International Journal of Clinical Medicine Research
2014; 1(1): 5-10
Published online March 10, 2014 (http://www.aascit.org/journal/ijcmr)

Keywords
Myocardial Motion,
Myocardial Deformation,
Strain Imaging,
Myofiber Mechanisms of the Myocardium

Received: February 12, 2014
Revised: March 03, 2014
Accepted: March 04, 2014

An essential calculated power formula as a new index to study myocardial function of heart

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Citation
Mersedeh Karvandi, Saeed Ranjbar, Seyed Ahmad Hassantash. An Essential Calculated Power Formula as a New Index to Study Myocardial Function of Heart.*International Journal of Clinical Medicine Research.* Vol. 1, No. 1, 2014, pp. 5-10

Abstract
This paper serves to study an arbitrary piece of the myocardium while is mechanically contracted, from the end-diastole to the end-systole. This finding can be posed to gain a good understanding of the local contractility, in contrast with chemical and electrical reasons of the cardiac muscle contraction. This assessment would be followed by a new notion of the power of myocytes resulting in the sufficient energy to the cardiac motion and deformation. This power was measured through 50 normal cases for each muscle volume samples in the myocardium at peak systolic. Case data included velocity, displacement, strain and strain rate. For instance, passing 0.2s from end diastole for a case in a piece with unit volume of his/her base lateral, 2.72w power is needed to get 4.5cm/s, 0.83cm, 25% and 1.85 1/s, in velocity , displacement, strain and strain rate respectively. This option is also considered as a remodeling index of the ventricles that as an example of this include due to vavular stenosis or regurgitation, coronary artery disease with associated decreased contractility and fibrosis leading to ventricular dilatation, genetic abnormalities resulting in hypertrophy, and progressive development of local fibrosis, severe hypertension, conduction abnormalities, etc. in fact this remodeling is a response to a problem with either the muscle itself or the environment in which it has to work and is an attempt to keep on fulfilling the heart's task-circulating the blood. However, since this is an abnormal situation with inherent mechanical disadvantages, in the long term, this will lead to irreversible damage to the muscle which evolves into ventricular dysfunction and heart failure. The early detection and follow-up of change in cardiac function and myocardial properties is thus of major importance.

1. Introduction

Current clinical methods for evaluation of cardiac function by echocardiography are mainly qualitative or semiquantitative. One of the most commonly used is the qualitative assessment of regional myocardial motion and deformation. Several new techniques have been proposed to provide quantitative motion information, such as the analysis of endocardial border displacement and the recently introduced Doppler Tissue Imaging (DTI) technique. This new imaging modality allows for the measurement of velocities at any point in the ventricular wall during the cardiac cycle, providing information about subtle wall motion abnormalities which are difficult to

observe just by visual analysis [1-2]. Several studies have been carried out to determine normal values of myocardial velocities and to show its sensitivity to detect wall motion abnormalities in several pathologies [4-7].

However, tissue velocities represent both local contractile and elastic properties and whole heart translation movement and tethering effects. In order to separate local and global effects a new parameter called 'Strain Rate' has been proposed to measure the local contraction and deformation. Ultrasound strain rate has recently been introduced by Heimdal et al [8]. and preliminary studies have been carried out to evaluate its precision and clinical use [9-10]. Strain Rate Imaging can be obtained by calculating the local in-plane velocity gradients along the ultrasound beam from Doppler Tissue velocity data. However, Strain Rate calculations are very dependent on the image noise and artifacts, and different calculation algorithms may provide inconsistent results. It has been shown, both in the early animal lab work based on microcrystal measurements, and more recently using the non-invasive based methodologies, that analyzing myocardial velocities and deformation, especially when combined with the response to a dobutamine challenge, enables the assessment of myocardial dysfunction in a wide range of cardiovascular pathologies (among which: coronary artery diseases and stress echo [11-14], vavular diseases [15-16], hypertension [17-18], hypertrophic cardiomyopathy [19], cardiac resychoronization thrapy [20]). In fact regional myocardial velocities and deformation prove to be a powerful tool to understand and quantify myocardial (dys-) function. Several cardiac conditions are associated with very specific changes in motion and deformation, which can be quantified using echocardiographic techniques. Analyzing myocardial deformation has provided important insight in cardiac mechanics and in the understanding of changes induced by a range of cardiac pathologies. Although none of the current techniques for ultrasound deformation assessment is perfect, they still provide insight for assessment of the cardiac function in individual patients. So for a proper interpretation of velocity and deformation data in a clinical setting, it is required to understand the power of cardiac mechanics in normality and pathologies, combined with knowledge on how intrinsic power of cardiac influences motion and deformation.

2. Background

2.1. Strain

The strain-stress relationship is a common concept used to investigate the mechanical properties of materials and has recently been applied to the analysis of cardiac tissue properties. The concept of strain (ε) corresponds to the deformation of an object as a function of an applied force stress (s). It represents the precentage of change of the unstressed dimension after the application of stress. And hold for both expansion (positive strains) and compression (negative strains). Considering a one dimensional object. The possible stress deformations are lengthening and shortening. In this case the strain can be expressed as the difference between the original length (L_0) and the length (L) after deformation. Normalized by the original length (L_0). As shown in the following equation:

$$\varepsilon(t)=\frac{L-L_0}{L_0} \tag{1}$$

As shown, the strain is a dimensionless parametr and, by convention, positive correspond to lengthening and negative strains to shortening.

In some cases the length of the object is known during the deformation process, in that case we can define the instantaneous strain as:

$$\varepsilon(t)=\frac{L(t)-L(t_0)}{L(t_0)} \tag{2}$$

Where (L(t)) is the length at a given instant (t) and (L(t_0)) is the original length.

2.2. Strain Rate

The strain rate is measurement of the rate of deformation and corresponds to the velocity of the deformation process. Taking into account equation (1) and (2) the latter definition, the instantaneous strain rate $\dot{\varepsilon}(t)$ is expressed as the temporal strain derivative:

$$\dot{\boldsymbol{\varepsilon}}(\mathrm{t})=\frac{\boldsymbol{d\varepsilon(t)}}{\boldsymbol{dt}}=\frac{\boldsymbol{dL(t)}}{\boldsymbol{dt.L(t)}}=\frac{\boldsymbol{L'(t)}}{\boldsymbol{L(t)}} \tag{3}$$

With $L'(t)$ being the rate of deformation and L(t) the instantaneous length. The strain rate units are s^{-1} . Compared with the rate of deformation unites m.s^{-1}. As a conclusion, both strain rate and strain are closely related and they can be derived one from each other as we will discuss later.

Different approaches have been proposed to calculate the strain and strain rate parameters; some preliminary studies have been carried out to evaluate their precision and clinical usefulness [8-10]. Two main methods have been proposed: the Crosscorrelation method and the velocity gradient method. The Cross-correlation method is based on the principles of elastography where two consecutive radiofrequency signals are compared to extract information about the tissue elasticity properties [21-22]. Considering two consecutive radiofrequency signals applied to a tissue under deformation, the resultant backscatter signals received have similar patterns, except for a temporal shift related to the actual deformation. Cross-correlation analysis provides the temporal shift or delay introduced due to the object motion. From this analysis, different parameters such as the change in distance, local motion, velocities, etc., can be derived. The principal limitation of this technique is its

high computational cost and the need of very high temporal resolution to avoid noisy estimates. That limits its application to M-mode acquisition. On the other hand, Fleming et al [23].and Uematsu et al [24]. introduced the concept of myocardial velocity gradient as an indicator of local contraction and relaxation. This concept is directly related to the already formulated strain rate parameter (3) under the assumption of linear and uniform strain (homogeneous, isotropic and incompressible material) as can be shown in the following expression:

$$\dot{\varepsilon}(t)=\frac{d\varepsilon(t)}{dt}=\frac{dL(t)}{dt.L(t)}=\frac{L'(t)}{L(t)}\approx\frac{v_1(t)}{L(t)}-\frac{v_2(t)}{L(t)}. \quad (4)$$

where $v_1(t)$and $v_2(t)$are the local instantaneous velocities at two myocardial points separated an $L(t)$ distance. This formulation allows to compute myocardial strain rate as the spatial gradient of myocardial velocities, which can be obtained by Doppler echocardiography. Since the computational load is not high it can be implemented as a post-processing step after acquiring Doppler Tissue images or in real-time from digitally stored tissue velocity information [8]. The axial natural strain component can be calculated from the strain rate curve time-integration expressed by:

$$\varepsilon(t)=\int_{t_0}^{t}\dot{\varepsilon}\,(t)dt \quad (5)$$

3.Method

When we are using myocardial motion and deformation to assess left ventricular function, it is important to dedicate the relation between intrinsic power and the resulting motion and deformation. How much mechanical power is necessary to make these dynamical deformations for each cardiac muscle volume? This problem would give the best information of the cardiac function. In summary, the main factors influencing to the cardiac function mechanically are: 1) Regional myocardial motion and deformation. 2) Intrinsic power, i.e. the sufficient energy force developed by myocardium, resulting in motion and deformation.Having applied ViVid7 GE echocardiography system for 50 normal cases, we acquired velocity, displacement, strain and strain rate at peak systolic phase, for each part of the myocardium, i.e. at the base, middle and apical of septal, lateral, anterior and inferior.All data sets comprise intrinsic power values at peak systolic, for each local part of the myocardium. (Figures 1 and 2) show these value:

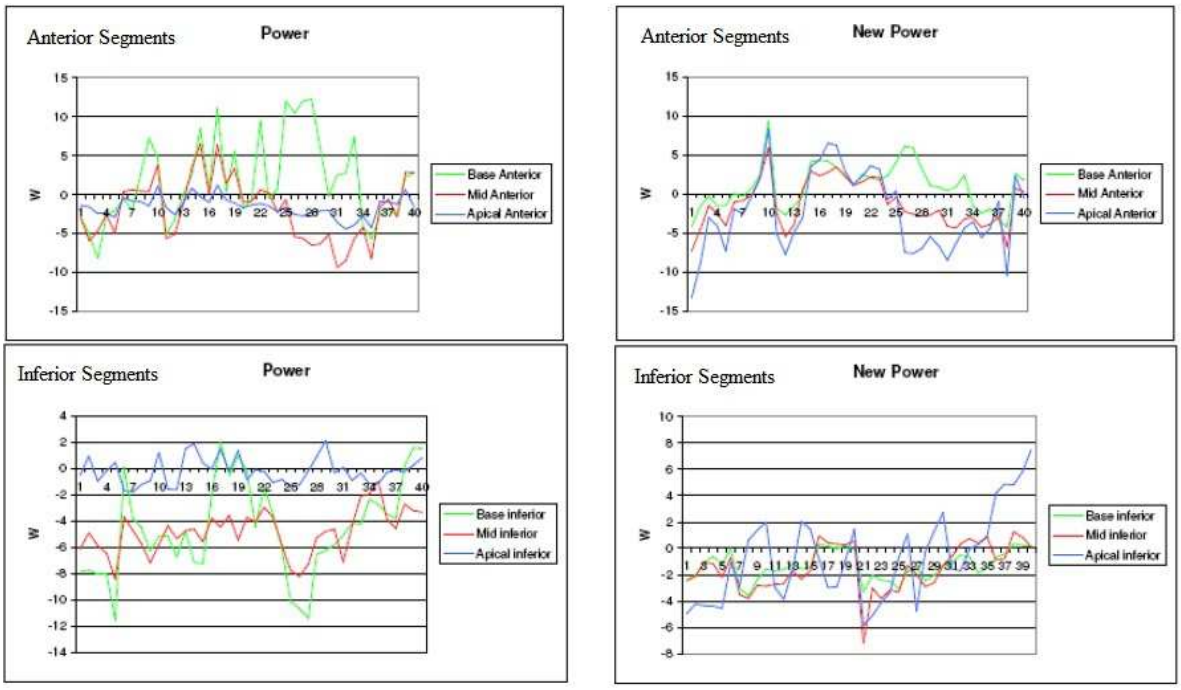

Figure 1.*The left side diagrams show "The Power" for each anterior and inferior echocardiographic segmentat peak systolic phase. This new variable states what power is desired to regard a deformation and a displacement that occurs in normal cases.The right side diagrams show intrinsic powers but without the heart translation consideration.*

3.1. Remark

The value of this new option at the base septal for a case is 6.5w. This means 6.5w is required to make a necessary motion and deformation of this part at the peak systolic phase. Removing the general motion of the heart, this value for the same case at the base septal is less than 6.5w. This states tethering helps a piece of myocardium to cause for the desirable motion and deformation.

4. Result

We pick a myocardial muscle volume sample, for instance from the basal septum and follow-up its motion and deformation within the end diastole to the end systole.

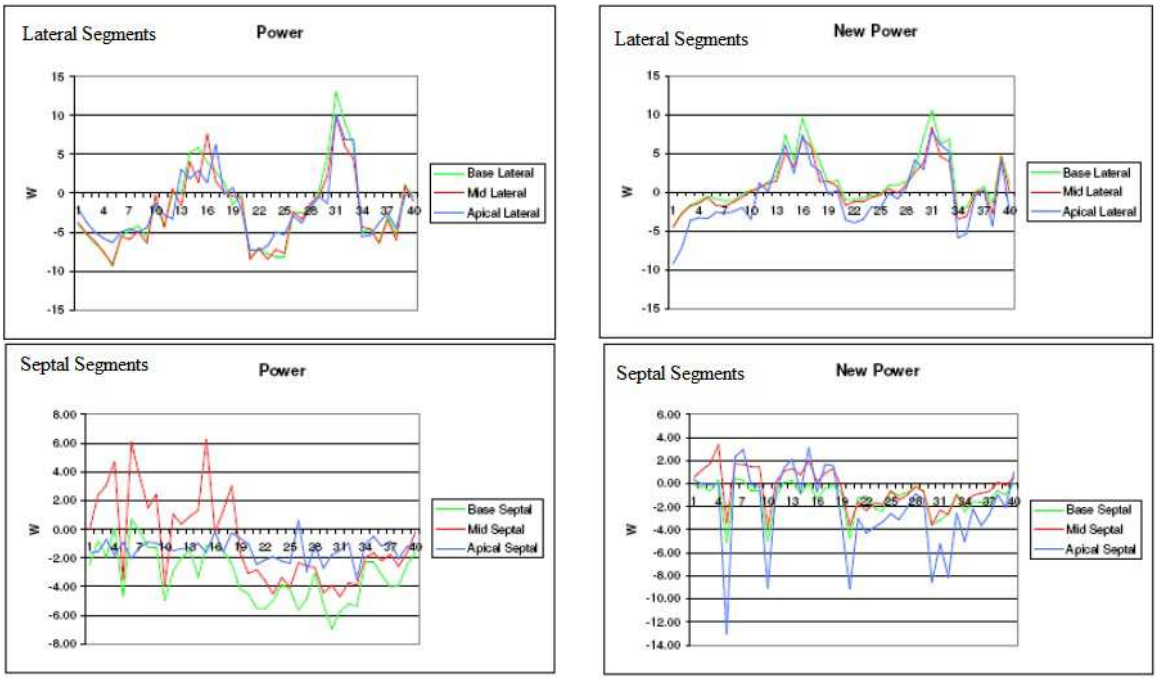

Figure 2.*The left side diagrams show "The Power" for each lateral and septal echocardiographic segment at peak systolic phase. This new variable states what power is desired to regard a deformation and a displacement that occurs in normal cases. The right sidediagrams show intrinsic powers but without the Heart translation consideration.*

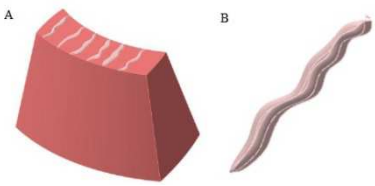

Figure 3.*A) A muscle volume sample at end-diastole B) a fiber which is named AB of the same muscle volume sample*

Over the time" t" after the end diastole, radial strain rate for the fiber AB (Figure 3B) is:

$$strainrate = V_B(t) - V_A(t)/L_{AB}(t)$$

Let D_t *and* W_t are displacement and velocity of the above muscle volume element at time t.

By classical mechanic we set [25]:

$$D_t + L_{AB}(t) - L(t_0) = \frac{1}{2}a_t t^2 + W_t \mathrm{t}$$

t_0 is the time and $L(t_0)$ is the length of the fiber AB respectively at the end diastole.

Having used 1D deformation definition, we rewrite the above formula by the following way:

$$\frac{D_t}{t} + \frac{L_{AB}(t)}{t} - \frac{L(t_0)}{t} = \frac{1}{2}a_t t + W_t$$

By radial strain rate and strain of the fiber AB at time "t" ,we have:

$$\frac{D_t}{t} + strainrate(t).(strain(t)+1)L(t_0) = \frac{1}{2}a_t t + W_t$$

Thus we can reformulate a_t in below:

$$a_t = 2\frac{D_t}{t^2} + 2strainrate(t).\frac{(strain(t)+1)L(t_0)}{t} - \frac{2W_t}{t} \quad (6)$$

Now if μ be density and volume(t) (8) of our muscle volume sample after the contraction during the time "t". Power which is needed to result in this motion and deformation at time "t" would be:

$$P(t) = \mu.\mathrm{Volume(t)}.\mathrm{a_t}.\mathrm{D_t}/_t \quad (7)$$

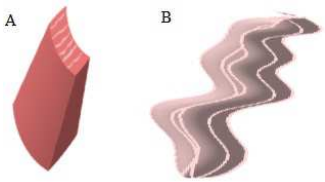

Figure 4.*A) The muscle volume sample at time "t". B) Wall thickeningof the same muscle volume sample.*

5. Discussion

Decreased or increased in power, for a muscle volume sample can be caused a maker of myocardial ischemia, LV dysfunction, or LV hypertrophy. As intrinsic power has simultaneously been contributed by some codependent factors such as motion and deformation, it is take in as a remodeling index to keep up normal conditions. For instance, essential wall thickening (at radial deformation) and lengthening/shortening (at longitudinal deformation) of a fibercan be made by its power. Having increased in wall

thickening of a piece of left ventricle, motivate us to inhibit widely dilate with a hypertrophic wall, by a reduction in power of that part. Particularly when power tends to zero for a muscle volume, it may be a factor that shows myofibers of that region, have inoperative. So it would be a criterion, we can make use of stern cell method to improve myocardial infarction zone?

On the other hand, this power shows its influence on the red blood cell flow by change in volume. In fact, when a unit volume of red blood cell (RBC) is interacting with a piece of ventricle, has a simple description of its change in volume, i.e. it can be calculated by:

ΔV(t)= Local change in volume of RBC with unit volume over time:

$$\Delta \mathbf{V}(\mathbf{t}) = \left(\mathbf{Radial\ deformation}(\mathbf{t})\right) \times \left(\mathbf{Longitudinal\ deformation}(\mathbf{t})\right) \times \mathbf{Displacement}(\mathbf{t}) \quad (8)$$

From this point of view, one can locally compute myocardial pressure which is a very valuable measure for many normal and pathological conditions of the heart, particularly right ventricular function and interrelationship of the right and left heart. This pressure is directly measured by the following way:

$$\text{Local myocardial pressure}\ (\mathrm{t}) = P(t).\ t / \Delta V(t) \quad (9)$$

If we follow myofiber transactions from the end diastole to the end systole, they would show us curves [26-27] that intrinsic power is made to move myofibers on these bands. One of applications of power is related to artificial heart. because having known the rout of myofiber transactions, the power is strongly needed to be applied on the wall motion of artificial heartto keep transactions on their curves. Since local myocardial pressure (9) is an index which presentsa direct relationship of the blood fluid and the myocardium, both can be used in a mechanical and dynamical modeling of the heart at the same time.

Considering that a myofiber in the myocardium has a recursive property [21-22], it means that it tries to hold itself to normal condition, after a remodeling on it. For a fiber this remodeling shows itself, in a lengthening or a shortening. This recursion is guarantied by the intrinsic power of myofiber. The other application of power at this manuscript is a special case that we can call it critical boundary. For a myocardial fiber, the “critical boundary” means maximum and minimum powers which are needed to result in a Max or Min for the deformation. Now if the necessary power of a fiber come down of Min or come up of Max then that fiber would basically beinoperative.

6. Conclusion

This novel formula may facilitate noninvasive quantification of ventricles in clinical and research settings.

Acknowledgement

The authors are really grateful to Drs. Mahnoosh Foroughi.

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