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Meeting abstract

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143 Multidimensional turbulence mapping in mitral insufficiency

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Introduction

Blood flow turbulence may contribute to the progression and hemodynamic consequences of valvular heart disease, but cannot be measured by current clinical methods.

Purpose

How does the high velocity jet of systolic mitral regurgitation relate to regional turbulence in the left atrium during systole and diastole?

Methods

Time-resolved, three-dimensional phase-contrast MRI (PC-MRI) data were acquired in three perpendicular directions in a 73 year old male with severe mitral regurgitation due to a posterior leaflet prolapse, using a clinical 1.5 T scanner. The mean velocity field was computed by conventional phase subtraction. Using a recently described approach to quantify the fluctuating velocities associated with turbulent flow from the magnitude components of the PC-MRI signals, the turbulent kinetic energy (TKE) was obtained for each voxel. The TKE is a direction-independent measure of turbulence intensity and thereby insensitive to measurement directions. Using commercially available visualization software, the velocity field was assessed by computing streamlines and the extent and degree of turbulence intensity was examined by creating clip planes color-coded according to scales of TKE.

Results

In systole, the turbulence map demonstrates a marked increase in TKE along the course of the high velocity regurgitant jet (left, Figure 1), directed along the atrial septum. In diastole, regional increases in TKE are seen along the open mitral valve leaflets, and near the origin of the left lower pulmonary vein (LLPV, middle, Figure 1). Concurrent streamlines demonstrating the flow from the LLPV reveal that this diastolic turbulence arises at the site where the pulmonary vein inflow collides with the blood volume in the left atrium, resulting in an acute change in the direction of inflow (right, Figure 1). In contrast, the right upper pulmonary venous inflow, which transits the left atrium along the interatrial septum without abrupt turns, does not demonstrate turbulence, despite having higher peak velocity than the left vein (0.9 vs 0.6 m/s).

Conclusion

Turbulence accompanies significant valvular disease not only in the expected high velocity jet regions, but also at other areas and at different times in the cardiac cycle. Localizing turbulence at different phases and sites may improve the understanding of the pathophysiology of valvular diseases by identifying how they disrupt both global and regional hemodynamics and flow. It is known that endothelium in regions subjected to turbulence becomes activated, promoting interaction with platelets and increased atherogenicity. Turbulence also identifies regions at risk for endocarditis. In vivo maps of turbulent kinetic energy may therefore provide a new perspective for assessing the progression of valvular disease and a basis for comparison of treatment options.

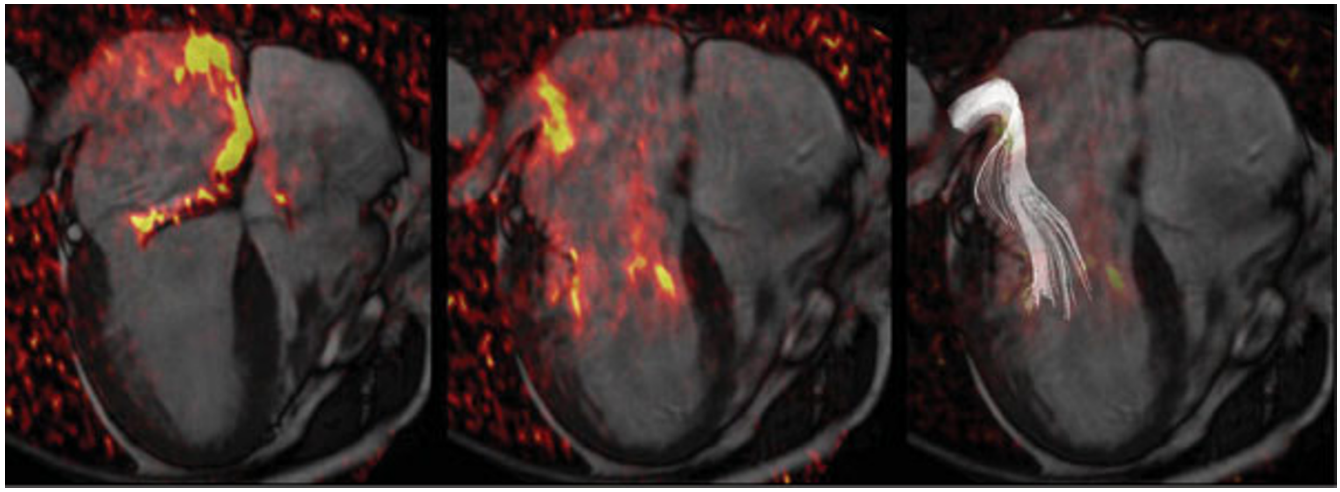

Figure 1
Generalizing phase-contrast MRI to map the effects of turbulence in the setting of mitral insufficiency. Novel data are presented on the relationship between flow regurgitation and areas of turbulent flow.