Quantitative Hemodynamic Evaluation in Children with Coarctation of Aorta: Phase Contrast Cardiovascular MRI versus Computational Fluid Dynamics

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Abstract. Pressure gradient across coarctation of aorta (CoA) is conventionally computed from phase contrast magnetic resonance imaging (PC-MRI) by applying the Bernoulli equation to the peak blood flow velocity measurement obtained just distal to the aortic narrowing. In order to test the validity and accuracy of the Bernoulli flow assumptions of negligible viscous forces in assessment of pressure gradients across the coarctation, we sought to determine pressure information from patient-specific computational fluid dynamics (CFD) simulation, modeling Newtonian, viscous, incompressible blood flow under steady and pulsatile inflow conditions. The transient high velocity jet observed though a moderate thoracic aortic coarctation model (65% area reduction) reconstructed from magnetic resonance angiography scans of an 8-year old female patient provided for the 2012 STACOM CFD challenge, was studied over a cardiac cycle under patient-specific flow conditions. Descending aorta hemodynamics was contrasted with a geometrically and dynamically comparable normal aorta simulation. The peak velocity of the modeled CoA jet (6.99 m/s) was observed to occur ~2 cm distal to the site of coarctation. The magnitude of this velocity was found to be similar to appropriately dynamically scaled clinical observations (6.00±0.6 m/s) of peak velocity obtained from PC-MRI data on three pre-surgical CoA patients, evaluated at Children's Hospital of Pittsburgh. Bernoulli pressure gradient across the CoA computed using the CFD velocity field at the peak-systole instant of pulsatile flow grossly overestimated the true gradient predicted from CFD (30 mm Hg) when unsteady jet wake effects were more pronounced, but underestimated the CFD pressure gradient at steady time-averaged inflow conditions (5.8 mm Hg). Based on this pilot study, CFD determined flow fields are a more reliable clinical indicator of pressure gradient which considers viscous flow and complex jet wake interactions affecting hemodynamics downstream of CoA.

Keywords: Coarctation of Aorta, Computational Fluid Dynamics, Phase Contrast Magnetic Resonance Imaging, Pressure Gradient.

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1 Introduction

Coarctation of aorta (CoA), one of the most commonly encountered congenital cardiovascular diseases, is characterized by aortic narrowing, resulting in differential blood flow and systolic pressure gradient between the upper and lower extremities. Significant pressure-loss across the coarctation, turbulent jet flows [1], and hypertension in the proximal circulation characterize this condition, which could potentially result in major adverse cardiovascular events if left undetected. Therefore, CoA requires comprehensive qualitative and quantitative evaluation of pressure gradients, collateral flow, and velocity across the coarctation in order to assess its severity for timely disease management. Morphological evaluation of coarctation and functional assessment of collateral flow at various levels of the descending aorta (DAo) is routinely performed [2] with phase contrast magnetic resonance imaging (PC-MRI). Pressure gradient (in mm Hg) is computed from peak blood flow velocity measurements (in m/s) obtained just before and after the aortic narrowing, using the Bernoulli's equation. In order to test the validity and accuracy of the Bernoulli flow assumptions of negligible viscous forces in assessment of pressure gradients across the coarctation, given that turbulent jet flows are observed, we sought to determine pressure information from patient-specific computational fluid dynamics (CFD) simulation, modeling Newtonian, viscous, incompressible flow. We examine the transient jet flow observed over a cardiac cycle though a moderate thoracic aortic coarctation model (65% area reduction) reconstructed from magnetic resonance angiography scans of an 8-year old female patient (BSA = 0.94 m^2), as provided for the 2012 STACOM CFD challenge.

The paper is further organized as follows. In Section 2, the computational methods are briefly explained followed by the nature of the supporting clinical study that was conducted. In Section 3, pressure gradient results obtained with CFD are presented along with analyzed flow structures in the DAo, drawing attention to differences that exist between the flow structures seen past the CoA and those seen in a normal aorta. Section 5 concludes with a short note on the limitations of this study and the focus of present studies.

2 Methods

2.1 Computational Methods

Direct numerical simulation (DNS) was performed to solve the Navier–Stokes equations using a second-order accurate, finite difference method; this solver has been used extensively for image-based hemodynamic modeling and incorporates a validated multi-grid artificial compressibility numerical solver [3, 4] simulating incompressible and Newtonian blood flow with constant hemodynamic properties (ρ = 1000 kg/m^3, $\mu = 4 \times 10^{-3}$ Pa.s). Flow is simulated on a high-resolution unstructured Cartesian immersed boundary grid. In this study, the resulting 3D CFD grid had ~200,000 uniformly spaced nodes, with an average node spacing resolution of 0.03 mm, which was generated after immersing the surface model in a Cartesian grid of 228 x 118 x 387 cubical elements. This solver has been employed for DNS jet flow characterization studies in the past [5] for a range

of flow regimes (laminar to turbulent) which were validated against particle image velocimetry (PIV) quantitative flow measurements and flow visualization techniques [6]. The temporal resolution was considered as 0.01 simulation time units i.e. $O(10^{-4})$ sec. Computations were performed using normalized spatial and temporal units. The mean aortic annulus diameter of 1.61 cm was used to achieve spatial normalization, and the mean inflow Reynolds number (Re = 1070) was used in order to obtain temporal normalization. The cardiac cycle was discretized into 21 steps per cycle (0.7 seconds, ~86 BPM) and a second order interpolation scheme was employed in order to obtain inlet conditions based on the input discrete cardiac cycle data. Simulations were conducted using a Poiseuille inflow velocity profile having a mean-velocity varying as per the PC-MRI obtained cardiac output waveform (3.245 L/min, seen in Figure 1) , without extending the inlet. A mass-flow split type outflow boundary condition is employed in the solver, with a 42.2% - 58.8% flow split between the head-and-neck vessels and the DAo. Simulation data was gathered for the 5th cardiac cycle, in order to ensure damping of initial transients.

As a case control, a similar steady inflow hemodynamics simulation was conducted for a normal aorta model (idealized model) which was dynamically scaled (by inlet Re) to match the steady time-averaged inflow of the simulated CoA case, considering the same outflow boundary splits and similar grid resolution. The jet across the coarctation was analyzed for downstream velocity and helicity and compared with the normal model in order to visualize alteration of vortical flow structures in the DAo. Further, the pressure gradient in the normal model, across a length equivalent in size and location to the simulated CoA case, was computed in order to arrive at a qualitative as well as quantitative comparison between normal and pathological cases.

2.2 Clinical Study

Following approval from the Institutional Review Board, we collected retrospective cardiac MRI data on children pre-repair for CoA, evaluated at Children's Hospital of Pittsburgh. PC-MRI velocity field data in children between the ages 5 and 18 were collected over a period of 2 years. For this study, three cases having focal CoA with narrowing of the isthmus and proximal DAo were examined. Pressure drops across the coarctation were computed based on Bernoulli principle using peak velocity measurement, V, obtained from PC-MRI data as $4 \times V^2 = \Delta P$ [7]. The results of the patient study were dynamically scaled to match the flow regime and peak-systole cardiac output of the CFD modeled CoA case. Dynamically scaling of the collected patient-specific velocity data was achieved in a two step procedure: a) First the velocity at the coarctation was scaled up or down appropriately by scaling the extent of narrowing (area as a percentage of area at the hiatus) to be equivalent to the 65% narrowing seen in the simulated CoA case, given the inverse relationship between velocity and the square of diameter at a cross-section; let us reference this computed velocity as $v_{65\%}$. b) Next, since each patient had a different peak flow cardiac output, the velocity expected at the inlet to the ascending aorta (AAo) was computed for a diameter of 16.1 mm (i.e. diameter of inlet to the CoA CFD model) and the ratio of

this computed velocity with the inlet profile-average velocity of the CoA CFD model (0.99 m/s at peak systole) was obtained, while accounting for the applied 58.8% mass-flow split boundary condition at the DAo. This ratio was applied to $v_{65\%}$ in order to finally obtain the velocity expected at the coarctation given that the patient had an identical cardiac output at peak-systole as modeled in the CFD simulation.

3 Results and Discussion

Based on analysis from the clinical investigation, patients with ~30% focal extent of coarctation were expected to have peak velocity distal to the CoA of 1.75±0.25 m/s i.e Bernoulli pressure drop of 12.25±0.25 mm Hg with collateral flows ranging between 5 and 20 mL/beat, depending upon the extent of collateral vessel development during the time of diagnosis. Extrapolating this clinical investigation using the principles of dynamical scaling, patients with a similar focal extent and location of CoA as the modeled patient (65%) are expected to have peak velocity distal to the CoA of 6.00±0.6 m/s (see Table 1) i.e. pressure drop of 144±1.5 mm Hg by the $4\times V^2$ formulation. The velocity range was close to the peak systole velocity observed distal to the CoA from CFD, whereas the pressure gradient was far greater than the CFD result.

Table 1. Summary of clinical study along with dynamical scaling procedure

Patient no	Age	Sex	Collateral Flow (mL/beat)	Peak Velocity at CoA (m/s)	Peak Velocity at Hiatus (m/s)	Isthmus (cm)	Hiatus (cm)	% CoA narrowing
CoA001	11 yr 7.6 mo	F	5.00	1.30	0.40	0.40	1.50	73
CoA002	12 yr 8 mo	M	6.80	1.75	0.80	1.00	1.36	26
CoA003	13 yr 0 mo	M	20.00	1.50	0.90	1.30	1.45	31

Dynamical Scaling			
Patient no	Peak velocity expected at 65% narrowing ($v_{65\%}$, m/s)	AAo peak velocity at 16.1 mm inlet - given 58.8% DAo split (m/s)	Peak velocity at 65% CoA matched to modeled patient CoA peak cardiac output (m/s)
CoA001	0.75	0.14	5.47
CoA002	7.72	1.15	6.66
CoA003	5.82	0.98	5.86
		Mean +- Standard Deviation =	***6.00 +- 0.60 m/s***

The peak systole and the running average (equivalent to a time-averaged steady inlet flow simulation) flow fields for the simulated CoA case are presented for comparison in Figure 1. Using the instantaneous pulsatile CFD pressure field at the peak systole, pressure drop across the CoA computed at two reference planes (depicted in Table 2), was ~30 mm Hg. At end diastole, 0.36 mm Hg pressure drop was observed. In comparison, peak systole pressure drop computed using Bernoulli principle across the coarctation using the peak CFD velocities observed in the plane distal

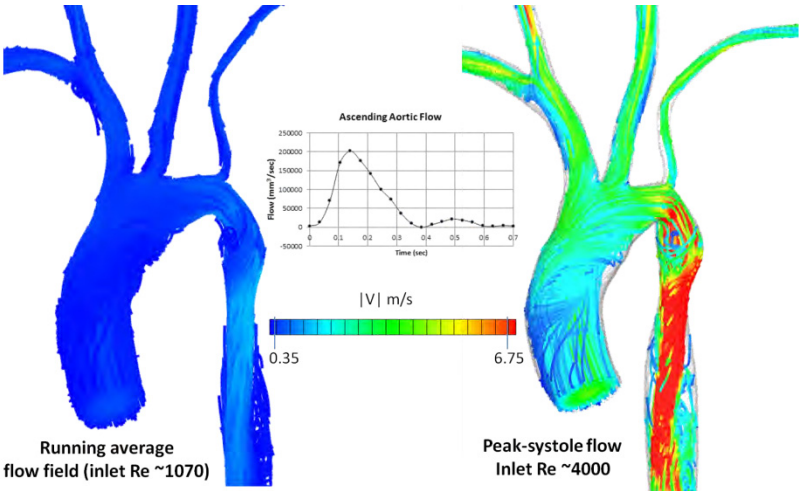

Fig. 1. Stream traces computed by time integration of the velocity field in Tecplot (Bellevue, WA) colored by velocity magnitude, |V|, for a mean ascending aortic velocity of 0.265 m/s, Re 1070 (left) obtained from the running average pulsatile flow field; and at the peak systole instant of the prescribed pulsatile cardiac output (right) having average inlet velocity of ~1 m/s.

to the level of the coarctation (6.78 m/s from CFD) and proximal to it (4.93 m/s from CFD), was ~81 mm Hg, which was again a gross overestimation of the true CFD pressure gradient.

The pressure fields at peak systole and end-diastole are colored onto the observed flow stream traces plotted at their respective times instants of the cardiac cycle, in Figure 2A. These pressure results have also been summarized in Table 2.

Table 2. Peak and time-averaged CFD pressure gradient between two reference planes in the CoA model. AAo (inlet) pressures corresponding to peak systole and end-diastole pressures of 115 and 65 mm Hg proximal to the CoA, respectively, are also indicated.

Peak pressure difference	30.02 mm Hg	*Peak Systole (mm Hg)*
Mean pressure difference	5.81 mm Hg	
Flow splits in supra-aortic and DAo	25.6/11.3/4.3/58.8 (%)	
Pressure in AAo (Systolic/Diastolic)	147/65.5 mm Hg	87 115 143 171

The peak velocity observed from CFD data in the peak-systole jet was 6.99 m/s, which was ~10% higher than that observed at the neck of the coarctation. The running average flow field obtained from pulsatile flow simulation and the corresponding steady inflow centerline velocity and pressure profiles were found to match with a corresponding maximum CoA jet velocity of 1.45 m/s, indicating that the quasi-steady assumption is a reasonable approximation for mean flow of the cardiac cycle.

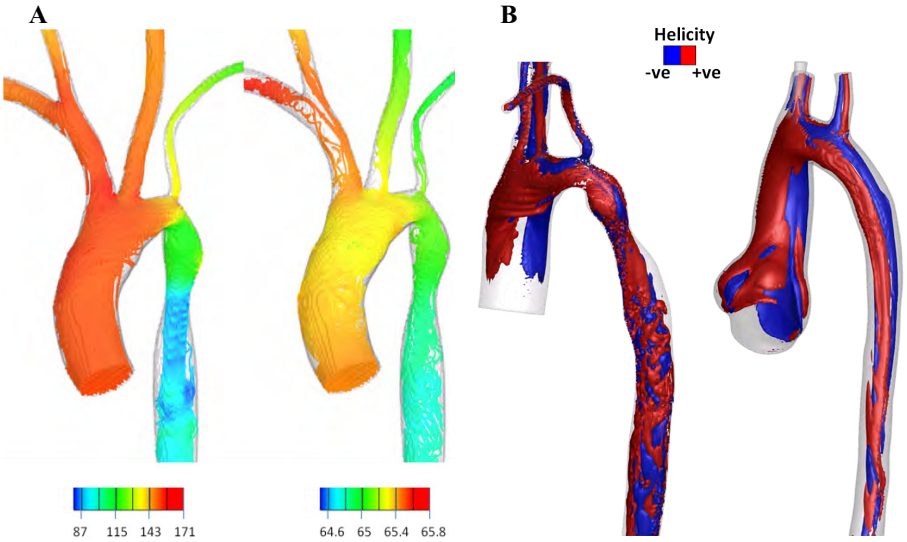

Fig. 2. A) Stream traces computed by time integration of the velocity field colored by pressure at end-systole (left) and end-diastole (right), in mm Hg. B) Helicity isocontours computed at peak systole flow instant of the CoA case (left) and the normal aorta case simulated at steady Re 1070 inflow conditions. Helical flow patterns transform into a turbulent jet wake past the CoA whereas they remain helical at the DAo of the normal aorta, therefore indicating a striking disparity in downstream hemodynamics.

In order to evaluate the influence that CFD inflow velocity profiles prescribed at the inlet of the aorta models have in hemodynamics observed, peak systole velocities were compared for a parabolic Poiseuille flow (peak velocity = 2x mean) and a plug flow boundary velocity profile. Peak velocities past the CoA at multiple cross sections were found to match within +-0.4 m/s.

The pressure drop obtained from the time-average steady inflow CFD model (~5.8 mm Hg) were underestimated by the Bernoulli estimates computed from the same velocity field (~1 mm Hg). This is expected on account of neglected viscous frictional losses in the Bernoulli formulation. The corresponding CFD pressure drop observed across the isthmus of the normal aorta model, considering similar reference planes for data extraction, was ~5 mm Hg, for the identical steady inflow Re.

In order to compare the nature of vortical structures in the DAo flow field between the CoA case and the normal aorta case, helicity was computed as a flow derived parameter and rendered as isocontours (see Figure 2B) for time-averaged inflow conditions (Re 1070). Helicity was computed as the normalized magnitude of the dot product between vorticity and velocity vectors at each node in the computed flow field. Positive helicity (highlighted red) indicates right handed vortical structures and negative helicity (highlighted blue) indicates left handed vortical structures. Helicity in the DAo is regarded as a contributor to optimizing naturally occurring transport processes in the cardiovascular system, avoiding excessive energy dissipation. Further, statistically significant differences are known to exist in the helical content of aortic flows at different phases of the cardiac cycle. Helical flow patterns were observed in the AAo and transverse arch of both CoA and normal cases, but these patterns were more pronounced in the DAo of the normal aorta model. In contrast, the DAo past the coarctation case did not demonstrate such coherent helical flow but rather complex shedding vortical flow characteristic of a turbulent jet wake.

Flow induced wall shear stress (WSS) is believed to play an important role in the initiation and progression of vascular diseases. Increased WSS was seen at the isthmus of the normal aorta and pathological CoA CFD models. A comparison was made between the running time average pulsatile WSS field in the CoA case and the normal aorta simulation at steady inflow conditions (Figure 3). The increased WSS in the CoA case was nearly twice as high at the coarctation (46-48 dynes / cm^2) than that expected in the normal aorta.

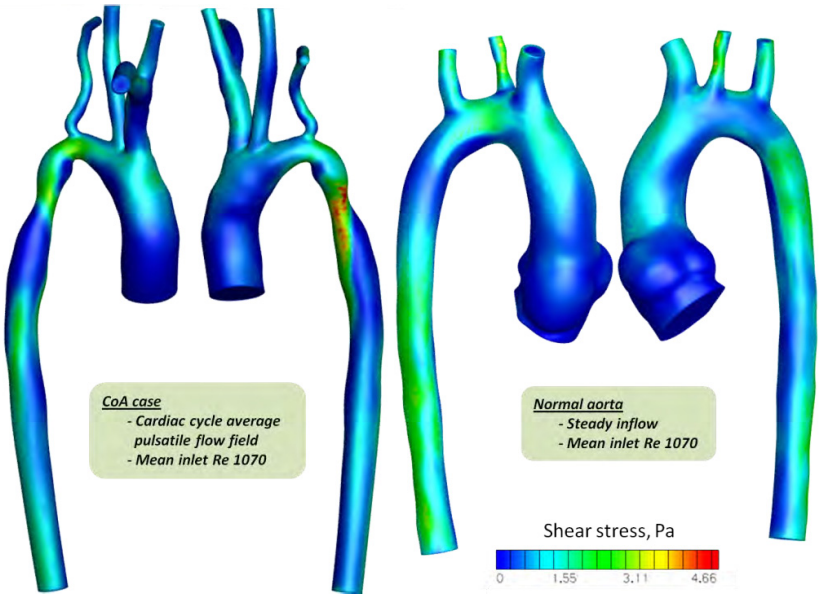

Fig. 3. Surface plots of the time averaged WSS field obtained from the pulsatile CoA simulation (left) and the steady inflow normal aorta simulation, at the corresponding time-averaged Reynolds number (right)

4 Conclusions

CFD is a powerful tool for simulation of altered hemodynamics in pathological anatomies and is adopted in this study as a diagnostic technique for evaluating blood velocity and pressure gradients across a patient-specific CoA. The downstream hemodynamics and helicity observed in the DAo of the normal aorta model were significantly different than that observed in the jet wake of the CoA case. The peak velocity of the modeled CoA jet was observed to occur ~2 cm distal to the site of coarctation and the magnitude of this velocity was found to match clinical observations after appropriate dynamical scaling. The 65% stenosis in the simulated CoA led to a multifold increase in expected pressure gradient and peak DAo velocity in comparison with a normal aorta. The primary conclusion of this study is that Bernoulli estimated pressure drops overestimate the true pressure drop computed using CFD at the peak-systole instant of the pulsatile flow model when complex jet wake effects are pronounced, and underestimate it when compared using a time-averaged steady flow field. Based on this pilot study, CFD determined flow fields are a more reliable clinical indicator of pressure gradient that consider viscous flow and complex jet wake interactions affecting hemodynamics downstream of CoA. Patient-specific CFD modeling for a larger sample size is required in order to confirm this hypothesis. Since collateral flow is an important clinical indicator for determining severity and timing of surgical management, future CFD studies on anatomical models that include reconstructions of major collateral vessels along with their respective PC-MRI determined mass-flow splits, is warranted for more realistic examination of DAo hemodynamics.

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