Anatomy & Physiology: Current Research

ISSN: 2161-0940

Trainini, Anat Physiol 2015, 5:S4
DOI: 10.4172/2161-0940.S4-001

Research Article Open Access

Electrophysiological Bases of Torsion and Suction in the Continuous Cardiac Band Model

Jorge Trainini [1], **Benjamín Elencwajg** [1*], **Néstor López Cabanillas** [1], **Jesús Herreros** [2] **and Noemi Lago** [1]

[1]*Hospital Presidente Perón, Buenos Aires, Argentina*

[2]*Universidad Católica San Antonio (UCAM), Murcia, Spain*

[*]**Corresponding author**: Benjamín Elencwajg, Department of Surgery, Hospital Presidente Perón, Buenos Aires, Sanchez de Loria 1865, 1241 CABA Argentina; Tel: +54 11 49114472; E-mail: belencwajg@yahoo.com

Rec date: Jun 01, 2015; **Acc date:** Jul 07, 2015; **Pub date:** July 15, 2015

Abstract

Objective: Torrent Guasp concept, that postulates that the ventricles are formed by a continuous muscle band, provides the basis for two aspects of left ventricular dynamics: The torsion mechanism and the rapid early diastolic filling by the suction effect. We investigated the electrophysiological basis of this mechanism, by electroanatomical left ventricular endoepicardial mapping (EEM) in five patients, during radiofrequency ablation.

Results: The apical loop is activated by a simultaneous depolarization of the distal descending and proximal ascending band segments. At the point of crossing of both bands a radial activation spreads from the descending to the ascending band. From this point, begins a simultaneous and opposing activation of the proximal and distal ascending band. The activation of the distal ascending band segment is the latest.

Conclusions: The activation sequence of the continuous band provides an electrophysiological basis for the ventricular torsion and suction mechanism.

Keywords: Torrent Guasp; Myocardial band; Ventricular torsion; Ventricular suction; Active isovolumic diastolic phase; Three phases heart; Electrophysiologic activation

Abbreviations

EEM: Endoepicardial mapping, LV: Left ventricle

Introduction

The Torrent Guasp concept postulates that the ventricles are formed by a continuous muscle band that originates at the level of the pulmonary valve and extends to the aortic root, limiting in this way the two ventricular chambers. Two muscle segments can be differentiated: the endocardial descending and the epicardial ascending band segments describing 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 ascending band segments cross at a point that we called "crossing of band segments" (Figure 1).

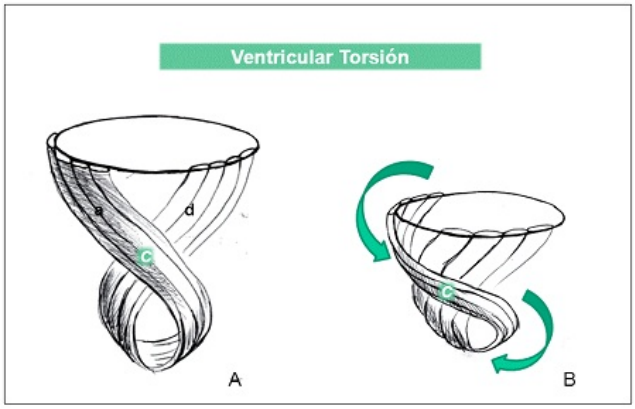

Figure 1: Ventricular torsion. **A:** Resting ventricular state. **B:** The subsequent mechanical contraction produces torsion in reverse directions between the ventricular apex and base. **C:** crossing of band segments; d: descending band segment; a: ascending band segment.

This specific anatomical arrangement would support the interpretation of two fundamental aspects of left ventricular dynamics:

1) The torsion mechanism and

2) The physiology of rapid diastolic filling by the suction effect [1-3].

Although these mechanisms are currently well characterized, there are as yet no studies to clarify their electrophysiological basis in humans [4-7]. The purpose of this work was to study the electrical activation sequence of the left ventricular (LV) endocardial and epicardial band segments that support this concept.

Material and Methods

The sequence of LV endocardial and epicardial electrical activation was studied by 3D electroanatomical mapping (EAM) with the Carto (Biosense Webster, California, USA) navigation and mapping system, which allows a three-dimensional representation with activation maps and electrical propagation. Isochronic and activation sequence maps were correlated with the surface ECG. Ventricular activation maps were built with 50 ± 8 endocardial and epicardial points, providing high density detailed recordings. Apical, lateral and basal views were obtained.

The study was performed at the Hospital Presidente Perón, in Buenos Aires, Argentina. Five patients with informed consent previously approved by the Institutional Ethics Committee were included in the study. All patients had sinus rhythm, normal QRS and no structural cardiomyopathy by Doppler echocardiography and resting and exertion gamma camera studies. No nuclear magnetic resonance or coronariography studies were performed, as there was no history of cardiomyopathies or ischemic heart disease.

The 3D EEM was performed during radiofrequency ablation of arrhythmias associated to probable abnormal epicardial pathways. Mapping was performed at the beginning of the studies, followed by routine ablation procedures. No complications were encountered. Presence of abnormal pathways did not interfere with the mapping, since baseline sinus rhythm was maintained during the whole procedure.

As the descending band segment was endocardial and the ascending one epicardial, two approaches were used to perform mapping. The endocardium is accessed by a conventional transseptal puncture and the epicardium by a percutaneous pericardial approach according to the technique described by Sosa et al. [8]. Mapping of the pericardial cavity was done using an ablation catheter (Navistar® F curve. Biosense Webster, California, USA).

Endocardial and epicardial mappings were successively performed. They were then superimposed, synchronizing results with electrocardiographic temporization, to obtain the simultaneous mapping of both ventricular surfaces.

Results

Isochronic mapping

Three-dimensional mapping allowed detailed activation recordings. Although the activation sequence varied in its details, the general aspects were similar in every case, establishing an endo-epicardial depolarization matrix. As EEM corresponded to the left ventricle, the previous activation wave originating in the right ventricle was not recorded.

Activation sequence

Figures 2 to 4 show propagation of endocardial and epicardial activation. In all the figures, the left panel shows the right lateral projection and the right panel the simultaneous left anterior oblique projection. Activated zones at each moment are indicated in red. The vertical white line in the reference QRS illustrates the activation moment corresponding to each figure. The electrograms (green traces) correspond to the reference temporal electrogram (above) and to the activated area at that moment (below). The lateral part represents band segment activation in the Torrent Guasp loop (chord model). In this figure, the depolarized area at that moment is represented in red and those areas previously activated and in refractory period are represented in blue.

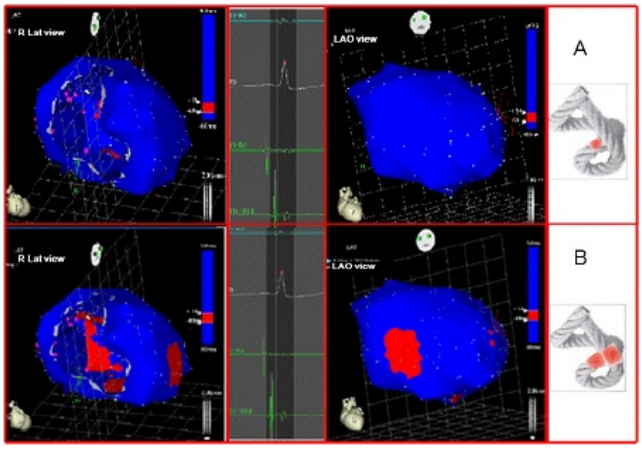

Figure 2: A: Onset of left ventricular activation. The left panel shows interventricular septum depolarization, corresponding to the descending band segment. In the right panel, the ventricular epicardium (ascending band segment) has not been activated yet. B: "Crossing" of band segments. The activation progresses in the left ventricular wall through the descending band segments (axial activation) and simultaneously propagates towards the epicardium (radial activation) activating the ascending band segment.

Left ventricular activation is initiated in the interventricular septum (Figure 2A). 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 called "crossing of band segments" (Figure 2B). This finding, as stated in Discussion, partly modifies the Torrent Guasp model and would constitute the electrical basis of the ventricular mechanical torsion phenomenon. After the "crossing of band segments", the activation loses its unidirectional character and becomes more complex. Figure 3 A shows 3 simultaneous wave fronts:

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 from the crossing point towards the opposite end.

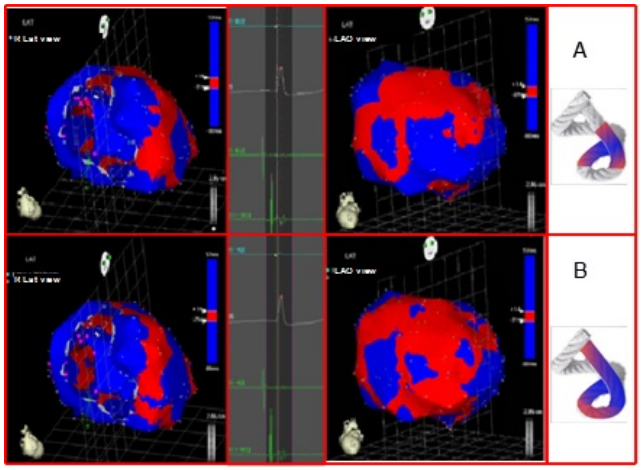

Figure 3: A: Bidirectional activation of the apex and the ascending band segment. The panel shows the end of septal activation, extending towards the apex, synchronously with the epicardial activation in the same direction. At the same time, the epicardial activation is directed towards the base of the left ventricle. **B:** Propagation progression. The panel illustrates the progression of activation in the directions of the previous panel.

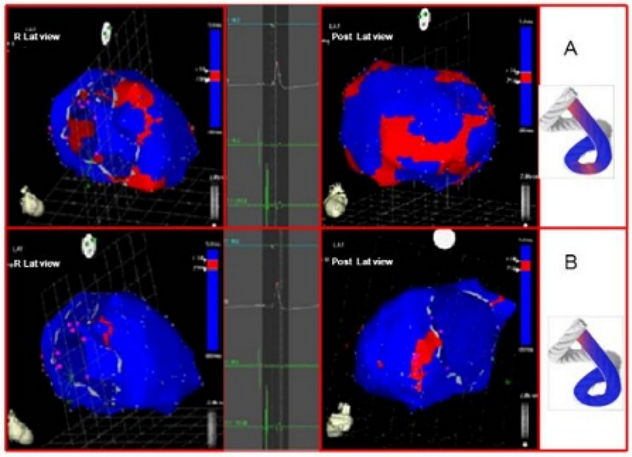

Figure 4: A: Late activation of the ascending band segment. At this moment, corresponding to approximately 60% of QRS duration, subendocardial activation (descending band segment) is already complete. The distal portion of the ascending band segment (epicardial segment) depolarizes later. This phenomenon 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.

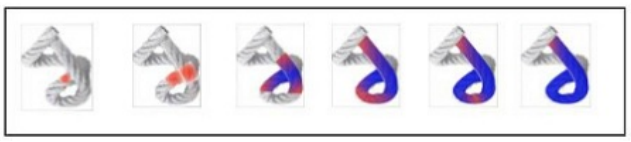

Figure 5: A: Chord model. Activation sequence of the Torrent Guasp muscle band.

Figures 3B to 4A and 4B show the progression and end of this process. It can be seen that endocardial activation 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 ventricular suction mechanism.

Discussion

Once the primary objective of the investigation -the activation sequence- was analyzed, it had to be correlated with two of the fundamental mechanical processes of the heart, as systolic torsion and diastolic suction. Regarding torsion, the Torrent Guasp concept of a continuous myocardial band 3 provides an attractive alternative to the classic anatomical and functional concept. According to this theory, cardiac mechanics implies a succession of muscular movements that take place one after the other along the muscle band, producing LV stretching, shortening, torsion, lengthening and expanding movements during the cardiac cycle. Torrent Guasp expresses "the succession of functional events related to cardiac mechanics, represent unequivocal proof of the longitudinal diffusion of stimuli along the ventricular myocardial band" 3.

However, this "peristaltic" sequential activation does not explain some currently well-known fundamental phenomena, such as opposing clockwise and anticlockwise LV apex and base torsion, mainly responsible for its mechanical efficiency [9-11].

Ventricular torsion is defined as the reverse rotational movement of the base and apex. This work shows a hitherto not described activation sequence that would explain ventricular torsion. 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, and more precisely, from approximately the mid-third of the descending band segment to the mid-third of the ascending one. From the anatomical point of view, this passage could be mediated by "inter band segment fibers" (Torrent Guasp "aberrant fibers") [12].

From this point, the ascending band segment depolarizes 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:

The apical loop depolarizes from the crossing point of both band segments with two simultaneous wavefronts (from the descending and the ascending band segments) probably generating their synchronized contraction.

The activation of the ascending band segment extends from the crossing point in two opposing directions: towards the apex and towards the base (Figure 5). The ensuing contraction will also have a reverse sense, giving origin to clockwise and anticlockwise rotations of

the apex and base. This would explain the high pressure that allows ejecting the greatest blood volume at the onset of ventricular ejection during a period that involves only 20% of this phase (Figure 1).

Ventricular filling is usually considered a passive function, produced by ventricular relaxation, the elastic fibers´ action and the "vis a tergo" [13] some authors, however, question that these mechanisms are sufficient to explain rapid ventricular filling, especially of the left ventricle, during the first milliseconds after atrioventricular valve opening. An anatomo-functional mechanism has been postulated, by which during the initial phase of diastole (isovolumic diastolic phase) the ventricle actively aspirates blood. This effect would be produced by apex-base lengthening during this phase with the consequent abrupt decrease of intraventricular pressure, causing or favoring the initial rapid filling phase by a suction mechanism similar to a "plunger", followed by diastolic relaxation of the ventricular wall and slow filling. The rationale for this mechanism would be the persistent contraction of the ascending band segment during the initial phase of diastole, called the isovolumic diastolic phase. Although Torrent Guasp presents convincing anatomical evidence, his works lack functional studies to similarly evaluate his concept [4-14].

The activation of the ascending band segment occurs after depolarization of the descending band segment, a delay that is due to the propagation of activation from the endocardium to the epicardium (Figures 2A and B).

We have shown in this study that the endocardium depolarizes completely during the first part of the QRS. In turn, Buckberg et al. [10] 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, the ventricular contractile state will last approximately 400 ms. If ventricular systole lasts around 300 ms, the remaining 100 ms correspond to the isovolumic diastolic phase (sometimes called isovolumic relaxation). Briefly, during the initial part of diastole, the ascending band segment would remain contracted due to the depolarization occurring during the course of the QRS. Thus, the explanation of this delayed contraction does not require depolarizations after the QRS.

In the present study, the final part of the QRS corresponds to the activation of the ascending band segment, which would enable its persistent contraction during the isovolumic diastolic phase, generating a suction mechanism through the "plunger effect" (Figure 5). According to these results, we may infer:

Endo-epicardial 3D mapping shows an electrical activation sequence in the area of the apex loop in agreement with the synchronic contraction of the descending and ascending band segments.

The simultaneous and opposing activation of the ascending band segment to 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 (ventricular torsion mechanism).

Late activation of the ascending band segment, compatible with its persistent contraction during the initial phase of isovolumic diastole, takes place without need of postulating electrical activations after the QRS.

The novel activation sequence of the Torrent Guasp muscle band found in the present work, would explain the previous process triggering ventricular torsion and the suction mechanism. Moreover, it establishes that the activation of the ascending band segment completes the QRS. This finding explains the persistent contraction of this muscle segment during the first part of diastole, ruling out the traditional concept of passive relaxation.

With respect to clinical implications, the study postulates a "three-phase heart": systole, suction and diastole. We consider that the data obtained are particularly important as they were recorded in humans, with structurally normal hearts and in physiological conditions (not experimental). It remains to be seen what happens in different pathologies.

During the course of the last years, the pathology of ventricular filling and diastolic failure has become important. In this regard, a great number of studies have focused on the altered passive properties of the myocardium.

It is possible however, that at least some of these pathologies are due to ventricular contractile dysfunction during diastole. Similarly, systolic alterations could be attributed in some cases, to changes in the apical loop activation. The corroboration of this phenomenon could have clinical and therapeutic consequences. It would be possible to develop heart failure classifications based on the suction phase, as well as pharmacological or surgical therapies or treatments with any device that consider the regulation of the persistent diastolic contraction of the ascending band segment, the temporization and/or synchronization of contraction of both band segments, etc. Actually, the pathophysiological basis of cardiac resynchronization therapy could correspond to this phenomenon. We have also rescued the value of the apex in the resolution of ventricular reduction techniques as well as anatomical speculations of the great band on the different functions of the basal and apical loops, the first one more involved with the ejection phase and the second one with the filling and suction phase [15,16]. The path remains open, especially since we know that a dilated heart does not have an adequate suction phase and hence an efficient contraction. The active mechanism of the great myocardial band on diastolic effect opens a wide field for surgical repair techniques, both of the shape and volume, and hence the function of the left ventricle [17].

References

1. Wiggers CJ, Katz LN (1922) The contour of the ventricular volume curves under different conditions. Am J Physiol 58: 439-7
2. Brecher GA (1954) Cardiac variations in venous return studied with a new bristle flowmeter. Am J Physiol 176: 423-430.
3. Torrent-Guasp F (1998) [Structure and function of the heart]. Rev Esp Cardiol 51: 91-102.
4. Coghlan C, Hoffman J (2006) Leonardo da Vinci's flights of the mind must continue: cardiac architecture and the fundamental relation of form and function revisited. Eur J Cardiothorac Surg 29 Suppl 1: S4-17.
5. Armour JA, Randall WC (1970) Structural basis for cardiac function. Am J Physiol 218: 1517-1523.
6. Robb JS, Robb RC (1936) The excitatory process in the mamalian ventricle. Am J Physiol 115:43-52.
7. Sallin EA (1969) Fiber orientation and ejection fraction in the human left ventricle. Biophys J 9: 954-964.
8. Sosa E, Scanavacca M, d'Avila A, Pilleggi F (1996) A new technique to perform epicardial mapping in the electrophysiology laboratory. J Cardiovasc Electrophysiol 7: 531-536.
9. Cosín Aguilar J, Hernándiz Martínez A, Tuzón Segarra MT, Agüero Ramón-Llin J, Torrent Guasp F(2009) Estudio experimental de la

llamada fase de relajación isovolumétrica del ventrículo izquierdo. Rev Esp Cardiol;62:392-9.

10. Buckberg GD, Coghlan HC, Torrent-Guasp F (2001) The structure and function of the helical heart and its buttress wrapping. V. Anatomic and physiologic considerations in the healthy and failing heart. Semin Thorac Cardiovasc Surg 13: 358-385.
11. Buckberg G (2004) Ventricular structure and surgical history. Heart Fail Rev 9: 255-268.
12. Torrent Guasp F, Buckberg G, Carmine C, Cox J, Coghlan H et al. (2001) The structure and function of the helical heart and its buttress wrapping. The normal macroscopic structure of the heart. Seminars in Thorac and Cardiovasc Surg 13:301-19.
13. Sonnenblick EH (1980) The structural basis and importance of restoring forces and elastic recoil for the filling of the heart. Eur Heart J Suppl A: 107-110.
14. Zarco P (2001) [The ventricular rapid filling phase: a muscle relaxation or contraction process?]. Rev Esp Cardiol 54: 1031-1032.
15. Trainini JC, Herreros J (2011) ¿El corazón es una bomba de succión? Rev Argent Cardiol:79:39-46.
16. Mandinov L, Eberli FR, Seiler C, Hess OM (2000) Diastolic heart failure. Cardiovasc Res 45: 813-825.
17. Brutsaert DL, Sys SU, Gillebert TC (1993) Diastolic failure: pathophysiology and therapeutic implications. J Am Coll Cardiol 22: 318-325.

This article was originally published in a special issue, entitled: **"Physiology"**, Edited by Dr. Robert A Walker