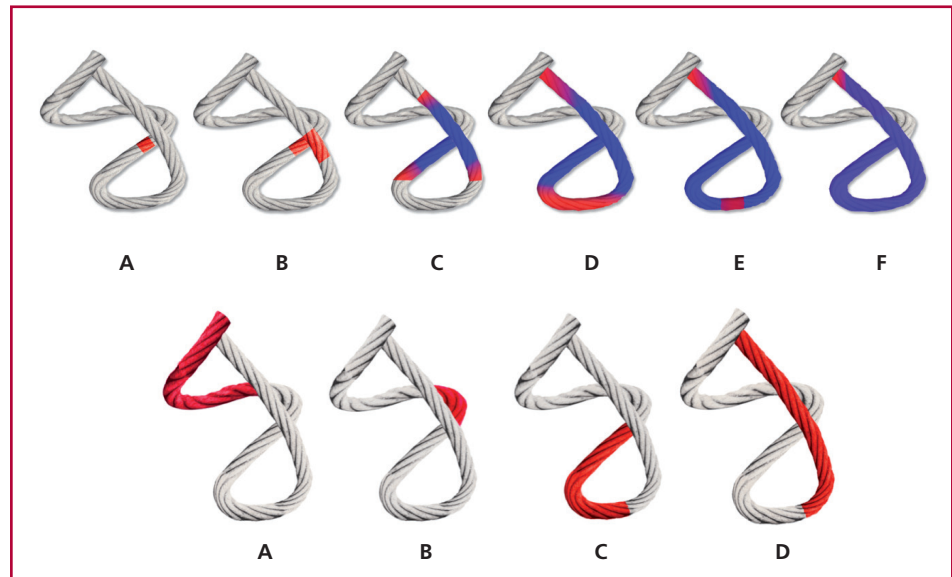
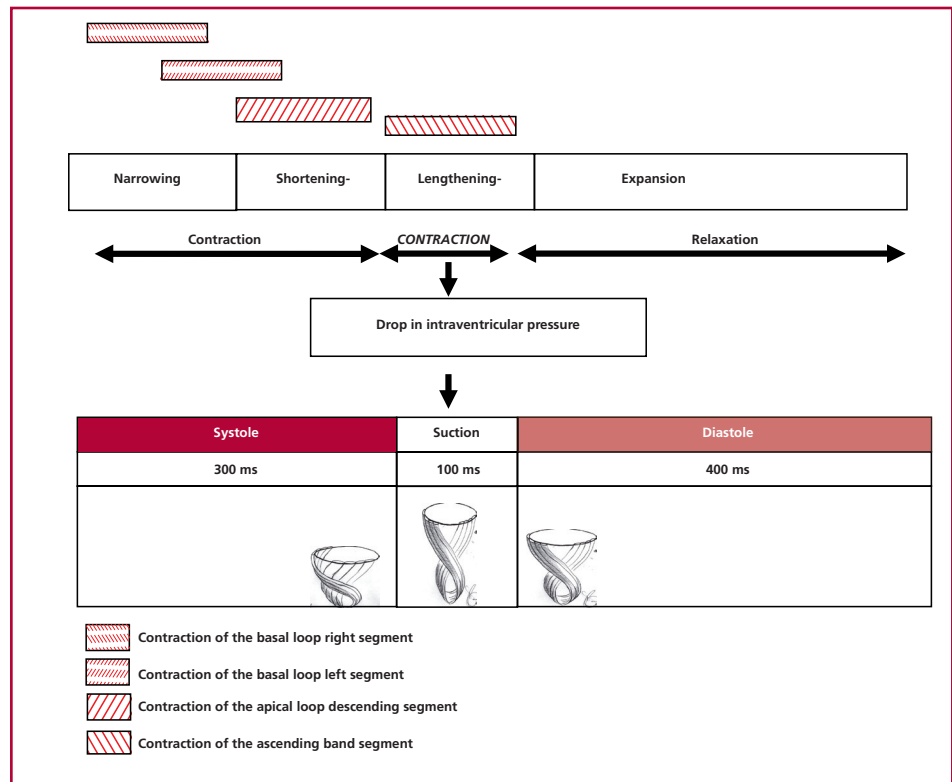


Fig. 7. Effects produced by cardiac excitation-contraction coupling.



band explains heart function (Figure 7). (2) However, this “peristaltic” sequential activation does not correlate with some currently well-known fundamental phenomena, such as opposing clockwise and counterclockwise torsion of the left ventricular apex and base, which are mainly responsible for its mechanical efficiency (Figure 6). The electrical activation of the heart is the consequence of the propagation of stimuli through the muscular structure of the heart, including Torrent Guasp’s ventricular myocardial band and

the rest of the myocardial fibers. The cardiac mechanism of suction and ejection requires an integrated structural-functional relationship interpreting several aspects of its dynamics due to the propagation of excitation. This explains that the isovolumic diastolic phase is an active process of contraction, during which the ventricle actively sucks blood by a suction mechanism similar to a “plunger”, (10) until a certain level of intraventricular pressure opens the atrioventricular valves producing the rapid filling phase.

Stimulus propagation and left ventricular torsion

In this study, three-dimensional endocardial and epicardial EAM confirms the model of activation propagating across the descending and ascending band segments. Figures 3 to 5 help to elucidate the sequence of activation of the contractile areas and how this sequence leads to cardiac dynamics in relation to the propagation of the excitation wavefront with a coordinated pattern according to muscular structure.

We found that the activation sequence is different from that described by Torrent Guasp, but explains ventricular torsion, defined as the reverse rotational movement of the base and apex. At the crossing point of both band segments, activation spreads from the endocardium towards the epicardium (radial propagation), that is, from the descending to the ascending band segment. From the anatomical point of view, this passage could be mediated by “interband segment fibers” (Torrent Guasp’s “aberrant fibers”). (11)

From this point, the ascending band segment is depolarized in a dual direction; towards the apex and towards the base, at the same time as the descending band segment completes its activation towards the apex (Figure 5). Thus, two fundamental phenomena take place:

1. The apical loop is depolarized from the crossing point of both band segments with two simultaneous wavefronts (from the descending and the ascending band segments) generating their synchronized contraction.
2. The activation of the ascending band segment extends from the crossing point in two opposite directions: towards the apex and towards the base (see Figure 5). The ensuing mechanical contraction will also have a reverse sense, giving origin to clockwise and counterclockwise rotations of the apex and base.

For Lewis and Rotschild, (12) conduction of stimuli occurred from the endocardium to the epicardium through the ventricular walls. On the contrary, in 1936 Robb (13) reported that the excitatory process was conducted axially, and in 1942 he wondered: “Why is it possible that the impulses are conducted from the endocardial surface to the epicardial surface if the ventricular wall has well differentiated bundles separated by connective tissue sheaths?” (14) Surprisingly, in experimental studies, Armour and Randall (1970) concluded that in the left ventricular anterior wall the impulse is conducted from the endocardium to the epicardium. (15) This local situation in the left ventricular anterior wall contrasts with previous ideas and with the rest of the muscular mass in which the electrical activity of the subepicardial muscle bundles precedes that of the subendocardial muscle bundles. In 1980, Torrent Guasp reported: “the onset of fiber contraction is earlier at endocardial layers because they are activated by the descending band segment, while the subepicardial layers are activated later by the ascending band segment” (16) and in 1998 he confirmed: “the descending

band segment is activated before the ascending band segment”. (17) By 2001, Buckberg and Torrent Guasp corroborated the hypothesis that excitation runs unidirectionally along the ventricular myocardial muscle band (Figure 6). (18)

Our investigation modifies these concepts, as the activation is simultaneously axial and radial. This sequence of activation has an important anatomic component in cardiac architecture that was not evidenced by Torrent Guasp; (2) what he considered as “aberrant fibers”, we prefer to call them “interband segment fibers”, as they may be the anatomical site of radial activation from the descending band segment to the ascending band segment.

The phase of systolic narrowing (isovolumic contraction phase) starts with the contraction of the right and left segments of the basal loop. The shortening phase is produced by the descending movement of the ventricular base with simultaneous ventricular torsion. This longitudinal contraction starts at the basal ring and continues towards the apex. The mechanism of descent and ascent of the ventricular base makes the apex remain motionless. This is explained because the ascending segment, which is stiff during systole and the initial phase of diastole, acts as a tight rod keeping the apex motionless. The pressure generated to eject the greatest amount of blood at the beginning of the ejection phase and for a time period that occupies only 20% of systole is feasible due to ventricular torsion. This action is possible because the electrical activation progresses towards the descending band segment (axial activation) and simultaneously activates the ascending band segment (radial activation). Although the electrical impulse propagates through the ventricular myocardial band, the radial activation towards the ascending band segment plays a key role in ventricular torsion because it allows opposing contraction forces in the longitudinal axis which generate the intraventricular pressure necessary for rapid blood ejection. The interband fibers connecting the descending and ascending band segments would be responsible for conducting the impulse from one band segment to the other. This mechanism of torsion is similar to “wringing a towel”. (19)

The meaning of electrical activation and contraction was not considered in the historical description of systole and diastole (20), and only the hemodynamic concept of ejection and ventricular filling were taken into account. We must find a relationship between activation and mechanics. The explanation lies in the trajectory of the simultaneous axial and radial activation reaching the crossing of band segments and in the spatial orientation of the myocardial fibers (the subendocardial fibers to the right and the subepicardial fibers to the left). (21) This orientation is consistent with the evolutionary loop of the circulatory system giving origin to the two ventricles in birds and mammals. Ventricular torsion, defined as the reverse rotational movement of the base and apex generates: a) in-

creased pressures, b) reduced ventricular stress, and c) homogenization of its distribution in wall thickness.

Torrent Guasp stated that “the sequence of ventricular muscle action of the different ventricular regions takes place along the ventricular myocardial band”, (17) resembling a peristaltic movement (Figure 6). Then, how could the ventricle provoke its torsion movement if this action requires two simultaneous and opposing forces? Unidirectional activation does not explain ventricular torsion or the evolutionary development of a structure designed to exert a force capable of ejecting the ventricular volume at a velocity of 300 m/s with low energy cost. We have found the answer in the simultaneous axial and radial activation.

Active suction in the isovolumic diastolic phase

There was no evidence to support ventricular filling as an active phenomenon, and we have found that it is produced by myocardial contraction with apex-base lengthening of the left ventricle after the ejection phase causing a suction mechanism similar to a “plunger”. The rationale for this mechanism would be the persistent contraction of the ascending band segment during the isovolumic diastolic phase.

We have found that the endocardium is completely depolarized during the first part of the QRS. In turn, Buckberg et al. found that the mechanical contraction triggered by this electrical phenomenon starts 50 ms later and persists for approximately 350 ms. If the depolarization of the ascending band segment starts 50 ms after that of the descending one and its contraction persists for the same period of time, ventricular contraction will last for approximately 400 ms. If ventricular systole lasts around 300 ms, the remaining 100 ms correspond to the isovolumic diastolic phase (sometimes called isovolumic relaxation time, although ventricular contraction does exist). Briefly, during the initial part of diastole, the ascending band segment would remain contracted due to the depolarization occurring over the course of the QRS. Thus, the explanation of this delayed contraction does not require depolarizations after the QRS, as Pedro Zarco assumed. (22)

In our investigation, the final part of the QRS corresponds to the activation of the ascending band segment, (see Figure 5) which would enable its persistent contraction during the isovolumic diastolic phase to generate a suction mechanism (“plunger effect”). With the beginning of ventricular untwisting during the isovolumic diastolic phase, progressive lengthening of the ascending band segment generates a negative intraventricular pressure while the ascending band segment is still contracted (active process), a residual energy of the torsion process.

Interpretation of the active suction phase

The sequence of activation here analyzed explains the presence of a phase of active suction between systole and diastole, with muscular contraction, energy

expenditure and abrupt decrease of intraventricular pressure (Figure 7). This effect allows blood to enter the ventricular chamber due to a pressure difference with the peripheral circulation, and accounts for 70% of ventricular filling in only 20% of the filling time.

This phase of active suction between systole and diastole lasting 100 to 200 ms is due to muscular contraction with a drop in intraventricular pressure below zero (see Figure 7). Sonnenblick (23) and Tyberg (24) reported that after balloon occlusion of the mitral valve in the dog, left ventricular diastolic pressure declines below zero. As a result, the heart is a dynamic suction pump that requires a limitation of elastic recoil to allow the subsequent systole to be effective. This mechanism of left ventricular suction has been demonstrated by the efficacy of right heart bypass (Fontan-Kreutzer procedure), (25) as well as by mechanical support with univentricular assist devices where blood is drawn from the left ventricle into the aorta. (26-28)

In the traditional model, ventricular filling is only determined by venous pressure. In fact, atrial pressure is too low to explain this situation. Based on this “key doubt”, Torrent Guasp developed the concept of a suction pump supported by the physiological and muscular structure that he described. (29) The contraction of the final portion of the ascending segment generates the mechanism of blood suction towards the left ventricle. The high inflow velocity at low ventricular pressures demonstrates that suction is an active phenomenon.

Residual systolic blood volume

Residual systolic blood volume represents 30% of the total end-diastolic blood volume. From the point of view of fluid mechanics, between systole and diastole, the left ventricle is a closed chamber filled up with incompressible blood so that its volume cannot change regardless of the degree of muscle contraction. Therefore, this phase is isovolumic, and as muscular contraction cannot modify the blood volume, intraventricular pressure decreases favoring diastolic filling (“plunger effect”). There is a range of residual systolic blood volume which is optimal for suction. If blood volume is higher, muscle contraction must increase to create the necessary fall in blood pressure. On the contrary, if blood volume is lower, the interaction between the walls will hinder the suction mechanism and the boundary layer phenomena in diastole will alter ventricular filling.

Study limitations

The results of the present study are clear but the low number of patients is a limitation. Yet, the physiological processes do not need a high number of experiences as in clinical studies. In addition, these results could be reinterpreted with studies with a larger number of cases or with experimental studies in animals with better control of variables. Despite this may be

a limitation, the fact that this study was performed in humans and not in animals makes it more reliable. Another aspect to consider is that the discussion could be considered speculative, as anatomic or hemodynamic interpretations were based on an electrophysiology study without performing morphological dissections or measuring pressures, volumes or dimensions. In fact, it should be mentioned that Torrent Guasp conducted the first phase of this experience with anatomical data and that Buckberg's interpretations were based on pressure measurements. However, it was necessary to prove the electrophysiological aspect to determine a new cardiac physiology, as the direction of electrical conduction determines the real function. All these considerations were cleared up in the present study and should be continued in future investigations.

CONCLUSIONS

This study demonstrated the existence of a "three-phase heart": systolic phase, suction phase and diastolic phase. The information here presented is particularly relevant as it was obtained in humans with structurally normal hearts and under physiological conditions. According to the study findings, we may conclude that:

1. Three-dimensional endo-epicardial mapping demonstrates an electrical sequence of activation in the area of the apical loop in agreement with the synchronic contraction of the descending and ascending band segments.
2. The simultaneous and opposing activation of the ascending band segment from the starting point of its radial activation from the descending band segment is consistent with the simultaneous reverse rotation of the apical and basal areas (mechanism of ventricular torsion).
3. The late activation of the ascending band segment is consistent with its persistent contraction during the initial period of isovolumic diastole (untwisting and suction, and takes place without need of postulating further electrical activations after the QRS complex.

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Conflicts of interest

None declared

(See author's conflicts of interest forms in the web / Supplementary Material)

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