## Assessment of Left Ventricular 2D Flow Pathlines during Early Diastole Using SPAMM-PAV: a study in normal volunteers and canine models with reperfused infarcts

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Introduction: The functional mechanisms involved in adverse left ventricular (LV) remodeling caused by reperfusion injury (RI) are unclear [1]. We have developed a new high temporal resolution MR imaging technique, SPAMM-PAV (SPAtially Modulated Magnetization with Polarity Alternated Velocity encoding) that provides simultaneous regional assessment of flow velocities and myocardial strains during early diastole [2]. Using this method, we performed flow pathline analysis, (1) to study the impact of RI on the nature of filling patterns and (2) to develop a novel index of filling efficiency, characterized by the kinetic energy (KE) of flow pathlines during early diastole.

Flow pathline and kinetic energy: As illustrated in Fig. 1, mitral inflow velocity curve is an average of the flow velocity within a region circled around the mitral valve. At each time instant during rapid filling, a fixed number of virtual emitter particles, proportional to the area under the inflow velocity curve, are released from the mitral valve plane to generate a set of flow pathlines for each time instant [3]. The KE of each set of flow pathlines, as illustrated in Fig. 2, can be calculated based on the knowledge of the volume covered by each pathline during each time step, its velocity and the density of the blood, with the derived formula,  $KE = mv^2/2 = (\rho AD)(l/\Delta t)^2/2$ , where  $\rho$  is human blood density (assumed to be constant at 1060 kg/m<sup>3</sup>), A is

**a** 480 (ms)

Repid Filling

Repid Filling

Repid Filling

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Fig. 1. Number of emitter particles released at a given time instant corresponds to the area under the mitral inflow velocity curve.

3.6 mJ **b** 

the constant horizontal cross-sectional area, 
$$D = \sum\limits_{n=1}^{100} \sqrt{\left(x_{n+1} - x_n\right)^2 + \left(y_{n+1} - y_n\right)^2} \ (\textbf{\textit{x}}_n, \ \textbf{\textit{y}}_n \ \text{were latitudinal}$$
 and longitudinal coordinates respectively), I is the linear

distance between two interpolated time points, and  $\Delta t$  is the time-span between points. Averaging the KE of all the pathlines corresponding to a set of emitter particles at a given time instant, one can get the overall average KE of the blood entering into the LV at that time instant, indicative of the efficiency of diastolic filling.

**Experiments and Results:** Studies were performed in six normal volunteers and three dogs following 5-6 hours of balloon occlusion of the left anterior descending artery (LAD) followed by reperfusion creating an antero-septal transmural infarct with reperfusion injury. SPAMM-PAV

(LAD) followed by reperfusion creating an antero-septal valve plane in a normal volunteer, with contransmural infarct with reperfusion injury. SPAMM-PAV dotted to reflect wavefront like propagation. measurements for both in-plane directions were conducted on a 1.5 T scanner on two long-axis slices, with imaging parameters set as follows: imaging matrix: 192×192. resolution: 1.5mm×1.5mm. slice

Fig. 2. (a) Time series (row1: acceleration, row 2: deceleration) of particle traces emitted from the mitral

Fig. 2. (a) Time series (row1: acceleration, row 2: deceleration) of particle traces emitted from the mitral valve plane in a normal volunteer, with color coded upon the instant KE. The top-left subfigure is red dotted to reflect wavefront like propagation. (b) Overlapped particle trace trajectories for all time points.

with imaging parameters set as follows: imaging matrix: 192×192, resolution: 1.5mm×1.5mm, slice thickness: 8mm, views per cardiac phase: 3, tag separation: 8mm, Venc: 120-150 cm/s, temporal

resolution: 14-15ms.

-■- NormalVol -o-- Dog Baseline 0.16 Inf. Dog 1 Inf. Dog 2 Inf. Dog 3 0.12 Kinetic Energy, mJ 0.08 0.00 Rapid Filling Phase n 50 100 150 200 Time, ms

Fig. 4. Comparison of the overall average KE of normal volunteers, dog baseline and infracted dogs. Only the frames in rapid filling phase are investigated. The limited standard deviation for normal volunteers demonstrates a close KE of them.

The temporal evolution of the trajectories of a normal volunteer obtained from a 4-chamber slice, shown in Fig. 2a, follow a specific pattern, with uniform distribution of blood throughout the LV in the early time frames followed by a more centric filling pattern restricted to the basal regions and away from the walls during later time frames. This is more clearly visualized when the pathlines are overlapped using the temporal color scheme shown in Fig. 2b. A slight physiologic difference in flow

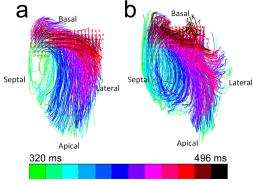


Fig 3. Overlapped blood emitter particle flow pathways for (a) normal canine and (b) canine with reperfused infarction (imaged 3 days post reperfusion), color-coded to indicate time of release of blood emitter particles.

pattern is observed between the normal volunteer (Fig. 2b) and a healthy dog, shown in Fig. 3a, where the flow pathline propagates mostly with a clockwise circulation through the basal region and then directly down to the apex although maintaining fairly uniform distribution. Relative to the canine baseline, the flow pathlines of an acute infarcted dog, Fig. 3b, shows abnormalities. During the early stages of rapid filling, the emitter particles close to the spetal wall form a significant vortex extending from basal to mid walls avoiding the apex (region of infarct), while

emitter particles close to the lateral wall propagate down to the apex along the lateral wall. This asymmetric vortex pattern may be responsible for increased wall stresses in the left ventricle. As illustrated in Fig. 4, the differences between the kinetic energy curve (each point on the curve represents the average kinetic energy for a set of pathlines corresponding to emitter particles released at that time point) of normal volunteers, dog baseline and acute infarcted dog are pronounced. With only the cardiac phases during rapid filling investigated, the KE curves display a typical bell shape. The normal-volunteer KE curve, an average of all the six 6 normal volunteers, exhibits a higher value than the canine baseline, which indicates a larger flow velocity in healthy human than canine. The three infarcted dogs exhibit much higher KE than the baseline animal. We believe that this increase in KE is due to increased velocities due to the swirling flow required to maintain the vortex formed in the septal-basal regions, and maybe responsible for late remodeling.

Conclusion: In conclusion, 2D pathline analysis provides a direct regional and quantitative assessment of diastolic filling patterns following myocardial infarction.

References: 1. Pfeffer MA, et al., Lancet, 362 (2003) 759-766; 2. Zhang Z, et. al., Magn Reson Med; 2011, DOI:10.1002/mrm.22965; 3. Zhang Z, et al, ISMRM annual meeting, Montreal, Canada, May 7-13, 2011.