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Quantification of 4D left ventricular blood flow organization in normal and failing hearts

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Objective

To measure the volume and distribution of separate flow components in normal and dysfunctional left ventricles (LV).

Background

The forces created by blood flow dictate a continuous remodeling of cardiac structures. In the normal heart this mechanism interactively creates an optimal geometry for efficient flow [1]. In various cardiac disorders such as LV dysfunction, however, the normal flow organization may be altered, leading to negative remodelling. The description and quantification of the true 4D (3D+time) blood flow organization in dysfunctional LVs remains incomplete.

Method

Seven dilated cardiomyopathy (DCM) patients (4 female, aged 52 ± 14 years [mean ± SD]) and six healthy subjects (3 female, aged 58 ± 4 years) were studied. Three-directional, 3-dimensional cine phase-contrast-MRI velocity data and morphological b-SSFP long- and short-axis images were acquired on a clinical 1.5 T MRI scanner (Philips Achieva). The LV endocardium was segmented (http://segment.heiberg.se, [2]) from the short-axis images at the times of isovolumetric contraction (IVC) and isovolumetric relaxation (IVR). Pathlines were emitted from the IVC LV blood volume and traced forward and backward in time until IVR, thus covering the entire car-

diac cycle. The IVR volume was used to determine if and where the traces left the LV. This information was used to automatically separate the pathlines into four different LV flow components [3,4](Table 1, Figure 1).

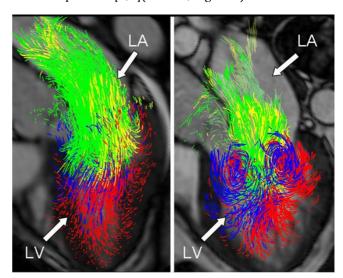
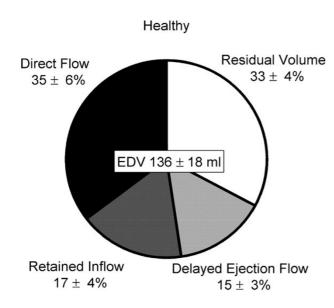


Figure I
Pathline visualization of LV blood flow organization at early diastole in a healthy 58 year old female (left panel) and a 61 year old female with dilated cardiomyopathy (right panel). Green, direct flow; yellow, retained inflow; blue, delayed ejection flow; and red, residual volume. LA, left atrium; and LV, left vemtricle.

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Table I: Components of LV blood flow

Component	Description
Direct Flow	Blood that enters and leaves the LV within the same cardiac cycle
Retained Inflow	Blood that enters but does not leave the LV within the same cardiac cycle
Delayed Ejection Flow	Blood that leaves but does not enter the LV within the same cardiac cycle
Residual Volume	Blood that resides in the LV more than two cardiac cycles



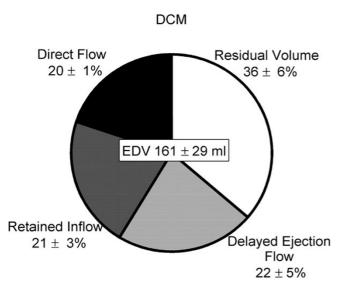


Figure 2
Illustration of the four LV flow components as a percentage of LV end-diastolic volume (mean ± SD) in the six healthy subjects and seven patients with dilated cardiomyopathy.

Results

There was no difference in heart rate between DCM and healthy subjects (58 ± 11 vs 64 ± 8 bpm, NS). The LV ejection fraction was smaller and LV end-diastolic diameter was larger in DCM compared to healthy subjects (43 ± 5 vs $59 \pm 2\%$, p < 0.001, and 60 ± 5 vs 45 ± 3 mm, p < 0.001, respectively). The direct flow/end-diastolic volume (EDV) ratio was lower and the delayed ejection flow/EDV ratio was higher in the DCM compared to healthy subjects (20 ± 1 vs $35 \pm 6\%$, p < 0.001, and 22 ± 5 vs $15 \pm 3\%$, p < 0.01, respectively) (Figure 2).

Conclusion

Multidimensional flow analysis can measure the compartmentalization of blood transiting the beating LV. The present findings imply that the highly organized flow in and out of normal LVs is demonstrably altered in DCM, even with only moderate LV dilation and dysfunction. Such measures may contribute not only to our understanding of the pathophysiology and diagnosis of disease, but also to the optimization of therapy.

References

- I. Richter Y, Edelman E: Circulation 2006, 113:2679-2682.
- 2. Heiberg E, et al.: Comput Cardiol 2005, 32:599-602.
- 3. Eriksson J, et al.: ISMRM flow workshop 2009:47.
- 4. Bolger AF, et al.: JCMR 2007, 9:741-747.