Research Article

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Ventricular Complementarity

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Abstract

Objective: *Through research that we have carried out continuously in recent years and that includes the sequence of cardiac activation, anatomical studies, physiological experiences on the protodiastolic phase, ventricular septal interdependence, clinical and experimental research on negative intraventricular pressure and the suction mechanism, we reach the understanding of biventricular complementarity.*

Methods: *The endo- and epicardial electrical activation sequence of the left ventricle has been studied using three-dimensional electroanatomical mapping with a Carto navigation that allows a three-dimensional anatomical representation, including five patients.*

Results: *Diastole is divided into two phases. In the first, the ventricular volume does not vary, it is isometric, but with energy expenditure its morphology changes generating intraventricular negative pressure, call protodiastolic phase of myocardial contraction. During the second phase, ventricular filling occurs without energy expenditure.*

Conclusions: *The asynchrony between the RV and LV cycles allows the interaction between expulsion and loading of each of them.*

Keywords: Cardiac suction; Myocardium; Proto-diastolic phase; Complementarity ventricular.

Introduction

The mechanical activity of the heart is complex because it is the result of integrating its ejective, suction and filling properties through the helical continuous myocardium under different successive and related phases. Anatomical texts describe the heart globally, but devote scant attention to the arrangement adopted by the muscle fibers in the spatial myocardial construction. It is impossible to interpret cardiac function if the morphological structure within its organizational pattern is not taken into account. (Trainini et al., 2023).

Through the continuous research we conducted in recent years, including left ventricular electrical activation sequence, anatomical studies and physiological experiences on protodiastolic phase of myocardial contraction (PPMC) (isovolumetric relaxation), septal ventricular interdependence, clinical and experimental research on negative intraventricular pressure and suction mechanism, the cardiac cycle, left ventricular (LV) and right ventricular (RV) filling curves, the comparative echocardiographic study between normal patients and those with heart failure with preserved ejection fraction (HFpEF), it is possible to understand the biventricular complementarity in which the active suction of both ventricles is a fundamental point. (Trainini et al., 2023; Trainini et al., 2019; Trainini et al., 2022; Trainini et al., 2024).

According to our investigations, as both ventricles have two active phases, suction and ejection, that must be fulfilled by a single chamber each, complementarity, the result of pressure interaction at the beginning of the cardiac cycle, plays an elementary role in its functional continuity. At this point we can discern that the previous suction during the PPMC, improperly called isovolumic relaxation phase, is what allows in its filling the subsequent expansion of the ventricles. This phase is in complementarity with the systolic property of plunger developed by the opposite ventricle. The objective of this work is the study between expulsion of one ventricle and suction of the opposite one in circulatory movement. The circulatory movement requires the dynamic relationship between ejection of one ventricle and suction of the opposite. As there are two circuits available: systemic and pulmonary, complementarity acts between the LV impulse and the RV suction in the systemic circulation, while the RV impulse coerces with LV suction in the pulmonary circulation.

Material and Methods

The endo and epicardial electrical activation sequence of the LV has been studied by means of three-dimensional electroanatomic mapping (3D-EAM) with a Carto navigation and mapping system that allows a three-dimensional anatomical representation, with activation maps and electrical propagation. Isochronic and activation sequence maps were generated, correlating them with the surface ECG. Endo and epicardial ventricular activation maps were also produced, obtaining detailed high-density recordings with apical, lateral and basal views. The study was conducted at Presidente Perón Hospital (Buenos Aires Province, Argentina) including five patients (4 males) (Table 1) who provided their informed consent. All patients were in sinus rhythm, with a normal QRS and did not have evident structural heart disease by Doppler echocardiography and in gamma camera stress and rest studies. Three-dimensional 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 with normal QRS complexes, both in duration and morphology, without antegrade preexcitation.

Patient	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: Patient characteristics

As the muscular structure of the LV is made up of an endocardial layer (descending segment) and an epicardial layer (left and ascending segments), two approaches were used to carry out mapping. The endocardial access was performed through a conventional atrial transseptal puncture. The epicardial access was obtained by means of a percutaneous approach in the pericardial cavity (Sosa et al., 1996) with an ablation catheter. Endo and epicardial mappings were performed consecutively and immediately and were later superimposed, synchronizing them by electrocardiographic timing. Thus, a simultaneous mapping of both surfaces was obtained. In addition, the propagation times of electrical activation through the myocardium were measured in milliseconds (ms).

Until now, the theory of the continuous myocardium lacked an essential investigation, due to the absence of documentation on the electrophysiological mechanism that would support the mechanical activation sequence of the helical anatomical model. The advent of 3D-EAM managed to overcome this limitation, since it not only allows independent recording of the various ventricular zones, but also those of the endocardium and epicardium, either exclusively or integrated.

We mapped the activation of the left ventricle on its endocavitary and epicardial surfaces according to the methodology described. (Trainini et al., 2023; Trainini et al., 2019; Trainini et al., 2022; Trainini et al., 2015) Mapping

was performed simultaneously with the surface ECG. This provided a unified temporal frame of reference, allowing on the one hand to correlate both records, and on the other hand to obtain a synchronized view of the simultaneous activation observed in various electroanatomical incidents.

Results

Electrophysiological research on cardiac suction. One of the most debated aspects of the suction pump concept is its electrophysiological rationale, namely the alleged lack of correlation between the sequence of the mechanical activity proposed and the electrical activation observed.(Trainini et al., 2015) The advent in recent years of three-dimensional electrophysiological navigators has allowed the receipt of detailed and highly precise information in the activation sequence of the various cardiac structures, a situation that has allowed us to clarify in our research the electrophysiological activation of the continuous helical myocardium, the which we have already published. (Trainini et al., 2023; Trainini et al., 2019; Trainini et al., 2022; Trainini et al., 2015).

The right and left segments make up the basal loop, which, attached at its origin to the anterior face of the cardiac fulcrum (myocardial support) (Trainini et al., 2021; Trainini et al., 2024), is fundamentally made up of circular fibers in a descending direction. At the anterior border of the LV these fibers become the descending segment with a subendocardial position. Upon reaching the apex, they abruptly change their orientation to form the ascending segment to insert into the cardiac fulcrum, mainly on its inferior face (Figure 1).

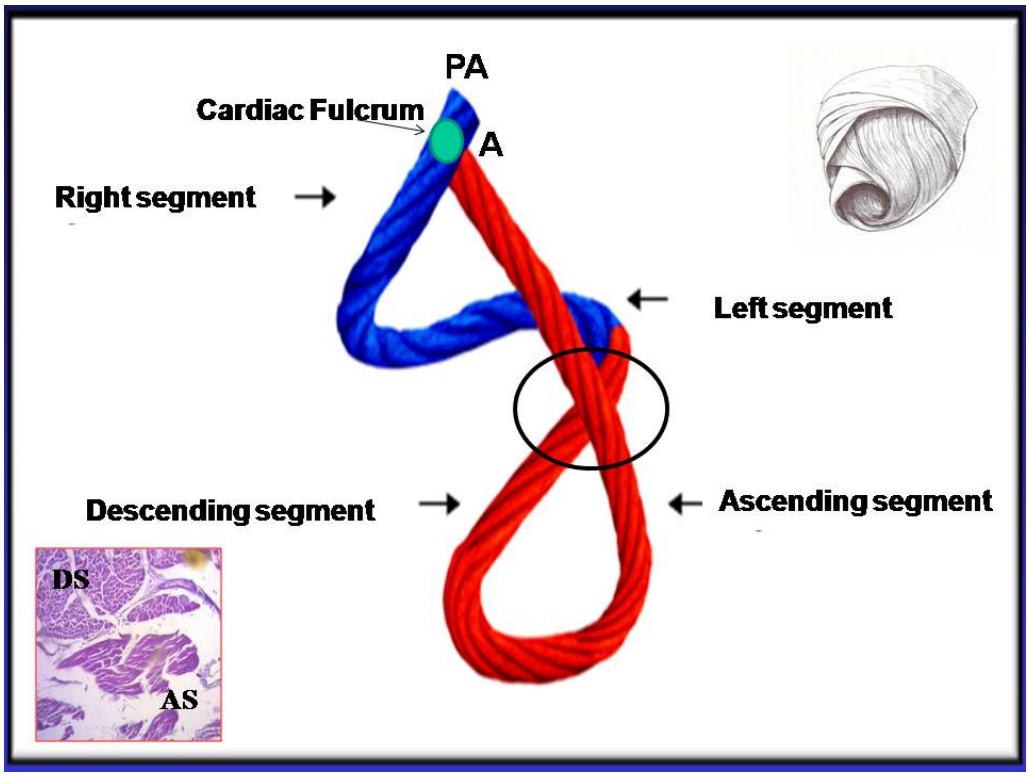

Figure 1: Helical myocardium in the cord model that simplifies the spatial structure. It shows the different segments that compose it. In blue: basal loop. In red: apical loop. PA: pulmonary artery; A: Aorta. The black circle details the site that we have called band cross-over, which is explained in the inset. The histology shows the different orientation of the longitudinal fibers (ascending segment, AS) in relation to the descending (transverse fibers, DS) (bovine heart). Given the different anisotropic orientations of the fibers, this zone corresponds to the beginning of the opposite helical movement that produces myocardial torsion. In the upper corner the spatial arrangement of the continuous myocardium is observed.

The central point consists in postulating that the PPMC constitutes an active phenomenon generated by late myocardial contraction, after closure of the aortic valve, that produces ventricular lengthening, separating the base from the apex.

A very important finding of this investigation was that endocardial activation is fully completed when the surface QRS has barely reached 60% of its duration. The rest, therefore, corresponds to epicardial activation. This late stimulation (lasting around 83 ± 16 ms in our investigations) (Trainini et al., 2023) at the epicardial level, corresponds to the apical loop ascending segment and results in its stiffening during the PPMC through an active process with energy consumption.

According to the records obtained with the three-dimensional mapping of all cardiac activity that we have published from 2015 (Trainini et al., 2015), the electrical impulse propagates along the helical myocardium in order to achieve two opposite forces that facilitate ventricular torsion (helical movement). To this end, the stimulation goes from the descending to the ascending segment with an average delay of 25.8 ms ± 1.483 (Table 2). During the progression of systole, transmission is

longitudinal, producing shortening of the base-apex and radial distance (transverse and anteroposterior) with narrowing of the chamber and coiling of the myocardium, determining ventricular torsion (Figures 2 and 3).

Time	Pat.1	Pat.2	Pat. 3	Pat. 4	Pat. 5	*X*	*SD*
Radial time from descending to ascending time	25	26	24	26	28	25.8	1.483

References. ms: milliseconds; Pat: Paciente; X: Mean; SD: Standart deviation

Table 2: Radial propagation time (in ms)

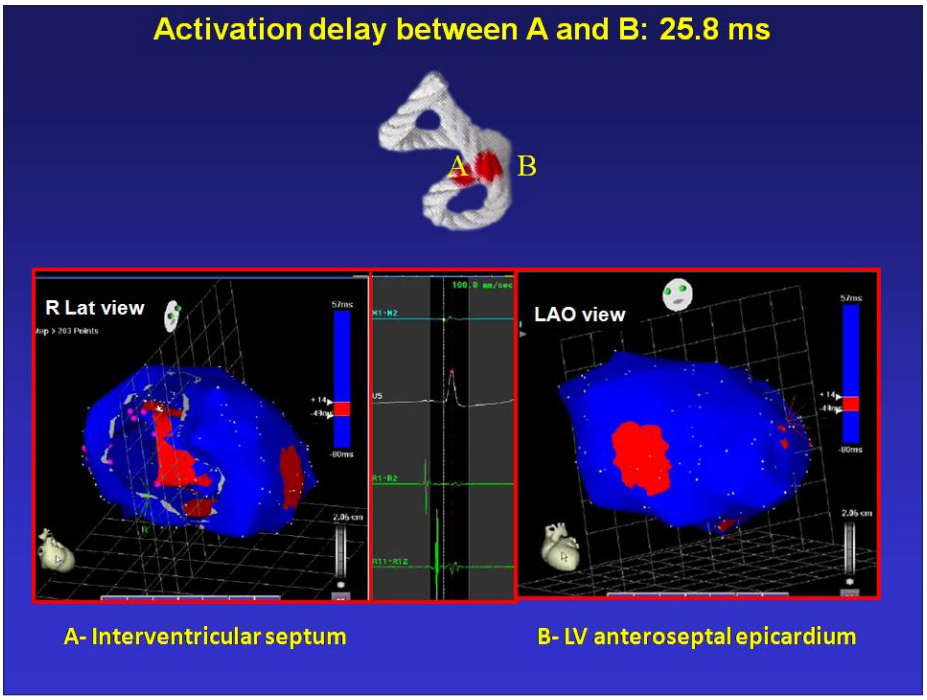

Figure 2: Transverse impulse transmission from the interventricular septum (A) to the left ventricular anteroseptal epicardium (B).

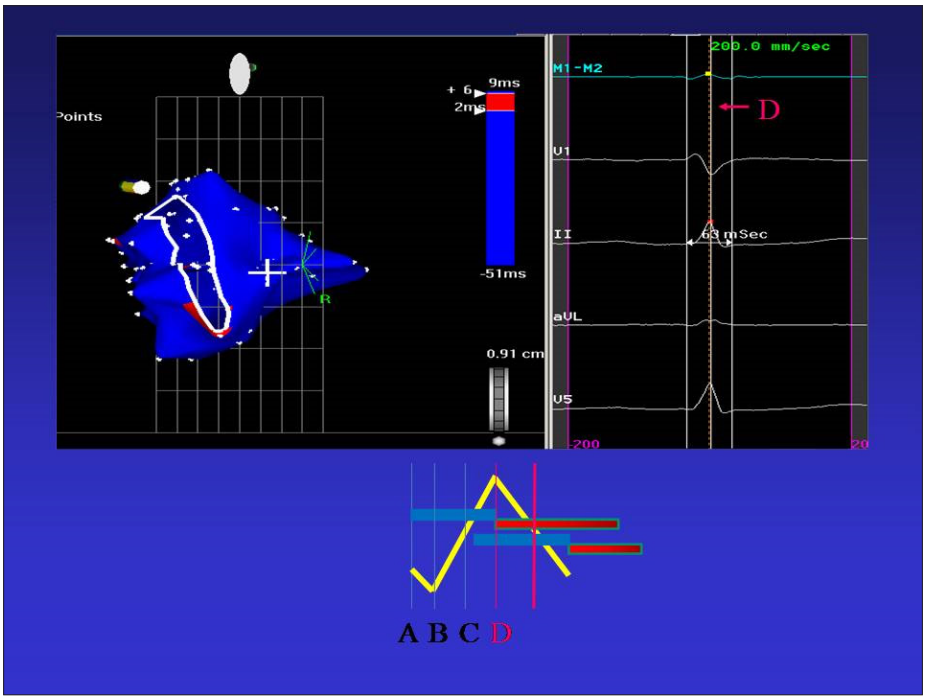

Figure 3: Continuation of the previous figure. The endocardial activation ends in the area corresponding to the mitral annulus. Note that all the endocardial activation "occupies" approximately 60% of QRS duration (D line in the right panel). Epicardial activation has started earlier, but its completion occurs during the final portion of the QRS. The whole sequence is coherent with the mechanical activation sequence described in Proposition 2 and provides its electrophysiological support.

In our studies on three-dimensional electroanatomical mapping we have an asynchrony with an average of 38.2 ms (35-40 ms ± 2.135) between the onset of RV systole with respect to left ventricular systole (torsional movement) (Trainini et al., 2023; Trainini et al., 2019; Trainini et al., 2022). The pulmonary valve (PV) opens first than the Aortic valve (AV). This result agrees with the literature consulted, estimating a delay of 40 ms. (Mangione et al., 2022).

Once systole is over, during the initial instant of the PPMC, the ascending band contracts and the descending band repolarizes. This late contraction of the ascending segment allows, in the PPMC, the corresponding mechanics to achieve the process of ventricular detorsion-lengthening and suction, generating the conditions of drop in intraventricular pressure necessary to achieve blood aspiration.

The basal loop (contraction of the right and left segments) determines ventricular narrowing, while contraction of the descending segment together with the ascending segment causes the shortening-torsion movement of systole. (Torrent-Guasp et al., 2001; Torrent-Guasp et al., 2005). All these physiological processes are required for the ejection phase. In the continuity of cardiac activity, the contraction of the apical loop ascending segment, by providing ventricular lengthening, establishes the PPMC process, which generates intraventricular negative pressure (suction phase) through a "suction cup"-like or suck-up mechanism. Ventricular expansion, last cardiac movement, is produced during the diastolic relaxation phase (diastolic filling). Within these concepts, cardiac function consists of three movements. Between systole (300 ms) and diastole (400 ms) there is a third contraction movement (100 ms) which represents a link between the other two, generating the intermediate cardiac suction phase.(Trainini et al., 2015; Torrent-Guasp et al., 2005) We have corroborated this PPMC duration time in echocardiographic studies in both the LV and RV. In 10 normal people (Trainini et al., 2023) we found that its duration in the LV was 83 ± 16 ms and in the RV was 30.8 ± 5 ms (Trainini et al., 2023).

It is important to recall that the helical arrangement of the myocardium matches the anisotropy of directions adopted by the cardiac muscle fibers in their sequential depolarization, that we have verified with a very slight activation delay (Ballester et al., 2008; Beaumont et al., 2017; Biesiadecki et al., 2014; Carruth et al., 2020) between the ascending and descending segments (Figure 4). The geometrical properties of the cardiac fibers have great importance in the ability to generate, through electrical propagation, the necessary forces for their function. The fibers of the descending segment on the anterior aspect of the LV, which run in depth through the mesocardium, crossing obliquely with the ascending segment at an angle of 75% are responsible for the opposite activation of the descending (endocardial) and ascending (epicardial) segments. The spatial orientation of myocardial fibers explains the torsion movement and functional efficiency of the heart throughout the cardiac cycle.

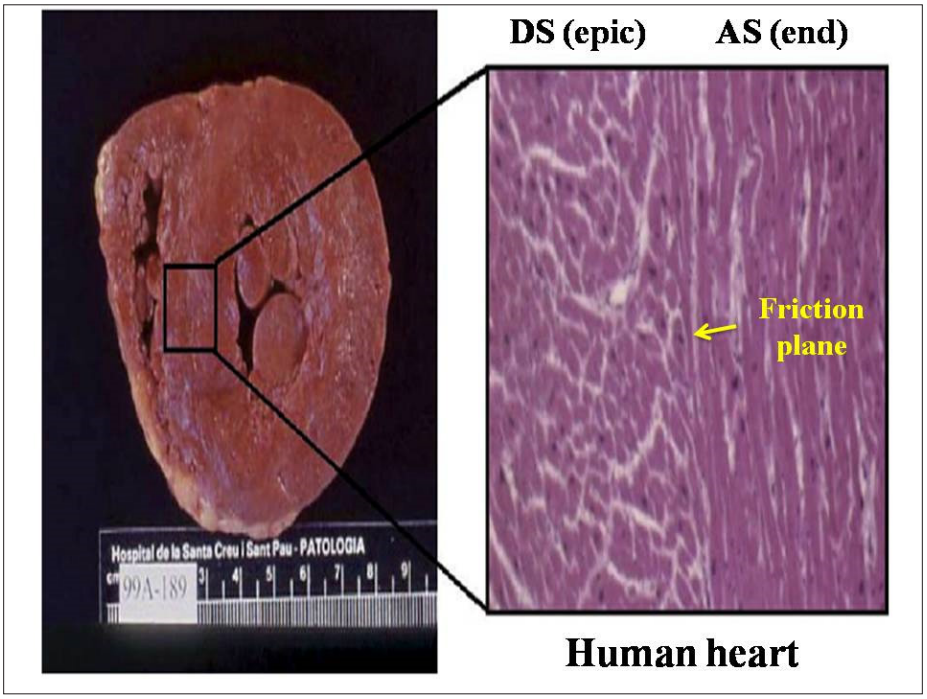

Figure 4: Microscopic view (right) of the interventricular septum medial segment in the human heart, clearly showing absence of transition circumferential fibers between the descending (right) and ascending (left) segments of the continuous myocardium. Also note that there is no fascia or anatomical structure located between the two fiber bundles. Moreover, in the macroscopic section (left), it can be seen how the sudden transition of the fiber angle change draws a line that can be perceived with the naked eye and that, in echocardiographic images, gives rise to the known medioseptal linear image generated by the acoustic interphase originating from by the abrupt change in angulation in this region of the septum. References: DS (epic) : descending segment (epicardic); AS (end): ascending segment (endocardic).

Discussion

Protodiastolic Phase of Myocardial Contraction (PPMC). A fundamental topic that we have investigated, which did not have no electrophysiological evidence, was to consider ventricular filling as an active phenomenon generated by myocardial contraction that tends to lengthen the left ventricular apex-base distance after the ejective phase, producing a suction effect similar to that of a "suction cup" (Trainini et al., 2019; Trainini et al., 2022; Trainini et al., 2015). This mechanism is explained by the persistent contraction of the ascending segment during the onset of the PPMC, as theoretically postulated by Torrent Guasp et al. (2004).

According to this investigation, diastole is divided into two very different phases. In the first, the size of the ventricle does not change; it is isometric. It changes its morphology (longitudinal, annular and helical torsion) and contraction of the myocardial walls is elicited with energy consumption, generating negative pressure. This is the phase termed PPMC, which lasts around 100 ms. During the second phase, the necessary conditions are produced to achieve ventricular filling with ventricular dilation due to blood repletion without energy expenditure throughout the remaining 400 ms.

In the traditional model, cardiac filling is only determined by venous pressure. Actually, atrial pressure is too low to explain this situation. The last contracted areas of the ascending segment produce a suction effect to draw blood towards the LV, generating negative pressure. The high filling velocity at low pressures establishes that this is an active phenomenon.

In the circulatory cycle, there is only one period that attains negative pressure in ventricular chambers at a defined moment. This phenomenon is only produced during the PPMC in which pressure drops to -3 mmHg, according to our measurements in patients. (Trainini et al., 2023). This phase is active and isovolumic. Between AV closure and mitral valve (MV) opening and hence, between PV closure and tricuspid valve (TV) opening, there is a sudden drop of intraventricular pressure with energy consumption that may reach negative values. It is during this phase that the muscle contraction of the final portion of the ascending segment in its insertion into the cardiac fulcrum –myocardial support- (Trainini et al., 2024) elicits myocardial lengthening-detorsion with the ventricular chambers closed. This contraction of the septum, as it is of ventricular interdependence, determines, as we shall see, the PPMC in both ventricles. This physiological action is the cause of the drop in intraventricular pressure ("depression") to the possibility of reaching negative values in both the RV and LV. After semilunar valves closure, the time the PPMC lasts depends on the pressure drop required to open the atrioventricular valves. And this implies, in addition to the corresponding circuit with its set of pressures, a faculty of the intrinsic muscular state of the myocardium to achieve the necessary detorsion in the generation of the intraventricular pressure drop. Due to this circumstance, the normal values of the PPMC may have a margin of variation.

Interpretation. Historically, the ventricular expansion process has not had the thorough analysis it deserves, the real meaning of diastolic filling being ignored in the physiological mechanism. In this regard, only systole has been given the category of being an active phase, with muscle contraction and energy consumption. (Zarco, 2001).

As expressed so far, how should we refer to diastolic filling? As cause or consequence of the expansion? As a result, would it be produced by venous pressure (vis a tergo) or by an active mechanism of myocardial suction?

Traditionally, venous pressure has been considered the cause of atrioventricular valve opening. However, the maximum pressure that occurs at the aorta and pulmonary artery outlet decreases constantly until minimum values are reached at the atrial entry. Here a necessary reflective point opens: can the RV have a suction phase despite having half the thickness of the LV? Let us recall that the relationship of pressure and resistance between the LV and RV is 6:1, that is, the RV is proportional to 15% of the LV. In the face of a lower resistance, the PV in relation to the force to which it is subjected, opens before the AV, which is fundamental in the complementarity of the ventricles in a dynamic interaction between ejection and suction.

This suction in the PPMC, due to the low gradients attained at the atrial entry, must be considered the fundamental element for venous return in complementarity with the systolic impulse. This is supported by the subatmospheric pressures ("depressions") recorded in these chambers during the PPMC. The driving role of the atria ("atrial kick") is minimal. Its power is 1% relative to that of the ventricle. Obviously, this low gradient is related with a need for active ventricular suction. (Brecher, 1958).

It is not the ventricles, mere cavities, that display this action but their muscular walls that make up the helical continuity of the myocardium as a single mass and that give it its leading role. The atria are outside this morphology and therefore lack adequate walls to propel their contents; they have a reservoir function and act as decompression chambers for the sharp blows produced by the sudden closure of the atrioventricular valves.

In the classical concept, there was only one active function in each ventricle, systole, with a positive pressure that determined it was the cause of venous return. This is not justified with what has been observed; on the contrary, venous pressure is fundamentally a consequence and not a cause of ventricular expansion, according to the values of the pressure gradient displayed by the venous circuit. This fact is supported by observing the moment in the PPMC in which the filling pressure, both in the systemic and pulmonary circuits, tends to have a suction action.

The pressure of the circulatory circuit decreases from the aorta to the venous end. This drop in pressure determines a very low

venous pressure, with a mean value between 0 and 2 mmHg in the venae cavae (Rushmer et al., 1953; Rushmer, 1964), so it is expressed in cm of water. The low venous pressure at the entry of both ventricles cannot create the force that propels the blood to enter the ventricles, but through the active suction of these chambers. The ventricles, made up of resistant muscles, do not expand with low venous pressure and much less due to atrial contraction, with their very thin walls, which also lack an adequate structure to prevent blood backflow. Venous pressure is not the cause of ventricular expansion but its consequence. As deduced from our investigations, we must admit that blood entering the ventricles is the result of suction due to the contraction of both ventricular walls during the PPMC.

A fluid moves along a tube in response to a pressure gradient. Therefore, the pressure gradient must be greater than 10 mmHg to generate flow, due to energy losses in the system. When the vascular duct lacks this gradient, flow ceases. This is called "critical closing pressure".

Blood pressure fluctuates around a mean value of 90 mmHg. Pulmonary pressures have an average of 22/8 mmHg with a mean pressure of 13 mmHg. On the other hand, the pressure difference between the pulmonary capillaries and the left atrial (LA) is 4-6 mmHg. Thus, a low-pressure gradient allows the same amount of blood to pass through the pulmonary circuit as through the general circuit, which has a gradient of 90 mmHg, due to the complementarity between ejection and suction energies. In addition, the power of the LA is only 1% that of the LV, so noticeable effects as pump function cannot be expected from it. The LA distends in the face of a LV suction deficit, but no notable effects of flow impulse can be expected. Obviously, this low gradient could not generate a sudden ventricular filling without the need for active ventricular suction. (Trainini et al., 2019).

Ventricular-septal interdependence. What we have seen in the electrophysiological study with a Carto navigation and mapping system is that endocavitary activation ends before the end of the QRS complex; therefore, its continuity corresponds to the late activation of the distal portion of the ascending segment after semilunar valve closure, justifying the persistence of contraction during the isovolumic diastolic phase, the basis of the ventricular suction mechanism of both ventricles which we call PPMC.

According to our echocardiographic investigation in patients without heart disease this stimulation lasts 83 ms in the LV and about 30 ms in the RV. (Trainini et al., 2023). Its epicenter occurs in the ascending segment of the apical loop with the result of producing lengthening of the septum during the PPMC, implying consequences in both ventricles. The echocardiographic work of Mora Llabata et al. (2018) finds a difference of 88±7.1 ms in the development of systolic distortion between the systolic and post systolic phases, which coincides with the duration of the ascending segment activation in the left ventricular protodiastolic phase myocardial contraction (LVPPMC) found in our studies. (Mora et al., 2016; Mora et al., 2018; Mora et al., 2021). Post-systolic longitudinal strain, indicating the contraction involved in left ventricular detorsion, is basically produced in the septal segments and in the anterior basal region which correspond to the anatomical location of the ascending segment.

The interventricular septum has a predominant value in myocardial function as its anatomical location is essential in biventricular interdependence. (Trainini & Herreros, 2008). It consists of a ventral and a dorsal part. The first portion is made up of the descending segment, the intraseptal band (final segment of the muscle band), and the anterior septal band. The first two belong to the LV and the other to the RV. The posterior region of the septum is made up of the descending segment (dependence of the LV) and the posterior septal band, corresponding to the RV. (Trainini et al., 2019; Trainini et al., 2022). In this way, septal contraction determines the PPMC in both ventricles, as this septum is a structural interdependence between both (Figure 5).

This is possible due to myocardial fiber orientation, with longitudinal predominance in the final part of the ascending segment, which become progressively more oblique as they descend, preserving the spiral fibrillary conformation in the general structure. The fibers, being able to expand longitudinally, achieve an elongation effect and generate a drop in intraventricular pressure when the atrioventricular valves are closed, while the rest of the fibers are relaxed. This anatomical interdependence between both ventricles controlled by the septum, not only implies the LVPPMC but also that of the RV, which is a scarcely known situation.

This contraction of the interventricular septum that persists after semilunar valve closure also causes a drop of intraventricular pressure in the RV ("depression"), with the remaining walls relaxed. The intraseptal band (final portion of the ascending segment), located between the anterior septal band and the descending segment, takes part in this mechanism. This is confirmed through echocardiography by demonstrating that post systolic strain occurs later in the segments of the interventricular septum (Mora et al., 2016), which implies finding during the right ventricular protodiastolic phase of myocardial contraction (RVPPMC) a subatmospheric pressure at the beginning of diastole estimated in about -2mmHg.

The consequence of PPMC in both ventricles is a drop in intraventricular pressure, which implies generating the mechanical conditions for subsequent diastolic filling. Thus, this phase has myocardial contraction and energy consumption. This physiological situation in both ventricles is feasible, given that by sharing the interventricular septum, the chambers are subject to a process of anatomical and functional interdependence.

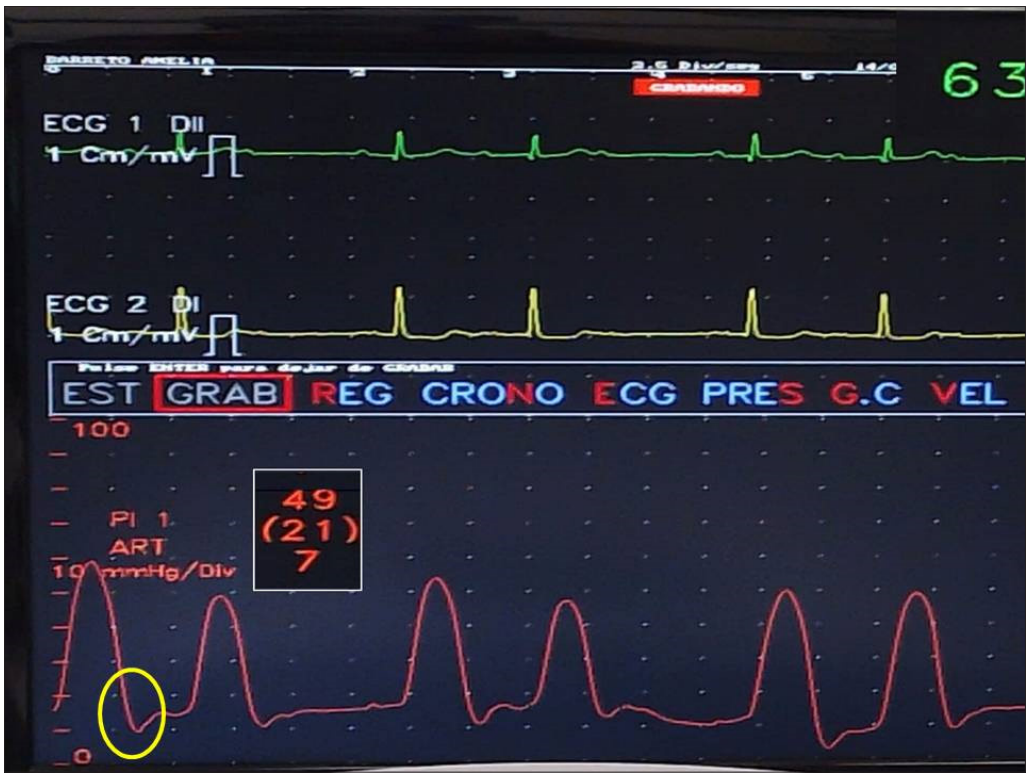

Figure 5: Right ventricle recording. The drop in intraventricular pressure during the RVPPMC is observed in one patient (yellow circle).

Protodiastolic contraction of the left ventricle: The basal loop (contraction of the right and left segments) determines ventricular narrowing and the beginning of the cardiac cycle, while contraction of the descending segment together with the ascending segment causes the shortening-torsion movement of LV systole. All these physiological processes are required for the ejection phase. As it corresponds to a greater resistance bed, the AV opens after the PV, an asynchrony that, as we will see, corresponds to the last period of left ventricular diastolic filling.

This cardiac cycle model is based on the fact that the right intraventricular pressure, during the RVPPMC represents the minimum pressure of the venous system, while the left intraventricular pressure in the LVPPMC implies the minimum pressure of the arterial system. Both pressures during the PPMC involve a suction mechanism to achieve diastolic filling. In this way, circulatory mechanics works in a complementary manner between the alternate phases of suction and ejection of both ventricles and not between the classic division between RV and LV. In this investigation, the interrelationship between the cardiac phases becomes critical for the correct interpretation of the circulatory movement.

The activation analyzed in our electrophysiological investigations (Trainini et al., 2023; Trainini et al., 2019; Trainini et al., 2022, Trainini et al., 2015) explains the insertion of a suction active coupling phase between systole and diastole, with muscle contraction, energy consumption, and drop of intraventricular pressure. This effect draws blood into the ventricular chamber by means of a difference between ventricular and peripheral pressure, and is responsible that 70% of the total filling volume is achieved in only 20% of the total filling time, in stage 1 of diastole. Tyberg et al. (1970), using MV balloon occlusion in a dog, showed that during diastole the LV decreases its pressure below zero. This was also seen by Sabbah in dogs. (Sabbah & Stein, 1981).

An opposite rotation between the apex and the base of the heart is produced as a consequence of the oblique myocardial fiber arrangement around the LV, a situation allowing ventricular torsion (systolic contraction) and the subsequent detorsion (suction mechanism, "suction cup" effect). Apical rotation (counterclockwise, seen from the apex is considered positive, while basal rotation (clockwise, seen from the apex) is negative. To calculate twist, the ultrasound machine algorithm performs an algebraic sum (it adds the positive value of apical rotation to the negative one of basal rotation). (Mora et al., 2018; Dong et al., 2001). In our experience, the value in normal subjects is around +19º ± 9º (3), always with predominant apical rotation. These studies document the concept that the apex is the one that "commands". Although the terms twist and torsion are usually indistinctly used, actually twist corresponds to the algebraic difference of basal and apical rotations, while torsion corresponds to twist divided by end-diastolic apex-base distance, so in essence torsion would be twist normalized by left ventricular longitudinal diameter, with a measurement that reaches 13.1± 4°/cm in investigations published. (Trainini et al., 2019; Mora et al., 2018).

Protodiastolic contraction of the right ventricle. The septum has a fundamental importance in myocardial function as its mechanics is decisive in biventricular interdependence. In the RV, the longitudinal component predominates over the

circumferential one. In fact, practically all the methods of right ventricular function assessment (except the 3D volumetric method) are based on longitudinal parameters (TAPSE, tissue Doppler velocity, longitudinal strain of its lateral wall). (Trainini et al., 2023) Even when estimating surface shortening fraction, the fundamental component is longitudinal. However, by means of cardiac magnetic resonance with DENSE technique myocardial tagging sequences, it has been possible to verify that there is some rotation in the RV. How to explain it if there are no oblique fibers, as the RV is formed at the expense of the basal loop? It can be explained through the transmission of torsion applied by the LV on the RV especially at the level of the interventricular septum. Actually, if we make a cone with elastic material forming two cavities, it is easy to verify that if torsion is generated in the internal cavity, it is also produced in the external one.

There is a very brief period at the beginning of systole in which the base and apex rotate counterclockwise. This situation, called inertial state -in this case cardiac- has a physical explanation, as bodies have the property of trying to remain in their resting state. It implies the resistance that matter opposes when changing its state of movement including changes in the speed or direction of movement. Then, they differ in their sense, being clockwise at the base and counterclockwise at the apex.

Septal contraction zones that are still active at semilunar valves closure allow the development of a depression zone in the right ventricular chamber (pressure drop), as the free semilunar-shaped wall of the RV is relaxed. The intraseptal band, final segment of the continuous myocardium, which runs between the anterior septal band and the descending segment, would be responsible for this action. Echocardiographic studies have confirmed that the last post-systolic strain occurs later in the segments of the interventricular septum. (Mora et al., 2018). This situation can create a subatmospheric pressure or "depression" at the beginning of diastole in the RVPPMC that is estimated to be around -2 mmHg. Figure 5 from our laboratory shows the marked pressure drop in the right intraventricular pressure curve during the RVPPMC. In the case of suction, the invasive pressure record that demonstrates its drop is of indisputable value. (Nagueh, 2018).

The result is the corresponding active suction that occurs in the right ventricular diastolic isovolumic relaxation phase, which should more appropriately be called RVPPMC. This suction is evident because when the venous return flow is tagged there is acceleration in the final sections of the venae cavae. (Rushmer, 1972). This flow is slow during systole and suddenly accelerates to enter the right ventricular chamber. The fact that the right segment that makes up the right ventricular free wall is relaxed at that moment adds to its support; therefore, the intraseptal contraction together with the relaxation of this wall would act in the same way as between the contraction of the ascending segment in left ventricular protodiastole and the rest of myocardial relaxation during the LVPPMC (Figure 6). Thus, when the semilunar valves close, the septum is still contracting, causing a drop in right intraventricular pressure, and generating active suction with energy consumption.

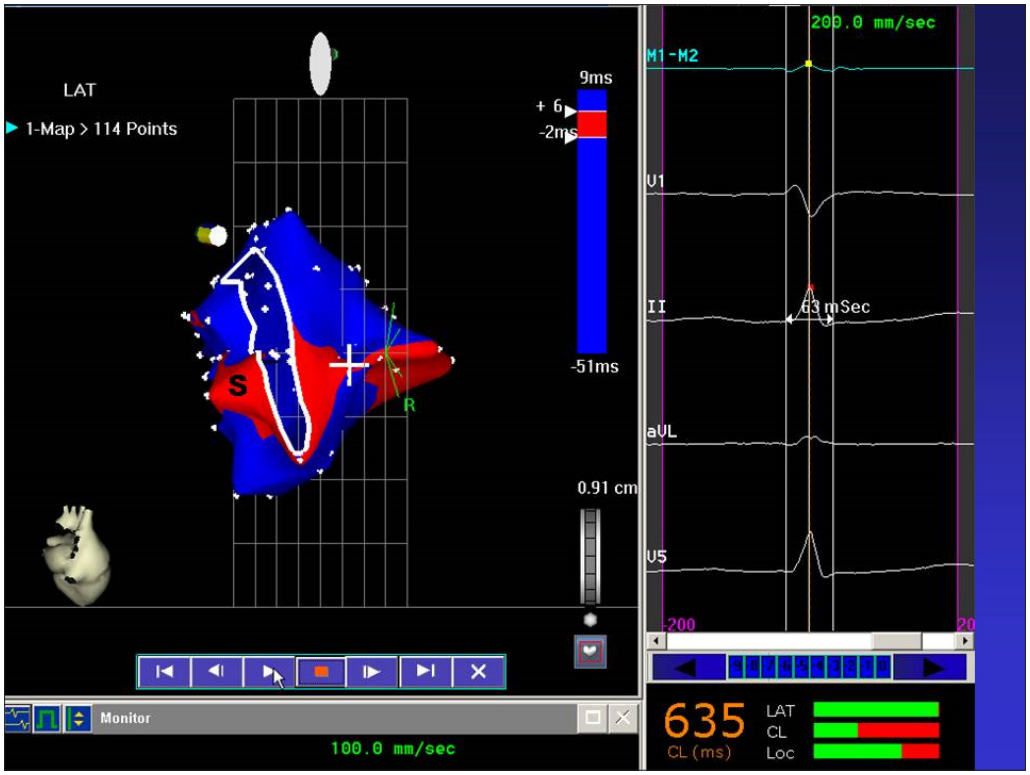

Figure 6: Propagation sequence map with 3D electroanatomical mapping in patients. As the pulmonary valve closes, the blood volume within the right ventricle remains constant. However, during this period, the interventricular septum at the level of the intraseptal band continues to contract. This causes a drop in right intraventricular pressure with the consequent subsequent suction of the systemic circuit. Ref. S: septum.

Complementariedad Biventricular

The initial asynchrony between the RV and LV in semilunar valve opening explains the necessary functional complementarity of the circulatory movement and gives support to the anatomo-physiological structure of the continuous helical myocardium that constitutes the heart. The general and pulmonary vascular beds are connected in series to form a continuous circuit through the active, ejection and suction phases of each ventricle, as, while the first generates positive pressures; the second one tends to negative ones.

As each ventricle has a single chamber to fulfill two active phases of opposite functions, suction and ejection, and a passive phase of diastolic filling, we must consider that to complete its unidirectional circulatory system it needs an asynchrony between its phases so that blood can flow in one direction and with an effective sequence. Although systemic circulation and pulmonary circulation seem to be independent circuits, the synchronized activation between ejection and suction of both ventricles is in line with the acceptance of the active concept of suction that they present, endorsed by recently published research studies. (Trainini et al., 2023; Trainini et al., 2019; Trainini et al., 2022; Trainini et al., 2015) There is an evolutionary instance that supports the complementarity between ventricular suction and ejection. In anurans, the right and left atria act alternately. This allows blood to remain unmixed and be ejected sequentially by the only undivided ventricle they have in order to comply with the two circuits. This is analogous in the evolutionary strategy with each human ventricle, which with a single cavity must exercise the load and expulsion destined for its specific circulation.

In this way, the circulatory system constitutes two subsystems in which ejection and suction of the different ventricles act in that alternate complementarity, with both phases in active form, energy consumption and muscle contraction:

- Systemic subsystem: Left ventricular ejection + right ventricular suction;
- Pulmonary subsystem: Right ventricular ejection + left ventricular suction

The histological architecture present in the venae cavae-RA and pulmonary vein-LA sequence should be interpreted as passive ducts that allow blood transport to the ventricles. Due to their structural characteristics, they are not capable of enough propulsion to fill them. The histology of (figure 7) shows that the venous structures from the venae cavae to the LA have no propulsion capacity. The different histology of the pulmonary artery, capable of propulsion, is what would allow the embolus function of the RV towards the suction of the LV.

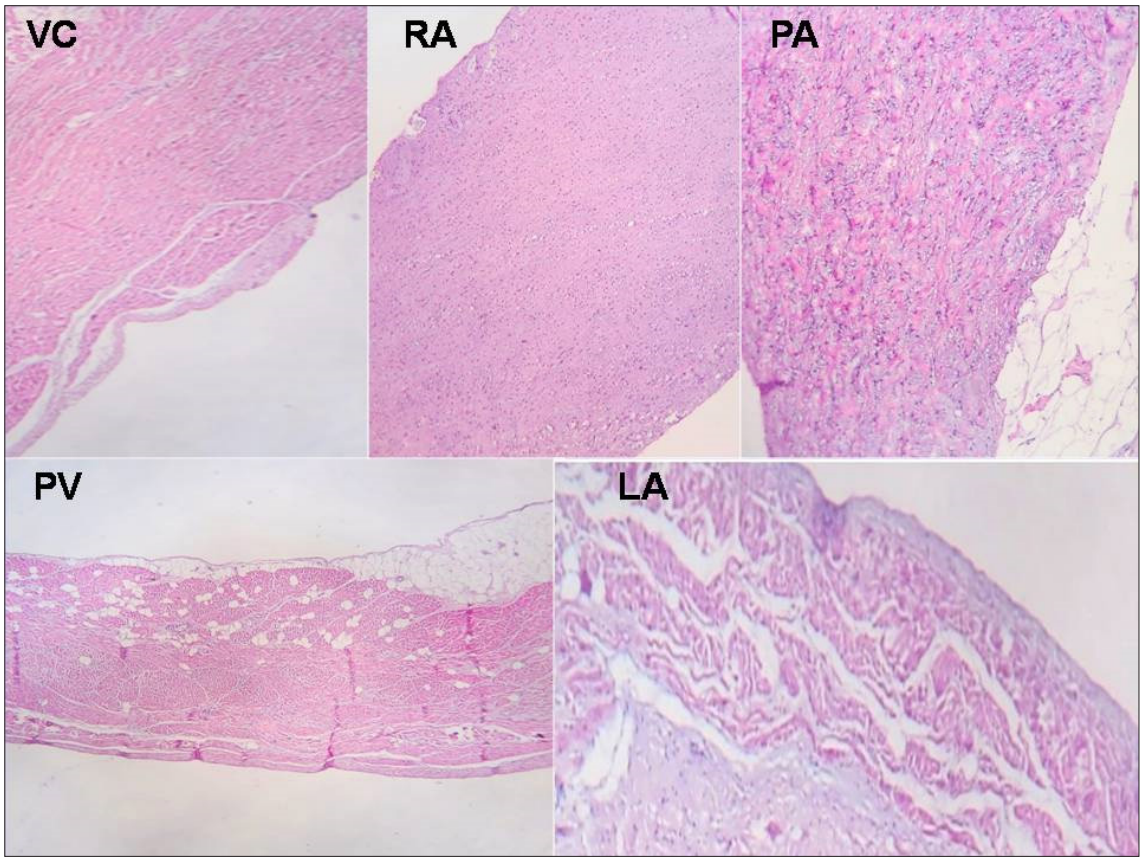

Figure 7: VC. Vena cava: a 3-mm-thick vena cava structure is shown formed in its middle layer by smooth muscle cells in parallel bundles. RA. Right atrium: wall formed by myocardial muscle fibers approximately 4 mm thick. PA. Pulmonary artery: myocytes formed by smooth muscle cells with a spiral structure are observed in a loose stroma. PV. Pulmonary vein: structure similar to the pulmonary artery with myocytes made up of smooth muscle with a fasciculated structure. LA. Left atrium: layer of myocardial muscle fibers approximately 4 mm thick. (All histological sections belong to bovine heart stained with H&E 10x).

The Cardiac Cycle

Right atrial pressure is very low and its small changes cannot explain the control of ventricular filling, especially if we take into account that variations in the position of the body and breathing cause larger fluctuations in the pressure it displays.

In our studies on 3D-EAM there is around 38.2 ms asynchrony between the onset of right ventricular with respect to left ventricular systole. (Trainini et al., 2022) The PV opens before the AV. (Mangione et al., 2022). In addition, the time between TV closure and PV opening is shorter than that between MV closure and AV opening, as it depends on the time it takes to acquire the necessary pressure, related with the opening power.

This difference of 40 ms between the anticipated opening of the PV in relation to the AV is logical, since with the two ventricles ejecting and filling at the same time the circulation would be practically interrupted, without the necessary continuity, falling to even lower levels than the gradients displayed by the normal circulatory return. The two active functions of ejection and suction synchronized between both ventricles would correspond to the circulatory continuity. Here the question arises: how is the early opening of the PV in relation to that of the AV explained? The ventricles take advantage of the asynchronous impulse of about 40 ms, that is, 5% of the 800 ms duration of the cardiac cycle, for one of them, the RV, to load the other, the LV, through the complementarity between ejection and suction.

The RV begins its systolic activity 12.4 ms before the LV. Pulmonary valve opening begins as soon as its intraventricular pressure rises to 8-10 mm Hg. The RV input region contracts very early. This right ventricular ejection starts prior to left ventricular systole. Only at an average of 38.2 ms after the beginning of the cardiac cycle with RV contraction, the ascending and descending segment are simultaneously stimulated, which would imply at that moment left ventricular ejection when the AV opens.

In our investigations, during this estimated interval with an average of at least 38.2 ms between PV and AV opening, the last left ventricular filling phase occurs (stage 3 of diastole), a specific point in the cardiac cycle that we will explain later.
Once left ventricular systole has started, it lasts 300 ms. When the AV closes, the LVPPMC begins with active muscle contraction and energy consumption. How could the intraventricular pressure drop adequately to open the MV, if faced with a closed isovolumic chamber without flow there is no strain of the muscle walls with active muscle contraction? In this situation there is energy transmission from the muscle to the fluid in an isovolumic condition. This has been demonstrated in electrostimulation studies, with invasive measurements, and with tissue strain in ultrasound imaging. (Trainini et al., 2023; Trainini et al., 2019; Trainini et al., 2022).

A fluid moves along a tube in response to a pressure gradient. When all the blood is perfused in the vascular system, the pressure gradient must be greater than 10 mmHg to generate flow, although from physical theory it would suffice for it to be greater than the loss of load. The pressure difference between the pulmonary capillaries and the LA is 4-6 mmHg. (Rushmer, 1972). This can be solved from hydraulics if we consider that the right ventricular ejection phase and the left ventricular suction phase work as a unit to achieve the necessary power. Even in the absence of the RV, as we have seen in the experimental laboratory, the value of left ventricular suction is evident. (Trainini et al., 2019). Thus, the situation of left ventricular suction is solved, adding to its comprehension:

- The contractile activity in the LVPPMC found in the electrophysiological studies with a Carto navigation system.
- The myocardial tissue strain found during this phase in echocardiographic studies.
- Curves of left intraventricular pressure drop reported in resynchronized patients.

In this regard, we have found a longer duration of the LVPPMC in patients with heart failure with (HFpEF). This is because muscle power decreases and therefore the time to transmit the same energy and produce the necessary pressure change increases. This increase is due to the need for a longer time to open the MV in order to achieve a sufficient drop in intraventricular pressure. In our ultrasound studies we have found a LVPPMC with an average duration of 83 ms in normal patients, well below the 134 ms presented by patients with HFpEF. (Trainini et al., 2023).

In essence, LV suction represents the complementary link of the circulation between both ventricles in order to move the blood content through the pulmonary system. In the same way, as we will see later, RV suction helps blood filling after LV ejection and passing through a zone of resistance such as the systemic capillary circuit with a pressure drop to values between 0 and 2 mmHg.

How is stage 3 of diastolic filling achieved in the LV, after the first and intermediate stages of diastasis? According to what we have investigated, after PV opening and before AV opening, an average of 38.2 ms elapses. (Trainini et al., 2015). During this time, the last stage of LV filling occurs in complementarity between RV ejection and LV suction.

Left ventricular filling curve: The LV filling curve has two filling peaks and an intermediate one called diastasis (separation), in which the intraventricular volume does not change (1-2% of total filling). Thus, diastolic filling (around 400 ms) consists of three stages. The two filling stages, the first and the third, within the 400 ms of diastole, coincide with forces that propel blood into the LV. The first is a consequence of the LVPPMC whose duration of 80-100 ms allows the drop in intraventricular pressure prior to MV opening. After diastasis, the intermediate stage in which flow ceases, the second LV diastolic filling occurs (stage 3), synchronized with right ventricular ejection that occurs about 40 ms before the estimated opening of the AV, indicating the complementarity between right ventricular ejection and stage 3 of left ventricular

diastole. The RV begins its ejection while the LV is in the final phase of diastole. This fact favors the power to generate the second filling, as the LA by itself does not have the necessary strength for this task.

Physiological interpretation postulated that the atrium could exercise this function in isolation, historically called "atrial kick" (its power is 1% in relation to that of the LV), but this stage 3 of diastolic filling is actually an energy consequence of RV ejection with plunger power in the appropriate time.

In addition, atrial histology does not guarantee energetic propulsion in developing the necessary pressures, as the absence of valves is added to their thin walls to prevent blood recoil. Therefore, during the second filling of the LV (stage 3), this movement is synchronized with the ejection of the RV, some 40 ms before AV opening occurs.

Right ventricular filling curve: Between ejection and suction of both ventricles, in an alternate and synchronized manner, enough time has to elapse for blood flow passage associated with the systemic and pulmonary circuits, expressed with Hagen-Poiseuille's theorem factors. After AV opening, with a velocity of approximately 200 cm/s, blood circulates in the aorta at a mean speed of 40-50 cm/s and falls to 0.07 cm/s in the capillaries. Eighty percent of the pressure drop occurs in the arterioles due to vascular resistance. The velocity of venous blood is not the same as that of arterial blood; it only approximates its value.

The veins contain most of the blood volume (75%), the capillaries 5% and the arteries 20%. Blood volume passing through the transverse lines of the circulatory territory per unit time is the same as the amount of blood that enters and leaves the system, as if it were a single duct. Five liters of blood pass through each section line. As blood volumes are equal, changes in the cross-sectional area affect blood velocity.

Blood flows by a pressure gradient. In its passage it loses impulse (friction + resistance) The force that returns blood to the right atrium (RA), called "systemic filling pressure", is around 7 mmHg and reaches 2 to 4 mmHg in the RA; therefore, this minimum gradient does not allow flow, as it is below the 10 mmHg required for circulation ("critical closing pressure"). This physiological circumstance explains the active intraseptal band contraction in the RVPPMC with RV free wall relaxation after PV closure to improve the active filling of this chamber. Therefore, blood that enters the RV comes not only from the RA, but is also sucked from the venae cavae. (Rushmer, 1972).

Mean pressure in the venae cavae is between 0 and 2 mmHg. Pulsations in the veins suggest RV contractile phenomena, which correlates with the active period in the RVPPMC. This activity in the venae cavae resembles the pressure waves in the RA. As AV closure is prior to that of the PV, and TV opening before that of the MV, RVPPMC duration is obviously shorter than that of the LVPPMC. This phase lasts 30.8 ms in the RV, while in the LV it lasts an average of 83 ms, according to echocardiographic investigations we performed in control patients. (Trainini et al., 2023). The values of RVPPMC duration vary in the literature. (Brechot et al., 2008). A confirmation of this point is the increased blood velocity in the final segment of the venae cavae, because as the pressure drop in the circulatory system is constant reaching very low values in the venae cavae, this acceleration has to be related with RV suction in the RVPPMC. Thus, at the beginning of diastole, even negative values can be detected.

As explained, this phase is active and with energy consumption. Its action is analogous to the same phase in the LV. Aortic valve closure prior to that of the PV represents a plunger effect of the LV more adequate in time to the moment in which the RVPPMC is produced.

Conclusions

In circulatory physiology we must consider fundamental investigated facts:

The asynchrony between the right and left ventricular cycles allows the interaction between ejection and load in each chamber. Myocardial contraction, active and with energy consumption, during the PPMC in both ventricles enables blood flow during venous return, replacing the lack of adequate gradient between the vascular beds and these chambers.

Complementarity, consequence of the asynchrony between PV and AV opening and AV prior to PV closure, allow the overlapping action between left ventricular ejection with right ventricular suction, as well as right ventricular ejection with left ventricular suction.

Beyond their morphological continuity, each atrium and its corresponding ventricle cannot be considered as a functional unit in the active ejective and suction mechanism. This functional pattern is constituted by the alternate and synchronized phases of both ventricles, that is, RV ejection-LV suction and LV ejection-RV suction. The classical concept hampered the knowledge of cardiac development and understanding the myocardium as a continuous muscle with a support we have called cardiac fulcrum. (Trainini et al., 2021; Trainini et al., 2022).

The systemic and pulmonary vascular beds are connected in series to form a continuous circuit. We must speak of RV-pulmonary circulation and LV-systemic circulation as a single circulatory pathway in which the pressure differences and vascular resistances determine the continuous blood circulation. The ventricular chambers and the vascular circuits are two distinct systems with characteristic dynamics, whose purpose is to keep circulatory flow. This concept compels considering the active suction during the PPMC of both ventricles as an essential element in the physiology of the circulatory system, as it is the continuity link between the pulmonary and systemic circulations.

It is necessary to consider that the system of circulatory movement would be established by the complementary

functional phases (ejection-suction) of each ventricle and not by their spatial condition (right-left).

Abbreviations

AV : aortic valve
HFpEF : heart failure with preserved ejection fraction
LA : left atrium
LV : left ventricle
LVPPMC: left ventricular protodiastolic phase of myocardial contraction
MV : mitral valve
PPMC : protodiastolic phase of myocardial contraction
PV : pulmonary valve
RA : right atrium
RV : right ventricle
RVPPMC : right ventricular protodiastolic phase of myocardial contraction
TV : tricuspid valve
3D-EAM : three-dimensional electroanatomic mapping

Compliance with Ethical Standards

Compliance with Ethical Standards

Not applicable

Disclosure of potential conflicts of interest

Not applicable

Research involving Human Participants and/or Animals

Not applicable

Informed Consent

Not applicable

Availability of Data and Materials

Not applicable

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Ethical Approval and Consent to Participate

Not applicable

Consent for Publication

Not applicable

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