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European Journal of Cardio-thoracic Surgery 25 (2004) 376–386

EUROPEAN JOURNAL OF CARDIO-THORACIC SURGERY

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Review

Systolic ventricular filling

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Received 21 October 2003; received in revised form 8 December 2003; accepted 15 December 2003

Summary

The evidence of the ventricular myocardial band (VMB) has revealed unavoidable coherence and mutual coupling of form and function in the ventricular myocardium, making it possible to understand the principles governing electrical, mechanical and energetical events within the human heart.

From the earliest Erasistratus' observations, principal mechanisms responsible for the ventricular filling have still remained obscured. Contemporary experimental and clinical investigations unequivocally support the attitude that only powerful suction force, developed by the normal ventricles, would be able to produce an efficient filling of the ventricular cavities. The true origin and the precise time frame for generating such force are still controversial.

Elastic recoil and muscular contraction were the most commonly mentioned, but yet, still not clearly explained mechanisms involved in the ventricular suction. Classical concepts about timing of successive mechanical events during the cardiac cycle, also do not offer understandable insight into the mechanism of the ventricular filling. The net result is the current state of insufficient knowledge of systolic and particularly diastolic function of normal and diseased heart.

Here we summarize experimental evidence and theoretical backgrounds, which could be useful in understanding the phenomenon of the ventricular filling. Anatomy of the VMB, and recent proofs for its segmental electrical and mechanical activation, undoubtedly indicates that ventricular filling is the consequence of an active muscular contraction. Contraction of the ascendent segment of the VMB, with simultaneous shortening and rectifying of its fibers, produces the paradoxical increase of the ventricular volume and lengthening of its long axis. Specific spatial arrangement of the ascendent segment fibers, their interaction with adjacent descendent segment fibers, elastic elements and intracavitary blood volume (hemoskeleton), explain the physical principles involved in this action. This contraction occurs during the last part of classical systole and the first part of diastole. Therefore, the most important part of ventricular diastole (i.e. the rapid filling phase), in which it receives $>70\%$ of the stroke volume, belongs to the active muscular contraction of the ascendent segment.

We hope that these facts will give rise to new understanding of the principal mechanisms involved in normal and abnormal diastolic heart function.

Keywords: Ventricle; Myocardium; Anatomy; Physiology; Myocardial mechanics

1. Introduction

Biological hysteresis cycle (i.e. natural tendency of any object to recover its previous form) within the ventricular myocardium [1] is sustained by successive shortening

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doi:10.1016/j.ejcts.2003.12.020

and lengthening phases, occurring at different organizational levels, from sub-cellular entities to the whole organ [2]. In the mathematical and biological world, this principle of 'self-similarity' (as the fundamental part of fractal design theories) [3] is well described and applied in ongoing attempts to create an integrative, 'physiomic' approach in analyzing complex structural and functional relationships within one organ or system [4].

Substantial progress has been done recently in elucidating subtle relationships at cellular and molecular levels [5–7], but only with the discovery of the ventricular myocardial band (VMB) [8,9] it became clear that unavoidable coherence and mutual coupling of form and function exist in the entire ventricular myocardium. With this new basic knowledge, it is now possible to understand the principles governing electrical, mechanical and energetical events within the human heart [10,11].

Etymologically, the terms *systole* and *diastole*, which would be frequently mentioned in this article, have been derived from the Greek's words 'stello' (reduction, shortening) and 'diastello' (augmentation, lengthening), respectively.

According to the classical mechanical conceptions [12,13], during the ventricular systole (i.e. from the initial increase of the ventricular pressure to the appearance of the aortic incisura), the initial isovolumetric contraction phase is followed by rapid and slow ejection, with consecutive reduction of the ventricular cavities volume and shortening of their long axes. Those two facts (i.e. volume reduction and long axis shortening), at first glance, may seem clearly coherent with the ventricular myocardial contraction. Similarly, during the classical diastole (i.e. from the appearance of the aortic incisura to the end of atrial contraction), the initial isovolumetric relaxation phase is followed by the auxotonic relaxation phases (i.e. rapid filling, diastasis and atrial contraction), with the increase of ventricular cavities volume and lengthening of their long axes. The mechanical events during this part of classical cardiac cycle (i.e. volume increase and axis lengthening), may also seem coherent with ventricualr myocardial relaxation (active and passive).

But, in reality, the volume increase of the ventricular cavities and consecutive lengthening of their long axes, begins as early as the last part of classical systole (slow ejection) and spans over the initial third of classical diastole (encompassing isovolumetric relaxation and rapid filling phases). Surprisingly, those two crucial actions responsible for the ventricular suction filling (i.e. ventricular volume increase and long axis lengthening) are apparently the results of ventricular myocardial contraction.

At first glance, this statement sounds like contradictio in adjecto. And really, it is difficult to relate causally any contraction (implying shortening) within ventricular wall, with subsequent increase of the ventricular volume and lengthening of its long axis.

The main purpose of this article is to elucidate the force, raised by myocardial contraction in the ventricular wall, capable of producing simultaneous increase of ventricular dimensions (i.e. volume and long axis). Our observations will help clinicians (cardiologists, cardiac surgeons) dealing with systolic and diastolic function of diseased hearts.

2. Historical background

The first idea about heart as a 'pressure-suction pump', exposed as early as in the 3rd century BC, has been attributed to the famous Greek physician, Erasistratus of Chios (304–250 BC). Subsequently, it has been supported by Galen of Pergamon (129–210 AD), who believed that heart contained a distinctive muscle, capable of spreading ventricular walls apart, after each systole [14].

Ever since that time, it was evident that 'vis a tergo' (the 'force from behind' carried out by the blood expelled from the left ventricle), described by William Harvey (1578–1657 AD), was not sufficient to explain venous return and filling of the ventricles [15].

Although the authorities, like Harvey and Albrecht von Haller (1708–1777), have persistently denied such possibility, Zugenbühler (1815) and Schubarth (1817) claimed that ventricles "must suck the blood in order to prevent the formation of an empty space". Wedemayer (1828) has experimentally proved the existence of 'atrial aspiration', connecting a catheter inserted in jugular vein of the horse with water-filled glass tube. Observing excised beating animal hearts, submerged in water (i.e. with no external pressure gradients), Johnson (1823) and Chassignac (1836) have noticed ejection of the fluid during systole and aspiration of the fluid during diastole. Reporting their results of intra-cavitary pressure measurements, Goltz and Gaule (1878) re-focused the attention on 'ventricular aspiration'. These and all subsequent theoretical and experimental works advocating the existence of 'vis a fronte' (the 'force from the front', attracting the blood to the heart) have initiated numerous controversies and emotional reactions. In a review of Gerhard Brecher's seminal book, 'Venous Return', it has been cleverly noted: "Whatever role cardiac suction of venous blood may play in determining circulatory dynamics, no one can deny that mention of this term has proven a most effective method of rising blood pressure in several generations of cardiovascular physiologists" [15,16].

Nowadays, almost 2300 years after Erasistratus, the debate is still going on. Early adherents of the active diastolic concept could not find another reasonable explanation for rapid ventricular filling during short diastolic periods, as during tachycardia and exercise in humans, or in a normal presence of extremely fast heart rates (>600 per min), as seen in some animals (mouse, humming bird). In spite of that, Harvey's position has been supported by many leading authorities at the beginning of past century. Von den Velden (1906), Straub (1910) and particularly Carl Wiggers (1928), could not accept the possibility of vis a fronte ventricular

filling. Almost funny situation arose in Wiggers' own laboratory, when Louis Katz (1930) had experimentally proved diastolic sucking action of the turtle's left ventricle. Similar conceptions, although with different explanations, have been later on (1954) independently elaborated by Torrent-Guasp (active muscular contraction) and Villa (elastic recoil) [15–17].

Two important facts, for proper understanding of this problem, came out from Purkinje's (1843) observations of 'punctum fixum' and 'punctum mobile'. Namely, he has observed that apex of the heart remains motionless, while atrioventricular junction moves toward and from it during ventricular systole and diastole, respectively. Later on (1935), this fact has been proved cinematographically by Benninghof [15].

A series of recently published explanations of ventricular structure and function [1,8–11] have finally shed a new light on this and many other very important issues. Current knowledge of the unique ventricular structure and functional relationships (presented at the NIH-NHLBI Workshop: 'Form and Function: New Views on Disease and Therapies for the Heart', April 25–26, 2002, Bethesda, MD), urges for reconciliation of some exceeded concepts in cardiovascular basic science and clinical practice. Here we offer some experimental evidences and theoretical backgrounds, which could be useful in understanding the phenomenon of the ventricular filling by the active muscular contraction.

3. Ventricular myocardial band—form

The architectural organization plan of the ventricular myocardial fibers is represented by a single muscular band (Fig. 1). This VMB describes two spirals in the space, during its trajectory from the pulmonary artery to the aorta, defining a helicoid which delimitates two cavities, the so-called right (RV) and left ventricles (LV) [1,8–10].

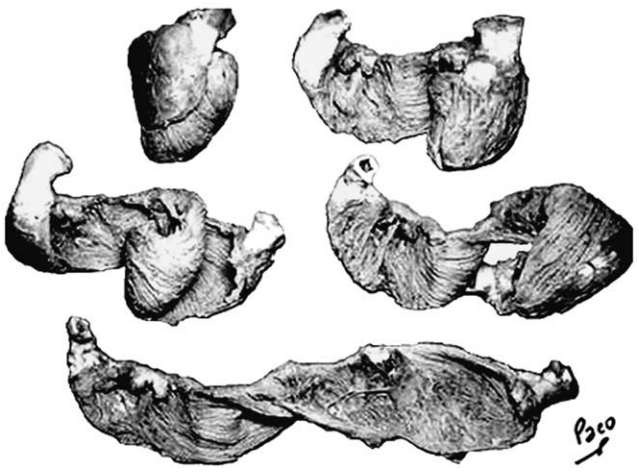


Fig. 1. Five bovine hearts with a similar size showing the successive stages of the ventricular myocardial band dissection. [Reproduced with permission of Ediciones Doyma S.L. From: Torrent-Guasp F. La Mecanica Ventricular. Rev Lat Cardiol 2001;22(2):50].

In relation to their location, the first spiral is designated as the basal loop (BL) and the second one as the apical loop (AL). In both loops, it is possible to distinguish two constitutive segments (Fig. 2): in the BL, the right segment (RS) which corresponds to the RV free wall and the left segment (LS) which participate in constitution of the LV free wall; in the AL, the descendent segment (DS) with fibers coming down from the ventricular base to the apex

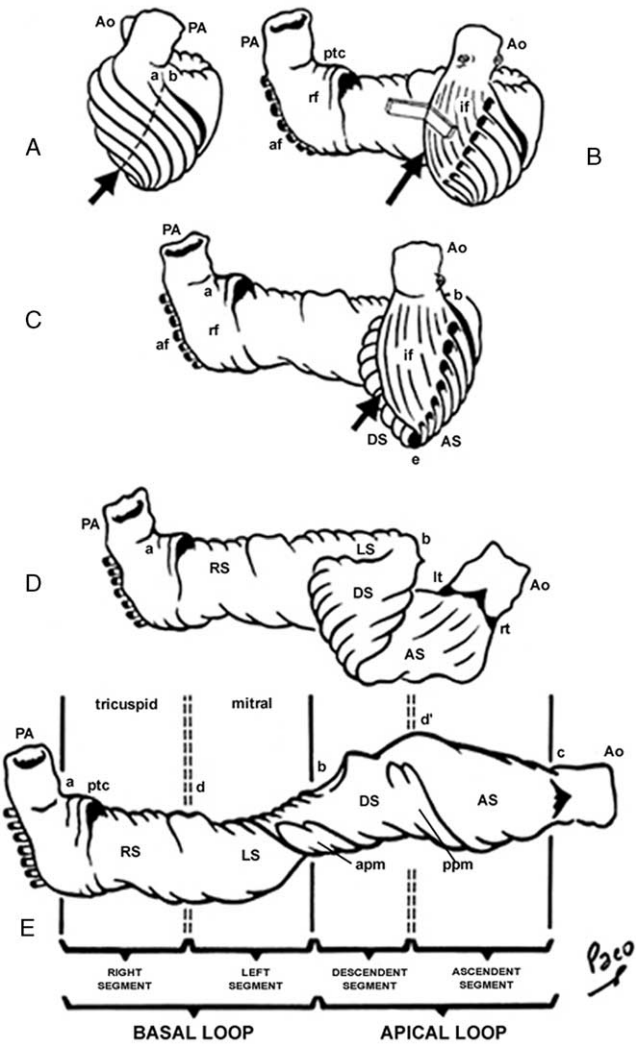


Fig. 2. Schematic presentation of the VMB (compare with Fig. 1). Ao, aorta; PA, pulmonary artery; RS, right segment; LS, left segment; DS, descendent segment; AS, ascendent segment; lt, left fibrous trigone; rt, right fibrous trigone; a, pulmonary artery root; b, VMB central fold; c, aortic root; d,d′, the posterior interventricular sulcus level in wrapped VMB (double dotted lines); e, vortex cordis; apm, anterior papillary muscle; ppm, posterior papillary muscle; ptc, pulmo-tricuspid fibrous cord; af, aberrant fibers coming from the AS (on diagram A they are cut along the dotted line, pointed out by an arrow, to permit the separation of the RV free wall); if, intra-septal fibers coming from the AS; rf, recurrent fibers coming from the RV free wall (RS). Tricuspid and mitral free-wall leaflets attachment line (RS and LS, respectively). [Reproduced with permission of Ediciones Doyma S.L. From: Torrent-Guasp F. La Mecanica Ventricular. Rev Lat Cardiol 2001;22(2):51].

and the ascendant segment (AS) with fibers going up from the apical regions to the basal ones.

In relation to their function, the BL, with fibers running in a transversal plane of the ventricles (i.e. perpendicularly to the ventricular long axis), behaves like the circular muscle embracing by its RS and LS, entire AL. On the contrary, DS and AS fibers of the AL, run in predominantly vertical direction (i.e. almost parallel with ventricular long axis), but with an opposite obliquities (with 'X' crossing at about 90°).

4. Ventricular myocardial band—function

Development of any muscular contractile activity is a function with a foreseeable result, depending on the predominant direction and spatial arrangement of constitutive muscular fibers [1,8–10,18]. Within a helicoid configured by VMB, it is possible to distinguish the four above-mentioned tracts (VMB segments), whose respective fibers adopt different directions along the VMB trajectory. This fact gives rise to a correspondent, specific mechanical result, when each of them contracts in a successive manner during the cardiac cycle. Accordingly, these four segments can be functionally individualized through the different actions they perform during contraction. Therefore, it must be noticed that four segments of the VMB have pronounced functional, rather than morphological personality. Respecting the evident coherence between the form and the function, which always exists in any organ, the double action performed by the ventricles (i.e. the ejection and the suction of blood), could now be explained by means of the above-described structure.

The underlying mechanism of ventricular function could be roughly compared [1,8–10] with mechanical performance of an internal combustion motor engine (Fig. 3A). The BL in that case represents a cylinder, meanwhile the AL would correspond to a piston. In spite of such morphological correspondence, there is a fundamental difference between the ways by which heart and motor engine perform their functions. This difference comes out from the earlier mentioned Purkinje's observations [15]. In the motor engine, the cylinder remains motionless during each working cycle. On the contrary, in the heart it is the cylinder (Fig. 3B), represented by the BL that moves up and down in each cardiac cycle. But, it must be said that the BL does not simply slides as a mobile metal cylinder slipping on the AL (as it will be explained later). It comes up and down accompanying the AL when it lengthens and shortens, respectively. In other words, it happens so with increase and decrease of the ventricular volume during each cardiac cycle, as it was clearly demonstrated by MR imaging of a beating heart in a longitudinal plane [19–21].

Thanks to the incontrovertible results provided from experimental electrophysiological studies by Cox et al. [22,23], and to the recent reports on fast Fourier analyses

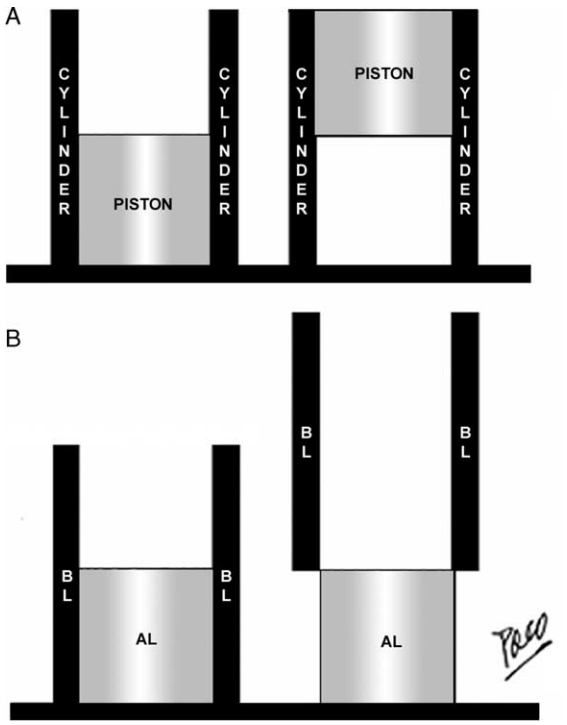


Fig. 3. In (A) the cylinder and the piston of a motor engine work by means of alternative ascending and descending displacements of the piston, meanwhile (B) in the heart it is the cylinder, represented by the BL, that moves up and down since the piston, represented by the AL remains motionless. This fact explains the alternative lengthening and shortening of the ventricular mass. [Reproduced with permission of Ediciones Doyma S.L. From: Torrent-Guasp F. Las razones de la estructura y mecanica ventricular. Rev Lat Cardiol 2000;21(5):163].

of gated blood pool ventriculography by Flotats et al. [24], it is evident that the RV free wall (i.e. the BL-RS) is the first region of the ventricular mass to be excited in each cardiac cycle. Since the excitation and consecutive contraction waves preferably follow the longitudinal axes of the myocardial fibers (cellular and tissue levels), as shown by Robb and Robb [25] and others [26–28], accordingly, it is evident that such progression will occur necessarily along the successive VMB segments (the whole organ level).

5. The contraction of the RS and LS

At the beginning of the classical systole (i.e. isovolumetric phase), successive contraction of the RS and LS, respectively, give rise to a tight embracement of the AL and consecutive increase of the intra-cavitary pressure. Although this kind of 'belt action' performed by the BL does not produce a significant decrease of the ventricular base inner diameter (because at this time, the ventricles are filled up with incompressible blood and the AV valves are closed), it does support and facilitate apropriate coaptation of the AV valve leaflets and the efficient closure of tricuspid and mitral orifices. This fact has been recognized, using different imaging techniques, as the initial change in size

and shape (perimeter and area) of the AV orifices, with the onset of systole [29].

6. The contraction of the DS

Contraction of the DS (Fig. 4B), as shown by tagged MR imaging [19–21], produces three simultaneous actions: shortening of the ventricular longitudinal axis, counter-clockwise rotation (viewed from the apex) of the ventricular base and additional change in size and shape of the AV orifices.

These three actions are causally related with particular spatial arrangement of the DS fibers at the onset of their contraction. Namely, due to their predominant *verticality*, ventricular base is forced to descend toward the motionless apex, thus producing a longitudinal shortening of the ventricular mass cone. Their slight *obliquity* gives rise to the counter-clockwise rotation of the ventricular base, which implicates a torsion of the ventricular mass cone (like 'wringing the wet towel or cloth', as been described long ago by Borelli [11], or like 'squeezing a lemon' as described recently). Finally their *conicity*, pertaining to the conical form of the entire ventricular mass (enforced by previous belt action of the BL), causes the consecutive reduction of the ventricular base dimensions and shape, since it is forced to descend toward narrower, apical part of the cone. It is obvious that these three actions of the DS (together with earlier actions of the BL), could produce an efficient ejection of blood to the great arteries.

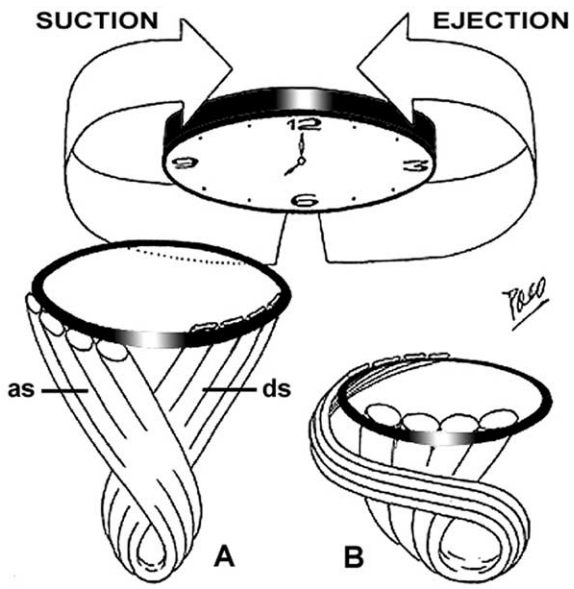


Fig. 4. The AL with its two segments (AS and DS). In (B) contraction of the DS (thickened fibers) obliges the base to perform a descent, a counter-clockwise rotation and a diameter reduction. In (A) the contraction of the AS (thickened fibers) obliges the ventricular base to perform an ascent, clockwise rotation and a diameter augmentation. [Reproduced with permission of Ediciones Doyma S.L. From: Torrent-Guasp F. La Mecanica Ventricular. Rev Lat Cardiol 2003;24(1):30].

7. The contraction of the AS—systolic ventricular filling

Finally, the ultimate part of the VMB that enters contraction during the cardiac cycle is the AS (Fig. 4A). This segment is a key for understanding all those 'secret forces' (commonly mentioned in literature) that are responsible for the ventricular filling [30]. Before any further explanation of AS mechanics, it is important to realize some anatomical facts and physical principles (the last being recently described in detail [31] and incorporated in forthcoming elegant mathematical model, created by the research team of Prof. Morteza Gharib at Californian Institute of Technology [personal communication]).

From the anatomical point of view, it is important to notice two facts. The first one is that the length of the AS is greater than that of the DS (as it has been shown in previous reports) [1,8–11,17,31], and the second one is that the degree of radial inclination (i.e. obliquity) of the fibers, pertaining to the AS (even in a completely reposed ventricular myocardium), overcomes that of the DS. The second fact is a direct consequence of the previous one, keeping in mind that both segments are wrapped around dynamic fulcrum represented by certain volume of blood, which is always present inside the ventricular cavities [32]. Therefore, the intra-ventricular blood does not only passively sustain forces generated by the surrounding myocardium, but also provides a support to it (as a fulcrum) in generating such forces. Because of this interdependence, the term 'hemoskeleton' was introduced to describe this specific role of the intra-ventricular blood. And really, it is more than evident from the clinical practice, that different loading conditions (i.e. dynamic changes of the hemoskeleton), have a great influence on ventricular performance [33–35].

From the physical standpoint, it is important to realize that dynamic hemoskeleton, applying the principles of the first class lever, could change the mechanical efficiency of contracting myocardial fibers by altering the distance and proportion of the curvilinear lever arms. The term 'dynamic', used here to describe the hemoskeleton, emphasizes that the support it gives to surrounding myocardium, varies according to consecutive changes of intra-ventricular blood volume (i.e. fulcrum radius) during the cardiac cycle. In general, the bigger the hemoskeleton, the smaller the lever effect and vice versa.

Specific spatial arrangement of the DS and AS fibers and the presence of dynamic hemoskeleton, impose their complex interplay during the hysteresis cycle. Verticality, obliquity and conicity, although not equally represented, could be regarded as the common denominations for both DS and AS fibers. Predominant verticality in DS and obliquity in AS fibers, along with their X-crossing within

the AL, evidently explain the opposite effects produced by their respective contraction.

Therefore, prior to its own contraction, the AS sustains progressive elongation by ongoing contraction of the DS (Fig. 4B). As mentioned above, predominant longitudinality of the DS fibers, along with presence of initially large hemoskeleton (i.e. end-diastolic blood volume), results in abrupt downward displacement of the ventricular base. At the same time, initially small but (as the ejection of blood progresses) increasing obliquity of the DS fibers, gives rise to counter-clockwise rotation of the ventricular base. This movement, apart from its 'wringing' effect, increases the curvilinear distension of the AS to the maximal point, in which the hemoskeleton is represented by the residual blood volume inside the ventricular cavity.

At this moment, the AS segment starts its own contraction. Here, it is of utmost importance to realize two facts. The first one is that predominant obliquity of the reposed AS has been additionally increased during previous contraction of the DS. The second one, that forthcoming contraction of curvilinearly distended AS, utilizes as a fulcrum the smallest possible hemoskeleton (i.e. residual blood volume), thus attaining the maximal efficiency from the lever mechanism. Therefore, the contraction of the AS necessarily implies simultaneous shortening and pronounced rectifying of its fibers (Fig. 4A). This kind of active biomechanical performance could explain an almost paradoxical fact: ventricular elongation, promoted by muscular contraction.

Putting all these observations together, the contraction of the AS produces three simultaneous actions: abrupt lengthening of the ventricular longitudinal axis, clockwise rotation of the ventricular base, and increase in size and change in shape of the AV orifices. As a net result of these actions, ventricles are rapidly increasing their volumes, and with all valves being closed during that time, a powerful suction force for the atrial blood is thus generated.

As shown by Katz in 1930 [30], the fact that intraventricular pressure drops ($dP/dt < 0$) even during the rapid filling phase, clearly points out that it has to be preceded by a large and rapid increase of the ventricular volume ($dV/dt > 0$), capable of producing the effective suction force ($dP/dV < 0$). Now, it is evident that such increase of the ventricular volume is produced by the contraction of the AS.

Another important question pertains to the chronological location of the AS contraction during the cardiac cycle. The contraction of the AS starts at the summit of the ventricular pressure curve and finishes at the end of the suction phase (i.e. rapid filling phase in classical conception). And really, the *effects* of the AS contraction, fulfill the etymological criteria defining diastole (i.e. augmentation, lengthening). But, in fact, the actual *mechanism* by which the AS attains its effects is the muscular contraction. Since the systole has been considered as a synonym for muscular contractile activity, applying this criterion, AS contraction could be regarded as a systolic event. Accordingly, it is clear that the title of this article has been intentionally composed in a manner to emphasize that ventricular filling is an active phenomenon, performed by the muscular contraction of the AS fibers.

8. The cardiac cycle

Since the first ideas about chronology of mechanical events presented by Wiggers (1915), and later on assembled in the first description of the cardiac cycle by Lewis (1920), there were many attempts to incorporate new knowledge in the old frame (Mejiler and Brutsaert, 1978; Zile, 1989; Gillebert, 1994) [36–38]. Although many clinicians believed that these efforts have had merely academic or didactic relevance, each day, more and more of them are changing this attitude. Two decades long, and yet fruitless efforts in resolving diastolic heart function (and failure), along with many other similar examples, have been the main reasons for such turnover.

After anatomical and functional explanations given here, it is almost natural to incorporate them in the logical sequence of the mechanical events during the cardiac cycle. The classical conception (Fig. 5B) about cardiac cycle is presented together with the new one (Fig. 5A) in order to make easier understanding of crucial differences between them.

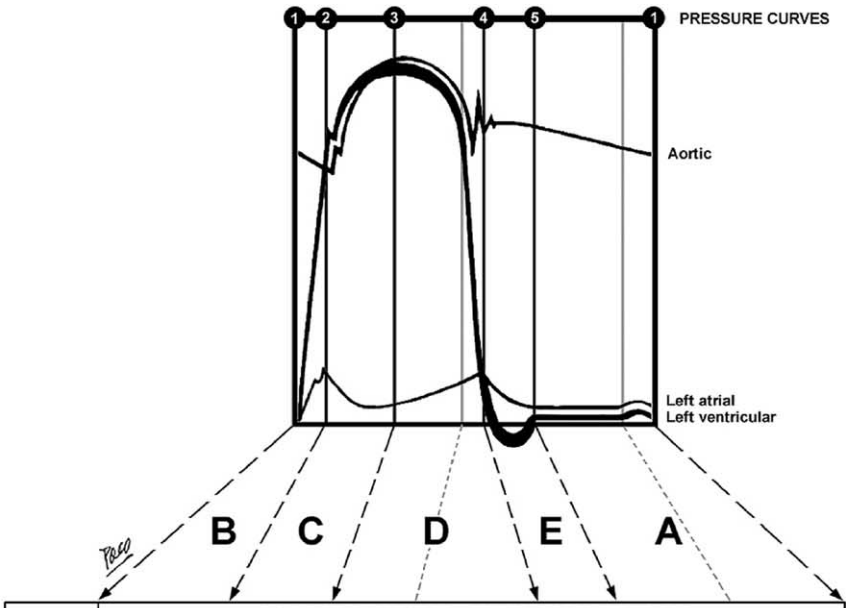
Successive contraction and relaxation of the VMB segments produces several fundamental movements of the ventricular mass: narrowing, shortening (with torsion or twisting), lengthening (with untorsion or untwisting) and widening. Chronological correlation of these movements with the successive phases of the cardiac cycle is shown in Fig. 6. During the cardiac cycle, ventricular wall also undergoes thickening and thinning. Both phenomena belong to the same hysteresis cycle, representing not only changes in single myocite diameter, but also the specific kinematic interaction between the contractile and elastic element within the ventricular wall.

8.1. Systole

8.1.1. Compression phase (Fig. 6B)

This phase starts at the end of diastole and runs until the first increase of the ventricular pressure finishes (black thick line at the LV pressure curve, diagram 2). The initial pressure increase is produced by the transversal, centripetal *narrowing* of the base of the ventricular cone. This movement occurs due to successive contraction of both segments of the BL (RS and LS). As a consequence of this *belt action of the BL*, occurs an abrupt apex dilatation, since at that time the apical region is still relaxed. The 'apex beat' produced in this manner could be easily felt on the anterior thoracic wall.

In the classical concepts, this period is considered as the *isovolumetric contraction phase*. The term 'isovolumetric',



A	CARDIAC CYCLE - NEW CONCEPTION				
	SYSTOLE		DIASTOLE		DIASTASIS
PHASES	COMPRESSION	EJECTION	DECOMPRESSION	SUCTION	DRAINAGE
MOVEMENT	NARROWING	SHORTENING TWISTING	LENGHTENING UNTWISTING		WIDENING
VOLUME*	DECREASING		INCREASING		NO ESTIMABLE CHANGE
ACTIVITY†	CONTRACTION - 0.5"				REPOSE - 0.30"
DURATION‡	0.05"	0.09"	0.25"	0.11"	0.30"

B	CARDIAC CYCLE - CLASSICAL CONCEPTION						
	SYSTOLE			DIASTOLE			
PHASES	ISOVOLUMETRIC CONTRACTION	RAPID EJECTION	SLOW EJECTION	ISOVOLUMETRIC RELAXATION	RAPID FILLING	DIASTASIS	ATRIAL CONTRACTION
					AUXOTONIC RELAXATION		
VOLUME*	NO CHANGE	DECREASING		NO CHANGE	INCREASING		
ACTIVITY†	CONTRACTION - 0.31"			REPOSE - 0.49"			
DURATION‡	0.05"	0.09"	0.17"	0.08"	0.11"	0.19"	0.11"

* - Ventricular volume.
† - Ventricular muscle activity with duration in seconds.
‡ - Cycle length of 0.8 seconds at heart rate of 75/min.

A) - After F. Torrent-Guasp (see references 1, 8-10, 17, 31)
B) - Phases from M.R. Zile (see reference 37)

Fig. 5. Cardiac cycle. (Upper tray) Pressure curves (aorta, LA, LV) with black vertical lines correlating to the new conception, and supplemental vertical gray lines depicting some phases specific to the classical conception. (Lower tray) Comparison (arrows) of the new (A) and the classical (B) conception of chronological sequences of mechanical events (compare with Fig. 6).

in fact, does not reflect the reality, since the increase of pressure, registered during this phase, as an incontrovertible condition, requires a certain (even slight) degree of ventricular volume decrease.

8.1.2. Ejection phase (Fig. 6C)

This phase starts at the end of the compression phase and runs along the second increase of the ventricular pressure up to its summit (black thick line at the LV pressure curve, diagram 3). This pressure increase is produced by *shortening* of the ventricular cone due to a descending movement of the ventricular base (see adjunct triangle to diagram 3). This movement occurs due to the contraction of the DS. Because of its spatial orientation, contraction of the DS simultaneously produces a *counter-clockwise rotation* of the ventricular base, which in turn gives rise to a *torsion or twist* of the entire ventricular mass. The ejection of blood to the aorta and pulmonary arteries, takes place during this phase.

In the classical concepts, this period is considered as the *rapid ejection phase*. It is believed that during this phase,

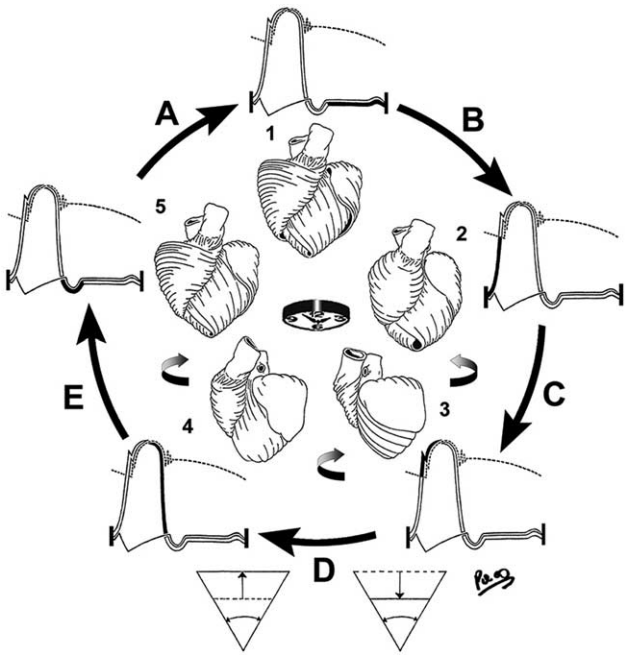


Fig. 6. The five successive phases of the cardiac cycle, each of them corresponding to the black tract of the double line of the LV pressure curve that appears with the aortic pressure curve (dotted line) and the LA pressure curve (simple continuous line). The five diagrams represent the respective configuration adopted by the ventricular cone at the end of those phases shown by the arc of five corresponding arrows: A, drainage phase; B, compression phase; C, ejection phase; D, decompression phase; E, suction phase (compare with Fig. 5). [Reproduced with permission of Ediciones Doyma S.L. From: Torrent-Guasp F. La Mecanica Ventricular. Rev Lat Cardiol 2003;24(1):31].

ventricular cavity suffers significant constriction (i.e. transversal shortening) in order to eject blood. This 'optical illusion' appearing on transversal plane MRI is produced by the reduction of the height of the ventricular mass cone (due to its descent toward the apex). As a proof of this statement, it has been observed on MRI that the angle of the ventricular cone vertex remains constant along entire cardiac cycle.

8.2. Diastole

8.2.1. Decompression phase (Fig. 6D)

This phase starts at the end of the ejection phase and runs until decrease of the ventricular pressure reaches the atrial pressure level (black thick line at the LV pressure curve, diagram 4). This pressure decrease is produced by *partial lengthening* of the ventricular mass cone, due to initial ascending movement of the ventricular base (see adjunct triangle to diagram 4). This movement is a consequence of the initial contraction of the AS. Because of its spatial orientation, contraction of the AS, simultaneously produces an *initial clockwise rotation* of the ventricular base, which in turn gives rise to *partial untorsion or untwist* of the entire ventricular mass. During this phase, the residual volume of blood within ventricular cavities is released from any myocardial compression.

In the classical concepts, this period encompasses the *slow ejection phase* and *isovolumetric relaxation phase*. Similarly as previous, the term isovolumetric does not suit the reality, since the decrease of pressure, registered during this period, neccessairly implies certain level of increase of the ventricular cavities volume.

8.2.2. Suction phase (Fig. 6E)

This phase starts at the end of the decompression and runs until the end of the second, successive increase of the ventricular pressure (thick line at the LV pressure curve, diagram 5). The pressure decrease in suction phase is produced by *complete lengthening* of the ventricular mass cone, due to the final ascending movement of the ventricular base (see adjunct triangle to diagram 4). This movement also occurs due to the contraction of the AS. Because of its spatial orientation, the AS simultaneously produces the *final clockwise rotation* of the ventricular base, which in turn gives rise to *complete untorsion* or *untwist* of the entire ventricular mass. During this phase occurs an active suction of the blood from the atria.

In the classical concepts, this period is considered as the *rapid filling phase*. It is said, that during this phase, occurs significant transverse enlargement of the ventricular cavities. But, as explained earlier (now in the contrapose), this is also an optical phenomenon evident on MRI produced by the ascending movement of the ventricular base along its conical path.

8.3. Diastasis

8.3.1. Drainage phase and atrial contraction phase (Fig. 6A)

The drainage phase, occupying the whole diastasis period, starts at the end of the suction phase and ends when atrial contraction occurs, just before the next compression phase (black thick line at the LV pressure curve, diagram 4). Low and uniform pressure during this period is a result of complete *relaxation and repose* of the entire VMB. The *widening* of the ventricular cavities, started with AS contraction in previous phases, is thus completed. Since the ventricles have been already filled up with blood, passage of the blood from the atria in this period is reduced to neglectedly low levels.

In the classical concepts, this period belongs to the diastole, and is designated as *diastasis phase*, which precedes *atrial contraction phase*. The term 'diastasis' [from Greek 'dia' (to go through) and 'stasis' (stagnation)] seems very appropriate, since it describes an interruption of the atrio-ventricular blood flow during this period. Nevertheless, it can be accepted that there is a slow dripping of blood during diastasis. The contribution of the atrial contraction to ventricular filling, even according to the classical conceptions, is disputable.

9. Discussion

Any unresolved problem in medical science, automatically becomes the unique kind of Rorschach's test. Structure and function of the ventricular myocardium is, perhaps, the best example for the previous statement. This problem has gathered (or divided) numerous experts from different branches. Today, it is not unusual to find that mathematician and engineer are talking about the heart in a very comprehensive manner. Paradoxically, even with very 'good team', coherent explanations of some very simple questions are still pending. It is upon us to see why, but it is unavoidable fact that the nature is not so complicated as the scientists are.

Anatomical facts about macroscopic structure of the ventricular myocardium explained in this article, came from the half century long experimental research on more than 1000 dissected hearts belonging to different species. The evidence of the VMB was not only an important structural fact, but also suggested numerous other solutions pertaining to the heart's mechanical and electrical physiology [1,8–10,17,26,31].

Development of new, highly sophisticated diagnostic techniques capable of producing dynamic, simultaneous images of ventricular cavities and ventricular walls as well as their powerful supporting hardware and software, have proved both structural and functional concepts derived from the VMB [19,20,21,34,43].

But still there are a lot of points to be clarified. The mechanisms responsible for ventricular filling have been, for a long time, very difficult to understand and explain even with the present knowledge of the VMB. It was fully accepted that left ventricular filling is facilitated by negative (suction) pressure, produced in the normal LV [30,32]. But who produces it? And how?

The forces capable of generating such a powerful suction have been attributed to many logical and other reasons. Kinematic interaction between the elastic and contractile element within the ventricular wall has been considered, so far, as a major source of diastolic suction forces. Systolic myocardial contraction, decreasing the ventricular volume below its equilibrium levels, produces the elastic deformation of the collagen network. There are opinions that potential energy, stored in stretched collagen fibers, could appear as powerful elastic recoil force in the second phase of the hysteresis cycle (i.e. during the diastole) [13,16,30,36]. Although this possibility sounds logical, there are numerous anatomical and functional data which make it hardly acceptable [30].

Myocardial collagen network arrangement (endo-, epi- and perimysial fibers) and function in different loading and contractile states has been excellently described by LeGrice [39,40]. But ventricular elastic 'machinery' does not exclusively pertain to the myocardial matrix. Recent insights into the specific role of intra-cellular and sarcomeral proteins (e.g. titin) have elucidated their importance in contractile and elastic kinematic interaction. This interaction is essential for the uniform distribution of stress and strain within ventricular wall, which in turn, maintains the structural (and functional) stability of the sarcomere [5–7,13,18,39–41]. And really, it has been proved that remarkable uniformity of sarcomere length (2.25–1.5 μm) exists in the individual as well as in adjacent myocytes. Whit sarcomere shortening capacity of 10–20%, the heart is able to produce the ejection fraction of up to 60%, and systolic wall thickening of up to 30% [5–7,18,23]. It is obvious from these facts that some kind of amplification factor has to be involved in this functional augmentation during the systole. Spatial organization of VMB segments, their sequential activation and interaction with dynamic hemoskeleton utilizing the principles of lever explains the anatomical and mechanical background applied in this functional amplification. It was also estimated that $<50\%$ of normal systolic wall thickening could be explained by the increase of myocyte diameters, and that $>50\%$ of it occurs due to the transverse shear ('slippage') along myocardial cleavage planes (layers) [18,40,42]. The reports on estimated percentage of systolic wall thickening varies according to the applied methodology [18,19,23]. Overestimation of this systolic phenomenon using MRI analyses [19–21], as mentioned earlier, could be explained by the descending movement of the BL along the ventricular mass cone (see adjunct triangles to diagrams 3 and 4 in Fig. 6). Furthermore, Hexeberg has shown a significant transmural anisotropy of thickness between different layers during the systolic contraction. He has proved that thickening of the distinct myocardial layer does not only reflect the work performed by its constitutive fibers, but also the work of all other transmural layers [42]. These observations are also concordant with architectural plane and sequential activation of VMB segments. Therefore, the thickening of the ventricular wall during the systole appears to be a consequence of several factors, occurring during successive, longitudinal contraction along VMB. Histological arrangement of the myocardial fibers and layers (i.e. their three-dimensional structure, achieved by interconnection of the cardiomyocite lateral anastomotic branches) clearly defines the limits of both thickening and thinning within a particular layer during the longitudinal slippage of its fibers [5–7]. This limitation is also applicable to collagen network surrounding the contractile elements. As a consequence, elastic deformation forces generated within the ventricular wall, do not posses kinetic capacities, compared to those generated by active muscular contraction. The best evidence of this statement is the velocity of the ventricular filling, which could not be explained by slow, passive elastic recoil [19–21]. Such a powerful (and fast) force may only belong to the muscular contraction. Moreover, even this conclusion may be insufficient in understanding the efficient ventricular filling during the extremely fast heart rates [15,16,28,43]. Further steps in ongoing research of the electrical

transmission along the VMB could offer some explanations in the near future [43].

Muscular origin of diastolic suction, since earliest Galenic observations, has been advocated and denied from time to time. Ongoing debate about ventricular long axis function, in the recent literature, indicates that numerous investigators have indirectly proved the muscular (active) origin of the ventricular suction filling, even without any intention to do so. Most of them did so by observing systolic and diastolic behavior of the LV long axis on echocardiography. Sanderson et al. [44] was trying to explain the strange elongation of the LV long axis, discordant with Doppler mitral velocities, during the early diastole. He concluded that LV recoil was dependent on the energy stored by previous systole, and that impaired LV contraction during systole will lead to reduced suction and early diastolic filling. Instead of muscular contraction (i.e. AS contraction) directly responsible for early diastolic lengthening of the LV long axis, he believed that such force belongs to the elastic recoil generated during previous ejection phase. In his and in others' opinions [30,32], only the powerful contraction capable of emptying the LV below its equilibrium volume could produce effective suction pressure. Since he had correctly noticed that LV long axis elongates due to the rapid upward movement of the ventricular base, his insisting on the elastic recoil forces was strange. Interestingly, very soon after the previous report, Sanderson et al. [45] had realized "that factors other than intrinsic recoil or stored energy from the previous systole, influence the left ventricular long axis velocity in early diastole." In a series of editorial articles about normal and abnormal long axis function, Henein and Gibson [46,47] have emphasized the importance of the 'longitudinally directed fibers' for LV systolic and diastolic function. Without referring any previous knowledge about VMB, their satement is concordant with important functions of the AL segments (DS and AS) described here. They have also realized that elongation of the LV long axis occurs due to the upward movement of the 'mitral ring', and moreover, they claim that such movement could not be regarded as a 'passive consequence'. Surprisingly, they have proposed the 'contraction of the atrial pectinate muscles' as a generating force, capable of pushing up (and very fast?) entire ventricular base. Another interesting observation was presented by Petrie et al. [48]. According to them, 'AV plane displacement' may be considered as the most sensitive parameter of the LV *systolic* function, and very reliable early predictor of poor prognosis in patients with *diastolic* (or 'subtle systolic') heart failure.

Since François-Franck's first suggestion (1877) that most of the ventricular filling occurred in early diastole, it has been shown by many others [16,23,30,32,37,38] that $>70\%$ of the LV stroke volume is received during the first third of diastole (i.e. during classical rapid filling phase). The new concept of the ventricular mechanical events chronology during the cardiac cycle (presented here in comparison with classical one) clearly points out that the period of maximal ventricular filling, is in fact, the period that belongs to the *contraction* of the AS. Therefore, it is very important to realize that healthy heart possesses strong capacities (defined by specific spatial arrangement of the VMB segments) to ensure its own efficient emptying (systolic function) and filling (diastolic function), by means of successive muscular contraction along the VMB.

10. Conclusion

Anatomical data, mechanical and physical principles presented here clearly demonstrate that the contraction of the AS, producing a paradoxical effect—uprising the ventricular base, lengthening the ventricular long axis and thus creating the suction force—presents that 'secret filling force', we were looking for so long time. This force uses the systolic mechanism (i.e. contraction) for ventricular filling during the diastole. Understanding this mechanism could be of particular importance in our efforts to prevent and treat diastolic heart failure.

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