Three-Dimensional Analysis of Regional Left Ventricular Endocardial Curvature from Cardiac Magnetic Resonance Images

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Abstract

Left ventricular (LV) remodeling is usually assessed using changes in LV volume, while disregarding regional changes that may occur independently of volume. We hypothesized that 3D analysis of regional endocardial curvature could provide useful information on localized remodeling. Cardiac magnetic resonance (CMR) images were acquired in 44 patients: 14 normal controls (NL), 15 with dilated cardiomyopathy (DCM), and 15 ischemic heart disease (IHD). LV endocardial surface was reconstructed throughout the cardiac cycle and used to calculate for each point the curvedness, normalized by instantaneous LV size (C_n). Normalized curvedness was compared between groups of segments: NL (N=401), DCM (N=255) and IHD (N=92). While in NL segments, C_n values were comparable in basal and mid-ventricular segments, they were higher in the apical segments ($p<0.05$). Also, % change in C_n was higher in mid and apical compared to basal segments ($p<0.05$). At all LV levels, C_n in DCM segments was lower ($p<0.05$) than in NL and IHD. In contrast, % change in C_n was lower in both IHD and DCM segments compared to NL ($p<0.05$). 3D analysis of regional LV endocardial curvature from CMR images provides quantitative information, which is consistent with the known pathophysiology, and may prove useful in the evaluation of LV remodeling.

1. Introduction

Left ventricular (LV) remodeling as a result of disease progression or in response to therapy is reflected by changes in ventricular size and shape. To date, assessment of LV remodeling has been predominantly described in terms of changes in LV volume and ejection fraction, while disregarding the simultaneously occurring changes in ventricular shape, because the development of tools for quantitative evaluation of ventricular shape has been lagging. Moreover, LV function has been studied both on a global and regional basis, but previous studies have focused on global LV shape, while only initial feasibility data have been published regarding local LV shape [1,2]. A recently proposed global LV shape analysis from real-time 3D echocardiographic images [3] was able to demonstrate independent changes in LV volume and shape as well as to identify differences between normal and abnormal ventricles.

In the current study, we focused on the quantification of regional endocardial curvature from dynamic 3D LV endocardial surfaces derived from cardiac magnetic resonance (CMR) images, assuming that such analysis could prove useful in the evaluation of LV remodeling. Accordingly, the aims of this study were: (i) to determine normal patterns of regional endocardial curvature and its changes throughout the cardiac cycle, and (ii) to test this technique on images obtained in patients with global and regional hypokinesis, in order to understand the effects of these conditions on regional LV shape.

2. Methods

2.1. Population

We studied 44 consecutive patients, referred for CMR imaging including 14 patients with normal to mildly abnormal LV function (NL group), 15 patients with regional wall motion abnormalities secondary to ischemic heart disease (IHD group) and 15 patients with dilated cardiomyopathy (DCM group). Exclusion criteria were: dyspnea precluding a 5-10 second breath-hold, atrial fibrillation or other cardiac arrhythmias, pacemaker or defibrillator implantation, claustrophobia and other known contraindications to CMR imaging.

2.2. CMR imaging

CMR images were obtained using a 1.5 Tesla scanner (Siemens) with a phased-array cardiac coil. Steady-state free precession dynamic gradient-echo mode was used to

acquire images using retrospective ECG gating and parallel imaging techniques during 5-10 sec breath-holds with a temporal resolution of 30 frames per cardiac cycle. Cine-loops of 8 mm thick slices with 2 mm gaps and 2.0x2.0 mm in-plane resolution were acquired in three long-axis planes representing the two-, three- and four-chamber views, and a stack of short-axis views from just above the ventricular base to just below the apex.

2.3. 3D endocardial surface detection

The entire set images was analyzed using prototype software (4D-LV Analysis MR, TomTec). End-diastole was identified as the first frame in the sequence and end-systole as the frame depicting the smallest LV cavity. For each of these frames, LV endocardial boundary was manually initialized in each of the three long-axis views (figure 1, left). Endocardial surface was then reconstructed throughout the cardiac cycle. Subsequently, this surface was superimposed on the original image set and its position was adjusted frame-by-frame by an experienced investigator wherever the surface did not accurately follow the endocardium. The resultant dynamic endocardial surface (figure 1, center) was exported as a connected mesh for analysis of LV endocardial curvature.

2.4. 3D curvature analysis

Custom software was used for analysis of regional LV endocardial curvature. First, for each node on the structured mesh representing the LV endocardial surface a quadric polynomial function was locally fitted to approximate a smooth surface as previously described [4]. Then, for each point, two values were calculated: maximum curvature k_1, defined as the inverse of the radius of the smallest circle that would fit into the surface at that particular point, and the curvature k_2, similarly defined in the perpendicular direction (figure 1, top right). Then local 3D surface curvedness, C, was calculated as the root mean square value of k_1 and k_2 [5] and then normalized by mean instantaneous LV curvedness, calculated by averaging curvedness at all nodes on the endocardial surface. This latter step was performed to compensate for changes in LV regional shape secondary to changes in LV size. Finally, normalized curvedness C_n was mapped onto the volume-rendered endocardial surface (figure 1, bottom right). Finally, the endocardial surface was divided into 17 segments. For each segment, regional 3D normalized curvedness was obtained by averaging local C_n-values measured in all nodes in the segment.

2.5. Segment classification

Short-axis images at basal, mid-papillary and apical levels were reviewed by an independent reader blinded to the results of 3D curvature analysis, who classified wall motion in each segment as normal or abnormal. These classifications were used to separate segments by pathology and compare endocardial curvature between NL segments at four different LV levels (basal, mid-ventricular, apical and apical cap) as well as between NL

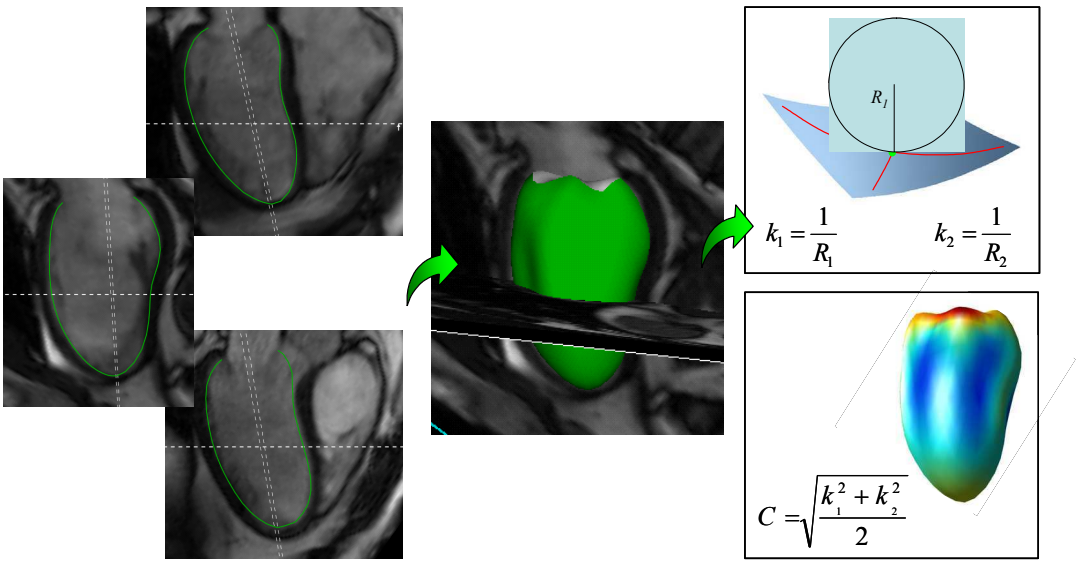

Figure 1. Schematic representation of the 3D analysis procedure: following manual initialization of the endocardial contours at end-systole and end-diastole in 3 radial long-axis views (left panels: 2-, 3- and 4-chamber views), 3D endocardial surface was reconstructed throughout the cardiac cycle and superimposed on the original images (center). For each voxel on the reconstructed endocardial surface, two values were calculated: maximum curvature k_1, and the curvature k_2 in the perpendicular direction (top right). Then local 3D surface curvedness, C, was calculated and mapped onto the volume-rendered endocardial surface (bottom right), which was encoded with colors ranging from dark blue used for low curvedness values to dark red for high curvedness.

and hypokinetic segments, while averaging separately segments from ventricles in patients with DCM and abnormal segments from patients with IHD.

2.6. Statistical analysis

Data were displayed in the box-plot format, wherein: the box represents the 1st quartile, median, and 3rd quartile values; whiskers extend to the extreme values within 1.5 times the inter-quartile range; and outliers are shown as "+" symbols. Differences in regional curvedness between three LV levels as well as differences between NL, DCM and IHD segments were tested using Kruskal-Wallis test. P-values <0.05 were considered significant.

3. Results

Figure 2 shows three examples of LV endocardial normalized curvedness, C_n, mapped onto the respective volume-rendered LV endocardial surface in a patient with normal LV function and in two patients with wall motion abnormalities, along with time-curves depicting variations in curvedness throughout the cardiac cycle. In the patient with normal LV function, C_n gradually increased during systole in all segments, reaching peaks at slightly different times in different segments, and then decreased during diastole (top). Similar behavior can be seen in the patient with DCM, despite the reduced magnitude of changes (middle). In contrast, in the patient with IHD, the time curves reflected a more disorganized pattern of changes in endocardial curvedness (bottom).

Figure 3 shows the summary of minimum, maximum and percent change in regional C_n for the NL segments, grouped by LV level. While both maximum and minimum values of C_n (top) were comparable in the basal and mid-ventricular segments, they were significantly higher in the apical segments and highest in the apical cap. In addition, percent change in C_n (bottom) was lower in the apical region compared to basal and mid segments.

Comparisons of these parameters between the three groups of segments: 401 NL, 92 IHD and 255 DCM segments showed that: (1) minimum C_n was comparable between groups; (2) maximum C_n was lower ($p<0.05$) in DCM segments than in NL and IHD segments, which were similar; (3) percent change in C_n was lower ($p<0.05$) in both IHD and DCM compared to NL.

4. Discussion and conclusions

We tested the feasibility of quantifying regional, LV size normalized, 3D endocardial surface curvature; used normal ventricles to establish normal patterns of curvature and its changes throughout the cardiac cycle, and studied two different pathologic conditions affecting LV shape, one global (DCM) and one more localized (IHD).

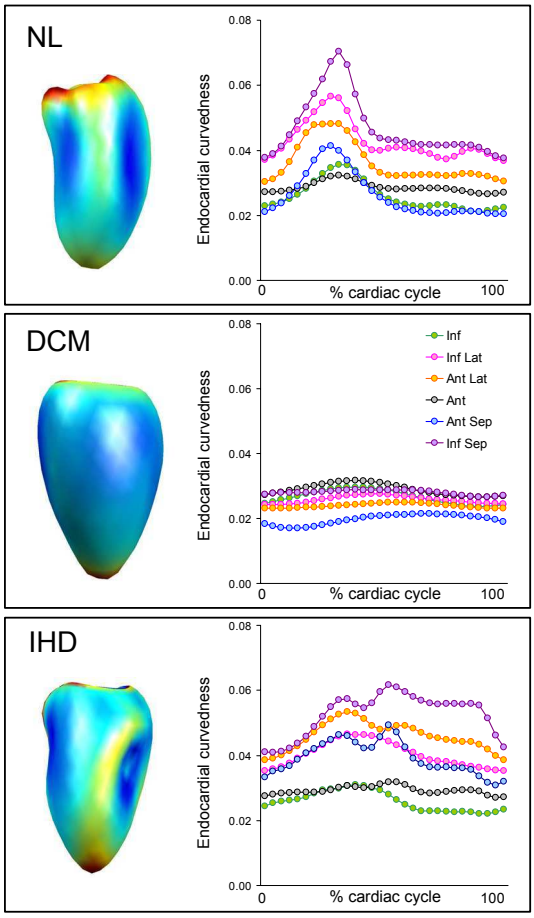

Figure 2. Examples of LV endocardial curvedness mapped onto the volume-rendered endocardial surface in a patient with normal LV function (NL, top) and in two patients with wall motion abnormalities: a patient with DCM (middle) and a patient with IHD (bottom), along with the 6 time-curves depicting variations in mid-ventricular regional curvedness throughout the cardiac cycle.

While our findings in normal ventricles demonstrating a systolic increase in endocardial curvature are expected and consistent with those previously demonstrated in a smaller group of subjects [6], our results confirmed the ability of this analysis to accurately detect subtle changes occurring frame-by-frame throughout the cardiac cycle (figure 2). Similarly, the increase in endocardial curvature toward the apex (figure 3) is intuitive, but our results have proved the ability to quantify these regional differences.

The reduced maximum regional endocardial curvature in DCM is expected and consistent with previous findings [7]. Since dilated ventricles are known to be more spherical than normal ventricles on a global level, and because ventricular dilatation in these patients is concentric and relatively uniform across segments, one could expect similar findings on a regional level.

Interestingly, DCM patients showed minimum value of normalized curvedness similar to the other study groups, while maximum values were increased. Since minimum values of C_n occur at end-diastole while maximum values are end-systolic, this finding is likely due to the expected reduced LV function and not only due to an increase in LV volume. Importantly, the normalization step allowed separating the effects of LV volume changes from the morphologic information, proving that this step is not redundant but rather complimentary to the standard LV volume analysis.

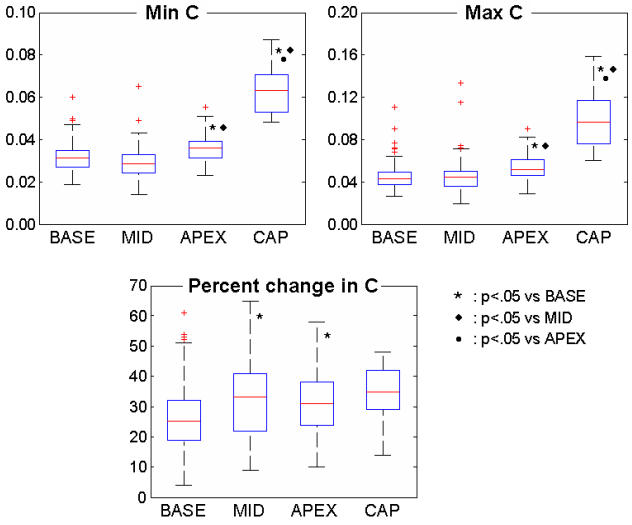

Figure 3. Box plots depicting normal variability in regional LV endocardial curvature from base to apex: minimum, maximum and % change in regional endocardial curvedness (Min C, Max C, and % change in C) calculated for the 401 normal segments, grouped by LV level. C-values expressed in m^{-1}. *p<.05 vs base, ◆p<.05 vs mid, •p<.05 vs apex, Kruskal-Wallis test.

The reduced percent change in endocardial curvature in segments that are hypokinetic as a result of either concentric dilatation or IHD is also an expected finding consistent with reduced function in these segments irrespective of etiology. Importantly, these results have proved the ability of this technique to provide quantitative information not only in normal ventricles, but also under pathological conditions, thus providing support for clinical feasibility of this new methodology.

One limitation of our technique is that it relies on the availability of a reconstructed LV endocardial surface, which is based on subjective multi-plane and multi-phase initialization, tracing and corrections, and is thus prone to considerable inter-measurement variability. In this study, we used commercial software for 3D surface reconstruction and did not test its reproducibility, since this study focused on the 3D analysis of regional endocardial curvature, which is fully automated once the endocardial surface is defined, and is therefore 100% reproducible.

Importantly, this study constitutes one of the first attempts to objectively describe regional LV shape and deformation based on 3D analysis of CMR images and test the clinical feasibility of this new methodology in a relatively large number of patients with different patterns of wall motion. The ability to quantify regional LV shape may prove especially important in clinical scenarios, in which LV shape is affected locally and thus the conventional global measures of LV volume and shape may fail to differentiate normal from abnormal ventricles. One example of such common pathology would be the paradoxical septal motion in patients with right ventricular volume overload secondary to pulmonary hypertension, which is easily visualized but has been difficult to measure quantitatively. Our results indicate that this approach provides quantitative information on regional ventricular function, which is consistent with the known pathophysiology, and may thus prove clinically useful in the evaluation of LV remodeling.

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