3D Cine Phase-Contrast MRI of Flow Patterns and Turbulent Kinetic Energy in Patient-Specific Models of Carotid Disease under In Vivo Mimicking Flow Conditions

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INTRODUCTION: Lumen narrowing plaques in atherosclerotic carotid disease often give rise to disturbed or turbulent blood flow, which in itself may be a causative factor in the disease progression [1]. 3D cine phase-contrast (PC) MRI enables comprehensive investigations of flow, but is hampered by long scan times when high resolution is needed [2]. We sought to assess the use of high-resolution PC-MRI for the assessment of flow in patient-specific models of atherosclerotic carotid disease under *in vivo* mimicking flow conditions.

METHODS: Two patient-specific carotid models, manufactured based on *ex vivo* MR angiography scans of excised carotid plaques [3], were used. Model #1 had a severe stenosis (>70%) in the internal carotid artery (ICA) and dilatation proximal to the stenosis. An oblong moderate-to-severe stenosis was present in the external carotid artery (ECA). Model #2 had a moderate-to-severe stenosis (70%) in the proximal ICA and narrowing of the common carotid artery (CCA) in the vicinity of the flow divider.

Table 1 – 3D cine PC parameter settings.

Spatial resolution	0.96x0.96x0.96 mm^3
VENC	100 cm/s
TR/TE	7.3/4.5 ms
Pixel bandwidth	237 Hz
Temporal resolution	58 ms
Signal averages	3

Pulsatile flow conditions mimicking those previously measured in these carotids *in vivo* were generated by a custom flow loop driven by a computer-controlled gear pump. 3D cine PC data were acquired on a clinical 1.5T scanner (Philips Achieva) (see table 1). Scan time was 45 minutes per model. Velocity maps were reconstructed by conventional phase-subtraction. Maps of turbulent kinetic energy (TKE) were reconstructed from the magnitude data using methods described previously [4]; TKE is a scalar measure of turbulence intensity permitting quantitative investigations of post-stenotic flow turbulence. Plots of average flow rate and total TKE were generated for the post-stenotic ICA in both models, as specified in Fig. 1. Flow patterns were studied by pathline analysis and the spatiotemporal distribution of elevated turbulence intensity was assessed based on TKE isosurfaces.

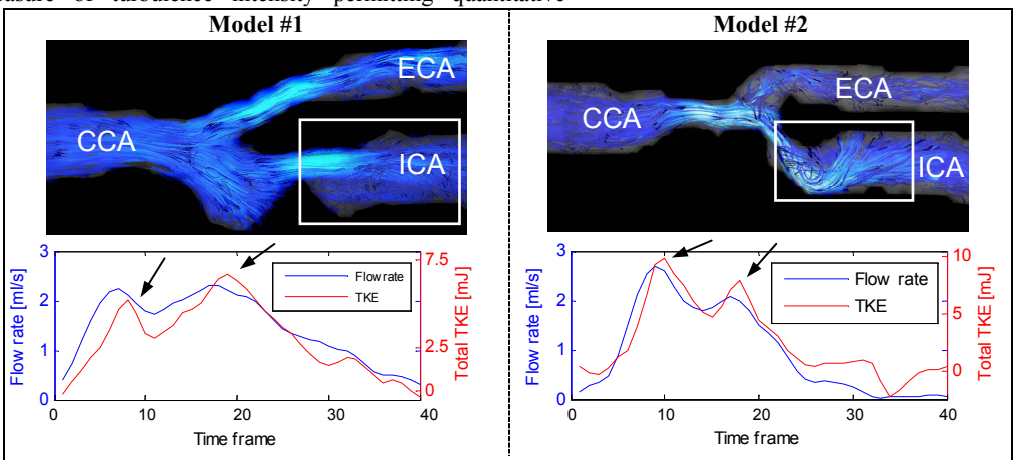

Figure 1. Isosurfaces of the geometries of the patient-specific models (grayscale) combined with pathline visualizations (blue) of the flow around peak systole (Upper row). The plots show the total ICA TKE within the marked rectangles and the ICA flow rate (lower row). Note that both the early and late TKE peaks occur later in time than the flow rate maxima (arrows).

RESULTS: Data demonstrated overall high quality, as determined by observers with several years of experience with 3D cine PC. The temporal evolution of TKE in the post-stenotic ICAs resembled that of the ICA flow rates; however, the TKE peaks (arrows) occurred later in time than the flow rate maxima (Fig. 1). The visualization of pathlines, color-coded by speed (brighter blue = higher velocities), drew our attention to several interesting flow features. In model #1, the most prominent flow feature was present in the dilated ICA sinus, which was filled by rotating flow throughout the cardiac cycle. Fluid from this ICA region flowed retrograde to the ECA (Fig. 2). TKE values up to 100 J/m^3 were measured downstream from the ICA stenosis. In model #2, complex retrograde flow was detected in the post-stenotic region of the ICA (Fig 3a). Peak TKE of about 225 J/m^3 was present at the outer wall of the post-stenotic ICA sinus (Fig. 3b).

DISCUSSION: This preliminary study demonstrated the potential of using 3D cine PC-MRI in patient-specific models of atherosclerotic carotid disease to provide detailed information of flow patterns and the spatiotemporal dynamics of turbulence intensity. Without the scan time restrictions associated with *in vivo* studies, data can be acquired with high resolution and SNR. In this way, potential transarterial pathways for emboli and intrastenotic flow can be studied in detail (Figs. 2&3), which is difficult *in vivo* [5]. The value of simultaneously obtaining TKE data is indicated in Fig. 1 where a temporal offset is seen between flow rate and TKE in the post-stenotic ICAs. This is consistent with fluid dynamical theory on the stabilizing and de-stabilizing effects of acceleration and deceleration, respectively, and warrants further non-invasive MRI studies of the dynamics of turbulence in post-stenotic cardiovascular flows.

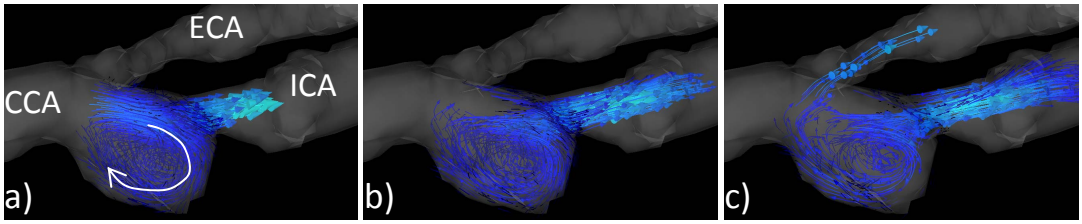

Figure 2. Retrograde flow in patient-specific model #1. Pathlines emitted from the proximal ICA sinus at systole and traced for a) 15 ms, b) 40 ms, and c) 80 ms reveal retrograde flow from the ICA to the ECA. This flow feature was persistent over the majority of the cardiac cycle.

REFERENCES: [1] Wakhloo AK, *et al*. J Vasc Interv Radiol 2004;15:S111-21. [2] Gatehouse P, et al. Eur Radiol 2005;15:2172-84. [3] Bale-Glickman J, *et al*. J Biomech Eng 2003;125:38-48. [4] Dyverfeldt P, *et al*. MRM 2006;56:850-58. [5] Harloff A, *et al*. MRM 2009;61:65-74.

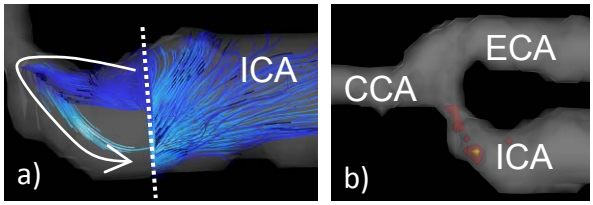

Figure 3. a) Pathlines emitted from a plane (dotted line) distal to the ICA stenosis in patient-specific model #2 and traced for 100 ms over systole reveal retrograde flow in the stenotic proximal ICA. b) Isosurfaces of TKE (red-yellow) at systole. Peak TKE was 225 J/m^3 (yellow).