Hemodynamics and Wall Shear Stress in the Pulmonary Arteries of Hypertension Patients using Phase Contrast MRI

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Introduction: A number of concomitant factors are thought to contribute to an increase in the mean pulmonary arterial pressure (MPAP) of pulmonary arterial hypertension (PAH) patients, such as increased pulmonary vascular resistance (PVR), increased blood flow (due to septal defects), and a decrease in wall distensibility.[1] This is in contrast to the normal pulmonary circuit, which is characterized by compliant artery walls and a low PVR, resulting in a low MPAP with little flow and pressure wave reflection. Since hemodynamic factors such as flow pulse waveform and wall shear stress (WSS) are known pathophysiological stimuli in the production of molecules that alter vascular tone and matrix properties,[2] we set out to quantify these values in the proximal left, right and main pulmonary arteries (LPA, RPA, & MPA) of control and PAH patients using phase-contrast magnetic resonance imaging (PC-MRI).

Materials and methods: Retrospective analysis was performed on cardiac PC-MRI images of the first and second generation pulmonary arteries of normotensive and PAH patients for this preliminary investigation. A fast low-angle shot (FLASH) gradient echo sequence was used to obtain retrospectively gated tissue intensity and through-plane phase velocity maps orthogonal to the longitudinal axis of the MPA, LPA, and RPA (Siemens Magnetom Avanto). The arteries were temporally segmented using clinically available flow software (ARGUS, Siemens Medical) and exported to a custom Matlab program developed for this study (Mathworks, Inc). Velocity mapping, flow quantification, bulk motion correction, and temporal/spatial axial WSS calculations were then computed (viscosity was assumed constant at .032 Poise).

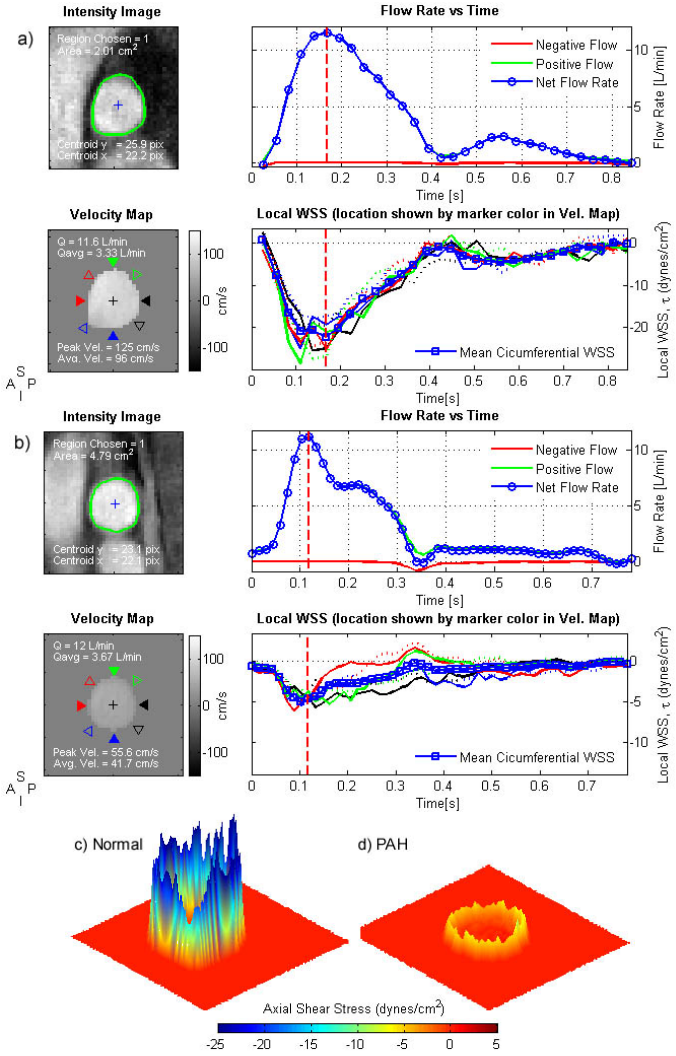

Figure 1. a) Normotensive and, b) PAH patient analysis of the RPA showing flow and shear waveforms. c-d) Patient WSS at systole.

Results and Discussion: The RPA tissue magnitude, velocity map, flow and WSS waveform for a normal 17 y/o male patient over the cardiac cycle are shown in Figure 1a. The flow/shear waveform shape and magnitude shown in this figure are representative of the LPA, RPA, and MPA of the normotensive patients measured thus far. The smooth dome shape contour of the flow waveform matches the characteristic shape of a healthy distal pulmonary vasculature with little wave reflection. The local and circumferential mean of the axial WSS over the cardiac cycle displays a symmetric distribution of WSS with a peak magnitude of 28.4 dynes/cm^2. These characteristics differed from the similarly aged 17 y/o male PAH patient RPA shown in Figure 1b. As with other PAH patients examined, the flow waveform demonstrates a mid-systolic notch attributed to wave reflection in partially obstructed distal arteries. In addition, the local and spatial average of the axial WSS was markedly lower in value when compared to the normotensive patients, with a peak magnitude of 6.1 dynes/cm^2, roughly 4.5 times lower than the measured normal value.

The drastic difference in WSS values between these two patient groups may be associated with the commonly observed occurrence of pulmonary artery dilation in PAH patients.[1] For the normotensive and PAH patient shown in Figure 1, the body surface area (BSA) normalized RPA sizes where measured at 1.1 cm^2/m^2 and 2.7 cm^2/m^2 - while the average flow rates where comparable at 3.3 L/min and 3.7 L/min, respectively. Yet the peak WSS magnitude in the normotensive patient was more than 4.5 times that of the PAH patient. This pattern has been observed in a number of patients in our study. The effect of reduced velocity gradients, and thus WSS, in the larger PAH arteries may prove an important effect on the cellular function of the proximal vasculature in PAH. Differences in WSS may also be related to age and the primary or secondary nature of the patient's PAH. Relatively older PAH patients, such as the example used in Figure 1b may be especially prone to dilation due to drug therapy intended to reduce PVR. We intend to further investigate these potential effects with additional patient groups ranging in age and pathology.

Conclusion: We have demonstrated the use PC-MRI to quantify hemodynamic WSS in PAH patients. Preliminary results indicate that WSS may be lower in PAH patients due to arterial dilatation. Degree of PAH (whether primary or secondary) and age may also pose a factor determining magnitude of WSS in the proximal arteries. Additional patients are being enrolled for further study.

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