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Potentials and limitations of ventricular torsion as indicator of cardiac function

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Abstract—**New non-invasive imaging techniques allow quantification of torsion of the left ventricle (LV). Presently, it is not well know what torsion can add as an indicator of left ventricular pump function. A frame work for understanding of cardiac motion has been designed that is based on general principles of mechanics. In this frame work experimental and clinical findings on torsion were related to various indices of LV function. The time courses of torsion and volume have much information in common. However, the rate of torsion during isovolumic relaxation provides important information on rate of LV pressure decay. Most importantly, assessment of the relation between torsion and the changing inner diameter during ejection renders unique information about the transmural gradient of contractile performance of the LV myocardium.**

I. Introduction

Left ventricular (LV) pump function is quantified conventionally by non-invasive measurement of arterial pressure and geometry of LV cavity and wall, as assessed by 2D echocardiography. Innovations in cardiac imaging, such as magnetic resonance imaging (MRI) of tags and ultrasound echo speckle tracking, offer new possibilities to estimate cardiac motion and deformation in 3D. Torsion is a specific component of deformation, representing rotation of the apex relative to the base around the long axis of the LV. Its quantification is suggested to be an important indicator of LV pump function [1, 2].

Already around 1500 Leonardo da Vinci [3] described the anatomy of the heart, in which he recognized helical pathways of muscular fibers. He speculated that such a helical structure is likely to induce beat to beat rotational motion. By viewing the beating heart in an open chest, the existence of this type of motion has been confirmed.

Although the whole heart is subject to torsion, we will focus on torsion of the LV only, because the LV represents the majority of ventricular mass and is nearly rotationally symmetric. In the present article, a framework for understanding of torsion will be presented that is primarily based on general principles of mechanics. Correlations between torsion and various indices of cardiac function, as found in experimental and clinical studies, will be discussed.

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II. Quantification of torsion

Torsion or twist is defined as the base to apex gradient of rotation of the LV wall around the long axis of the LV (SI-unit: rad/m). Although LV torsion has been observed for a long time, its quantification had to wait for advances in technology. Using MRI-tagging or speckle tracking, rotation can be measured in two or more short-axis cross-sections. Following the clinical standard, short-axis cross-sections of the left ventricle are projected as if they were seen from the apex, indicated in Fig. 1 by the X- and Y-coordinates for horizontal and vertical direction, respectively. By convention for a right-handed coordinate system, the Z-coordinate points in the base to apex direction. During ejection, when viewing the heart from the apex, the apex rotates counter-clockwise relative to the base [4], indicating that torsion, being the Z-gradient of rotation, is positive during this phase.

Above defined torsion appears inversely proportional with the size of the heart. To avoid dependency on size, torsion is normalized by multiplication with the equatorial radius of the LV. In a perfectly circular equatorial wall segment (Fig. 1), thus defined normalized torsion is equivalent with the axial to circumferential shear component. Normalized torsion is physically dimensionless, and appears to be the same for species of very different size, e.g. for man, dog and mice [4, 5].

Normalization of torsion may also be obtained by multiplication with the base to apex length of the ventricle [6]. Thus normalized torsion represents the difference in rotation angle between apex and base around the central long axis. In open-chest experiments on dogs, assuming that the base is fixed by the large vessels, directly measured apical rotation was used as approximation of torsion [6]. When observing cardiac motion more carefully, the base rotates substantially during the cardiac cycle [5, 7]. The above-introduced interpretation that rotation represents twist or torsion has resulted in a wide-spread erroneous use of the term twist for rotation of a ventricular short axis cross-section.

III. Torsion– function relation

The LV pressure-volume or pressure-diameter relation is often used for assessment of LV pump function. The question arises to what extent torsion can improve this

assessment. Models based on general principles of mechanics appeared of great help to understand the role of torsion. Although the LV is known to be ellipsoidal, for the analysis of torsion near the equator its shape was simplified to a cylindrical ring [8, 9]. In the incompressible thick wall, circumferential shortening of the inner layers exceeds that of the outer layers substantially. In absence of torsion, the myofibers are subject to the transmural difference in shortening. In the outer layers the myofibers follow a left-handed helical pathway (Fig. 1). In the inner layers this pathway is right-handed. As a result, positive torsion causes myofibers in the outer layers to shorten at the cost of lengthening of the myofibers in the inner layers. By precisely tuning torsion to volume ejection, transmural differences in myofiber shortening and myofiber stress disappear.

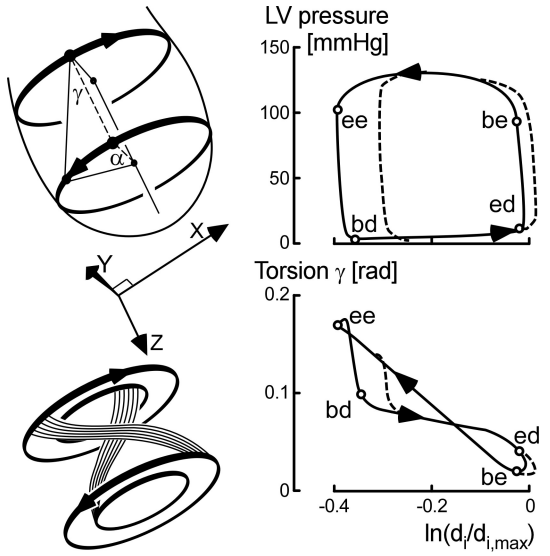

Fig. 1. Left panel: Torsion represents the base to apex gradient of rotation angle α. Torsion is normalized by multiplication with the equatorial radius, representing the axial to circumferential shear angle γ. The coordinate system has been indicated. The lower left panel shows that during systole torsion shortens myofibers in the outer layers and lengthens them in the inner layers. Right panel: LV pressure and torsion as a function of the logarithm of inner diameter during the cardiac cycle (derived from data by [10]). Letters bd, ed, be, and ee represent begin and end of diastole, and begin and end of ejection, respectively. The dashed curves represent diminished contractility.

In Fig. 1, LV pressure and torsion are plotted as a function of logarithmic LV cavity diameter, thus forming torsion-log diameter loops. The letters bd, ed, be and ee indicate begin and end of diastole, and begin and end of ejection, respectively. During ejection (Fig. 1, be→ee) the slope of the torsion-log diameter relation, representing the torsion to shortening ratio (TSR), appeared practically independent of volume load and ejection fraction, i.e. TSR=0.42. In normal dogs and normal humans, this ratio was found to be 0.43±0.04 [4] and 0.44±0.07 [11], as measured with 2D-echocardiography and MRI tagging, respectively. It was concluded that in normal subjects TSR was tuned so that myofiber shortening during ejection was uniformly distributed across the wall.

Torsion is directly related to the degree of anisotropy of the myocardium. If myocardium were isotropic, there could be no torsion. During diastole (Fig. 1, bd→ed), the change of torsion with a change in cavity volume is much less than in systole (be→ee) [6], showing that the myocardium is apparently more isotropic in diastole than in systole. Systolic and diastolic characteristics cross near the unstressed volume in diastole. During isovolumic contraction and relaxation torsion has to jump from the diastolic curve to the systolic curve (ed→be) and vice versa (ee→bd). During isovolumic relaxation, the jump represents recoil or untwisting that is fast and practically completed before mitral valve opening [12]. During isovolumic contraction, generally, the jump is less pronounced; it is slower and often extends to the ejection phase.

IV. Interpretation of Torsion

Torsion reflects the equilibrium of torque between the subendocardial and subepicardial myofibers, which have opposite handedness of their helical pathways (Fig. 1). Subepicardial myofibers force torsion to increase, whereas the subendocardial ones counteract torsion. The latter prediction is supported experimentally by the observation that TSR is increased in patients having severe aortic stenosis with coronary perfusion problems preferentially in the subendocardial layers [11]. In these patients, systolic contractile failure of the subendocardial layers causes an increase of torsion and a decrease of circumferential shortening, which results an extremely significant increase of TSR.

Thus, TSR provides unique information about transmural differences in contractile performance. In normal subjects, the relative standard deviation of torsion is about 0.28 [11], and that of TSR only 0.17. With aortic stenosis, the induced relative changes in torsion are smaller than those in TSR. Consequently, changes in systolic torsion are far less significant than those in TSR. The latter finding invalidates systolic torsion to be a reliable index of contractility, but show the strength of TSR. Another very important feature of TSR is that in normal subjects its value is a fixed dimensionless number, not dependent on species and ejection fraction. So, abnormality can be detected by a single measurement, not requiring comparison with values in the past.

With increasing age torsion increases [13, 14], whereas ejection fraction was not affected significantly. Thus, in elderly, TSR increases, which was interpreted to be caused by decrease of contractile function, preferentially in the subendocardial layers [14].

During isovolumic relaxation torsion shows clear recoil, often referred to as untwist. Untwist reflects the transition from the systolic to the diastolic working line (Fig. 1, ee→bd). Recoil is completed when diastolic forces exceed systolic forces, which is equivalent to entering the state of

diastole. The close relationship between decay time of LV pressure and rate of untwist has been confirmed experimentally by the finding of an inverse relationship between both quantities [1, 15]. Full diastole implies that LV wall stress and pressure are low enough for the start of LV filling. Indeed, opening of the mitral valve occurs after completion of untwist [12].

Untwist does not affect volume, but redistributes myofiber length. Subendocardial fibers shorten at the cost of stretching subepicardial fibers. Although it has been suggested widely that elastic energy of recoil is used for suction, the required balance of energy says differently. With recoil, elastic energy of the passive structures in the myocardium is released to bring back the shape of the heart to the diastolic state of equilibrium. Because the LV volume does not change during the isovolumic phase, no energy can be transferred to pump function. Instead, with decaying contractile force, there is a moment that passive forces stretch the still weakly active myofibers. So, most of the energy of elastic untwisting is lost as eccentric contractile work within the myocardium.

It should be noted however that many signs of heart failure tend to decrease speed and amplitude of untwist. Low contractility renders a low ejection fraction and dilatation. The resulting loop has been indicated in Fig. 1 as the dotted line. Because of the rightward shift of the torsion-diameter loop, the distance between systolic and diastolic working line (ee→bd) decreases, causing a decrease of the amplitude of untwist. Secondly, under these circumstances, the rate of relaxation decreases, which causes slowing down of the rate of untwist. Abovementioned changes are opposite with increase of contractility, for instance induced by dobutamine [1]. Then, end-systolic volume decreases, thus increasing the amplitude difference of torsion between systolic and diastolic working line. Moreover, both contraction and relaxation are generally faster, causing untwist to occur on a faster time scale.

Ejection fraction is highly correlated with torsion during ejection. With increased contractility or exercise conditions both ejection fraction and torsion increase [16]. In cardiac patients, ejection fraction is often low, resulting in low torsion. However, preservation of stroke volume in combination with heart failure may render ambiguous effects on torsion. If heart failure is caused by subendocardial contractile dysfunction due to coronary stenosis or aortic valve stenosis, torsion may increase. However, when the LV dilates simultaneously, the increase may be annihilated totally. This fact may explain why in some studies torsion does not correlate significantly with heart failure [17].

In summary, in the normal heart, torsion varies linearly with the logarithm of the inner diameter. In systole the relation is much steeper than in diastole. The difference in slopes causes fast changes of torsion at the transitions between systole and diastole, occurring during the isovolumic phases. With LV dilatation due to heart failure, both ejection fraction and torsion decrease. With increased contractility, ejection fraction increases and end-systolic volume decreases, resulting in increase of torsion during the ejection phase and increase of torsion recoil or untwisting. So far, measurement of LV volume and torsion provide similar information. Determination of the rate of LV relaxation requires measurement of LV pressure by a catheter. The rate of untwist may provide similar information with non-invasive means. Torsion adds major information on the transmural distribution of contractile performance, to be quantified most accurately by the torsion to shortening ratio TSR, where shortening represents the decrease of the logarithm of the inner diameter. Understanding of possibilities and limitations of the use of torsion as indicator of function has been facilitated considerably by the use of a frame work for heart mechanics, based on general principles of mechanics and physic.

V. References

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