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RESEARCH ARTICLE

LEFT VENTRICULAR SHAPE & SIZE BASED CONTRACTILITY INDEX (LVSCI), FOR EFFECTIVELY (I) DESIGNATING NORMAL WELL-CONTRACTING LEFT VENTRICLES, AND (II) DEPICTING CARDIOMYOPATHIC LEFT VENTRICLES AND RISK OF HEART FAILURE

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ABSTRACT

The heart's left ventricle (LV) has an intrinsically optimal ellipsoidal shape that efficiently contributes to its contractility. We start with this below figure, depicting the LV simulating geometrical model, as a prolate ellipsoid truncated 50% of the distance from equator to base. Based on this figure, we define:

LV shape factor: S = SA/LA (1)

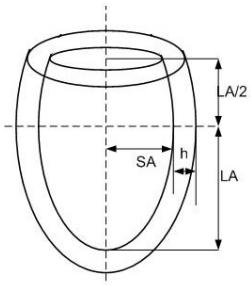

Figure. LV ellipsoidal model geometry: SA = AP/2 and LA = BA/1.5, where AP and BA are the major and minor axes of the LV ellipsoid.

The values of SA and LA can be determined from the echocardiographic monitored values of the LV volume (V) and myocardium volume (VM), given by:

$VM = 9\,\pi\,[2\,LA \times SA^2 + SA^2]h/8$ (2)

$V = 9\,\pi\,SA^2\,LA/8$ (3)

wherein V is LV volume, VM is myocardial volume, h is wall-thickness, LA and SA are endocardial major and minor radii. We then define $V^* = VM/V$. Based on the left ventricular ellipsoidal shell model, we can express the circumferential pressure-normalized LV wall stress σ^* (= σ /P), at the waist of the LV ellipsoidal model, as:

$$\sigma^* = \frac{2+S}{V^*}\left[1 - \frac{S^2(2+S)}{2(2+S)+V^*}\right] = f(V^*, S) \quad (4)$$

This equation provides σ^*as a function of shape factor S, for a given V*. Then the LV contractility index is given by dσ*/dtmaxwhich is a function of both *S* and V^* (= *VM /V*). We can thereby employ this LV contractility index which is also shown to be closely related to the conventional contractility index *dP/dtmax*. Now, based on the 3-d reconstructed surface images of normal LV and ischemic cardiomyopathic (ICM) LV, we have noted that a normal LV is more ellipsoidal in shape compared to the ICM LV. We have also noted that (i) the normal LV becomes more ellipsoidal from end-diastole (ED) to end-systole (ES)with greater reduction in shape factor (S), and (ii) also smaller in size (i.e. greater decrease V and hence greater increase in V*, compared to the ICM LV. In other words, the shape & size factor (S/V^*) decreases far more from the start of ejection (se) to the end of ejection (ee) in the normal LV compared to the ICM LV. So, then based on all these findings, we can now define a simplistic and yet very effective LV Shape &Size Factor-based nondimensional Contractility Index (LVSCI):

$LVSCI = [(S/V^*)se - (S/V^*)ee]/(S/V^*)se \times 100\,\%$ (5)

where se denotes start of ejection and ee denotes end of ejection. Based on thclinical studies data, it is shown that (i) for Group 1 with normal contractility, LVSCI = 70 and the traditional contractility index *dP/dtmax*= 1406 mmHg/s, whereas (ii) for Group 3, with poor contractility LVSCI = 47 and *dP/dtmax*= 948 mmHg/s. This represents a big testimony of the validity and novelty of our LV shape & size based nondimensional contractility Index LVSCI. which can be totally based on LV echocardiographic imaging of LV volume and its myocardial volume only.

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I. Introduction

How the Left Ventricle's intrinsic Anatomical shape is decisive for its Contractility performance

Contractility is the key mechanism of the left ventricular pumping role, and this is primarily due to its ellipsoidal shape. So, let us look into how this happens to be. In Figure 1, we are depicting the heart anatomy with its four chambers, right atrium, right ventricle, left atrium and left ventricle. We can notice that the left ventricle (LV) looks ellipsoidal in shape.

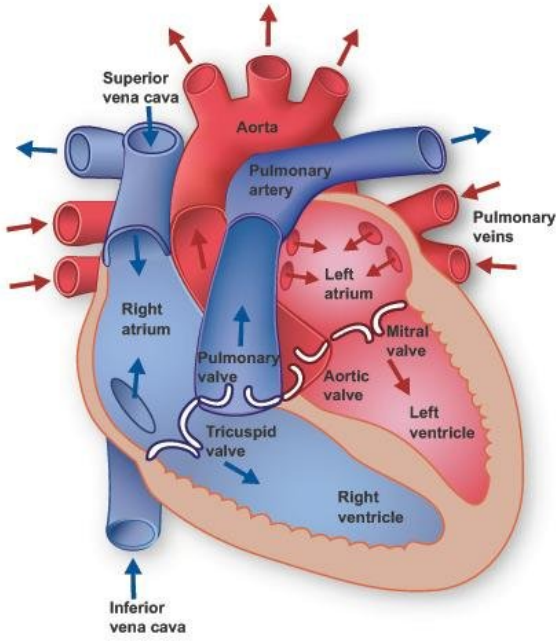

Figure 1. Picture of the heart, showing its four chambers, the valves, and the connecting blood vessels

The shape of the left ventricle (LV) is of clinical relevance for prognosis and diagnosis of heart patients. The left ventricle's myocardial wall has helically wound fibers. So, when these fibers are activated and contract, it causes twisting of the left ventricle from bottom upwards. This in turn generates LV wall stress and raises the intra-LV pressure to finally result in LV output into the aorta. Now this would not be possible if the left ventricle was cylindrically shaped. We have shown that a normal LV is more ellipsoidal in shape, and that a more spherically shaped and less-ellipsoidal shaped LV is associated with a failing heart [Ref. 1]. Now in this paper, we are providing big evidence of how a normal LV is more ellipsoidal shaped with a bigger contractility index than a pathological LV which is less ellipsoidal (and more spherical shaped) and less contractile. We then go on to (i) develop a novel shape &size-based contractility index LVSCI, (ii) compute its values for normal LV and pathological LV, and (iii) clearly show how LVSCI value is significantly different for normal and pathological LVs.

II. Left Ventricular Model Geometry and its Shape Factor

In Figure 2, we are depicting the LV simulating geometrical model, as a prolate ellipsoid, truncated at 50% of the distance from equator to base. The LV shape, as in figure 2, can be defined by the major and minor radii of its two surfaces: the endocardium of the LV and the septum, and a surface defined by the epicardium of the free wall. The overall longitudinal distance from the base to apex BA (=3LA/2) is thus 1.5 times the major radius of the ellipse. The simulated LV's geometrical parameters are SA = AP/2, and LA = BA/1.5, where BA (the LV long axis) is defined as the longest distance from the apex to the base of the LV, as measured on the four-chamber MRI view of the heart; AP is defined as the widest LV minor axis. Based on figure 2, let us define the

LV shape factor: S = SA/LA (1)

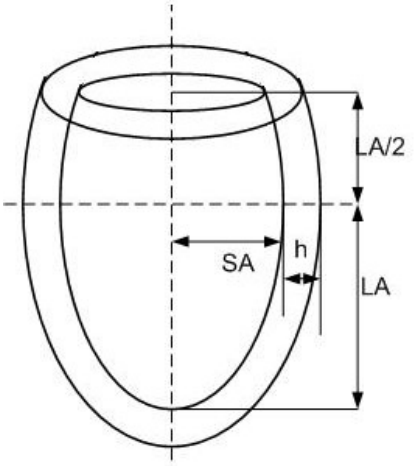

Figure 2. LV model geometry, showing the major and minor radii of the inner surface of the LV (LA & SA) and the wall-thickness (h): SA = AP/2, and LA = BA/1.5, where BA (the LV long axis) is defined as the longest distance from the apex to the base of the LV. Based on this figure, we can define the LV shape factor S = SA/LA. This figure is adopted from our journal paper [Ref. 1].

The values of SA and LA can be determined from the echocardiographic monitored values of the LV volume (V) and myocardium volume (V_M), given by:

$$VM = 9\,\pi\,[2\ LA \times SA^2 + SA^2]h/8 \quad (2)$$

$$V = 9\,\pi\,SA^2\,LA/8 \quad (3)$$

wherein V is LV volume, $V_M$is myocardial volume, h is wall-thickness, LA and SA are endocardial major and minor radii. We can then define $V^* = V_M/V$.

Now, LV volume (V), wall-thickness (h), and myocardial volume (V_M) can also be measured by MRI, but better still by 2-d or 3-d echocardiography. Hence, from Equations (2) and (3), we can determine SA and LA, and then the LV shape factor S = SA/LA in Eq (1).

III. Developing normalized LV wall stress σ* (= σ/P)

Based on an ellipsoidal shell model, the circumferential wall stress σθ (referred to as σ) at the waist of the LV ellipsoidal model, is given by Mirsky [Ref.2], as:

$$\sigma = P\frac{SA}{h}\left[1-\frac{SA(SA/LA)^2}{(2SA+h)}\right] = P\frac{SA}{h}\left[1-\frac{(SA/h)(SA/LA)^2}{2(SA/h)+1}\right] \quad (4)$$

From equations (2 & 3), we have, by putting S = SA/LA,

$$\frac{SA}{h} = \frac{V}{V_M}(2+\frac{SA}{LA}) = \frac{V}{V_M}(2+S) = \frac{2+S}{V^*} \quad (5)$$

where S = SA/LA constitutes the LV shape factor, and $V^* = V_M/V$. Combining equations (4) and (5), we can express the normalized LV wall stress σ^* (= σ/P), as:

$$\sigma^* = \frac{2+S}{V^*}\left[1-\frac{S^2(2+S)}{2(2+S)+V^*}\right] = f(V^*,S) \quad (6)$$

Equation (6) provides σ^* as a function of shape factor S, for a given V*. This is our non-invasively obtainable, pressure-normalized LV wall stress. The development of LV myocardial wall stress, by

activation of the LV myocardial fibers, enables the LV to contract during the isovolumic contraction phase, and to facilitate ejection of blood from the LV into the aorta.

IV. Left Ventricular Contractility Index, based on normalized LV wall stress

Our concept of a LV shape-based contractility index is that it is a measure of the capacity of the LV myocardial sarcomere to contract and generate wall-stress that will adequately raise intra-LV pressure to eject the blood. Now since the LV wall stress depends on its shape (as per equations 4 and 6), hence the LV contractile capacity also depends on the LV shape. This is the rationale behind the LV shape-based contractility index. Based on clinical observations, a healthy LV shape factor is more akin to the optimal-ellipsoidal shape factor, with a lesser value of S. However, it transforms into a more spherical shape with a greater value of S in a poorly contracting LV as well as in LV failure. Hence, our LV shape-based contractility index, expressed as $d\sigma^*/dt_{max}$ (based on equation 6) is meant to quantitatively express this clinical observation. Conventionally, the LV contractility is measured in terms of the LV pressure as dP/dt_{max}. Now in our paper [Ref.3], we had approximated the LV as a thick-wall spherical shell, consisting of incompressible, elastic material. We had employed the maximum circumferential wall stress (σ_θ) at the endocardium, to obtain:

$$\sigma^*(r) = \frac{\sigma_\theta}{P} = \frac{r_i^3}{r_e^3 - r_i^3}\left(1 + \frac{r_e^3}{2r^3}\right) \tag{7}$$

where r_i and r_e are the inner and outer radii, and P is LV intracavitary pressure [Ref.3]. Since the maximum wall stress occurs at the inner endocardial wall, we can obtain σ* at r = r_i, from equation (7) in terms of V and V_M as:

$$\sigma^*(r = r_i) = \left(\frac{V/(V_m + V) + 1/2}{1 - V/(V_m + V)}\right) = \left(\frac{3V + V_m}{2V_m}\right) = \left(\frac{3V}{2V_m} + \frac{1}{2}\right) \tag{8}$$

where P is LV intra-cavitary pressure; σ* is the wall stress; V (= $4\pi r_i^3/3$) denotes LV volume; V_m (= $4\pi(r_e^3 - r_i^3)/3$) denotes LV myocardial volume; r_i and $r_e$are the inner and outer radii of the LV, respectively. By differentiating equation (8) with respect to time, we get for the LV contractility index:

$$d\sigma^*/dt_{max} = \left|\frac{d(\sigma_\theta/P)}{dt}\right|_{max} = \frac{3}{2V_m}\left|\left(\frac{dV}{dt}\right)\right|_{max} \tag{9}$$

This contractility index is easily measured non-invasively, i.e. from echocardiography or magnetic resonance imaging, as explained in [Ref. 3]. We have also validated $d\sigma^*/dt_{max}$against the traditional contractility index dP/dt_{max} [Ref. 3]. Now in contrast to the normalized LV wall stress σ^*given by equation (9), our σ^*given by equation (6) incorporates the ellipsoidal shape of the LV. So we can now assume that the LV contractility $d\sigma^*/dt_{max}$ based on equation (6) can in fact provide as good or even better correlation with dP/dt_{max}, to hence serve as a more relevant non-invasive contractility index.

V. LV Contractility Index for the LV Ellipsoidal Model based on the Shape Factor

Now, as explained above, compared to the conventional invasively obtainable LV contractility index dP/dt_{max}, the LV contractility index can be expressed more intrinsically and non-invasively in terms of the maximum rate of generation of the LV normalized stress σ* (=σ/P), by using equation (6), to be (dσ*/dt) max, as given by this equation:

$$SFI = \left|\frac{d\sigma^*}{dt}\right|_{max} = \left|\frac{[\dot{V}(2+S)/V]+\dot{S}}{V^*} - \frac{S\left(\begin{matrix}S\dot{V}[16+8V^*+(24+8V^*)S+(12+2V^*)S^2+2S^3] \\ +\dot{S}[32+8V^*+(56+12V^*)S+(32+4V^*)S^2+6S^3]\end{matrix}\right)}{V^*(4+2S+V^*)^2}\right|_{max} = F(S,\dot{S},V,\dot{V},V^*) \tag{10}$$

Equation (10) indicates that corresponding to a patient's V(t) and V̇ (t) variations, the shape-factorbased contractility index SFI for that patient is a function of the shape factor (S) and V^*(= V_M /V) of a LV. Now we as well as cardiologists have been observing that an infarcted LV becomes less ellipsoidal as compared to a normally contracting LV shape, as demonstrated later in Figure 6. This resultant distorted shape of an impaired LV does not allow it to contract and deform in an optimal twisting mode, to efficiently perform its pumping function and deliver the requisite cardiac output efficiently. In accordance with this clinical observation, the shape factor index SFI (equation 10) incorporates the LV shape factor (S =SA/LA), and the influence of the distorted shape of an infarcted LV to its impaired pumping function.

The below Figure 3 demonstrates the validity of our dσ*/dt formulation, to determine its variation during the ejection phase of the cardiac cycle. It depicts how dσ*/dt can be computed for a subject, from the monitored pressure (P), volume (V), and computed LV dimensions (LA, SA, h, and S) during a cardiac cycle, by using the LV ellipsoidal model (as outlined above) . For this subject, the LV dimensions and the time-derivative of normalized stress (dσ*/dt) were calculated for every 20 ms during the cardiac cycle. Figure 3(g) also depicts the cyclic variation of the absolute value of (dσ*/dt) during the ejection phase. It is seen that the value of dσ*/dt becomes maximum about midway in the ejection phase.

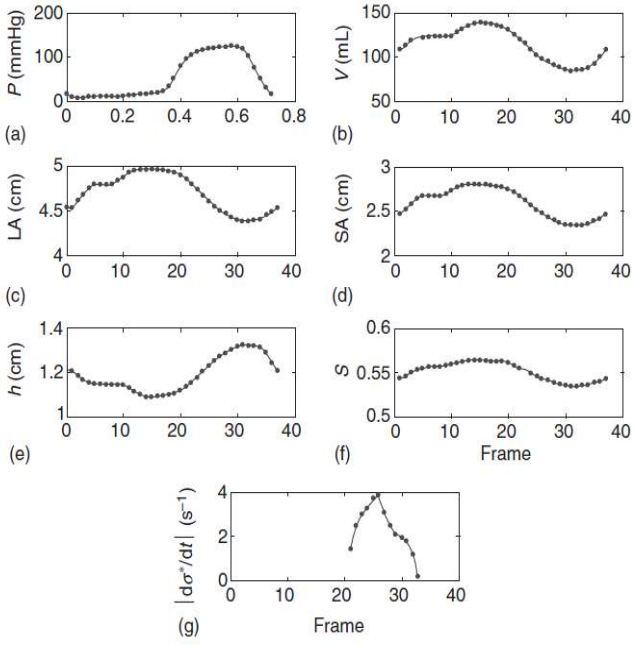

Figure 3. Depicting calculation of dσ*/dt. The figure shows measured pressure (P), volume (V), and dimensions (A, B, h, and s) during a cardiac cycle (by using LV ellipsoidal model), along with the absolute value of (dσ*/dt) calculated by using Equation (10) during the ejection phase for a subject HEL. (Adopted from Ref 1: Zhong, L., Ghista, D.N., Eddie, Y.K.Ng., Lim, S.T., Chua, T., and Lee, C.N., J. Biomechanics, 39, 2397, 2006)

VI. Evidence of a low value of shape factor (S) for a well contracting LV (SFI)

Now we will provide evidence of how a healthy LV should have as low a value of shape factor (S) as possible. The below Figure 4 illustrates the cyclic variations of h, S (= SA/LA), and σ*versus time during the ejection phase, for three patients. Now based on Equation (10), for maximum LV contractility as given by the LV Shape Factor Index SFI, we want dσ*/dt to be maximum. In the below Figure 4, it is seen that Patient DDM has the maximum value of dσ*/dt. Correspondingly, this patient also has the minimum value of S and the maximum value of dS/dt. So, this clinical data analysis confirms that for maximum contractility, we want S to be minimum and hence the LV should be as ellipsoidal as possible.

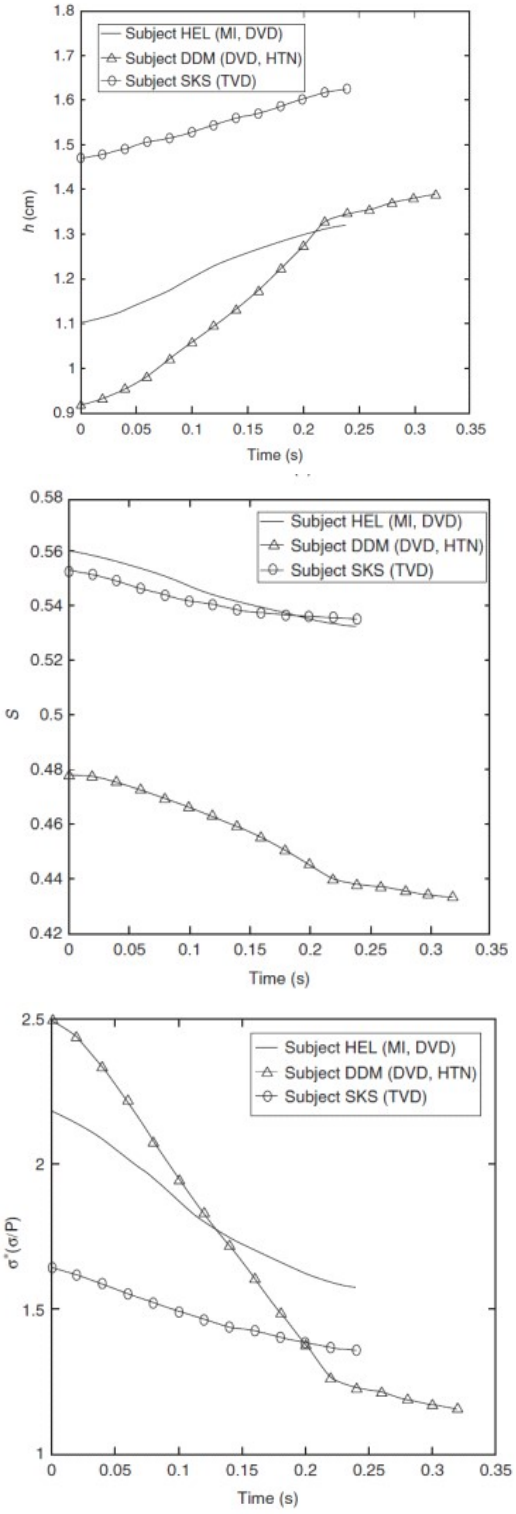

Figure 4. Variation of h, S, and σ* versus time during the ejection phase for (i) subject HEL with myocardial infarct (MI) and double vessel disease (DVD), (ii) subject DDM with DVD and hypertension (HTN), and (iii) subject SKS with triple vessel disease (TVD), during the ejection phase. Herein, t=0 represents the start-of-ejection. Subject SKS has the minimum generated σ*, while subject DDM has the maximum σ* during the ejection phase. (Adopted from Ref 1: Zhong, L., Ghista, D.N., Eddie, Y.K.Ng., Lim, S.T., Chua, T., and Lee, C.N., J. Biomech., 39, 2397, 2006)

VII. Optimal Left Ventricle Shape Factor

Let us designate the optimal shape factor S (= SA/LA) to be that value for which the generated myocardial wall stress (given by equation 6) for a given LV volume (at the start-of-ejection $V = V_{se} = V_{ed}$) is maximum for a specific value of V^*. This generated myocardial wall stress implies proper activation of the LV myocardial fibers during systole, to facilitate ejection of blood from LV into the aorta.

The concept of optimizing the shape factor is based on the formula of LV pressure $P = \sigma / \sigma^*$. During systole, the interaction of the act in–myosin filaments causes contraction of the myocardial fibers and generation of myocardial wall stress (σ).The resultant LV pressure generation is given by $P = \sigma / \sigma^*$, where σ^* is purely dependent on LV geometry and is a function of the shape factor (S) and volume ratio (V*), as given by equation (6). It is seen in Figure 5 that for a particular V^*, as S increases (i.e., as the LV becomes more spherical and less ellipsoidal) σ^* decreases, and hence the LV pressure increases. For an adequate amount of LV wall stress (σ) generated, we want the LV pressure to be maintained low, so that its oxygen demand is minimal. Hence, we want that for a specific V^*,the σ^* to be as high as possible and correspondingly S to be as low as possible, i.e., the LV must be more ellipsoidal in shape.

So, from equation (6), we can maximize with respect to S, as:

$$\frac{d\sigma^*}{dS} = \frac{1}{V^*} - \frac{1}{V^*}\frac{\left[(8S+12S^2+4S^3)(V^*+4+2S)-(8S^2+8S^3+2S^4)\right]}{(V^*+4+2S)^2} = 0 \quad (11)$$

Simplifying equation (11), we have:

$$6S^4+(4V^*+32)S^3+(12V^*+52)S^2+4(V^*+4)S-(V^*+4)^2=0 \quad (12)$$

from which we obtain the expression of the Optimal shape factor Sop as a function of V^*, as

$$S^{op} = 0.053V^* + 0.39 \quad (13)$$

for which σ* is maximum.

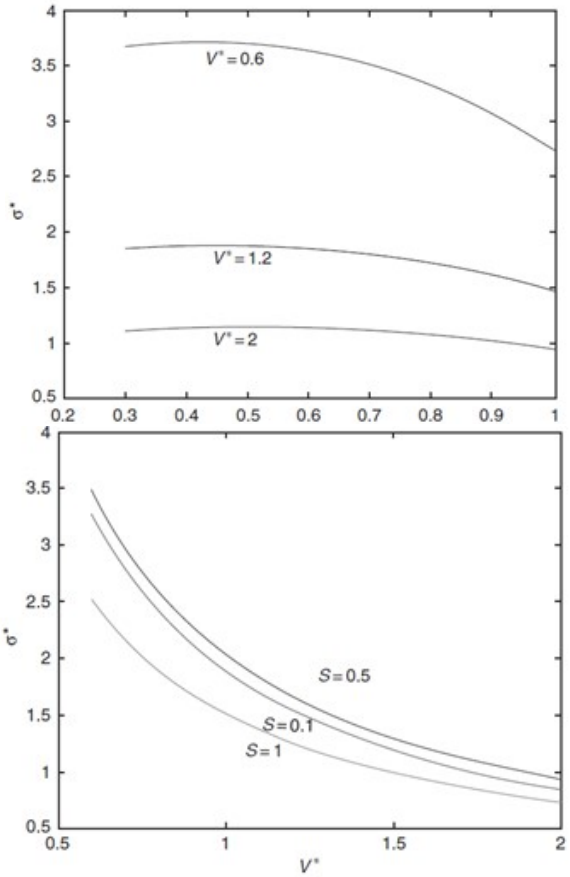

Figure 5. Variation of σ* with (a) S for different values of V*(= V_M /V) and (b) V* for different values of S. (Adopted from Ref 1: Zhong, L., Ghista, D.N., Eddie, Y.K.Ng., Lim, S.T., Chua, T., and Lee, C.N., J. Biomechanics, 39, 2397, 2006.)

This equation (13) can be referred to as the Optimal Shape Factor Index for maximizing LV contractility σ*. If we substitute expression (13) into $d^2\sigma^*/dS^2$, we get $d^2\sigma^*/ds^2$,to be negative. In other words, this optimal S function (of V*) maximizes σ*, in accordance with our

rationale. Equation (13) is represented in Figure 6 by the Optimal S vs V* line. The value of S > Sop is associated with a poor contractile heart; i.e., in this Figure 6, all calculated values of S and V* located far above this line are associated with a poor contractile LV. So, the significance of expression (13) is that one can adjudge the cardiac health state of a patient in terms of how close the shape-factor S (= SA/LA) corresponding to a patient's V* value (at the start of ejection) is to the optimal value. We do not even need to compute σ^* or $d\sigma^*/dt$ to evaluate how efficiently a particular LV is pumping. We will now proceed further and develop a proper formula involving S and V* for a well contracting LV.

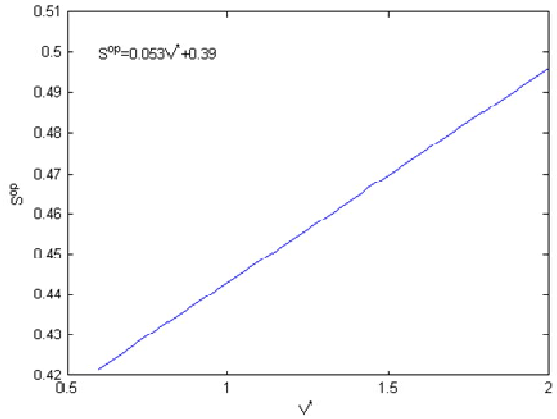

Figure 6. Optimal shape factor S versus V* at the start of ejection, represented by $S^{op} = 0.053V^* + 0.39$. This figure is adopted from Ref 1: Zhong, L., Ghista, D.N., Eddie, Y.K. Ng., Lim, S.T., Chua, T., and Lee, C.N., J. Biomechanics, 39, 2397, 2006.)

VIII. Clinical Studies showing association of LV shape with contractility

Now to further validate our association of LV ellipsoidal shape with its better contractility, we have performed clinical studies, which are described in detail in our paper [Ref. 1]. Conventionally, as we have indicated above, the LV contractility is measured as dP/dt_{max}. In this study, ten subjects, with EF = 0.63± 0.05 and dP/dt_{max}=1406±51 mmHg/s were selected to comprise Group1. They did not use nicotine, caffeine, or alcohol. The age profiles were similar and their anthropometric data, blood pressure, heart rate and ejection fraction (EF) were within the expected range. Ten other patients (with coronary and/or valvular disease), with EF=0.49± 0.13 and dP/dt_{max}=1183± 62 mmHg/s were classified into Group 2, having mean-age of 57.4 years. Finally, we have Group 3 of hospitalized patients, having EF=0.38± 0.12 and dP/dt_{max}= 948± 78 mmHg/s, with poor (clinically assessed) contractility. These subjects are listed in Table 1, showing the average values of monitored data for each group.

Table 1. Clinically monitored Data and Computed Parameters for Three Groups: Group1 (normal contractility), Group 2 (inadequate contractility), and Group 3 (poor contractility).

	Group 1	Group 2	Group 3
Age (years)	58.70 ± 6.65	57.40 ± 5.85	58.20 ± 9.11
dP/dt*$_{max}$*(mmHg/s)	1406.00 ± 51.00	1183.00 ± 62.00*	948.00 ± 78.00*
HR (beats/min)	72.69 ± 9.20	67.70 ± 10.04	74.02 ± 10.09
V_M (ml)	146.00 ± 43.00	189.00 ± 78.00	216.00 ± 80.00*
V(se) (ml)	119.26 ± 31.75	148.70 ± 68.32	177.41± 90.00
V(ee) (ml)	43.64 ± 9.87	79.45 ± 53.75*	116.73 ± 54.01*
S(se)	0.5	0.51	0.57
S(ee)	0.4	0.41	0.45
EF	0.63 ± 0.05	0.49 ± 0.13*	0.38 ± 0.12.00*

*p < .05 compared with normal contractility group

In this Table, S(se) and S(ee) denote the average values of the shape factor at start of ejection and end of ejection, for each group.

Now in the below Figure 7, we take the average values of V(se) and S(se) for each group, and then show how the corresponding LV shape looks like, for these three groups. It is noted from Table 1, that Group 1 LV (on the left of figure 7) has normal contractility, with lowest value of S(se), and high value of EF. Group 2 LV (in the middle) has poor contractility, higher value of S(se), and lower value of EF. Group 3 LV (on the right) represents a failing heart, with the lowest value of contractility, highest value of S(se), and lowest value of EF. Now we can also see in figure 7, that the left Group 1 LV has a distinctive ellipsoidal shape, while the middle Group 2 is less ellipsoidal, and the right Group 3 LV is more spherically shaped. Thus, it is clearly demonstrated that a more-spherical shaped LV with higher values of S(se) is associated with poor systolic function (i.e., low value of EF), and decreased contractility of the LV.

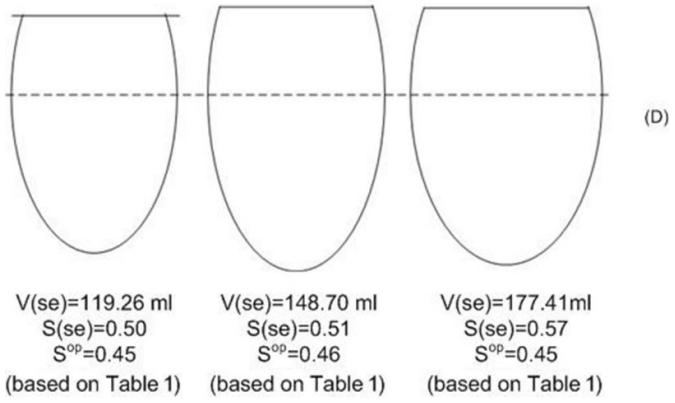

Figure 7. This figure provides the mean values of V(SE), S(SE), and S^{op}. The S^{op} is calculated by using equation (13). This figure clearly demonstrates that (i) a normal LV is more ellipsoidal shaped with a low value of shape factor S(se), and having high contractility value with high ejection fraction (based on Table 1); (ii) a more spherically shaped pathological LV has higher value of S(se), and is associated with poor systolic function (due to low value of ejection fraction) and decreased contractility value. This figure is adopted from our paper [Ref. 1].

IX. Development of a new LV Shape & Size based Contractility Index (LVSCI), and how effectively it can distinguish cardiomyopathic LVs with poor contractility

Now since the LV shape-factor based contractility index SFI (in equation 10) is a function of both S and V* (= V_M/V),let us look into how the shape & size factor (S/ V*) decreases from the start of ejection (se) to the end of ejection (ee) in normal and pathological LVs. Let us look at the above normal Group 1, in Table 1. At SE (or ED), Sse= 0.5; V* = Vm / Vse = 146/119 = 1.23. Hence, (S/ V*)se = 0.41. At EE (or ES), See = 0.4; V* = Vm / Vse = 146/43 = 3.4. Hence, (S/ V*)ee = 0.12. So (S/ V*) decreases from 0.41 at start of ejection to 0.12 at end of ejection. Now let us look at the above poor contractility Group 3, in Table 1. At SE (or ED), Sse = 0.57; V* = Vm/ Vse = 216/177 = 1.22. Hence, (S/ V*)se = 0.46. At EE (or ES), See = 0.45; V* = Vm/ Vse = 216/116 = 1.86. Hence, (S/ V*)ee = 0.24. So (S/ V*) decreases from 0.46 at start of ejection to 0.24 at end of ejection, which is much less decrease compared to the Group 1.

Based on this evaluation, let us now define our LV Shape & Size based Contractility Index, as

LVSCI = [(S/ V*)se - (S/ V*)ee]/ (S/ V*)se x 100 % (14)

This LVSCI Contractility index includes both the shape and size of the LV. Now based on the above computations and Table 1, we can compute that:

For Group 1, LVSCI = 0.29/0.41 = 0.70 or 70 %, traditional Contractility index dP/dt_{max}= 1406 mmHg/s, and EF = 0.63.

For Group 3, LVSCI = 0.22/0.46 = 0.47 or 47%, traditional Contractility index dP/dt_{max} = 948 mmHg/s, and EF = 0.38 (15)

So, this shows a direct correlation of LVSCI with the traditional Contractility index and Ejection Fraction (EF), namely greater the value of LVSCI, greater is the amount of cardiac contractility and higher is the EF. Also based on figure 4, for a healthier subject DDM's LV, (i) σ^* goes down from 2.6 to 1.2 from ED to ES in 0.3 secs, ie., $d\sigma^*/dt$ = 4.7 sec^{-1}, while (ii) S goes down from 0.48 to 0.43 in 0.3 secs, i.e., dS/dt = 0.16 sec^{-1}. On the other hand, for a more diseased subject SKS's LV, (i) σ^* goes down from 1.65 to 1.4 from ED to ES in 0.25 secs, ie., $d\sigma^*/dt$ = 1.0 sec^{-1}, while (ii) S goes down from 0.55 to 0.54 in 0.25 secs, i.e., dS/dt = 0.04 sec^{-1}. So we can state that a more normal LV has (i) a higher value of contractility index $d\sigma^*/dt$, (ii) lower value of shape factor S, and (ii) a higher value of dS/dt during the ejection phase than a more pathological LV.

X. Further Clinical evidence of correlation of Shape Factor S with Contractility

Now let us look at the below 3-d reconstructed surface images of normal LV and ICM LV in Figure 8.

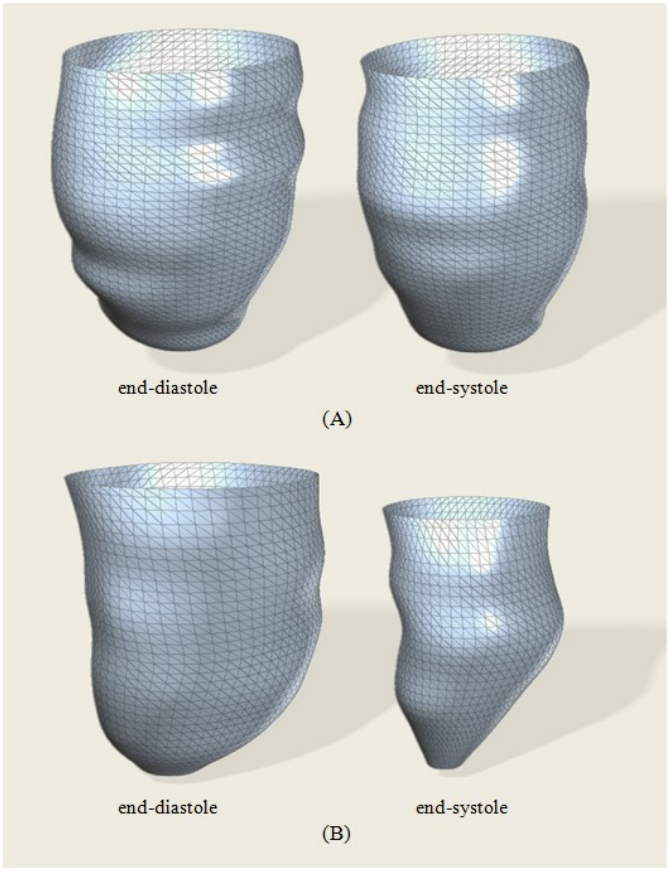

Figure 8. Three-dimensional reconstruction of LV endocardial surface at end-diastole and end-systole for (A) ICM (ischemic cardiomyopathic) LV and (B) normal LV. This figure shows how the normal LV can more effectively contract from ED to ES by becoming more ellipsoidal shapes and immensely decreasing its Shape Factor (S), compared to the ICM LV. Adopted from our paper Ref. 4.

It is seen that the normal LV (Fig. B) is more ellipsoidal than the ischemic cardiomyopathic (ICM) LV (Fig. A), and hence has a lower value of the shape factor S at end-diastole (or start of ejection). We can also note that the normal LV becomes more ellipsoidal from end-diastole (ED) to end-systole (ES), compared to the ICM LV. Hence the change (S_{SE} - S_{EE}) is greater for the normal LV, compared to that of the ICM LV, and the normal LV also shows a higher contractility. Now, based on actual calculation, in the normal LV, S decreases from 0.313 at start of ejection (SE) to 0.24 at end of ejection (EE). So, for the normal LV the normalized $\Delta S = [S_{SE} - S_{ES}]/S_{SE}$ = 0.223 or 22.3 %.On the other hand, in the ICM LV, S decreases from 0.36 to 0.313. Hence, for the ICM LV the normalized $\Delta S = [S_{SE} - S_{ES}]/S_{SE}$ = 0.131 or 13.1 %. In other words, for the normal LV, S decreases much more from SE to EE than for the ICM LV. The value of normalized $\Delta S$is higher for the normal LV than for the ICM LV. At the same time, we can also observe that for the normal LV, the volume V decreases a lot more from SE to EE, than for the ICM LV. Hence V* ($=V_M$ /V) increases much more from SE to EE for the normal LV, than for the ICM LV. So, for the normal LV, while S becomes much less from SE to EE, while V* becomes much greater from SE to EE. Hence, as calculated above, the factor (S/ V*) decreases much more in the normal LV than in the ICM LV, i.e., based on equation (14), the LVSCI contractility index value is greater for the normal LV than for the ICM LV. Hence, these actual images of normal and ICM LVs confirm the validity of our Shape & Size based Contractility Index

$$LVSCI = [(S/V^*)se - (S/V^*)ee]/(S/V^*)se \times 100\ \%$$
or
$$LVSCI = [(S/V^*)ed - (S/V^*)es]/(S/V^*)se \times 100\ \% \quad (16)$$

XI. Conclusion

We have developed a novel LV Shape & Size based Contractility Index,

$$LVSCI = [(S/V^*)se - (S/V^*)ee]/(S/V^*)se \times 100\ \%$$
as given by equation (14)

We have then shown by equation (15) that our LV shape &size based contractility Index LVSCI as well as the contractility measure dP/dt_{max} and Ejection fraction (EF) are much greater for the normal contractile LV Group 1 than for the poor LV contractile Group 3, based on Table 1.

This is also validated by the reconstructed LV endocardial clinical images of normal and ICM LVs in figure (8). Therein, we have shown that for the normal LV, (i) the shape factor S becomes much less from SE to EE than for the ICM LV, and (ii) V* (= V_M /V) increases much more from SE to EE for the normal LV, than for the ICM LV. So, for the normal LV, while S becomes much less from SE to EE, V* becomes much greater from SE to EE. Hence, as calculated above, the factor (S/ V*) decreases much more from SE to EE in the normal LV than in the ICM LV. Thus, based on equation (14), the LVSCI is greater for the normal LV than for the ICM LV. This represents a big testimony of the validity and novelty of our LV shape &size based contractility Index LVSCI. Now what we need to do is to determine the range of values for LVSCI for normal and pathological cardiomyopathic LVs. This LVSCI can enable more precision LV contractility measure, based on determination of LV shape factor S (= SA/LA) and size factor V* (= V_M /V) from just noninvasive echocardiographic monitoring of the LV volume and myocardial volume. Hence, by determining the ranges of LVSCI for different categories of patients, we can more precisely (and yet more simply) differentially diagnose patients with poorly contracting LVs, patients with cardiomyopathic LVs, and patents at risk of heart failure. **This will be the novelty of this LVSCI index**.

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