Medical Engineering & Physics 36 (2014) 275–284

Contents lists available at ScienceDirect

Medical Engineering & Physics

journal homepage: www.elsevier.com/locate/medengphy

Development of a patient-specific simulation tool to analyse aortic dissections: Assessment of mixed patient-specific flow and pressure boundary conditions

Mona Alimohammadi[a], Obiekezie Agu[b], Stavroula Balabani[a], Vanessa Díaz-Zuccarini[a,*]

[a] Department of Mechanical Engineering, University College London, WC1E 7JE, UK
[b] University College London Hospital, NW1 2BU, UK

ARTICLE INFO

Article history:
Received 5 February 2013
Received in revised form 10 October 2013
Accepted 6 November 2013

Keywords:
CFD
Patient-specific simulation
Aortic dissection
Windkessel model
Blood flow

ABSTRACT

Aortic dissection has high morbidity and mortality rates and guidelines regarding surgical intervention are not clearly defined. The treatment of aortic dissection varies with each patient and detailed knowledge of haemodynamic and mechanical forces would be advantageous in the process of choosing a course of treatment. In this study, a patient-specific dissected aorta geometry is constructed from computed tomography scans. Dynamic boundary conditions are implemented by coupling a three element Windkessel model to the 3D domain at each outlet, in order to capture the essential behaviour of the downstream vasculature. The Windkessel model parameters are defined based on clinical data. The predicted minimum and maximum pressures are close to those measured invasively. Malperfusion is indicated and complex flow patterns are observed. Pressure, flow and wall shear stress distributions are analysed. The methodology presented here provides insight into the haemodynamics in a patient-specific dissected aorta and represents a development towards the use of CFD simulations as a diagnostic tool for aortic dissection.

1. Introduction

Thoracic aortic diseases occur at a rate of approximately 9–16 cases per million annually in Europe and have a high mortality risk [1]. Aortic diseases arise from a combination of morphological factors, haemodynamic forces and mechanical properties of the tissue [2]. An aortic dissection (AD) is initiated when a cleavage forms in the intimal layer of the vessel wall. Blood flows between the media and adventitia, forming a false lumen (FL), and the force exerted by the extra-luminal blood causes the FL to propagate both axially and longitudinally [3]. Aortic dissections have a high mortality rate, at around 74% within the first two weeks following initial onset of the disease. Furthermore, the mortality rate increases by 1–2% per hour after symptoms become manifest [4]. AD is particularly prevalent in people with conditions such as hypertension, Marfan syndrome, Ehler Danlos syndrome and bicuspid aortic valve, and the likelihood of having AD appears to increase with age [5].

In type-B dissections (involving only the thoracic aorta), it is often possible to stabilise the patient with pharmacological therapy, but surgical intervention may be required in severe cases [4]. Chronic AD is commonly treated with open surgery, whilst endovascular approaches are used in acute cases [6]. Although expert consensus may provide some guidelines [7], no strict rules on whether or not to intervene are available. This is partly due to the fact that the mechanical and haemodynamic characteristics of AD are not fully understood [2]. Such an understanding could aid in predicting the progression of the disease and assist clinicians. Advances in computational and imaging techniques have shown great potential in understanding cardiovascular diseases [8–10].

One of the key issues in three-dimensional (3D) computational simulations for clinical purposes is to properly describe the boundary conditions (BCs) of the problem, as the simulation results are very sensitive to the choice of BCs. This is particularly important in the study of AD, wherein pathological flow reduction through one or more of the outlets is likely, as the distribution of flow is dependent on the BCs employed.

Various methodologies for defining BCs have been utilised, such as applying constant pressure [11,12] or specifying the flow passing through each of the domain boundaries at each time instance [13,14]. However, the pressure at each boundary in the real system changes throughout the cardiac cycle, as does the flow distribution. Another approach is to use measured flow and pressure waves reported in the literature [15–17], but in this case the results are not ideal for patient-specific studies. Coupling CFD simulations with zero-dimensional (0D) or Windkessel models provides an alternative methodology which allows for interdependent time-varying

* Corresponding author. Tel.: +44 2076793928.
E-mail address: v.diaz@ucl.ac.uk (V. Díaz-Zuccarini).

1350-4533/$ – see front matter © 2013 IPEM. Published by Elsevier Ltd. All rights reserved.
http://dx.doi.org/10.1016/j.medengphy.2013.11.003

flow and pressure distributions, which are more appropriate in the case of patient-specific simulations [18–23]. When coupling 3D and 0D domains, the problem of defective boundary conditions (as a result of a reduction in the parameter space, e.g. cross sectional area) must be addressed appropriately [24,25].

In this approach, Windkessel parameters need to be estimated for each domain outlet. There is no agreed methodology for parameter definition in RCR systems [26] with detailed descriptions of the methods used being surprisingly sparse. However, a large body of work has been devoted to fitting computational parameters to experimental data [27–29]. Notably, Bertoglio et al. [30] used a Kalman filter approach to define peripheral resistance for an idealised abdominal aortic aneurysm.

A number of studies have reported the results of thoracic repair using endografts [31,32]; however, few studies have reported on AD in patient-specific geometries in preoperative conditions [10,11,14,16]. To the best of our knowledge, Windkessel boundary conditions have not been applied previously to patient-specific ADs. This approach improves the accuracy of the simulation and is important for AD, as both the reduction of flow to limbs or organs (malperfusion) and regions of high pressure present serious risks for the patient.

In this paper, numerical simulations of the flow through a patient-specific type-B AD were carried out by coupling the 3D flow domain with 3 element Windkessel models at each boundary. The Windkessel parameters were tuned using invasive pressure measurements.

This study, initiated by the vascular service unit in University College Hospital, informs the early stages of a research effort to develop reliable, computationally efficient predictive tools that could aid clinicians to understand and manage the complex and patient-specific nature of AD in the future.

2. Methods

2.1. Geometry

The 3D geometry was reconstructed from 437 CT slices of a female patient with a Stanford type-B aortic dissection. The morphological information was extracted using the ScanIP (Simpleware Ltd., UK) image processing package. Different threshold and flood fill segmentation tools were used to create multiple masks to select the dissected aorta and its main branches.

The resolution of the scans was 0.7 mm/pixel, and the intimal flap between the two tears was generally 2–3 mm thick, hence it was possible to resolve the characteristics of the dissection. Smoothing operations were used to remove any pixelisation artefacts and dilation was used to counteract volume shrinkage due to smoothing.

Boundaries were cropped to provide a flat surface at each boundary. Fig. 1a shows the resulting model. Blood enters the ascending aorta (AA) into the aortic arch. The patient has a 'bovine aortic arch', i.e. the BT and LCC share a common trunk origin [33]. In the LS a large aneurysm (diameter approximately 30 mm) can be seen. At the distal arch, a coarctation is present as a result of the proximal expansion of the FL. The FL extends upwards from the entry tear and downwards from the re-entry tear such that it is present throughout the entire thoracic aorta and the luminal area of TL is reduced by more than 50% due to the dissection.

2.2. Computational fluid dynamics

Blood was considered to be an incompressible, Newtonian fluid with a density of 1056 kg m^{-3} and dynamic viscosity of 3.5 mPa s. The shear rate in the large arteries is sufficiently high to assume Newtonian behaviour [34] and the flow is considered to be laminar, which has been observed in both the FL and TL in a previous study of AD [35]. The mean Reynolds number was 800 and the Womersley number in the ascending aorta was approximately 1.9.

2.3. Boundary conditions

The inlet flow wave was not available for this patient, so flow from a dissected aorta in a previous study was utilised [36] and interpolated to the heart rate of the patient (70 beats per minute). The vessel wall was assumed to be rigid, and a no-slip condition was applied at the wall. Outlets were defined using Windkessel models (see Fig. 1), which use electrical analogues to describe hydraulic systems; pressure (P) and flow (Q) are analogous to voltage and current respectively. Solving the equation for the circuit shown in the inset of Fig. 1a yields:

$$P = (R_1 + R_2)\,Q - R_2 C \frac{dP}{dt} + R_1 R_2 \frac{dQ}{dt} \tag{1}$$

The derivative terms were approximated using the backward Euler method, for a time step of $\Delta t = 2.5$ ms, giving:

$$P_{n+1} = \frac{\left(R_1 + R_2 + R_1\beta\right) Q_{n+1} - R_1 \beta Q_n + \beta P_n}{1+\beta} \tag{2}$$

where $\beta = R_2 C/\Delta t$.

The Windkessel models were implemented via FORTRAN subroutines in ANSYS-CFX (ANSYS Inc., USA). For each internal solver loop, CFX passed the instantaneous flow rate to FORTRAN, calculated according to:

$$Q = \int_{A_i} \vec{u}.\vec{n}_i dA \tag{4}$$

where $\vec{u}$ is the velocity vector, $\vec{n}_i$ is the normal vector for interface i (constant for each interface) and A_i is the area of the interface. Hence, $Q_{3D,i} = Q_{0D,i}$ as required for the interface to be appropriate [9]. The instantaneous pressure, $P_{0D,i}(t)$ was calculated based on Eq. (2), using the pressure and flow from the previous time step, $P_{0D,i}(t-\Delta t)$ and $Q_{3D,i}(t-\Delta t)$ respectively, and the flow rate for the given solver loop, $Q_{3D,i}(t)$. The calculated pressure was passed back to CFX, and was applied as a uniform BC in the subsequent solver loop.

2.4. Data assimilation method

The Windkessel parameters (R_{1i}, R_{2i} and C_i) were tuned based on invasive pressure measurements. Informed consent and approval from the ethical committee were obtained by the managing clinician. All measurements were obtained from the standard clinical procedures undertaken by the clinical team. A transfemoral 5-French sized universal flush angiographic catheter (Cordis Corporation, USA) with a radio-opaque tip and multiple distal openings, was connected to a pressure transducer and anaesthetic monitor in order to capture pressure measurements, in the hybrid endovascular theatre at University College London Hospital (UCLH). Measurements were acquired in the distal arch, mid abdominal and each of the boundaries, with the exception of the LS, as it was deemed unsafe by the clinician to take measurements so close to the aneurysm. For the same reason, pressure measurements were not acquired in the FL. For each measurement location, a minimum of 5 cycles was averaged and the minimum and maximum pressures were recorded. It was not possible to record the complete pressure waves for the present patient, and hence only the minima and maxima of the pressure at each location were available for this investigation.

The measured maximum and minimum pressure at each location are shown in Table 1. The measured pressures are relatively