ANALYSIS OF THE ACTIVATION PROPAGATION ON BOTH VENTRICLES USING TAGGED MRI

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Abstract – **MRI tagging created new opportunities for motion analysis within the body. With tagged cardiac MRI, detailed motion analysis has been previously shown; e.g. measurements of local strain of the heart could be obtained easily for normal and pathological hearts. In this study, we calculate the activation times using the local time-strain relationship and compute the propagation velocity of the activation for both chambers of the heart. Here, in this initial analysis, we obtained propagation velocities for four canine hearts paced either at the atrium or right ventricle. The first case provides a condition similar to the normal physiological activation. We show here for the first time an activation propagation speed over the right ventricle *in vivo*. The overall method provides a unique noninvasive method to examine the underlying electrical activation of the heart, but further studies are needed to establish its true clinical value.**
Keywords – **Cardiac MRI, cardiac mechanics, activation time**

I. INTRODUCTION

With the progress of new MRI techniques, faster and functional imaging of the heart is becoming feasible. One of the promising methods is MR tagging, which creates the opportunity to observe and measure the movement of tissues specifically the heart [1]. In MR tagging, temporary magnetic marker lines are created on the tissue by use of specific RF waves.

Mathematical methods can be employed to gather detailed quantitative motion parameters (displacements, velocities or strains) from a set of tagged cardiac images. We have used here the results of the local strain analysis and aim to map the activation profile, display the propagation of activation and compute the velocity of the activation wave over both ventricles.

II. METHODOLOGY

Data used is obtained from MRI recordings of canine heart, which was paced from the atrium or the right ventricle. GE Signa 1.5T scanner was used with segmented *k*-space acquisitions during breath hold periods. The scanning parameters used were a 28- to 32- cm field of view, a time to repetition of 6.5 ms, a time to echo of 2.1 ms, a 256×96 acquisition matrix, a ±32-kHz bandwidth, two to three readouts per movie frame, an in-plane spatial resolution of 1.25×3 mm, and a slice thickness of 6-7mm.

Three orthogonal directions of tags and a mathematical motion model are needed to assess the 3D movement of tissues [2]. We employed a motion analysis method based on a four-dimensional B-Spline tensor representation [3]. Local deformation gradients were obtained using this parametric model and 3D strain values using finite strain tensor analysis was computed over a mesh located at mid myocardium of right and left ventricles. The motion field, activation time and velocity calculations are done automatically once the tags have been detected. Two ventricles are modeled differently; the left ventricle (LV) was modeled as a deformed cylinder, and the right ventricle (RV) as a deformed half cylinder. A mesh is defined on these cylinders, and from the motion analysis, we obtain the time series of strain on each mesh point. A Matlab toolbox that had been developed earlier for LV analysis is improved for this study [4]. Four-dimensional analysis enables us to observe strain changes at each mesh point during the contraction (Fig. 1 and 2). We have selected to use circumferential strain (Ecc) values in our analysis because of the orientation of the major muscle fibers in mid-myocardium are known to be in that direction [5].

The activation has been chosen as the time of peak Ecc strain value. When no peak is present neighborhood based correlation is employed as in [4]. Once the activation times over the whole heart is computed, the velocity of the activation wave propagation at each location is found. For this calculation, activation time of each patch is used along the real x, y, z coordinate values of the patches. Since activation propagation velocity is obtained by time and spatial derivative calculations, it tends to accentuate the noise in the data. Before velocity calculations 2D median filters are used to eliminate salt pepper noise in activation times due to occasional miscalculation of the strain peaks. The calculated velocity values are further scanned for local activation wave direction inconsistencies to eliminate velocity computation at sink or source locations. RV and LV analysis are done independently.

III. RESULTS

For a sample heart, the evolution of the strain for a mid-myocardial mesh of the LV and RV is shown in Fig. 1 and Fig. 2. Fig. 1. displays the local strains for the LV and Fig. 2. for RV, for both pacing conditions. In these graphs, the calculated activation point of each mesh point is displayed by a small vertical line. The small plots in each row represents mesh points arranged circularly in a short axis cut of the heart. Coming down on each column represents moving from the base of the heart towards its apex. These graphs are sub-sampled dramatically in both directions for display purposes. Only contraction part (systole) is shown.

Another helpful display method for showing the activation times over the both ventricle is the bulls-eye

Report Documentation Page

Report Date	Report Type	Dates Covered (from... to)
25 Oct 2001	N/A	-

Title and Subtitle Analysis of the Activation Propagation on Both Ventricles Using Tagged MRI	**Contract Number**
	Grant Number
	Program Element Number
Author(s)	**Project Number**
	Task Number
	Work Unit Number
Performing Organization Name(s) and Address(es) Institute of Biomedical Engineering Bogazici University Istanbul Turkey	**Performing Organization Report Number**
Sponsoring/Monitoring Agency Name(s) and Address(es) US Army Research, Development & Standardization Group (UK) PSC 802 Box 15 FPO AE 09499-1500	**Sponsor/Monitor's Acronym(s)**
	Sponsor/Monitor's Report Number(s)

Distribution/Availability Statement
Approved for public release, distribution unlimited

Supplementary Notes
Papers from 23rd Annual International Conference of the IEEE Engineering in Medicine and Biology Society, October 25-28, 2001, held in Istanbul, Turkey. See also ADM001351 for entire conference on cd-rom., The original document contains color images.

Abstract

Subject Terms

Report Classification unclassified	**Classification of this page** unclassified
Classification of Abstract unclassified	**Limitation of Abstract** UU

Number of Pages
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graph (Fig. 3 and Fig. 4). In this graph apex of the LV is the central ring, basal part is the most outer ring. RV is shown on the right side with partial rings, color represents the activation time from a reference time in imaging. It provides a quick display of the overall activation pattern of the heart. This is shown for atrial activation in Fig.3. and for ventricular activation in Fig. 4.

With the help of these activation times and the coordinates of the mesh points in 3D, propagation speed of the activation are computed in four canine hearts for both pacing conditions over the RV and LV. Preliminary statistics of these velocities are shown in Table 1. The average of the mean velocity of the activation propagation for RA pacing and RV pacing was 1.2 and 0.99 m/s for the LV and 1.18 and 0.81 m/s for the RV respectively.

IV. Discussion

If we compare the time-strain plots for both pacing conditions, for atrial pacing, we obtain similar local strain time graphs over the LV and RV. Strains decrease almost linearly during ventricular systole. On the other hand, for ventricular pacing, we observe double peaks due to initial or delayed stretch, definitely a more complex time-strain pattern with the start of contraction is delayed at certain locations.

In bulls-eye plots, for atrial activation, we see relatively small activation time range (134 ms) over both ventricles. Mechanical activation seems to start at several locations at once and proceeds in various directions. Some of the basal portion of the LV is activated last. These findings are compatible with physiological excitation through the Purkinje system. This is also supported mostly by higher propagation speed than ventricular pacing (Table 1). Although we should state that a definite distinction between propagation over the Purkinje system or myocardium itself can not be made definitely using this data.

On the ventricular paced heart clearly a propagation wave can be identified, starting from the RV and proceeding towards the LV. This and slower speed of activation wave propagation results in a higher range of the activation times for the LV (206 ms).

The high standard deviation values for all ventricles and pacing protocols, even after the use several neighborhood based smoothing approaches, prevents us employing statistical analysis. On the other hand, there is a trend of decreased velocity when the ventricle is activated locally as opposed to the case when it is activated through the Purkinje system. The activation propagation , on average, seems to be a little lower for the RV, in comparison to the LV (Table 1).

In the previous work of Wyman et al, the activation time has been calculated only over the LV. In that study, the heart motion is described with a LV-specific approach. The values for the LV are similar to the values obtained in [4], in spite of using a completely different mathematical motion model here. In our previous work, we had some preliminary results on activation times on both ventricles [6-7]. The improved method here, smoothing and eliminating the outliers based on the neighborhood relationships, resulted in better and consistent results and calculation of propagation velocities.

Neighborhood relationships among the patches can be utilized further to develop a more comprehensive model of the activation propagation in the future. This might improve the sensitivity and resolution of our method. At that time, it could be possible to investigate the relationship between local velocity of the activation wave and the mechanical status before contraction, a critical question in fetal arhythmiogenesis.

ACKNOWLEDGEMENT

The authors wish to acknowledge all the help and support of E.R. McVeigh Ph.D.

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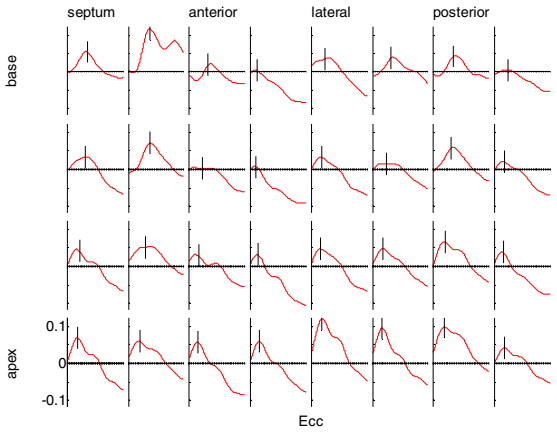
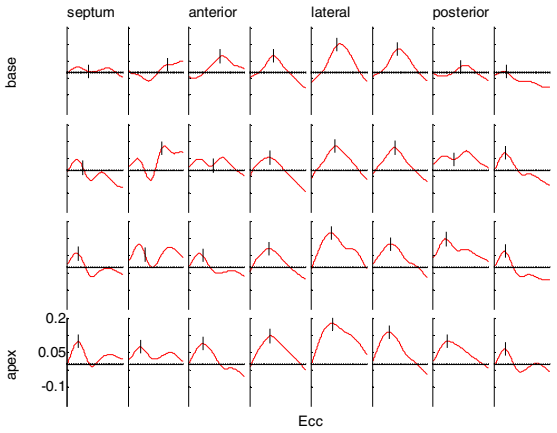
TABLE I
Velocity values for both ventricle with two different pacing protocols (m/s)

	RA/LA paced LV			RA/LA paced RV			RV paced LV			RV paced RV		
	Mean	Median	STD	Mean	Median	STD	Mean	Median	STD	Mean	Median	STD
1	1.1947	0.7182	1.1010	0.6862	0.5137	0.5805	0.9008	0.8254	0.6574	0.8408	0.4635	1.1227
2	0.7599	0.5190	0.7275	1.2483	0.6298	1.5233	1.0498	0.5123	1.2443	0.8578	0.7926	0.7372
3	1.3732	1.2602	0.5875	1.2721	1.0242	1.0093	0.7167	0.2900	1.3328	0.8930	0.7391	0.7638
4	1.5805	0.8522	1.9488	1.5325	0.7489	2.4146	1.3040	0.6569	2.0388	0.6430	0.3682	0.9567



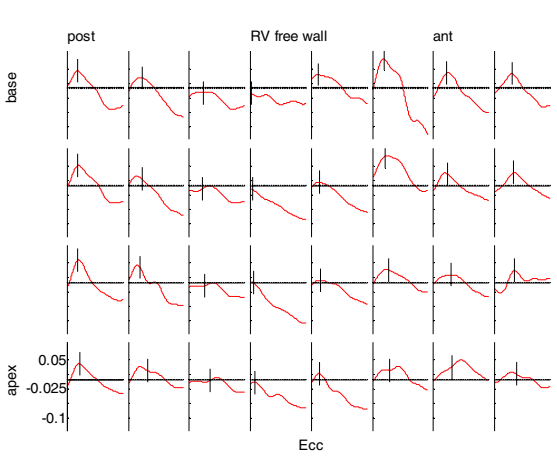
(a)

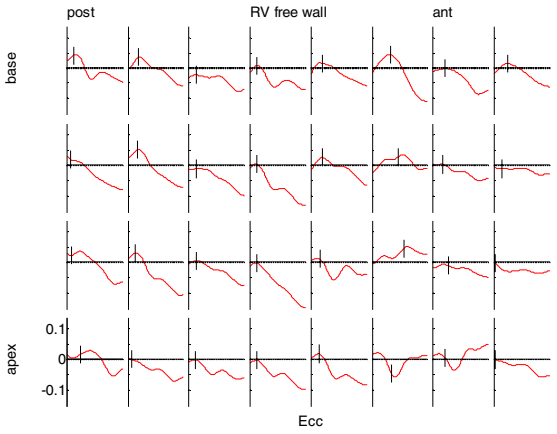


(b)

Fig 1. Strain values over the left ventricle during systole of a canine heart(#1) (a)atrial excitation, (b)RV excitation. Calculated activation times at each mesh point is displayed. Y-axis represents strain; -0.1 means 10% shortening, positive values represent stretching.

(a)





(b)

Fig 2. Strain values over right ventricle during systole of a canine heart(#1) and calculated activation times (a)atrial excitation, (b)RV excitation.

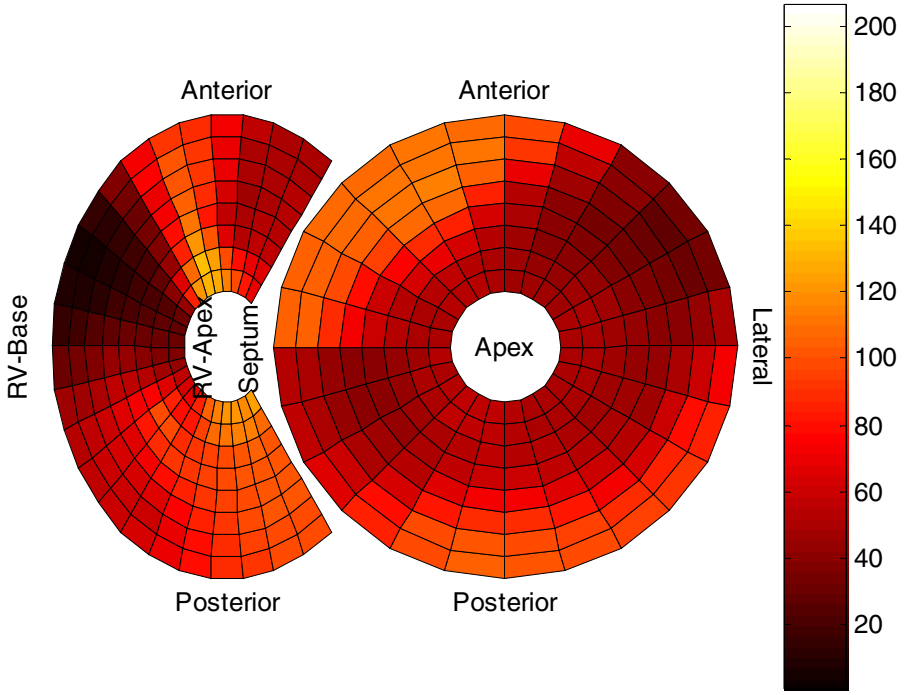


Fig3. Bi-ventricular Bulls-Eye Plot of activation times. Atrial activation

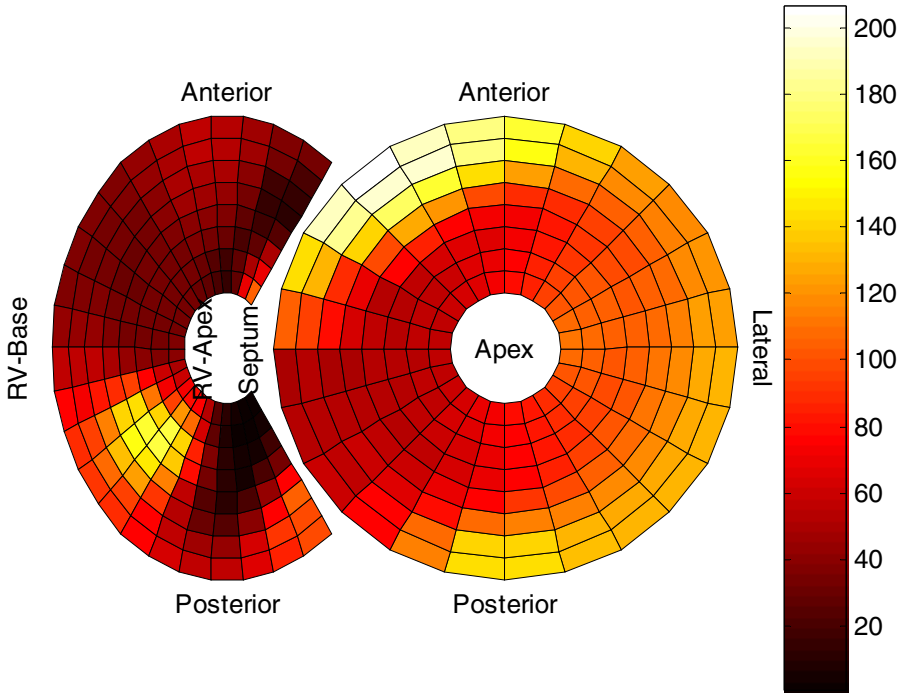


Fig4. Bi-ventricular Bulls-Eye Plot of activation times. RV pace