Published in final edited form as:

Comput Methods Biomech Biomed Engin. 2013 January ; 16(1): 66–72. doi:
10.1080/10255842.2011.607442.

An elongation model of left ventricle deformation in diastole

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Abstract

A numerical method of the left ventricle (LV) deformation, an elongation model, was put forth for the study of LV fluid mechanics in diastole. The LV elongated only along the apical axis, and the motion was controlled by the intraventricular flow rate. Two other LV models, a fixed control volume model and a dilation model, were also used for model comparison and the study of LV fluid mechanics. For clinical sphere indices (SIs, between 1.0 and 2.0), the three models showed little difference in pressure and velocity distributions along the apical axis at E-peak. The energy dissipation was lower at a larger SI in that the jet and vortex development was less limited by the LV cavity in the apical direction. LV deformation of apical elongation may represent the primary feature of LV deformation in comparison with the secondary radial expansion. The elongation model of the LV deformation with an appropriate SI is a reasonable, simple method to study LV fluid mechanics in diastole.

Keywords

elongation; left ventricle deformation; prolate spheroid; diastole

1. Introduction

In the numerical study of blood flow in the left ventricle (LV), especially vortex structure in the LV filling, to model the LV deformation is a problem that should be addressed first. One approach is the immersed-boundary method, solving the fluid–structure interaction between the LV wall in a Lagrangian coordinate system and the blood in an Eulerian coordinate system (Peskin and McQueen 1992; Watton et al. 2008; Yin et al. 2010). Combining LV deformation components such as apical and radial expansion, the method of fluid–structure interaction also combines the effects of each deformation component on LV blood flow. It is

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not known how a single deformation component influences the LV flow. Another limitation of this method is that difficulties with large LV deformation exist.

To prescribe LV deformation is an alternative, simple method without the complexity of fluid–structure interaction. One approach is based on the concept of fixed control volume (CV): taking the LV as a fixed CV, the real LV wall deformation is replaced by a velocity distribution given on the control surface (Maisano et al. 1999; Redaelli et al. 2001). The accuracy of the method relies on proper choice of the CV and specific velocity on the control surface, which is often problematic. Indirectly describing the LV deformation, the fixed CV method is usually used to investigate inflow through a mitral valve (MV), rather than fluid mechanics in a whole LV.

Another method to prescribe LV deformation is a dilation model put forward by Pedrizzetti and Domenichini (2005) and Pedrizzetti et al. (2010). They assumed that the LV is a deformable semi-prolate-spheroidal system, and the LV dilates with a constant (or varying) ratio of its two axes in diastole. This dilation model agrees with general observation on LV deformation, and can reveal such LV fluid mechanics as vortex development.

Recently, the research of Claessens et al. (2007) and Stoylen et al. (2001) discovered that the diastolic LV deformation started from the LV base and propagated towards the apex as a stretch strain wave. Their research suggested that LV stretching along the apical axis could be a major component of LV deformation. Therefore, we came up with an LV elongation model, assuming that the LV elongates only along the apical axis in diastole. We validated it by comparing with two other current models of the LV, the fixed CV model of Redaelli et al. (2001) and the dilation model of Pedrizzetti et al. (2010), as mentioned earlier. Meanwhile, we would interpret characteristics of the LV deformation and shape, and then their clinical indications.

2. Method

2.1 LV–MV models

The LV–MV geometry used here was based on the study of Redaelli et al. (2001). The MV had a single orifice, and the whole LV–MV geometry was axisymmetric about the LV apical axis, as the symmetric half shown in Figure 1(a). Three orifice areas were used: 1.5, 2.3 and 3.0 cm². The MV annulus diameter was 3.0 cm and the MV leaflet length was about 1.6 cm. The anterior and posterior leaflets were part of a half prolate-sphere wall and fixed at the maximum opening in diastole. The MV chordae, papillary muscles and other substructures were neglected. The 3.2 cm diameter atrium was truncated 2.0 cm above the LV base (Redaelli et al. 2001).

2.1.1 The elongation model—Considering the LV elongation along the apical axis may be a significant deformation component (Stoylen et al. 2001; Claessens et al. 2007); we put forth the LV deformation model with elongation only along the apical axis. We partitioned the LV–MV system into three zones from the atrium to the apex. Zone I included the atrium and the MV, Zone II was a cylinder as the middle part of the LV and Zone III was a half prolate spheroid as the lower part of the LV with the apex, as shown in Figure 1(b). In diastole, Zone I was fixed without motion; Zone II was elongated along the apical axis with the same velocity as Zone III, moving downward as a rigid body. Thus, only the apical axis length, H , was a function of time. The velocity of Zone II elongating and Zone III descending was derived from the intraventricular flow rate Q (in l/s) (Redaelli et al. 2001), as shown in Figure 1(d): $dH/dt = Q/A_L$, where A_L was the cross-section area of Zone II. The diastole lasted for 0.6 s. The maximum flow rate was 0.27 l/s at E-peak ($t = 0.1$ s). In this elongation model, the initial LV volume was 50 ml, and the final stroke volume was 74.5

ml. For all the LV models to be used here, we defined the ratio of the apical axis length to the LV base diameter as a sphere index, $SI = H/D$, to describe LV roundness, as seen in Figure 1. In the elongation model, the SI rose from initially 0.9 to 1.9 at the end of diastole.

2.1.2 The dilation model—In the dilation model of Pedrizzetti et al. (2010), the LV was a half prolate spheroid. The LV base diameter, D , and the apical axis length, H , were both functions of time, as shown in Figure 1(c). During LV deformation in diastole, we fixed the SI to be constant. Noticing that the temporal derivative of the LV volume was the intraventricular flow rate Q , we acquired the temporal derivatives of the LV base diameter and the apical axis length: $dD/dt = 2Q/(\pi^2 SI)$, and $dH/dt = SI dD/dt$. If $SI = 1.5$, the initial LV volume and the final stroke volume were the same as those in the elongation model above. For $SI > 1.5$, the initial diameter of the LV base was set at 4.54 cm, and the final stroke volume was the same as that in the elongation model because the same intraventricular flow rate curve in Figure 1(d) was used. We studied the dilation model at five SIs: 0.5, 1.0, 1.5, 2.0 and 2.5. Although the cases with the SIs less than 1.4 or greater than 1.7 might have few practical indications, they were studied to offer a broader vision when we inspected real cases in a narrow SI range.

2.1.3 The fixed CV model—In the model of Redaelli et al. (2001), the LV was a half prolate spheroid, as shown in Figure 1(a). In diastole, the diameter at the LV base was fixed at 4.54 cm and the apical axis length at 6.95 cm. Thus, the LV volume was a constant of 75 ml, and the SI of 1.53. The following velocity distribution on the control surface was used to represent the LV wall deformation (Redaelli et al. 2001):

$$v(z, t) = (-0.164E + 38t^{40} + 0.65t) \times \left[5 - 1.9 \left(\frac{(1+z^9)}{7300^2} \right)^{1/9} \right] (1 + 0.004z),$$

in which E is the transmitral velocity at E-peak, t is the time and z is the coordinate measured downward from the atrium inlet. Results from this model were directly cited from the study of Redaelli et al. (2001).

2.2 Haemodynamics model

Blood was assumed to be an incompressible Newtonian fluid with constant density of $\rho = 1060 \text{ kg/m}^3$ and viscosity of $\mu = 0.0027 \text{ Pa s}$. The continuity and Navier–Stokes equations controlled the LV blood flow (neglecting gravity). No-slip conditions were set on all walls. Free inflow conditions were set at the atrium inlet with constant pressure and no velocity gradient. As shown in Figure 1(d), the inflow velocity was derived from the intraventricular flow rate curve. Initially, the flow field was at rest with zero pressure for convenience. The relative pressure was used with no specification.

The dissipation function $\Phi (\Phi = [\nabla \bar{v} + (\nabla \bar{v})^T] : \nabla \bar{v})$ describing the energy irreversibly transferred to heat due to viscosity, was introduced to estimate the LV energy loss. Its product with viscosity, $\mu \Phi$, indicates the energy dissipation rate in Pa/s. We set $\mu \Phi > 540 \text{ Pa/s}$ as a judgement to single out regions with high-energy dissipation when studying SI effects on energy dissipation at E-peak.

2.3 Numerical techniques

The geometry models were created in GAMBIT® (ANSYS, Inc., Canonsburg, Pennsylvania, USA). Zone II in the elongation model in Figure 1(b) was meshed with hex/wedge-elements. All hex/wedge-elements had the same height in the apical direction, forming equally spanned layers. Tet-elements were used to mesh the other regions in Figure 1(b),(c). We

used five mesh sizes (mesh side length), 0.5, 0.7, 1.0, 1.5 and 2.0 mm, to test mesh dependence under the same boundary and initial conditions in the elongation model. Relative to the case with the mesh size of 0.5 mm, the differences of the transmitral velocity (defined at the MV orifice centre) at E-peak were -0.3, -1.8, -2.5 and -63.5 percent for the mesh sizes of 0.7 to 2.0 mm, respectively. We chose the mesh size around 1.0mm in our computation. Such a mesh size yielded about 135K elements in the elongation model and 149K elements in the dilation model (at SI = 1.5) at the initial time step, both of which were more than the 100K tet-elements used in the fixed CV model (Redaelli et al. 2001).

The 3D unsteady problem was solved with the segregated solver in FLUENT® (ANSYS, Inc., Canonsburg, Pennsylvania, USA). The momentum equations were discretised with the second-order upwind scheme in space and the first-order implicit formulation in time (Barth and Jespersen 1989; ANSYS FLUENT 12.0 Documentation 2009). Pressure-implicit with splitting of operators was used for the pressure-velocity coupling (Issa 1986; ANSYS FLUENT 12.0 Documentation 2009).

The dynamic-mesh technique in FLUENT® was used to simulate the LV deformation in the two models in Figure 1(b),(c). For the elongation model in Figure 1(b), no dynamic-mesh was set in Zone I. Zone II was remeshed by the layering dynamic-mesh technique. When the layer adjacent to the moving bottom surface of Zone II increased its height to 1.4 times its original height, it was split into two layers: one with the original height, and the other (still adjacent to the moving bottom surface) with the increased height. Zone III moved apically downward as a rigid body without remeshing. For the dilation model in Figure 1(c), we used the dynamic-mesh technique of remeshing (using the spring-based smoothing method in FLUENT®) to deal with the LV deformation. We set the maximum/minimum element size and the maximum skewness (the shape difference between an element and an equilateral element of the equivalent volume in FLUENT®) at the initial time step as limits to control remeshing. When a tet-element size or skewness was beyond its prescribed limit, a new node was added within the tet-element to split it into four smaller ones, or an old node moved to adjust the sizes and skewnesses of the elements around it (ANSYS FLUENT 12.0 Documentation 2009). The LV base was defined as a deforming surface on the horizontal plane. At the beginning of every time step, Zone II in Figure 1(b) and the LV cavity in Figure 1(c) were remeshed, and Zone III in Figure 1(b) was displaced. For both of the deformation models in Figure 1(b),(c), user-defined files were compiled and hooked for the dynamic-mesh function in FLUENT®.

The time step was fixed at 1 ms, smaller than 3ms used in the fixed CV model (Redaelli et al. 2001). Six hundred time steps were needed to complete a diastolic simulation of 0.6 s. Calculations were carried out on DELL® Precision T3400 workstations.

3. Results

In Figure 2, pressure and velocity distributions along the apical axis were compared between the three LV models at E-peak and the three orifice areas. Here the SIs were 1.1, 1.5 and 1.53 in the elongation, dilation and fixed CV models, respectively. From the initial moment to E-peak, the apical axis length of the LV increased by 0.7 cm from 4.1 to 4.8 cm (by 4.6 cm to finally 8.7 cm at the end of diastole) in the elongation model, and by 0.4 cm from 4.6 to 5.0 cm (by 3.8 cm to finally 8.4 cm) in the dilation model at SI = 1.5 [in which the LV base diameter also increased by 0.5 cm from 4.5 to 4.9 cm (by 1.1 cm to finally 5.6 cm)], whereas it was a constant at 7.0 cm in the fixed CV model (Redaelli et al. 2001). For all three models in Figure 2(a), pressure along the apical axis decreased from zero at the atrium inlet to a minimum at a position between the MV and the apex, and then restored to a certain degree near the apex. In contrast, velocity in Figure 2(b) rose from a low value at the atrium

inlet to a maximum below the MV, and then dropped to zero at the apex. The minimum pressure and the maximum velocity appeared at nearly the same apical position. With the increase of the MV orifice area, the magnitudes of both the minimum pressure and the maximum velocity decreased. At the largest orifice area of 3.0 cm^2 , the pressure had the lowest drop of 5 mmHg (667 Pa) and the velocity had the smallest increment of 1 m/s. Relative to the fixed CV model, the largest difference of the minimum pressure, 20.5%, appeared in the elongation model at the orifice area of 3.0 cm^2 , whereas the largest difference of the maximum velocity, 7.7%, appeared in both the elongation model and the dilation model (at SI = 1.5) at the orifice area of 2.3 cm^2 , as listed in Table 1. Comparing the elongation model and the dilation model, however, their maximum difference was only 6.1%, appearing in the minimum pressure at the orifice area of 3.0 cm^2 .

Figure 3 shows the maximum pressure drop (measured from the atrium inlet, equivalent to the magnitude of the minimum pressure), maximum velocity and energy loss (estimated from the Bernoulli equation) scaled by those from the fixed CV model along the apical axis at six SIs, at E-peak and the orifice area of 2.3 cm^2 . Only at SI = 0.5, the maximum pressure drop and velocity were lower than those in the fixed CV model, whereas all energy loss shown here was lower than that in the fixed CV model. With the SI increasing from 0.5 to 2.5, the maximum pressure drop rose from 0.75 to 1.09 at SI = 1.1, and then slightly decreased to 1.03. Similarly, the maximum velocity increased from 0.91 to 1.07 at SI = 1.5 and then decreased to 1.04. In contrast, the energy loss increased from 0.52 to 0.98 at SI = 1.1, decreased to a lower value of 0.82 at SI = 2.0 and then increased somewhat to 0.89.

Figure 4(a),(b) shows velocity vector fields and distributions of the energy dissipation rate higher than 540 Pa/s on the symmetric plane at E-peak in the two dilation models at SI = 2.5 and 0.5. A vortex pair appeared around the jet. Compared with the vortex pair at SI = 2.5 in Figure 4(a), the vortex pair at SI = 0.5 in Figure 4(b) tended to be underdeveloped and restricted towards the LV base corner due to the limited LV space in the apical direction. The maximum velocity of 1.4 m/s at SI = 2.5 was about twice that at SI = 0.5. The energy mainly dissipated in the regions in which the vortex development was blocked by the LV wall, as in the denoted regions of high-energy dissipation ($\mu\Phi > 540 \text{ Pa/s}$) shown in Figure 4(a) (too small to be discernible) and Figure 4(b). The relative energy dissipation was much stronger at SI = 0.5 than at SI = 2.5. The percentage of high-energy dissipation ($\mu\Phi > 540 \text{ Pa/s}$) in the total energy dissipation at E-peak decreased from 85% at SI = 0.5 to lower than 15% at SI = 1.5, as shown in Figure 4(c).

Figure 5 shows that the energy dissipation rate (averaged in the whole LV) was higher at the smaller SI. The maximum energy dissipation rate decreased from 401 Pa/s at SI = 0.5 to 170 Pa/s at SI = 2.5. With the increase in SI, the time needed for the energy dissipation rate to reach the maximum increased from 0.14 s at SI = 0.5 to 0.26 s at SI = 2.5.

4. Discussion

The elongation model could be used to study the LV fluid mechanics. Current image observations show that the LV has the shape of a half prolate spheroid with a SI varying from normally 1.7 to pathologically 1.4 near the end of diastole (Hong et al. 2008). The elongation model maintained the shape feature of the LV at a larger SI through increasing the apical dimension in diastole. Result similarity shown in Figure 2 indicated that neglecting the radial deformation did not affect the primary fluid mechanics of the LV. Therefore, elongation might play a major role in LV deformation, as indicated in the study of LV stretching propagation (Stoylen et al. 2001; Claessens et al. 2007).

As shown in Figure 2 and Table 1, however, the LV geometry difference among the models, especially in the apical dimension, caused a difference in result. Relatively, the elongation model did not differ so much from the dilation model as from the fixed CV in Table 1, since the elongation model yielded an LV geometry much closer to that in the dilation model at SI = 1.5. Another reason for greater similarity between the elongation model and dilation model (SI = 1.5) was that they used the same time step, one-third of that in the fixed CV model. For the minimum pressure and maximum velocity in Figure 2, the former seemed more sensitive to the model difference by producing larger difference, as listed in Table 1. This phenomenon could be a result of the linear pressure and squared velocity relationship governed by the Bernoulli equation: theoretically, pressure uncertainty may be as much as two times velocity uncertainty when uncertainty exists. Figure 2 also shows a conclusion from the Bernoulli equation that the minimum pressure accompanies the maximum velocity at a position (here, neglecting gravity and assuming energy loss to be relatively small).

Different from Figure 2, in which the LV models had the comparable SIs around 1.5, Figure 3 shows the impact of the SI on the LV flow. The SI denoted the apical LV space for the blood jet to develop before impinging the LV wall. In general, higher velocity accompanied by higher pressure drop and lower energy loss is favourable for blood ejection in the following cardiac phase of systole. For SI as small as 0.5, the pressure drop and the velocity had little chance to reach larger magnitudes before the jet impinged the LV wall. Although the energy loss at SI = 0.5 was small, it did not guarantee the high dynamic efficiency of the LV. On the one hand, the low-energy loss, based on the Bernoulli equation, might be the result of mild pressure drop and slow blood flow. On the other hand, the energy loss in Figure 3 was investigated locally along the apical axis and transiently at E-peak, not globally in the whole LV as in Figure 4, or continuously during the whole diastole, as in Figure 5, in either of which the dilation model at SI = 0.5 showed the least dynamic efficiency of the LV in terms of the energy dissipation rate. The range of SI > 1.0 might meet the dynamic requirement to blood ejection. It was noted that the confinement of the LV wall to the blood flow was weakened quickly with the increase in the SI. At SI = 1.5, 2.0 and 2.5, the minimum pressure (at E-peak and the orifice area of 2.3 cm²) appeared nearly at the same position on the apical axis with a similar magnitude; so did the maximum velocity. The differences in the maximum pressure drop and maximum velocity were within 10% in the range of SI > 1.0, which could be a necessary SI condition to neglect result discrepancy caused by models (though the energy loss difference within 20% was relatively large, energy loss calculated here along a line of the apical axis drew little concern in clinical practice). Considering the real LV, the narrower range, 1.0 < SI < 2.0, was more reasonable for an LV model. In this range, the elongation model showed further agreement on energy loss with the fixed CV model and the dilation model at SI = 1.5.

Figure 4 (at E-peak) and Figure 5 (in the whole diastole) more directly reveal the effect of the SI on the LV flow in terms of the energy dissipation rate in that relatively less energy would be lost at the larger SI. The larger SI enabled the jet to develop in a larger apical dimension for a longer time before the jet impinged the LV wall. Consequently, the vortex accompanied with the jet dissipated less severely near the LV wall. Meanwhile, the appearance of the quickest energy dissipation was postponed, as shown in Figure 5. Using a power-law function to approximate the relationship between the energy dissipation percentage and the SI in Figure 4(c), we obtained an exponent of the SI about -3. The negative exponent indicated that the flow was more affected by the prolate-spheroidal shape of the LV at a smaller SI, which was often related to a pathological LV condition (Hong et al. 2008). Taking SI = 1.4 and 1.7 as the typical SIs for the pathological and normal LVs, we estimated that the percentage of high-energy dissipation ($\mu\Phi > 540$ Pa/s) at SI = 1.4 was 1.8 times than that at SI = 1.7 at E-peak. As SI > 1.5, little difference was observed in Figure

4(c). In terms of the energy dissipation rate, the SI of 1.5 could be a reference to distinguish the normal from abnormal LVs.

For the numerical techniques used in the LV models, the elongation model was relatively easy to realise. The elongation model used the layering dynamic-mesh technique in FLUENT® to remesh deformable Zone II, which could maintain the constant mesh quality of hex/wedge-elements in the process of computation by adding new layers of hex/wedge-elements of the same size. The skewness in the added layers was a constant about 0.45, much lower than the maximum skewness of 0.76 in the whole LV. As a comparison, the remeshing dynamic-mesh technique in the dilation model worsened the skewness of the deformed or added tet-elements, though limitation parameters of the element size and skewness were set to control the mesh quality. For instance, the skewness, going up to 0.9, would cause trouble in convergence. To control the mesh quality and obtain convergence, smaller mesh sizes and more iteration steps were chosen during the remeshing process in the dilation model, which doubtlessly increased computation time. At the end of diastole, the dilation model with SI = 1.5 increased the element amount by 25% to 186K, whereas the elongation model increased the element amount by only 2% to 138K to reach the same stroke volume. Thus, by ensuring better and quicker solution convergence, the simplicity of the layering dynamic-mesh technique applied in the elongation model brought advantages to the practice of computational fluid dynamics in the study of LV fluid mechanics.

There were several limitations in this study. Although the assumption of the axisymmetric LV was still made for the purpose of comparison with the fixed CV model (Redaelli et al. 2001), axisymmetry of the LV could cause errors under real conditions. It should also be pointed out that no deformation and no substructures of the MV could lead to discrepancy with real physiological conditions.

5. Conclusion

We put forth an LV elongation model for the study of LV fluid mechanics in diastole: the LV elongated only along the apical axis. By comparing with other two LV models, the fixed CV model and the dilation model, the LV elongation model yields similar LV fluid mechanics to the other two models at the SI between 1.0 and 2.0 (usually 1.5 for a normal LV). The LV elongation model can be used for numerical study of LV fluid mechanics due to its reasonability and simplicity in reduction of numerical uncertainty and computational cost.

Acknowledgments

This study was supported by an Exploratory/Developmental Research Grant Award from the National Heart, Lung, and Blood Institute (grant No. R21HL102526).

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Figure 1.

The sectional view of the LV models. (a) The fixed CV model (Redaelli et al. 2001). Both the LV base diameter, D , and the apical axis length, H , were constant. (b) The elongation model. The LV was composed of three zones: Zone I (including the atrium and the MV), Zone II (cylinder) and Zone III (half prolate spheroid). The LV base diameter, D , was a constant, and the apical axis length, $H(t)$, was a function of time. (c) The dilation model (Pedrizzetti et al. 2010). Both the LV base diameter, $D(t)$, and the apical axis length, $H(t)$, were functions of time. (d) The intraventricular flow rate as a function of time in diastole (Redaelli et al. 2001).

Figure 2.

(a) Pressure and (b) velocity distributions along the apical axis at E-peak and three orifice areas of 1.5, 2.3 and 3.0 cm², and in three LV models: elongation (solid line, SI = 1.1), dilation (solid line with hollow squares, SI = 1.5) and the fixed CV (hollow squares, SI = 1.53).

Figure 3.

The maximum pressure drop, maximum velocity and energy loss along the apical axis at E-peak and the orifice area of 2.3 cm². The values were scaled by those from the fixed CV model. SI = 1.1 was the SI of the elongation model at E-peak; the other five, SI = 0.5, 1.0, 1.5, 2.0 and 2.5, were the SIs of the dilation model.

Figure 4.

The velocity vector fields and high-energy dissipation distribution (the denoted region by shadow) on the symmetric plane at E-peak in two dilation models at SI = (a) 2.5 and (b) 0.5. In the denoted regions, the energy dissipation rate $\mu\Phi > 540$ Pa/s. (c) The percentage of energy dissipated in the denoted regions ($\mu\Phi > 540$ Pa/s, near the LV wall) to the total energy dissipated in the entire LV. SI = 1.1 was the SI of the elongation model at E-peak; SI = 0.5, 1.0, 1.5, 2.0 and 2.5 were the SIs of the dilation model. The orifice area was 2.3 cm².

Figure 5.

The energy dissipation rates (volume averaged) in terms of time in the dilation model at SI = 0.5, 1.0, 1.5, 2.0, 2.5, and in the elongation model at SI = 0.9–1.9. The orifice area was 2.3 cm².

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Table 1

Relative differences of minimum pressure and maximum velocity among the elongation, dilation (SI = 1.5) and fixed CV models.

Relative difference (%)					
Minimum pressure			Maximum velocity		
Orifice area (cm ²)	Model	Relative to fixed CV	Relative to dilation	Relative to fixed CV	Relative to dilation
1.5	Elongation	4.2	4.6	4.5	4.5
	Dilation	-0.4		0	
2.3	Elongation	12	0.9	7.7	0
	Dilation	11		7.7	
3.0	Elongation	20.5	6.1	4.3	3.2
	Dilation	13.6		1.1	

The values were defined on the apical axis at E-peak for three MV orifice areas of 1.5, 2.3 and 3.0 cm².