A Direct Calculation of Hemodynamic Energy Loss in the Presence of Abnormal Aortic Flow

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Purpose: Aortic valve disease (AVD) in the form of stenosis, insufficiency, or congenital defect will disrupt normal function beyond the valve itself. This includes an increase in cardiac afterload and a drastic alteration in post-valvular 3D blood flow patterns[1, 2]. The current AHA/ACC standard-of-care guidelines, however, assess disease severity based on simplified measurements local to the valve, such as: peak velocity, effective orifice area, regurgitation, aortic diameter and transvalvular pressure gradient[3]. Paradoxically, it is known that similarly classified AVD patients under these guideline metrics can exhibit radically divergent outcomes - implying an incomplete characterization of the disease[4]. For this reason, functional assessment and risk-stratification may benefit from a robust methodology capable of quantifying the energetic load placed on the left ventricle (LV) due to the presence of AVD. The measurement of viscous energy loss, a parameter which is directly responsible for increased cardiac afterload and is independent of pressure recovery effects, is a promising candidate to quantify LV loading. With this in mind, the 4D flow technique (time-resolved 3D phase-contrast MRI with all principal velocity directions encoded) provides the necessary information to calculate this parameter. Therefore, we present a theoretical basis for the use of 4D flow MRI to characterize *in-vivo* energy loss and apply the technique in a pilot study of patients with aortic valve stenosis or aortic dilation (n=16) as compared to normal controls (n=10).

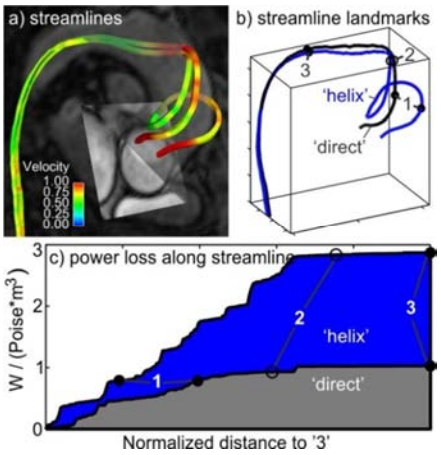

Fig 1. (a-b) *Effect of two aortic flow paths on* **(c)** *cumulative energy loss in an aortic valve stenosis patient. The 'helix' feature demonstrates greater energy lost along landmarks 1-3 compared to a particle traveling along the 'direct' path to point 3.*

Methods Theory: Viscous energy loss manifests in the form of viscous dissipation. The dissipation term, Φ_v, can be calculated from an energy reformulation of the viscous stress divergence in the incompressible Navier-Stokes equations. Knowledge of the velocity field (v_i), allows for the computation of Φ_v, i.e.:

$$\Phi_v = 2\left[\left(\frac{\partial v_x}{\partial x}\right)^2+\left(\frac{\partial v_y}{\partial y}\right)^2+\left(\frac{\partial v_z}{\partial z}\right)^2\right]+\left[\frac{\partial v_y}{\partial x}+\frac{\partial v_x}{\partial y}\right]^2+\left[\frac{\partial v_z}{\partial y}+\frac{\partial v_y}{\partial z}\right]^2+\left[\frac{\partial v_x}{\partial z}+\frac{\partial v_z}{\partial x}\right]^2-\frac{2}{3}\left[\frac{\partial v_x}{\partial x}+\frac{\partial v_y}{\partial y}+\frac{\partial v_z}{\partial z}\right]^2 \quad (1)$$

As a result, the rate of net energy lost in a volume of interest at a given moment in the cardiac cycle is $\dot{E}_{loss} = \mu \sum_{i=1}^{num\ voxels} \Phi_v V_i$, (2) where μ is the dynamic viscosity of blood (assumed Newtonian, μ=3.2 cP, V_i is the voxel volume)[5]. Imaging: 4D flow MRI velocity fields were obtained at 1.5T (Espree, Avanto, Siemens AG, Germany) in a sagittal oblique 3D volume covering the thoracic aorta using prospective ECG gating and a respiratory navigator placed on the lung-liver interface. Pulse sequence parameters were as follows: TE/TR=2.3–3.4/4.8–6.6 ms, flip angle α=7–15° and temporal resolution=38.4–52.5 ms; the field of view was 340–400×200–300 mm, with a voxel size of 1.8–2.1×1.8–2.1×2.0–2.8 mm^3. Velocity encoding was adjusted to minimize velocity aliasing (1.5-3.0 m/s). Patient Enrollment: AVD is often concomitant with aortic dilatation, thus it was necessary to control for aortic size using a dilated aorta cohort, which was defined as a mid-ascending aorta (MAA) diameter > 4cm. Therefore, 3 subject groups were retrospectively evaluated with IRB approval: Group A) healthy volunteers with normal aortic valve function and thoracic aorta geometry (n=10, 37±9 years, MAA=3.2±0.3); Group B) patients with normal aortic valve function and a dilated MAA (n=10, 54±8 years, MAA=4.3±0.3 cm); Group C) patients with mild/moderate aortic stenosis (AS) (n=6, 47±12 years, MAA=4.5±0.6 cm). Data Analysis: After eddy current correction, a level-set 3D segmentation of the thoracic aorta (vmtk, Orobix) was produced for each subject at flow systole, which was defined as peak flow. The aortic segmentation was then used to mask the velocity field and systolic energy loss was calculated using eqn. 1 and 2. Intergroup comparisons of mean values were analyzed with a Wilcoxon rank-sum test.

Results and Discussion: Both the dilated aorta cohort and AS cohort had abnormal helical and vortical flow (as observed with systolic streamlines emitted from the aortic valve plane). Fig. 1a-c illustrates the higher energetic cost of abnormal flow in a stenosis patient, as calculated for a massless particle traveling along a helix, compared to a direct route to the same vascular landmark. Fig 2 shows how these abnormal flow features, such as prominent systolic flow jets impinging on the aortic wall (Fig. 2a, see '**'), result in a statistically elevated $\dot{E}_{loss}$ for dilated (0.08±0.02W, p=0.011) and AS patients (0.81±0.42W, p<0.001), compared to healthy volunteers (0.05±0.02W). Additionally, local regions exhibiting flow separation (Fig 2a, '*'), high velocity flow jets (Fig 2a, '**') were co-located with elevated viscous dissipation. The energy loss results demonstrate the sensitivity of viscous dissipation to detect differences between patient cohorts. The markedly elevated AS energy loss values agree well with large pressure gradients known to be present in stenotic patients. However, the measurement of pressure gradients (i.e. potential energy loss) is often confounded by a recoverable energy exchange with kinetic energy, and is often observed as pressure recovery distal to AS. Thus, pressure recovery can reduce the utility of the pressure gradient as a risk stratification metric (as was recently documented when examining AS severity misclassification)[6]. These challenges highlight certain advantages of this technique, i.e.: 1) invasive pressure measurements are avoided, 2) the technique is not susceptible to pressure recovery, 3) regions of permanent energy loss are easily visualized. A potential disadvantage is error introduced by variability in the volumetric segmentation. For this reason, intra-observer reliability was assessed and showed an acceptable error of 6% when calculating $\dot{E}_{loss}$.

Conclusion: Increased aortic energy loss measured in the patient populations indicates that the LV must work harder to overcome inefficiencies introduced by abnormal aortic flow. Measuring energy loss may prove useful for risk stratification and functional assessment of aortic dilation, AVD, and possibly as a metric for interventional success. Longitudinal data collection is ongoing to understand the implications of viscous energy loss to clinical outcome.

References: **[1]** Barker, AJ, *et al.* Circulation. Cardiovascular imaging, 2012. 5(4): p. 457-66. **[2]** Briand, M, *et al.* J Am Coll Cardiol, 2005. 46(2): p. 291-8. **[3]** Bonow, RO, *et al.* Circulation, 2008. 118(15): p. e523-661. **[4]** Minners, J, *et al.* Heart, 2010. 96(18): p. 1463-8. **[5]** Venkatachari, AK, *et al.* Magn Reson Imaging, 2007. 25(1): p. 101-9. **[6]** Bahlmann, E, *et al.* Jacc-Cardiovascular Imaging, 2010. 3(6): p. 555-562.

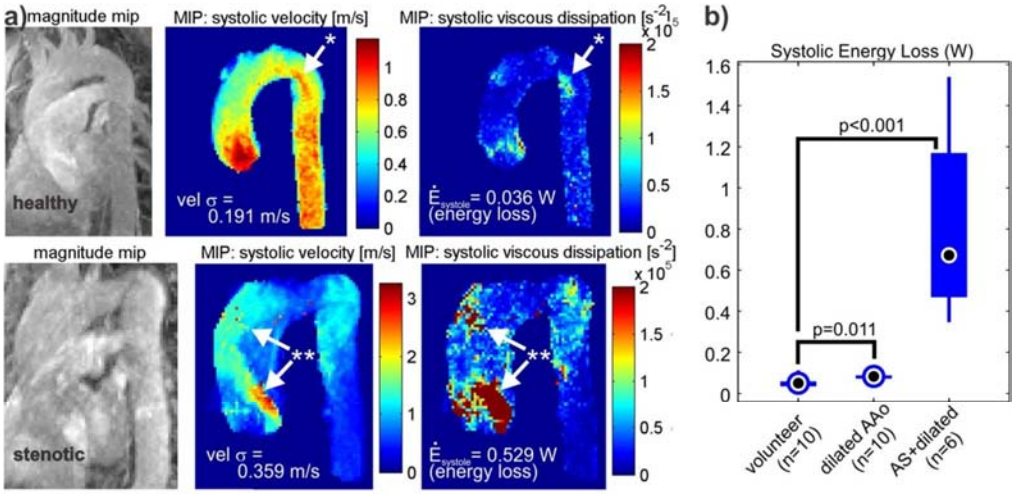

Fig 2. (a) *Sagittal 3D MIP projections of the intensity magnitude, systolic velocity field, and systolic viscous dissipation in the thoracic aorta of a healthy control (top) and a stenotic aneurysm patient (bottom).***(b)** *Systolic energy loss in volunteer, dilated aorta, and the stenotic, dilated aorta cohorts.*