

A Comparison Between Patient Specific Blood Flow and One Dimensional Wave Forms

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ABSTRACT

At present, modelling blood flow in the extensive network of arteries cannot be achieved in 3D. However, 1D models are able to capture the important features of arterial haemodynamics [1]. An assumption of 1D blood flow models is that vessels have a circular cross-section and that radial velocity profiles have a simple shape (e.g. flat or parabolic). However, real vessels have complex geometries and flow dynamics, especially near vessel branching points. The carotid bifurcation is one branch that has commonly been studied in 3D, initially using idealised geometries [2], and more recently using *patient-specific* geometries obtained from CT or MRI scans [3]. The aim of this study was to compare waveforms from 1D and 3D patient-specific models of the normal carotid bifurcation.

3D Model: The 3D geometry was extracted from a set of anonymous computed tomography (CT) images (Singleton Hospital, Swansea, UK). Simulations were performed using an in-house computational fluid dynamics solver, which uses the Characteristic Base Split (CBS) method [4] to solve the transient incompressible Navier-Stokes equations in 3D (vessel walls assumed rigid). A fully-developed unsteady (Womersley) velocity profile (obtained from *in vivo* data [5]) was imposed at the boundary of the common carotid artery and was discretised into 50 time steps. To investigate mesh convergence, the number of elements (N_e) was approximately doubled until little change occurred (final $N_e = 35,863$).

1D Model: A full description of the 1D model can be found in [1]. The governing equations in 1D can be expressed as:

$$\frac{\partial \mathbf{U}}{\partial t} + \frac{\partial \mathbf{F}}{\partial x} = \mathbf{S}, \quad \mathbf{U} = \begin{bmatrix} A \\ u \end{bmatrix}, \quad \mathbf{F} = \begin{bmatrix} uA \\ \frac{u^2}{2} + \frac{p}{\rho} \end{bmatrix}, \quad \mathbf{S} = \begin{bmatrix} 0 \\ -8\pi\mu \frac{u}{A} \end{bmatrix} \quad (1)$$

where c is wave speed and μ is blood viscosity and the independent variables are A (cross-sectional area), u (axial velocity), and p (pressure). p is related to A via $p = p_{ext} + \beta (\sqrt{A} - \sqrt{A_0})$, where external pressure p_{ext} is assumed zero, A_0 is the unstressed cross-sectional area and β is the wall stiffness parameter. The branches of the carotid bifurcation were modelled as truncated cones such that the cross-sectional areas at the ends closely matched the 3D model. While vessels walls in the 1D model are elastic, a very high value of β was used for comparison with the 3D model's rigid walls. The input

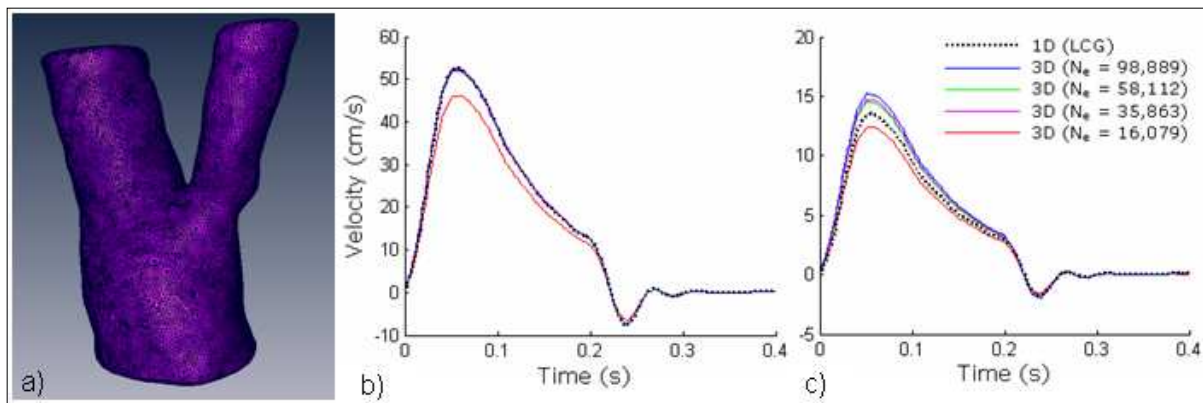


Figure 1: a) 3D patient-specific carotid bifurcation geometry. b) Internal and c) external carotid artery outlet mean velocities, showing the convergence of the 3D model as mesh density is increased, compared with the 1D model.

velocity waveform was equal to the spatial average of the 2D velocity profile. At the outlets of both the 1D and 3D models, a zero-pressure boundary condition was enforced. The bifurcation was considered to be reflectionless in the forward direction. The governing equations were solved using the Locally Conservative Galerkin method [1,7].

Results: Figure 1 compares the 1D and 3D models using the (mean) velocity waveforms at the two outlets. Given the assumptions in the 1D model, the agreement between the two models is striking. This suggests that 1D modelling is generally adequate for representing flow in normal branching vessels. It may be noted that the amplitude of the 1D velocity waveform for the external carotid branch (Figure 1c) does not quite match that of the waveform from the 3D model, whereas little difference is seen for the internal branch. One possible explanation for this is that the internal carotid is relatively cylindrical while the external carotid has a much less uniform geometry, particularly just after the branching point. This suggests that 1D modelling may not produce accurate results for highly irregular geometries, such as are seen in aneurysm and stenosis.

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