Blood Flow in the Healthy Aorta: Turbulent or not?

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Introduction: Turbulence and velocity fluctuations of the blood flow are believed to play a role in hemolysis, platelet activation and thrombus formation (2). Previous invasive studies have already assessed turbulence in-vivo based on catheter hot-film anemometry or perivascular Doppler ultrasound in animals (2-3) and in humans (4,5). Although velocities fluctuation has been shown to result in MR signal loss (6), it is nevertheless possible to measure the bulk velocity in presence of turbulence using MRI (7) and the effect may additionally be used to assess the turbulent kinetic energy with MRI (8). The Reynolds number has long been used to define critical values for turbulence. Nerem et al. (3) defined in-vivo a critical peak Reynolds number proportional to the Womersley number. More recently, those results were amended by a systematic analysis of the influence of Reynolds, Womersley and Strouhal numbers on the development of turbulence in-vitro for physiologically realistic pulsatile flow (9). To assess turbulence in the healthy aorta, this work investigates the spatial distribution of in-vivo Reynolds, Womersley & Strouhal numbers based on phase-contrast MRI.

Methods: 2D CINE phase-contrast (PC) MRI with 3D velocity encoding was performed in 30 healthy volunteers (age range = 20-36 years, 9 females) and 8 analysis planes along the aorta (Fig.1, right). The resolution was 1.24×1.82×1.25-1.82×5mm³ spatially and 24.4ms temporally. The data was segmented using a quantification tool based on B-spline interpolation and Green’s theorem for quantification of flow and wall parameters (10). The dimensionless numbers of Reynolds (Re), Womersley (αt) and Strouhal (St) were subsequently calculated based on the PC-MRI data and the heart rate (see Table 1). Based on the work of Peacock et al. (6), the critical peak Reynolds number, indicating the transition to turbulence, was derived:

\[ Re_{\text{peak}} = 169 \alpha t^{0.3} St^{-0.27} \]

Results: The time-resolved and critical peak Reynolds numbers are shown in Fig. 1. Table 2 provides the Womersley and Strouhal numbers as well as mean, peak and critical peak Reynolds numbers. The measured peak Reynolds numbers were used in conjunction with the calculated critical peak Reynolds numbers (9) to define turbulence and turbulence-free regimes (Fig. 2).

Discussion: Assessing turbulence in-vivo and non-invasively remains difficult. This study is limited by the Reynolds number itself that was originally defined for laminar flow in a rigid pipe and not for pulsatile blood flow in compliant arteries. In addition, the calculation of the Reynolds number depends on the blood viscosity and density that may be source of errors. Finally, the presence of turbulence itself may limit the quality of the MR-measurements by introducing signal losses and artifacts (6-8). Nevertheless, our measurements are in accordance with physiological ranges (9). According to our measurements and the model by Peacock et al. (9), even in normal non-pathological young volunteers onset of turbulence are present in the ascending aorta (planes 1,2,3) and in the descending aorta (plane 8). No significant turbulence patterns in the aortic arch were found (planes 4,5,6). The data of 30 young volunteers presented here provide a reference standard for further studies of age or disease related changes in turbulence. The investigation of changes in the level of turbulence and their correlation with the location and severity of cardiovascular disease may help to evaluate the role of turbulence in the development of aortic pathologies.


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**Table 1: Dimensionless numbers for pulsatile flow**

<table>
<thead>
<tr>
<th>Reynolds number</th>
<th>Womersley number</th>
<th>Strouhal number</th>
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<tbody>
<tr>
<td>Inertial forces / viscous forces</td>
<td>Unsteady forces / viscous forces</td>
<td>Dimensionless stroke volume</td>
</tr>
<tr>
<td>( Re(t) = \frac{Dv(t)D(t)}{\mu} )</td>
<td>( \alpha = \frac{Dw}{2} \sqrt{\frac{\rho - 2\pi f}{D} m} )</td>
<td>( St = \frac{Dw}{2} \sqrt{\frac{f}{v_p - v_s}} )</td>
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</table>

with: Density: \( \rho = 1055 \text{ kg/m}^3 \); Dyn. viscosity: \( \mu = 4.5 \cdot 10^{-3} \text{ cp} \) (1); Mean cross-sectional velocity: \( v(t) \); Diameter: \( D(t) \); \( Dw = \text{mean} \left( D(t) \right) \); \( v_p = \text{max} \left( v(t) \right) \); \( v_s = \text{mean} \left( v(t) \right) \); Heart rate: \( f \); and: \( Re_{\text{max}} = \text{max} \left( Re(t) \right) \); \( Re_{\text{peak}} = \max \left( Re(t) \right) \).

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**Fig. 1:** Left: Time-resolved and critical peak Reynolds number in 30 volunteers at 4 analysis planes: Average (continuous lines) and standard deviation (error bars & dashed line). Right: position of analysis planes.

**Fig. 2:** Boxplot of the difference between the measured peak Reynolds number (Re_peak) and the calculated critical peak Reynolds number (Re'_peak). The median (red line), lower/upper quartiles (blue box), most extreme values within 1.5 interquartile range (dashed lines) and the outliers (red points) are shown over the 30 volunteers and for each analysis plane defined in Fig. 1.

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