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Research Article

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Protodiastolic Phase of Myocardial Contraction

Trainini Jorge[1]*, Beraudo Mario[2], Trainini Alejandro[3], Wernicke Mario[4], Lowenstein Jorge[5], Herrero Efraín[6] and Hernández López Mayra Patricia[7]

[1]*Presidente Perón Hospital, National University of Avellaneda, Argentina*

[2]*Clínica Güemes, Luján, Argentina*

[3]*Presidente Perón Hospital, Argentina*

[4]*Clínica Güemes, Luján, Argentina*

[5]*Instituto de Diagnóstico de Investigaciones Médicas, Buenos Aires, Argentina*

[6]*Clínica Güemes, Luján, Argentina*

[7]*Centro Especializado del Corazón. Hospital MAC, México*

***Corresponding author:** Jorge Carlos Trainini MD, PhD, Presidente Perón Hospital. National University of Avellaneda, Argentina.

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Abstract

Introduction: 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 protodiastolic phase in both ventricles enables blood flow during venous return, replacing the lack of adequate gradient between the vascular beds and these chambers.

Material and Methods: These reflections are based: 1) Dissection of eighty-four hearts; 2) Anatomical, histological and histochemical studies; 3) Left ventricular endocardial and epicardial electrical activation in humans; 4) Echocardiography.

Results: Complementarity, consequence of the asynchrony between pulmonary valve and aortic valve opening and aortic valve prior to pulmonary valve 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, right ventricular ejection-left ventricular suction and left ventricular ejection-right ventricular 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*.

Conclusions: The systemic and pulmonary vascular beds are connected in series to form a continuous circuit. We must speak of right ventricle-pulmonary circulation and left ventricle-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 protodiastolic phase of myocardial contraction 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).

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Keywords: Cardiac suction, Complementarity ventricular, Cardiac cycle

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; PA: Pulmonary Artery; 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

Introduction

Right Atrial (RA) 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 [1,2]. The Right Ventricle (RV) is designed to eject large volumes of blood against low resistance, owing to its semilunar-shape with large surface area, but cannot do so at high pressures [3,4]. Pulmonary arterial resistance is half that of venous resistance. In systole, the RV shortens towards the apex. Unlike the Left Ventricle (LV), where blood is transferred from the mitral orifice to the apex before being directed to the aorta, in the RV blood is sent directly from the tricuspid orifice to the Pulmonary Artery (PA) following a central washout line that forms a 90°angle. This article is based on our extensive research on the subject [4-12]. Its purpose is to demonstrate that the cardiac filling phase is due to active suction during the Protodiastolic Phase of Myocardial Contraction (PPMC), classically called the diastolic isovolumetric phase.

Material and Methods

The methods used in this study to explain the hypothesis of the anatomo-functional integrity of the heart were:

1) Dissection of eighty-four hearts from the morgue, slaughterhouses, and breeding farms (for anurans) were used: a) 54 two-year-old cattle weighing between 1300 and 1900g (average 1650g); b) 17 humans (three at 8, 16, and 23 weeks of gestation, respectively; four infants at 30 days, 36 days, 10 weeks, and 27 weeks; one 4-year-old child; one 10-year-old child; and eight adults with an average weight of 300g); c) three porcine hearts (400g); d) 10 anuran hearts.

2) Anatomical, histological and histochemical studies were performed. The hearts were fixed in 10% buffered formalin. Histology was performed with hematoxylin and eosin, Masson's trichrome staining, and 4-micron sections. 10% formalin was used as a buffer. Immunostaining (s100-neurofilaments) was also performed.

3) Left ventricular endocardial and epicardial electrical activation in humans (five adult patients). The left ventricular endo and epicardial electrical activation sequence was studied using three-dimensional electroanatomic mapping (3D-EAM) with a navigation system and Carto mapping, enabling three-dimensional anatomical representation, with activation and electrical propagation maps. Isochronic and activation sequence maps were performed, correlating them with surface ECG.

4) Echocardiography to corroborate previous studies and the usefulness of these knowledge in clinical practice.

Results

In our studies on 3D-EAM [4] there is around 38.2ms asynchrony between the onset of right ventricular with respect to left ventricular systole. The PV opens before the AV [3]. Since the left ventricular muscle structure is composed of an endocardial and an epicardial portion, or descending and ascending segments, respectively, in the anatomical classification (Figure 1), two access routes were used to perform the mapping. The endocardial access was achieved by conventional atrial transeptal puncture and the epicardial access by percutaneous approach in the pericardial cavity with an ablation catheter. Endocardial and epicardial mapping were immediately and consecutively performed. In addition, the electrical activation propagation times of the muscle band were measured in milliseconds(ms) [13-15] (Figure 1).

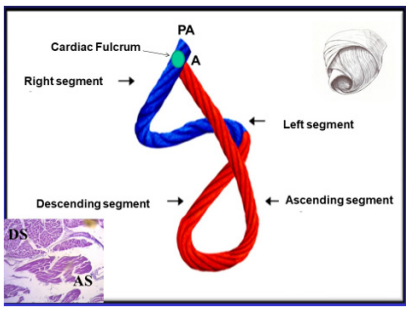

Figure 1: Site of the cardiac fulcrum in the continuous myocardium (cord model).
PA: pulmonary artery; **A:** aorta. The right inset shows the three-dimensional helical arrangement of the continuous myocardium. The histology shows the different orientation of the longitudinal fibers (ascending segment, **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 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. Therefore, the sequence of valve opening and closing at the first heart sound is as follows: 1) MV closure; 2) TV closure; 3) PV opening, 4) AV opening. The MV closes first, followed by the TV because high-pressure beds always close earlier. This statement is only valid with equivalent closing pressures and power between both valves and both ventricles. In this sense, the AV closes before the PV. This period between the closure of the TV and the opening of the PV; and opening of the MV and opening of the AV, classically referred to as the isovolumic diastolic phase, is referred to in this study as the PPMC because it is active with energy expenditure [4]. It extends in the LV for about 83ms, while in the RV it reaches about 30ms, according to our research [5]. This statement is only valid with equivalent closing pressures and powers between both valves and both ventricles. The general circulation is a high resistance circuit while in the pulmonary circuit this decreases, as well as pressure, to 1/6 of the systemic value. Thus, the left ventricular volume output is approximately equal to that of the RV, but they do not have the same power. The capacity of each ventricle must be evaluated not by volume but by the power it develops [6].

This difference of 40ms 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 40ms, that is, 5% of the 800ms duration of the cardiac cycle, for one of them, the RV, to load the other, the LV, through the complementarity between ejection and suction. In our investigations the RV begins its systolic activity 12.4ms before the LV [7].

Pulmonary valve opening begins as soon as its intraventricular pressure rises to 8-10mm Hg. The RV input region contracts very early. This right ventricular ejection starts prior to left ventricular systole. Only at an average of 38.2ms 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 begins, it lasts for 300 ms. When the AV closes, the LVPPMC begins. This phase is characterized by active muscle contraction and energy expenditure. How could intraventricular pressure fall sufficiently to open the left ventricle if, in a closed isovolumetric chamber with no flow, there were no deformity of the muscle walls with active muscle contraction? In this situation, energy is transmitted from the muscle to the fluid in an isovolumetric condition. This has been demonstrated in electrostimulation studies [7-12], with invasive measurements [6], and with tissue deformity in ultrasound [13,14].

Discussion

As we have seen above, the human research study conducted by us [7,8] on the propagation of electrical activity in the continuous myocardium verifies contraction of the ascending segment in early diastole as an interface between systole and diastole (Figure 2). This 80-100ms period is what we have called LVPPMC for better conceptual comprehension than the usual denomination of isovolumic relaxation phase. A fundamental subject we have investigated, that did not have 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 ejection phase, producing a suction effect similar to that of "sucking". This mechanism is explained by the persistent contraction of the ascending segment during the onset of the LVPPMC, as postulated by Torrent Guasp [15]. Here we must understand that this phase produces left ventricular suction with the consequent facilitation of stage 1 of rapid diastolic filling (Figure 2).

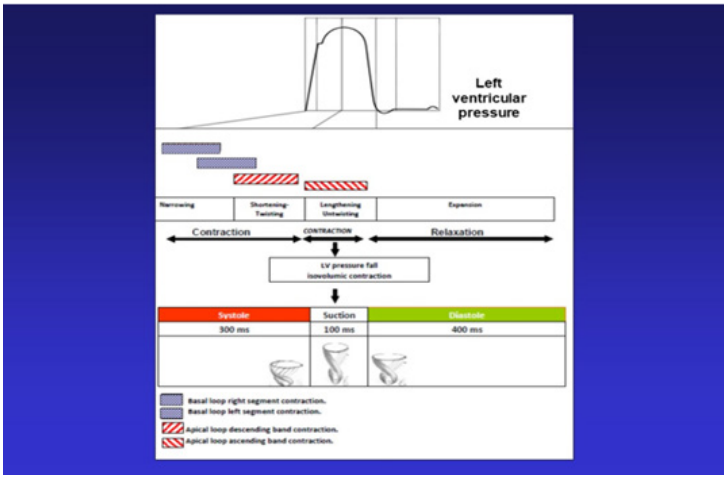

Figure 2: Effects of the cardiac activation-contraction mechanism. Correlation with left intraventricular pressure.

The RV impulse raises pulmonary artery pressure, after its constant steady drop since the beginning of its course after left ventricular systole. In any case, the gradient between the pulmonary circulation and the Left Atrium (LA) is still low to achieve left ventricular filling. 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 10mmHg 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 4-6mmHg. 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 [16]. Thus, the situation of left ventricular suction is solved, adding to its comprehension:

1) The contractile activity in the LVPPMC found in the electrophysiological studies with a 3D-EAM [2].

2) The myocardial tissue strain found during this phase in echocardiographic studies.

3) Curves of left intraventricular pressure drop reported in resynchronized patients (Figure 3). In this regard, we have found a longer duration of the LVPPMC in patients with Heart Failure with Preserved Ejection Fraction (HFpEF) [17]. This is because muscle power decreases and therefore the time to transmit the same energy and produce the necessary pressure change increases.

4) 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 83ms in normal patients, well below the 134ms presented by patients with HFpEF [5,17] (Figure 3).

Figure 3: Left intraventricular pressure with resynchronization therapy.
A. Resynchronization device turned off. **B**. In the same patient, a drop in left ventricular diastolic pressure is observed after resynchronization is restarted. 1) The yellow circle points out the increase of systolic arterial pressure in B with respect to A. 2) The red circle shows the negative intraventricular pressure in B.

The Figures 4 and 5, of tissue strain obtained by echocardiography, correspond to the LVPPMC. Figure 4 belongs to a patient without cardiac pathology in which the tissue strain curve is indicated in this phase of the LV, with analogous duration of 80ms. Figure 5 details the same LV phase but in a patient with HFpEF. The difference between patients shown by the LV tissue strain curve in the PPMC is clear. In the patient with abnormality, its slope is fractured with a 160ms duration that greatly exceeds normal values. This data correlates with the measurements made of left ventricular mass and relative wall thickness in these same patients, as in normal patients they have a value of 67g/m^2 and 0.33% respectively vs. 106g/m^2 and 0.49% in the patient with HFpEF Figure 4 and Figure 5.

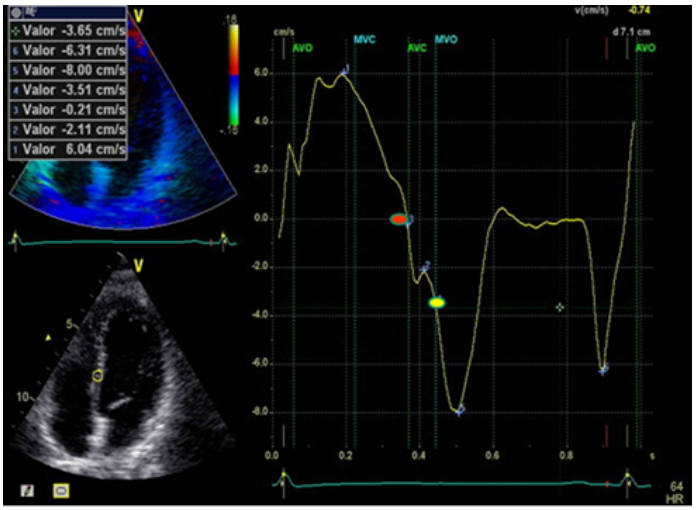

Figure 4: LVPPMC curve in a normal patient. The red dot indicates the beginning of the phase and the yellow dot its completion. Duration: 80ms; left ventricular mass: 67gr/m^2; relative wall thickness: 0.33%.

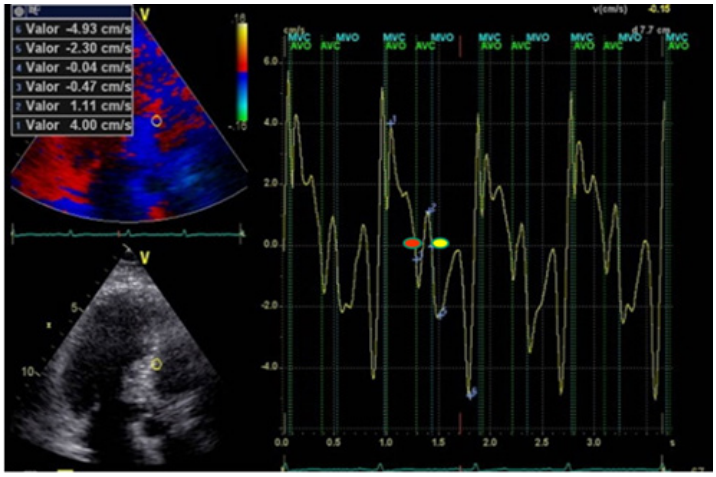

Figure 5: LVPPMC curve in a patient with HFpEF. The red dot indicates the beginning of the phase and the yellow dot its completion. Duration 160ms; left ventricular mass: 106gr/m^2; relative wall thickness: 0.49%.

This analysis implies considering the LVPPMC as the physiological moment of ventricular lengthening and detorsion of the cardiac cycle where the alteration would be found in patients with HFpEF. This "sucking" force acts on the LVPPMC by lengthening the ascending segment fibers to generate the negative pressure that allows MV opening to produce rapid ventricular filling. In echocardiographic studies, the last postsystolic strain occurred later in the segments of the interventricular *septum* (basal and apical anteroseptal segments) [18]. There are factors that influence filling. In fact, changes in wall thickness condition ventricular filling and the diastolic blood inflow that takes place in the coronary arteries may participate in active myocardial expansion. The increase in coronary reservoir during diastole distends the myocardial mass as if the ventricle were a corpus cavernous generating an erectile mechanism that prolongs the ventricular chamber [19]. The LVPPMC is an important mechanism to complete pulmonary capillary circulation towards the LV, and therefore, essential to maintain the continuity between the two circulations, systemic and pulmonary. The venous pressure in both circulations is similar; therefore, left ventricular suction plays a fundamental role to sustain circulation as we have seen in our experiments with exclusion of the RV. Impaired left ventricular suction energy can lead to regurgitation in which there is pulmonary congestion with normal ejection fraction, as this is only a proportion of end-diastolic volume.

Systemic vascular resistance is a faculty of the vascular system, while the term "output volume" refers to the power of the heart. Therefore, the concept that blood pressure is determined by the properties of the vascular system and cardiac power is accurate, but that "output volume" is synonymous of cardiac power is physically inaccurate [20]. Therefore, ejection fraction does not reflect the true state of cardiac capacity [6]. In essence, left ventricular 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, right ventricular suction helps blood filling after left ventricular ejection

and passing through a zone of resistance such as the systemic capillary circuit with a pressure drop to values between 0 and 2mmHg. The resistance of the two systems (pulmonary and systemic) and the pressures developed by the ventricles are proportional, which entails the same relationship in energy consumption, but not in the times, which will depend on their respective power. It is necessary for flow (output), i.e. the volume moved in a certain time, to be the same in both circulations in order to achieve continuity [21,22]. Therefore, the total power of both circulations has to be adjusted to ensure continuity.

In cardiac morphology, the ventricular chambers are cavities inserted within a single muscle mass made up by the continuous helical myocardium. Therefore, the muscle that generates the drive-suction in one ventricle shares part of its anatomy with the other ventricle (septal ventricular interdependence) [23], which will vary the relationship between the drive and suction power in the system. This leads us to a very important issue. We should not talk about cardiac energy but cardiac power, because that is how we take into account phase duration. The ratio is 6:1, that is, the RV is proportional to 15% of the LV. How can the RV with lower energy achieve to have the same consumption as the LV? The difference in resistance between the systemic and pulmonary circulation is added to the energy complement that sucks the pulmonary circulation during the LVPPMC in the 80-100ms after systole. With respect to systemic vascular resistance, it increases from the lowest value in the aorta to the highest value in the arterioles, and then decreases up to the RV [24-26]. The same occurs in the pulmonary circulation with maximum increase in the pulmonary arterioles. 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 38.2ms elapses [27]. During this time, the last stage of LV filling occurs in complementarity between RV ejection and LV suction.

1) 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 400ms) consists of three stages (figure 6). The two filling stages, the first and the third, within the 400ms of diastole, coincide with forces that propel blood into the LV. The first is a consequence of the LVPPMC whose duration of 80-100ms 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 40ms 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 (Figure 6). 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 right ventricular 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 (Figure 7). Therefore, when the LA contracts during the second filling of the LV (stage 3), this movement is synchronized with the ejection of the RV, some 40ms before AV opening occurs. In conclusion, in LV diastole we have (Figure 6):

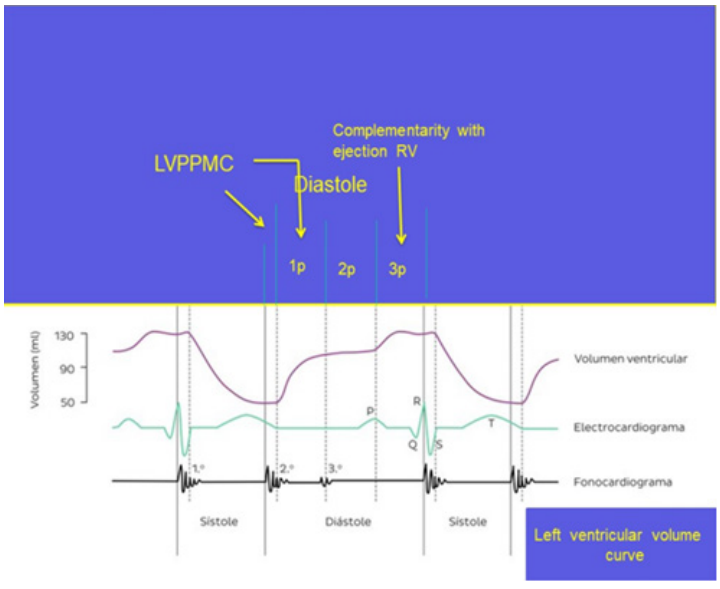

Figure 6: Left ventricular volume curve. Ref. LVPPMC: Left ventricular protodiastolic phase of myocardial contraction, which allows rapid filling of stage 1 of diastole; **1p**: first fast-filling phase; **2p**: diastasis; **3p**: second fast filling phase.

Stage 1: Rapid ventricular filling due to LVPPMC. Its volume corresponds to 70% of filling.

Stage 2: Diastasis. Filling drop to its minimum representing 1% of total volume

Stage 3: Increased flow due to the complementarity derived from the RV, as right ventricular systole is taking place during this period, due to prior opening of the PV before LV diastolic filling ends and the AV opens. This asynchronous timing lasting about 40ms with earlier RV contraction facilitate the stage 3 of left ventricular diastole, which implies 30% of the total volume. There is a curious fact in medical history regarding this topic. It was reported in 1837 by a doctor named King, who belonged to the London Hospital: "*In early systole of the right ventricle, the bulging of the septum towards the right cavity, through a bellows effect, contributes to the expulsion of the RV, favoring the slow filling of the LV*" [28] (Figure 7).

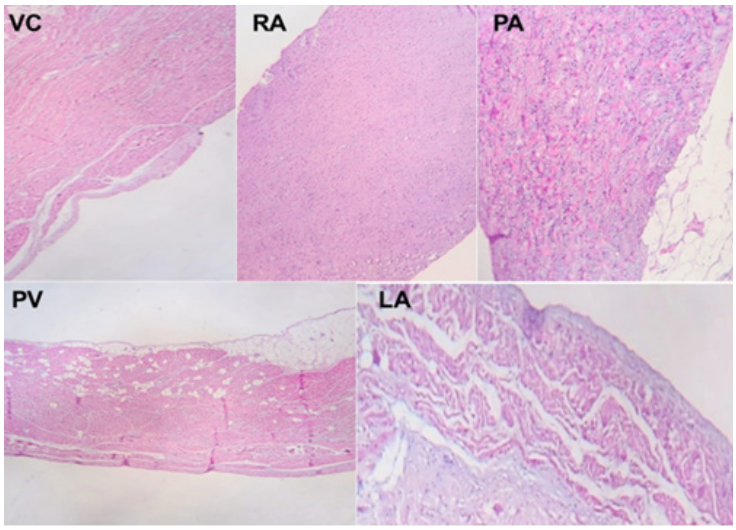

Figure 7: VC. Vena cava: a 3-mm-thick vena cava structure is shown formed in its middle layer by smooth muscle cells in parallel bundles. **RA. Right atrium:** wall formed by myocardial muscle fibers approximately 4mm thick. **PA. Pulmonary artery:** myocytes formed by smooth muscle cells with a spiral 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 4mm thick. (All histological sections belong to bovine heart stained with H&E 10x).

2) 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 200cm/s, blood circulates in the aorta at a mean speed of 40-50cm per second and falls to 0.07cm/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 [24]. 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 RA, called "systemic filling pressure", is around 7mmHg and reaches 2 to 4mmHg in the RA; therefore, this minimum gradient does not allow flow, as it is below the 10mmHg required for circulation ("critical closing pressure"). This physiological circumstance explains the active intraseptal band contraction in the Right Ventricular Protodiastolic Phase of Myocardial Contraction (RVPPMC) with right ventricular 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 [26]. Mean pressure in the venae cavae is between 0 and 2mmHg. Pulsations in the veins suggest right ventricular 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 30.8ms in the RV, while in the LV it lasts an average of 83ms, according to echocardiographic investigations we performed in control patients [5,17]. The values of RVPPMC duration vary in the literature [29]. 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 analo-

gous 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, right ventricular ejection-left ventricular suction and left ventricular ejection-right ventricular 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* (Figure 8) [30-33].

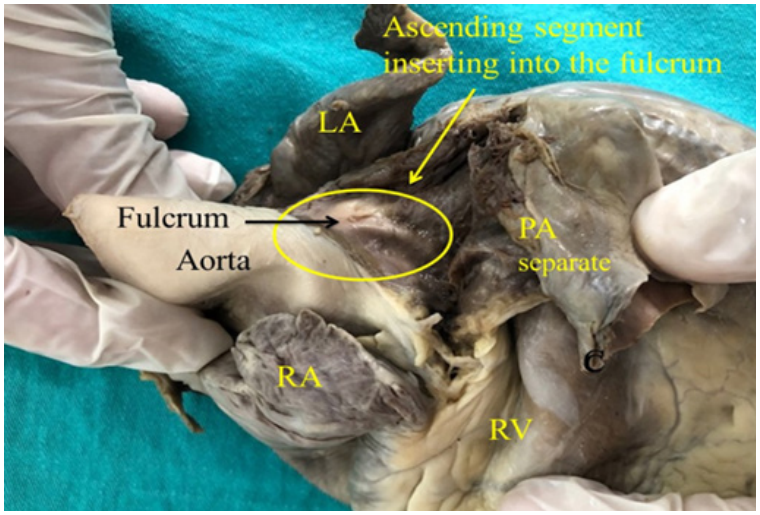

Figure 8: Adult human heart. The ascending segment can be seen inserting into the *cardiac fulcrum*. References: **LA:** Left atrium; **RA:** Right atrium; **PA:** Pulmonary artery; **RV:** Right ventricle.

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).

Acknowledgements

None.

Conflict of Interest

None.

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