Understanding Arterial Pressure from PIV data

Patrick H. Geoghegan¹*, Paul D. Docherty¹, Mathieu Sellier¹, Mark C. Jermy¹
1: Dept. of Mechanical Engineering, University of Canterbury, New Zealand
* Correspondent author: patrick.geoghegan@canterbury.ac.nz

Keywords: Biomedical flow, Arterial Hemodynamics, Compliant, Pressure

ABSTRACT

Cardiovascular disease is a major cause of disease around the world and is estimated to cost developed countries roughly 2% of their GDP annually. Critically ill patients with hemodynamic dysfunction exhibit reduced stroke volume, compliance, and diffusion of oxygenated blood to the organs, which leads to organ failure. Current treatment techniques include inotrope therapy and fluid resuscitation which are treated with independently selected doses based on mean arterial pressure and heart rate. This lacks concordance amongst clinicians regarding the optimal dosing levels. If the spatial variance in the pressure and flow fields in hemodynamic dysfunction could be determined, the confidence intervals for model-based capture and titration of therapy could be developed. In this work stereoscopic particle image velocimetry is used to obtain the velocity fields in a simplified model of a stenosis (a phantom). This thin walled compliant phantom is manufactured so that the mechanical response is equivalent to in-vivo conditions. The experimental set-up imparts a pulsatile flow on the phantom ensuring in-vivo conditions by matching both Womersley and Reynolds number. The pressure fields are obtained via integration of the Navier-Stokes equation and correlated to the upstream pressure signal. By applying the Navier-Stokes equations to the velocity field data from the PIV analysis and in contrast to typical computational fluid dynamics (CFD) there was no need to calibrate and iterate between pressure and velocity domains to satisfy a series of boundary conditions. The transient pressure domain information was obtained by the simple determination of the partial derivatives of the velocity. The pressure values were stable, smooth, in the expected order of magnitude, and intuitively correct. The results show that a pressure difference across the stenosis can be observed, and that this is related to the severity of the stenosis. This provides a pathway into stenosis diagnosis using pressure measurement equipment currently used in medical practice, after some further analysis and investigation has been carried out.

1. Introduction

Cardiovascular Disease (CVD) caused 30% of all deaths in New Zealand in 2012 (Ministry of Health 2015). CVD has been estimated to cost ~2% of GDP in developed countries (Adeyi et al. 2007). Critically ill patients with hemodynamic dysfunction exhibit reduced stroke volume, compliance, and diffusion of oxygenated blood to the organs (Davies et al. 2007). This ultimately
leads to organ failure, and exacerbation of the disease state. Typical therapies for hemodynamic dysfunction in critical care include inotrope therapy (Bhattacharya et al. 2010) and fluid resuscitation (Durairaj and Schmidt 2008; Rivers et al. 2010). These therapies are currently titrated against the patient condition using independently selected doses based on indicators such as mean arterial pressure and heart rate. This lacks concordance amongst clinicians regarding the optimal dosing levels. It is possible that model-based analysis could be used to determine patient-specific optimal doses of inotropes and fluid resuscitation (Marik et al.; Hoff et al.). However, before model-based analysis can form the basis of decision support frameworks, patient-specific models must be calibrated to aortic pressure measurements that are taken as the standard of care in most ICU settings. To date, some preliminary hemodynamic modelling has been undertaken (Chase et al. 2010; Hann et al. 2010; Hann et al. 2011; Revie et al.; Sundaresan et al. 2010; Kamoi et al.). If the spatial variance in the pressure and flow fields in hemodynamic dysfunction could be determined, the confidence intervals for model-based capture and titration of therapy could be developed. Clinicians are able to interpret some elements of hemodynamic pressure signals to ascertain certain information about the hemodynamic dysfunction a particular patients has (Marik et al. 2011; Hoff et al. 2009; Mayer et al. 2010; Marik et al. 2009). However, the ambiguity of the location of the pressure sensor in the artery leads to variance in interpretation and confounds some efforts to standardize care. Pulse pressure and mean venous pressure are generally measured in the Intensive Care Unit (ICU), and may contain the information needed to locate and diagnose certain arterial geometry deformations.

A stenosis is a buildup of plaque and inflammation that alters the internal geometry of an artery. Stenoses generally reduce internal diameter of the artery and induce local high velocity and pressure differentials (Geoghegan et al. 2013; Pielhop et al. 2012; Kung et al. 2011; Geoghegan et al. 2009). The high velocity leads to higher wall shear stress. Wall shear stress has been noted as a major source of damaged for the endothelium structure. MRI scanning is often used to diagnose, measure and locate stenosed arteries. Since, arterial pressure signals are more accessible than MRI scans, they may offer a new tool to diagnose and locate stenosis.

This work will determine whether a simple linear pressure response model can locate a stenosis using only pressure signals. Pulsatile fluid flow through a compliant axisymmetric stenosis model was determined using particle image velocimetry (PIV). Pulsatile pressure fields were obtained using velocity field data via integration of the Navier-Stokes equation and correlated to the upstream pressure signal to observe whether the stenosis affected the pressure in a distinct manner from the healthy regions of the artery.
2. Experimental Methodology

The thin walled compliant flow phantom (Fig. 1) was produced by the method developed in Geoghegan et al. (2012) and was constructed at a scale of 3.2 times life size from silicone (Dow Corning Sylgard 184). The main properties of the phantom (with a symmetric stenosis of 50% by diameter) along with that of the common carotid artery (CCA) are given in Tab. 1. The phantom internal geometry and the region of interest (ROI) studied is also shown. The physiological flow wave was obtained from phase contrast magnetic resonance imaging (MRI) measurements of the time dependent in-vivo flow rate in the CCA of a healthy male volunteer (Buchmann and Jermy 2010) with maximum, minimum and mean Re of 939, 379 and 632, respectively and a Womersley number $\alpha = 4.54$ (Fig. 2).

The phantom was ported to a previously developed flow system (Geoghegan et al. 2013; Geoghegan et al. 2010). The working fluid was an aqueous glycerine solution of 39:61 ratio by weight, which provided a refractive index that matched the silicone phantom ($n=1.141$) (Geoghegan et al. 2012). The fluid had a kinematic viscosity ($\nu$) of $10.2 \times 10^{-6}$ m$^2$s$^{-1}$ and a density ($\rho$) of 1150 kgm$^{-3}$ at 20°C. The physiological flow wave was produced by means of a piston pump driven by a stepper motor attached to a ball screw. An in-house National Instruments LabVIEW code controlled the piston system with an electromagnetic flow meter (Krohne Optiflux 1300) providing a real-time feedback of flow rate to the CPU. A pressure transducer was located upstream to record the inlet pressure variation.

Fig. 1 (left) Silicone flow phantom representing the common carotid artery with symmetric stenosis. (right) Magnification of the flow phantom ROI with a 50% symmetric stenosis (Geoghegan et al. 2012)
Images were recorded using two Dantec Flowsense 2MP cameras with a CCD array of 1608×1208 pixels. The 60mm Nikon lenses had an aperture size set to f=8 and a magnification of 0.2 to 0.15. This gives an estimated depth of field of 3 to 4.9mm and a diffraction limited particle size of 12.5 to 11.9 µm. The flow was illuminated with a 40mm high, 0.5mm thick light sheet from a New Wave Solo 120 XT laser with a wavelength of 532nm at 120mJ/pulse using a series of spherical and cylindrical lenses. The flow was seeded with nominally 10 µm diameter hollow glass spheres with a density of 1.1 g/cm³. The PIV measurements were phase locked, recording 22 image pairs per waveform over 50 waveforms. For time oscillating flow waves the laser pulse time delay selection is a compromise due to the difference in maximum pixel displacement for maximum and minimum flow rate. For this analysis a time delay of 650 µs was selected, which produced <3% invalid vectors at each phase. After minimum background subtraction, intensity normalisation
and masking, the images were processed with an in-house code implementing 2D FFT cross-correlation (Buchmann and Jermy 2007) with iterative window sizing and displacement. The data was validated using the signal-to-mean ratio filter and normalised median test (Westerweel and Scarano 2005).

The internal pressure derivative fields were determined using simplified cylindrical Navier stokes equations assuming full rotational symmetry.

\[
\begin{align*}
\frac{\partial p}{\partial r} &= -\mu \left( \frac{1}{r} \frac{\partial}{\partial r} \left( r \frac{\partial u_r}{\partial r} \right) + \frac{\partial^2 u_r}{\partial z^2} - \frac{u_r}{r^2} \right) - \rho \left( \frac{\partial u_r}{\partial t} + u_r \frac{\partial u_r}{\partial r} + u_z \frac{\partial u_r}{\partial z} \right) \\
\frac{\partial p}{\partial \phi} &= 0 \\
\frac{\partial p}{\partial z} &= -\mu \left( \frac{1}{r} \frac{\partial}{\partial r} \left( r \frac{\partial u_z}{\partial r} \right) + \frac{\partial^2 u_z}{\partial z^2} \right) - \rho \left( \frac{\partial u_z}{\partial t} + u_r \frac{\partial u_z}{\partial r} + u_z \frac{\partial u_z}{\partial z} \right)
\end{align*}
\]

where \( p \) is the local pressure; \( r \) and \( z \) are the radial and axial directions, respectively; \( u_i \) is the velocity component in the \( i^{th} \) direction; \( \rho \) is the density; \( \mu \) is the dynamic viscosity.

The pressure at some location \( k \) in the field can thus be calculated by integrating the Navier Stokes equation with respect to the radial and axial directions:

\[
\begin{align*}
p(r_k, z_k) &= p(r_0, z_0) + \int_{r_0}^{r_k} \frac{\partial p}{\partial r} \, dr + \int_{z_0}^{z_k} \frac{\partial p}{\partial z} \, dz \\
p(r_k, z_k) &= p(r_0, z_0) + \int_{r_0}^{r_k} -\mu \left( \frac{1}{r} \frac{\partial}{\partial r} \left( r \frac{\partial u_r}{\partial r} \right) + \frac{\partial^2 u_r}{\partial z^2} - \frac{u_r}{r^2} \right) - \rho \left( \frac{\partial u_r}{\partial t} + u_r \frac{\partial u_r}{\partial r} + u_z \frac{\partial u_r}{\partial z} \right) \, dr + \ldots \\
&\quad + \int_{z_0}^{z_k} -\mu \left( \frac{1}{r} \frac{\partial}{\partial r} \left( r \frac{\partial u_z}{\partial r} \right) + \frac{\partial^2 u_z}{\partial z^2} \right) - \rho \left( \frac{\partial u_z}{\partial t} + u_r \frac{\partial u_z}{\partial r} + u_z \frac{\partial u_z}{\partial z} \right) \, dz
\end{align*}
\]

If \( p(r_0, z_0) \) is declared to be at the wall beside the inlet, the gauge pressure at this point can be determined by linking the local pressure to the distension of the elastic wall:

\[
p(r_0, z_0) = \frac{2r_0 - D_N}{D_N} 2EhD_N^{-1}
\]

where \( r_0 \) is the distended radius of the phantom at \( z=0 \), \( E \) is the Young’s modulus of the phantom wall, \( h \) is the wall thickness, \( D_e \) is the natural internal diameter of the unstretched phantom.
Analysis was performed using MATLAB (Mathworks, Natick, MA, United States of America). The derivatives were calculated using the function ‘gradient.m’. Integrals were calculated using the trapezium rule within the function ‘cumtrapz.m’. The trapezium rule effectively utilizes the noise mitigation effects of integrals to remove some of the sharp behaviors that occur due to the differentiation of noisy experimental data. Hence, there was no need to using velocity smoothing routines to mitigate the effect of noise on the outcome.

4. Results and Discussion

The instantaneous velocity fields and in-plane streamlines at selected points in the flow waveform through the stenosis throat is shown in Fig. 3. Between $-2 \leq x/D \leq -1$ there was an increase in centerline ($y/D = 0$) velocity due to the decrease in cross sectional area of the phantom geometry. As flow passes through the stenosis throat two distinct flow regimes were observed; a central high velocity jet, and a low velocity recirculation region either side of the jet. These two flow patterns were separated by a shear layer. The peak flow velocity (1.9 m/s) observed is 2.6 times larger than the 0.72 m/s previously observed in a straight (i.e. unstenedosed, healthy) geometry (Geoghegan et al. 2016).

The velocity fields were combined with the Navier-Stokes equations in their cylindrical form (Eq.1-3) to calculate the pressure profiles. The corresponding phase locations for the data in Fig. 3 are shown in Fig. 4. The pressure profiles are cyclic due to the cyclic experimental input that was calibrated to achieve a particular flow wave. The history of input pressure and pressure at selected
locations are shown in Fig. 5. p4 and p5 lie in the high velocity regions post-stenosis jet. These pressures are identical at all times i.e. there is no change in pressure over a distance of one diameter downstream from the stenosis throat. p1 and p2 lie in the low velocity regions near the wall, outside the jet, one upstream and one downstream of the stenosis. These pressure histories match each other, but for a slight difference in amplitude. The pressure history on the centerline one diameter upstream of the stenosis (p3) is very similar to that at p1 and p2, with a close match to p2.

In a complaint, straight, healthy artery, there will be no pressure variation axially, except for phase lag, which is minimal over the distances experienced in the arterial system. From the results shown here, this is not the case with a stenosed artery. With the use of a pressure transducer attached to a catheter, a stenosis could potentially be detected by pressure drop alone. The exact relationship between pressure difference and stenosis severity will require an extensive analysis of different artery shapes and stenosis to provide the sensitivity required for pressure drop.

![Fig. 4 Pressure (kPa) assuming radial symmetry at selected phase locations (Note the non-zero scale limit – Blue=−5kPa)](image)
By applying the Navier-Stokes equations to the velocity field data from the PIV analysis and in contrast to typical computational fluid dynamics (CFD) there was no need to calibrate and iterate between pressure and velocity domains to satisfy a series of boundary conditions. The transient pressure domain information was obtained by the simple determination of the partial derivatives of the velocity. The pressure values were stable, smooth, in the expected order of magnitude, and intuitively correct. This analysis used an experimental input flow rate that was indicative of the flow wave observed in the carotid artery (Geoghegan et al. 2013). However, to achieve this flow wave in the experimental setup, the input pressure wave did not closely match pressure curves observed in humans, thus indicating complex upstream flow-geometry behaviors were not replicated in this experimental setup. However, this experimental setup rendered an accurate flow profile at the stenosis, and thus achieved the original goals of the study.

Future validation should incorporate more physiological aortic structures so that the upstream pressure is transformed in a more physiological way when it reaches the stenosis. This work was performed on a straight axis-symmetric geometry, which is representative of many arterial geometries. However, other stenosis geometries can include curvature or asymmetry. For such geometries, the method would require a different implementation of the Navier-Stokes analysis.

5. Conclusions
This work obtained pressure fields from PIV experimental results by integrating the cylindrical form of the Navier-Stokes equations. The results show that a pressure difference across the stenosis can be observed, which provides a pathway into stenosis diagnosis using current medical techniques with further analysis and investigation. A regressive cross-correlation of the input-output signals will be performed to determine if pressure response through the stenosis can be used as a marker of stenosis location. Further validation in a linear geometry is required to ascertain the effect of stenosis severity and stenosis wall angle on the pressure field. If flow separation downstream of the stenosis does not occur there would be a different pressure distribution downstream of the stenosis affecting data analysis. The assumptions used in the cylindrical form of the Navier-Stokes equations applied here are only applicable in axis-symmetric geometries. Further development of the analysis is required to provide a pressure analysis of more complex geometries.

6. References


