European Journal of Clinical and Biomedical Sciences
2017; 3(6): 129-133
http://www.sciencepublishinggroup.com/j/ejcbs
doi: 10.11648/j.ejcbs.20170306.15
ISSN: 2575-4998 (Print); ISSN: 2575-5005 (Online)

Communication

The Separate Effects of Wall-Thickening and Epicardial Volume as Determinants of Left Ventricular Ejection Fraction

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To cite this article:
Jijie Zhou. The Separate Effects of Wall-Thickening and Epicardial Volume as Determinants of Left Ventricular Ejection Fraction. *European Journal of Clinical and Biomedical Sciences*. Vol. 3, No. 6, 2017, pp. 129-133. doi: 10.11648/j.ejcbs.20170306.15

Received: October 12, 2017; **Accepted**: November 17, 2017; **Published**: December 13, 2017

Abstract: Ventricular torque magically pushes systolic ejection fraction, and such torsional moments originate in the Frenet frame of a curve of cardiac muscle. Since surgical treatments involve preoperative anatomy calculations of the cardiac muscle, the understanding of chamber geometric effects may guide left ventricular chamber-reduction surgery. We decomposed ejection fraction for an interpretation of geometric parameters in restoration and optimization of cardiac function. Three systolic geometric parameters--sphericity, contractility, ventricular torsion--were extracted for the ejection fraction from an epicardial view. The decomposing approach allowed us to estimate each factor's contribution to the total ejection fraction with the exception of the myocardium-to-chamber volume ratio, which represented the thickening effect during systole. All other effects took parts in 'shrinkage factor' (β), which corresponded the ejection fraction with an infinitesimal layer of muscle. Through comparing parameters for left ventricular ejection fraction, geometrical rearrangement of muscle bands for ventricular torsion is the most effective mechanical axis during partial left ventriculectomy, while ventricular shape factor only plays a less important role during systole in dilated cardiomyopathy, which often misleads the surgeon's assessment.

Keywords: Systole, Dilated Cardiomyopathy, Ventricular Torsion, Ejection Fraction, Stroke Volume

1. Introduction

Ventricular chamber enlargement and reduced ejection fraction characterize dilated cardiomyopathy. Despite remarkable growth in the understanding of myocardial function at the cellular, genetic, and molecular level, the effects of chamber shape and size and their influencing on the overall function of the left ventricle are not fully understood. If all other therapeutic approaches fail dilated cardiomyopathy, regardless of cause, at its late-stages manifests as terminal heart failure and may require left ventricular chamber-reduction surgery. The procedure, partial left ventriculectomy (PLV), aims at reduce left ventricular (LV) wall-tension (via laPlace's Law) by reducing the radius of the dilated LV chamber. Whether it improves global LV function remains controversial. [1-3] Although geometric effects have been noted to be important [4-7], there is no specific model or a model-based set of guidelines to guide surgeons in achieving the optimal shape and size through PLV. In this respect, surgeons use empirical approaches [8] in their approach to improve ventricular shape and size. Here we consider the role of individual geometric factors that determine ejection fraction in an effort to provide a quantitative method by which improvement of LV function via PLV can be understood.

2. Geometric Parameters

To investigate the role of each individual component, a strategic approach is to choose a model for each geometric parameter with an emphasis on its own effect while quenching the others. For example, the real contraction mode is somewhere between isotropic contraction and longitudinal

contraction. Isotropic contraction gives no shape effect and maximal contractility effect; longitudinal contraction exaggerates shape effect while minimizing contractility effect.

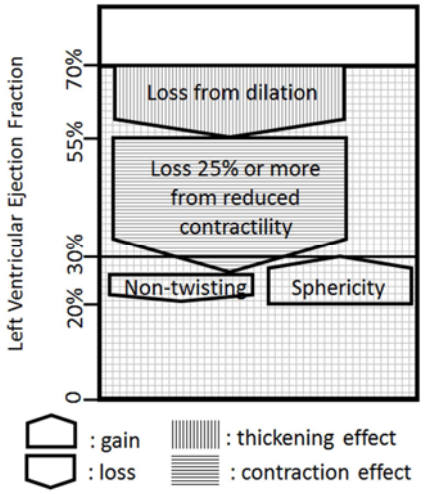

Figure 1. *Diagrammatic roadmap of decomposing ejection fraction for a dilated heart.*

Our analysis begins by switching from the traditional view of ejection fraction computed from volumes enclosed by the endocardial surface to volumes enclosed by the epicardial surface. The traditional numerator in the expression for ejection fraction (EF) is stroke volume. This numerator as in the following equation (where the superscript refers to the endocardial and epicardial surfaces respectively; *EDV* is the end-diastolic volume and *ESV* is the end-systolic volume.)

$$EF = \frac{EDV^{endo} - ESV^{endo}}{EDV^{endo}} = \frac{EDV^{epi} - ESV^{epi}}{EDV^{endo}} \quad (1)$$

is expressed in terms of epicardial volume change with the assumption that the myocardium is incompressible. Because individual myocytes and connective tissue are incompressible, it is known to good precision that left ventricular myocardial (tissue) volume (LVMV) remains constant throughout the cardiac cycle. There is a small change in volume between systole (emptying) and diastole (filling) due to coronary flow [9-10], but this small 5% volume change is can be neglected. The top of the chamber consists of the aortic outflow track and aortic valve with its annulus and the mitral annulus and mitral valve. For simplicity we approximate the plane of these valves by the straight line as shown.

Using the epicardial perspective, the ejection fraction can be re-expressed as the product of two dimensionless terms. They consist of: a purely epicardial "contraction" effect term; and a "wall- thickening" effect term. The latter incorporates the displacement of the endocardial surface due to muscle thickening:

$$EF = \frac{EDV^{epi} - ESV^{epi}}{EDV^{epi}} \cdot \frac{EDV^{endo} + LVMV}{EDV^{endo}} \equiv \beta \cdot \left(1 + \frac{y}{x}\right) \quad (2)$$

where $\frac{y}{x} = \frac{LVMV}{EDV^{endo}}$ is the left ventricular myocardium-to-chamber volume ratio. The term $(1+y/x)$ is the boundary displacement effect due to finite thickness of chamber wall. It represents the effect of wall thickening. The factor $\beta = \frac{EDV^{epi} - ESV^{epi}}{EDV^{epi}}$ represents contraction effect, which reflects volume change as measured via the epicardial surface resulting from ejection. Because, Stroke Volume $= \beta\, EDV^{epi}$, the shrinkage factor β (<1) can be thought of as the fraction of the epicardial end-diastolic volume that is converted to stroke volume by systole.

The geometric effect of wall-thickening, manifesting as radial (inward) displacement of the endocardial surface on the ejection fraction is contained in the myocardium-to-chamber volume ratio (y/x). Because the epicardial surface is displaced only slightly or not at all during systole, the ability to express LVEF in terms of y/x elucidates the role of myocardial mass in relation to EDV^{endo}. Furthermore, the myocardium-to-chamber volume ratio (y/x) accounts for the effect of wall-thickening during systole. We note that this effect is independent from other factors, such as sphericity, muscle contractility, and overall left ventricular torsion. All remaining factors other than wall-thickening are accounted for by the epicardial shrinkage factor β.

3. Discussion

3.1. Sphericity Effect

Myocardium circumferential wall stress is approximately 2.5 times larger than longitudinal wall stress. [11] Therefore, the heart has a tendency to dilate along its circumference rather than its meridian, and thus increase its sphericity. [12] Another consequence is that the heart contracts more along meridian than circumference when the ventricle is considered to be wrapped by a helical myocardial band. As explained before, longitudinal contraction is a typical model for the shape effect. Because the basal annulus does not contract substantially in dilated hearts, the corresponding annulus in our model is assumed non-contractile. For simplicity, the effect of sphericity is evaluated using a semi-ellipsoidal model and it is assumed that the ellipsoid contracts along its meridian. Sphericity has been defined in many ways, and some forms are considered independent of chamber size, such as (LV area)/(long axis)^2 in apical 4-chamber view.[13] Here we assume a fixed chamber size and myocardial volume, to exclude size effects, and express sphericity as (minor axis)/(semi major axis)=($2b/a$).

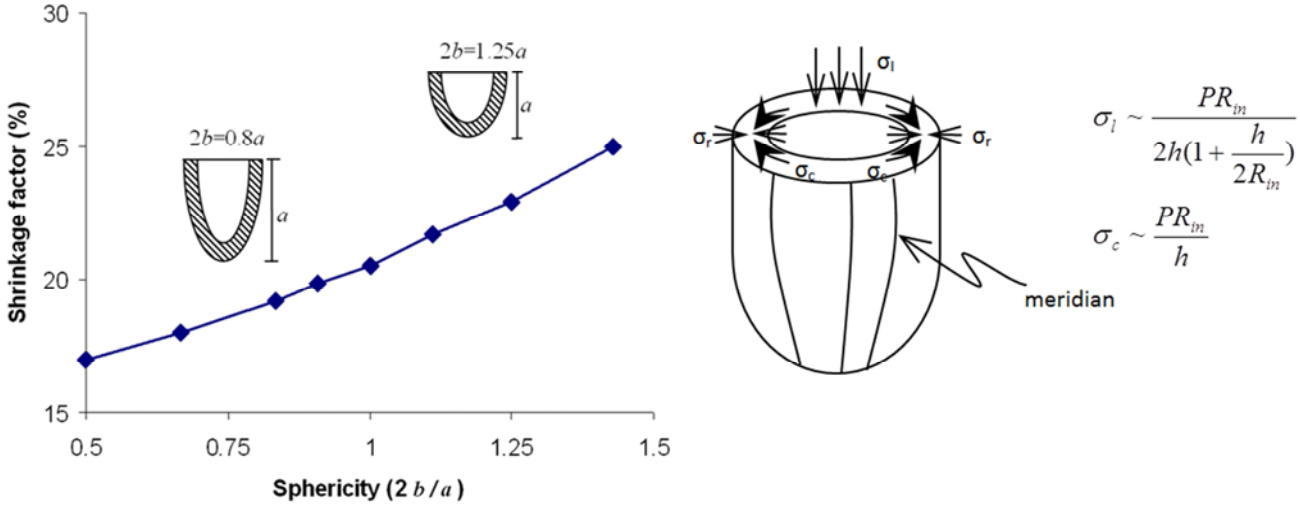

Figure 2. *Decomposition of wall stress and sphericity effect on the shrinkage factor β. The shrinkage factor increases as the shape of ventricle becomes more like a sphere than a baseball or ellipsoid, so does the ejection fraction, because the sphere is the most efficient shape to hold volume.*

In the semi-ellipsoidal model, epicardial major-to-minor axis ratio at end-diastole (a/b) is related with that at end-systole (a'/b) by contractility, where a' is the end-systolic major axis. For instance, 15% meridian contraction gives the relationship of a'/b to a/b by:

$$0.85\int_0^{\pi/2}\sqrt{a^2\cos^2\theta+b^2\sin^2\theta}\,d\theta=\int_0^{\pi/2}\sqrt{a'^2\cos^2\theta+b^2\sin^2\theta}\,d\theta \quad (3)$$

a'/b can be solved for any given ratio a/b by converting both sides into a complete elliptic integral of the second kind. Then, $\beta=(1-a'/a)$. For example, according to Figure 2 a more-elliptic heart with a/b=2.4 has EF=0.192(1+y/x) and a more-spherical heart with a/b=1.4 has EF=0.25(1+y/x). When the epicardial major-to-minor axis ratio (a/b) decreases, the sphericity (~2b/a) increases and the resulting ejection fraction *increases* with sphericity. This elevation of ejection fraction can reach up to 10% for dilated hearts as in the plot Figure 2. A straightforward explanation is that sphericity increases volume-to-surface ratio, so that the same surface contraction results in more volume change for the shape with higher volume-to-surface ratio. Note here that in obtaining this result, we have assumed that in increasing sphericity, the contractility of myofiber band remained intact.

3.2. Muscle Contractility

The individual fiber contractility is evaluated with the isotropic contractility assumption, i.e., assume that the epicardial shell shape from end-systole is geometrically similar to that of end-diastole. As a result, the shrinkage factor becomes $\beta=1-(1-k)^3$, where k is the shortening fraction on the epicardial interface. This is a conservative estimate since the isotropic contractility assumption overestimates the contractility effect. However, because the real heart contracts in all directions, the isotropic contractility assumption is more suitable in approximating the volume change than the circumferential or meridian contraction mode. This isotropic contractility assumption should induce no more than 10% error as shown in the Sphericity Effect section.

If a dilated heart with 100ml/m^2 chamber volume index contracts normally, say 12% on the epicardial interface, its ejection fraction will be higher than 54% and stroke volume would be even more than the normal heart (by similarity rules, and Figure 3). The ejection fraction of a sick dilated heart is less than 30%. Therefore, the contractility in dilated hearts must have been decreased in order to render the clinically observed ejection fraction values. This implies that in a dilated heart, the contractility is substantially impaired. For example, for a dilated heart with EF=30% the effective shortening fraction or contractility on the epicardial interface (Figure 4) can *not* be more than 6%. In this regard, the effective shortening fraction on the endocardial interface reaches 11.2% because of the thickening effect, which is consistent with clinical observation. [14-15]

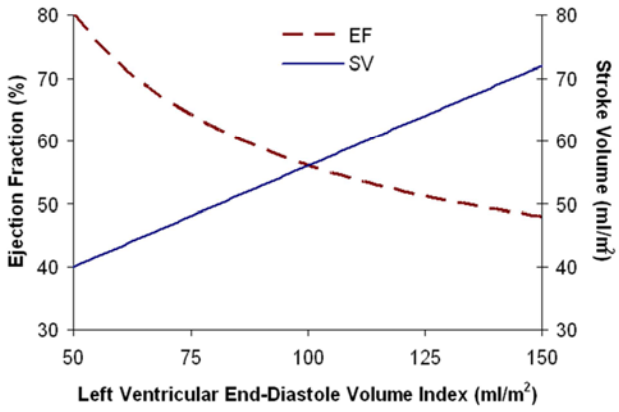

Figure 3. *Effects of left ventricle volume on ejection fraction and stroke volume. Where left ventricular myocardium volume index (LVMV) y is 76 ml/m^2 and effective epicardial contractility k is 12%. Stroke volume (solid line) increases with the ventricular volume, while the ejection fraction (dashed line) decreases with the ventricular volume.*

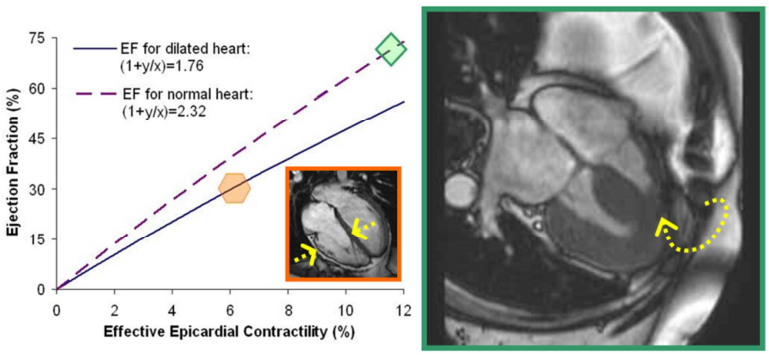

Figure 4. *Ejection fraction versus effective epicardial contractility for dilated and normal hearts. Normal hearts have 12% effective contractility and operate around the green diamond area; dilated hearts have only 6% effective contractility and operate around the orange hexagon area. This implies the dilated heart does not contract substantially.*

3.3. Overall left Ventricular Twisting

Previous studies show that the overall LV torsion, or the twisting motion during systole, plays a role in the ejection function and may even serve as a compensatory mechanism. [16-18] In contrast to some previous work [19-20], our model predicts a less important role for the twisting factor in delivering substantial values for ejection fraction during systolic phase. The following brief model places an upper bound of 5% on the twisting effect in ejection fraction; 5% is for normal heart mass ratio and 4% is for dilated heart mass ratio.

If a circular right cylinder of radius r and height h, wrapped by zero-thickness longitudinal muscle, is twisted while conserving the muscle length, the torsion alone results in the shrinkage factor

$$\beta = \left(1 - \frac{1}{3}(2+\cos\theta)\sqrt{1-4(r/h)^2\sin^2(\theta/2)}\right) \qquad (4)$$

where θ is the twist angle (i.e. two end cross-sections of the cylinder are twisted with respect to each other by θ degrees). Normal overall LV torsion is about 15°, but the heart may twist 25° to make up for the function loss from certain diseases. [18] If twist angle is less than 30°, the corresponding shrinkage factor β is less than 5% as illustrated in Figure.5. Therefore, from a systolic point of view, within the physiological range, the twisting motion contributes very little to ejection fraction. For example, a loss of 15° in torsion results in about 2% change in the shrinkage factor β.

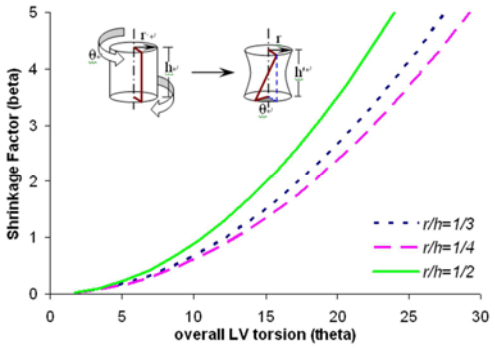

Figure 5. *Twisting effect on the shrinkage factor β is generally small and θ is normally around 15°.*

4. Conclusion

We have investigated the role of parameters that contribute to cardiac ejection fraction for a better interpretation of functional geometry. In this respect, we decomposed the systolic impact to four typical geometric cardiac parameters: (i) myocardium-to-chamber volume ratio (y/x), discussed in Part I; (ii) sphericity (2b/a); (iii) muscle contractility; (iv) overall left ventricular torsion. Our model, depicted as a roadmap diagram in Figure. 6, predicts that in a typical case of dilated cardiomyopathy, dilation of the ventricular chamber reduces the ejection fraction by 15%, which results in a reduction of ejection fraction from the normal value of 70% to 55%. Furthermore, a reduction in effective contractility results in another reduction of approximately 25%, which decreases the value of ejection fraction from 55% to 30% or lower in the roadmap. The loss of twisting motion in systole has a small effect of less than 5% which reduces ejection fraction in the roadmap to approximately 20%. A typical increase of 56% in sphericity increases the stroke volume by approximately 10% resulting in an elevation of the ejection fraction up to approximately 30% as exemplified in Figure. 1.

Myocyte cytoskeletal defects have been noticed to play an important role in the pathogenesis of dilated cardiomyopathy and heart failure. However, the prevailing concepts identify ventricular geometric changes as a primary contributing factor in the impairment of ejection fraction as well as other important factors such as loss of myocyte shortening capability, calcium cycling defect, and apoptosis. If muscle fibers contracted normally, our analysis shows that large and spherical hearts would have more stroke volume. In this regard, enlargement of the heart in dilated cardiomyopathy is responsible for partial compensation in the loss of stroke volume caused by the loss from diminished contractility. However, in an indirect path, an increase in the radius of the heart may induce large wall stresses, building up the residual stress, deranging muscle function and lowering muscle contractility. Geometric changes in muscle arrangement are not as important as the derangement of systolic muscle contractility present in dilated cardiomyopathy. For example, if we could preserve muscle contractility in an enlarged heart, the heart would be more efficient. It is important to note that

the geometric arrangement of muscle bands plays an important role during the diastolic phase. If diastolic function could be improved by other means, a spherical heart would have more advantage than other shapes. If the residual stress were not reduced during partial left ventriculectomy, the systolic function would worsen.

The major form of human heart failure is characterized by a progressive, uniform dysfunction of the entire myocardium. So the geometric parameters have to be coupled with residual stress and contractility in the consideration of a partial left ventriculectomy operation.

Acknowledgements

Thanks to Gerald David Buckberg, M. D. at UCLA Medical Center, for his discussion and safety elevation of open heart surgeries.

Disclosure Statement

The author has no conflict of interests to disclose.

Funding

This work is supported by Shanghai Leading Academic Discipline Project (Y0103)

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