

# A novel MRI-based data fusion methodology for efficient, personalised, compliant simulations of aortic haemodynamics\*

Catriona Stokes<sup>a,1</sup>, Mirko Bonfanti<sup>a,2</sup>, Zeyan Li<sup>b,3</sup>, Jiang Xiong<sup>c,4</sup>, Duanduan Chen<sup>b,5</sup>, Stavroula Balabani<sup>a,6</sup>, Vanessa Díaz-Zuccarini<sup>a,7</sup>

<sup>a</sup>*Mechanical Engineering Department, University College London, Torrington Place, London, WC1E 7JE, United Kingdom*

<sup>b</sup>*School of Life Science, Beijing Institute of Technology, Beijing, 100081, China*

<sup>c</sup>*Department of Vascular and Endovascular Surgery, Chinese PLA General Hospital, Beijing, 100083, China*

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<sup>1</sup>catriona.stokes.18@ucl.ac.uk, ORCID:0000-0002-9112-9568

<sup>2</sup>m.bonfanti@ucl.ac.uk, ORCID: 0000-0001-5548-3298

<sup>3</sup>3120181382@bit.edu.cn

<sup>4</sup>xiongjiangdoc@126.com, ORCID: 0000-0002-9884-1267

<sup>5</sup>duanduan@bit.edu.cn

<sup>6</sup>s.balabani@ucl.ac.uk, ORCID: 0000-0002-6287-1106

<sup>7</sup>v.diaz@ucl.ac.uk, ORCID: 0000-0003-3183-7463

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## Abstract

We present a novel, cost-efficient methodology to simulate aortic haemodynamics in a patient-specific, compliant aorta using an MRI data fusion process. Based on a previously-developed Moving Boundary Method, this technique circumvents the high computational cost and numerous structural modelling assumptions required by traditional Fluid-Structure Interaction techniques. Without the need for Computed Tomography (CT) data, the MRI images required to construct the simulation can be obtained during a single imaging session. Black Blood MR Angiography and 2D Cine-MRI data were used to reconstruct the luminal geometry and calibrate wall movement specifically to each region of the aorta. 4D-Flow MRI and non-invasive pressure measurements informed patient-specific inlet and outlet boundary conditions. Simulated wall movement closely matched 2D Cine-MRI measurements throughout the aorta, and physiological pressure and flow distributions in CFD were achieved within 3.3% of patient-specific targets. Excellent agreement with 4D-Flow MRI velocity data was observed. Conversely, a rigid-wall simulation under-predicted peak flow rate and systolic maximum velocities whilst predicting a mean Time-Averaged Wall Shear Stress (TAWSS) 13% higher than the compliant simulation. The excellent agreement observed between compliant simulation results and MRI is testament to the accuracy and efficiency of this MRI-based technique.

*Keywords:* Aorta, Computational Fluid Dynamics (CFD), Fluid Structure Interaction, Patient-specific simulation, Haemodynamics

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## 1 **1. Introduction**

2 Computational Fluid Dynamics (CFD) and phase-contrast Magnetic Res-  
3 onance Imaging (PC-MRI) techniques such as Four-Dimensional Flow MRI  
4 (4D-Flow MRI) are used to analyse arterial haemodynamics, providing valu-  
5 able insights to support clinical decision-making. Due to limitations in spatio-  
6 temporal resolution, 4D-Flow MRI cannot accurately capture small-scale flow  
7 features such as the fluid boundary layer, and hence cannot accurately es-  
8 timate clinically-relevant indices such as Wall Shear Stress (WSS) that are  
9 implicated in the onset and development of various cardiovascular diseases  
10 (Castagna et al., 2021; Mazzi et al., 2020; Miyazaki et al., 2017; Piatti et al.,  
11 2017; Zimmermann et al., 2018). Informing CFD simulations with medical  
12 imaging data can facilitate both high resolution and patient-specific accu-  
13 racy, yielding higher-quality haemodynamic data than any individual modal-  
14 ity could provide. However, simulation accuracy depends on the choice of  
15 modelling assumptions such as vessel wall compliance.

16 Modelling wall compliance is fraught with difficulties, so a rigid-wall as-  
17 sumption is commonly used. Unfortunately, this assumption has been shown  
18 to significantly affect the accuracy of haemodynamic metrics such as WSS  
19 (Lantz et al., 2011; Miyazaki et al., 2017; Qiao et al., 2019). Fluid-Structure  
20 Interaction (FSI), a technique that couples the structural dynamics of the  
21 vessel wall with the flow solution, is traditionally used to simulate compli-  
22 ance. However, the complex, inhomogeneous material properties of the aortic  
23 wall cannot be directly measured *in-vivo*. Instead, a uniform literature value  
24 of Young's Modulus ( $E$ ) is often assumed (He et al., 2021; Ryzhakov et al.,  
25 2019; Saitta et al., 2019; Tang et al., 2020). This assumption fails to ac-

26 curately capture vessel wall movement throughout the aorta, even when a  
27 range of  $E$  values are assessed.

28 A Moving Boundary Method (MBM) was developed by Bonfanti et al.  
29 (2017) to circumvent the structural assumptions and computational cost as-  
30 sociated with FSI. Using CT data to reconstruct the aortic lumen and 2D  
31 Cine-MRI data to compute vessel wall compliance locally in each region of  
32 the aorta, the MBM can accurately capture wall movement throughout the  
33 aorta, unlike FSI. MBM simulations of Type-B Aortic Dissection (TBAD), a  
34 severe pathology characterised by a tear in the innermost layer of the aortic  
35 wall, agreed closely with an equivalent FSI simulation yet required only half  
36 the simulation time (Bonfanti et al., 2018). However, CT images are not  
37 always available, for example in healthy patients where the exposure to high  
38 doses of ionising radiation are not clinically justified. MRI-based techniques  
39 have therefore been developed, but typically employ a rigid-wall assumption  
40 (Bozzi et al., 2017; Madhavan and Kemmerling, 2018; Youssefi et al., 2017).

41 Several MRI-based FSI studies of healthy aortae have appeared in the  
42 literature, including those of Lantz et al. (2011), Boccadifuoco et al. (2018)  
43 and Pons et al. (2020). Each used a uniform value of  $E$  throughout the  
44 aorta, requiring multiple FSI simulations for its calibration. Lantz et al.  
45 (2011) and Boccadifuoco et al. (2018) used several literature values of  $E$  in  
46 the physiological range, whereas Pons et al. (2020) iteratively adjusted  $E$  by  
47 calibrating the pulse wave velocity (PWV) using estimations from 4D-Flow  
48 MRI. Comparisons against MRI data found that FSI failed to accurately  
49 capture wall movement throughout the aorta when uniform  $E$  was observed.  
50 Thus, there remains a need for cost-efficient, MRI-based CFD methods to

51 accurately characterise non-uniform arterial compliance.

52 This study presents a novel MBM-based methodology to construct patient-  
53 specific, compliant simulations of a healthy thoracic aorta using a unique  
54 MRI data fusion process. MRA with 2D Cine-MRI is used to segment the  
55 aorta and calibrate region-specific wall movement, while 4D-Flow MRI with  
56 non-invasive pressure measurements informs patient-specific boundary con-  
57 ditions. Compared with FSI, the proposed CFD methodology can provide  
58 accurate results in significantly less time, using data from a single MRI ac-  
59 quisition session. This technique could facilitate shorter clinical decision-making  
60 timescales and reduce clinical resource requirements.

## 61 **2. Methods**

### 62 *2.1. Clinical Data*

63 Three MRI sequences of the thoracic aorta of a healthy volunteer were  
64 acquired using a Philips Achieva 3.0 TX multi-source MRI scanner at Beijing  
65 Institute of Technology, Beijing, China (Philips Medical Systems, Holland).  
66 MRA images of the thoracic aorta at diastole were acquired with a resolution  
67 of  $0.48 \times 0.48 \times 1 \text{ mm}^3$ . 2D Cine-MRI images were acquired as transverse  
68 planes with a resolution of  $1.25 \times 1.25 \times 4 \text{ mm}^3$  and a timestep of 38 ms.  
69 Finally, sagittal 4D-Flow MRI images were acquired at 24 points across the  
70 cardiac cycle with a voxel size of  $2.5 \times 2.5 \times 2.8 \text{ mm}^3$  and a single velocity  
71 encoding (VENC) of 1 m/s. A heart rate of 68 beats per minute and systolic  
72 and diastolic brachial blood pressures ( $P_{sb}$ ,  $P_{db}$ ) of 117 mmHg and 72 mmHg  
73 were measured using a sphygmomanometer after MRI acquisition. This work  
74 was ethically approved by the Institutional Review Board of the Chinese PLA

75 General Hospital (S201703601).

## 76 *2.2. Geometry and Meshing*

77 [Figure 1 about here.]

78 The simulation workflow is shown schematically in Figure 1. A patient-  
79 specific luminal geometry was reconstructed by manual segmentation of MRA  
80 data using Mimics (Materialize NV, Leuven, Belgium) (Figure 1a). The  
81 segmentation was bounded by an inlet at the ascending aorta (AA) and an  
82 outlet at the abdominal aorta (AbAo). Proximal sections of the supra-aortic  
83 branches, the Brachiocephalic Trunk (BT), Left Common Carotid (LCC),  
84 and Left Subclavian Artery (LSA), were included. MRA could not resolve the  
85 small arteries that branch from the descending thoracic aorta, so they were  
86 omitted. The cross-sectional area of the segmented aortic arch and proximal  
87 descending aorta was approximately 30% smaller than the equivalent 2D  
88 Cine-MRI areas. This is likely due to separation-induced flow stagnation  
89 in this region, which can reduce image contrast and erroneously indicate the  
90 presence of wall tissue (Henningsson et al., 2020). Fusing MRA and 2D Cine-  
91 MRI, selected regions of the segmented aorta were dilated by up to 2 mm in  
92 the surface-normal direction using Simpleware ScanIP (Synopsis Inc., CA,  
93 USA) to match the cross-sectional area measurements from 2D Cine-MRI  
94 whilst preserving the morphology of the lumen. The segmented geometry  
95 was used to construct a tetrahedral computational mesh with 525k elements  
96 using Ansys Fluent 20.0 (Ansys Inc., PA, USA) (Figure 1b). Details on  
97 prismatic layer settings are provided in Appendix A. Mesh refinement was  
98 determined using a mesh independence study described in Appendix B.

### 99 2.3. Boundary Conditions

100 The choice of inlet and outlet boundary conditions are critical to re-  
101 produce patient-specific physiological flow and pressure distributions. The  
102 inlet flow rate waveform was extracted from 4D-Flow MRI data at the as-  
103 cending aorta using GTFlow (GyroTools LLC., Zurich, Switzerland). Using  
104 spline interpolation in MATLAB (MathWorks Inc., Natick, Massachusetts,  
105 USA), a smooth waveform with 1 ms increments,  $Q_{SI}(t)$ , was derived from  
106 these measurements and used to compute a uniform velocity inlet profile  
107  $v_{inlet}(t) = Q_{SI}(t)/A_{inlet}$  (Figure 1c). The application of a measured veloc-  
108 ity inlet profile has been shown to produce equivalent results beyond two  
109 diameters of the inlet to uniform inlet profiles with the same inlet flow rate  
110 (Armour et al., 2020; Goubergrits et al., 2013; Madhavan and Kemmerling,  
111 2018; McElroy and Keshmiri, 2018; Pirola et al., 2018).

112 Three-element Windkessel (WK3) outlet pressure boundary conditions  
113 were applied at each CFD outlet to simulate the effects of the peripheral  
114 vascular system and minimise the incidence of non-physical pressure wave  
115 reflections (shown schematically in Figure 1). Patient-specific WK3 calibra-  
116 tion requires target values of inlet systolic and diastolic pressure ( $P_{sa}$ ,  $P_{da}$ )  
117 and mean outlet flow rates. Diastolic pressure remains relatively constant  
118 throughout the arterial tree whilst systolic pressure increases, so  $P_{da}$  was set  
119 equal to  $P_{db}$  and  $P_{sa}$  was derived from the brachial systolic pressure  $P_{sb}$  using  
120  $P_{sa} \approx 0.83P_{sb} + 0.15P_{db}$  (Westerhof et al., 2010). The target mean flow rate  
121 at each outlet was then derived from 4D-Flow MRI data. As one voxel ac-  
122 counted for 10-30% of the cross-sectional area of each branch, measurements  
123 of supra-aortic branch flow rates from 4D-Flow MRI incurred high uncertain-

124 ties. Instead, flow rate waveforms were first extracted at four planes in the  
125 descending aorta (DA1 - DA4 in Fig. 1). The total mean flow rate through  
126 the supra-aortic branches,  $\bar{Q}_{SA}$ , was then calculated as

$$\bar{Q}_{SA} = \bar{Q}_{inlet} - \frac{\bar{Q}_{DA1} + \bar{Q}_{DA2} + \bar{Q}_{DA3} + \bar{Q}_{DA4}}{4} \quad (1)$$

127 and  $\bar{Q}_{SA}$  was split proportionally between each branch based on their respec-  
128 tive diastolic cross-sectional area.

129 Each WK3 is an electrical analogue consisting of a proximal resistance  
130 ( $R_p^i$ ), a distal resistance ( $R_d^i$ ) and a capacitance ( $C_{WK3}^i$ ) whose values must  
131 be carefully calibrated. A 0D lumped parameter model of the aorta was used  
132 to tune the WK3 parameters (Figure 1e). The CFD domain was represented  
133 by a number of discrete segments ( $RLC$  units), each containing a resistor  
134 ( $R^i$ ), an inductor ( $L^i$ ) and a capacitor ( $C_V^i$ ) to simulate fluid pressure loss,  
135 inertance and volume compliance due to wall movement, respectively. A  
136 WK3 was connected to each outlet of this 0D model, and the inlet flow rate  
137 waveform  $Q_{SI}(t)$  was applied at the inlet. The resulting system of ordinary  
138 differential equations was solved numerically using 20-sim (ControllabProd-  
139 ucts B.V., Enschede, The Netherlands) by backward differentiation. For each  
140  $RLC$  unit,  $R^i$  were computed using the total pressure loss through each seg-  
141 ment from a steady-state CFD simulation at the mean inlet flow rate.  $L^i$   
142 were calculated using an expression for large arteries of length  $l^i$  and cross-  
143 sectional area  $A^i$ :

$$L^i = (4/3)\rho l^i \cdot A^{i-1} \quad (2)$$

144 where  $\rho$  is the density of blood (Westerhof et al., 2010). The volume compli-



145 ances  $C_V^i$  were calculated as

$$C_V^i = D^i \cdot V^i \quad (3)$$

146 where  $V^i$  is the volume of the segment.  $D^i$  is the local wall distensibility  
147 which is calculated from 2D Cine-MRI measurements of cross-sectional area  
148 at each segment as

$$D^i = \frac{\Delta A^i}{A_{min}^i} \cdot \frac{1}{P_{pulse}} \quad (4)$$

149 where  $\Delta A^i$  is the maximum cross-sectional area change across the cardiac  
150 cycle and  $A_{min}^i$  is the diastolic area. Because the supra-aortic branches were  
151 not resolved by 2D Cine-MRI, their respective distensibilities were calculated  
152 using an empirical relationship from Reymond et al. (2009):

$$D_{branch}^i = \rho^{-1} \cdot PWV^{i-2} \quad (5)$$

153 where  $PWV = 13.3d^{i-0.3}$  and  $d^i$  is the branch diameter.

154 To calculate  $C_{WK3}^i$ , the arterial network was first lumped as a single-  
155 WK3 analogue (Figure 1d). This allowed us to determine the total arterial  
156 compliance,  $C_{tot}$ , using a method described by Les et al. (2010). Using  $Q_{SI}(t)$   
157 as an input to this lumped arterial network, WK3 parameters were adjusted  
158 until target values of  $P_{sa}$  and  $P_{da}$  were met. The peripheral compliance  
159  $C_{per} = C_{tot} - \Sigma C_V^i$  was divided between each outlet proportionally to their  
160 mean flow rates  $\bar{Q}^i$ :

$$C_{WK3}^i = \frac{\bar{Q}^i}{\bar{Q}_{SI}} \cdot C_{per} \quad (6)$$

161 The total resistance of each outlet was calculated using:

$$R_{tot}^i = R_p^i + R_d^i = \frac{\bar{P}}{\bar{Q}^i} \quad (7)$$

162 where  $\bar{P} = 0.4P_{sa} + 0.6P_{da}$ . An initial guess of  $R_p^i$  was obtained to match the  
163 characteristic impedance of the vessel:

$$R_p^i = \frac{\rho \cdot PWV^i}{A_{branch}^i} \quad (8)$$

164 where  $A_{branch}^i$  is the cross-sectional area of the branch outlet. Finally, each  
165  $R_p^i$  was optimised so that the system achieved target values of  $P_{sa}$  and  $P_{da}$ .  
166 The final WK3 parameters are shown in Table 1.

167 [Table 1 about here.]

#### 168 2.4. Wall Compliance

169 In our moving mesh approach (Bonfanti et al., 2017), the structural dy-  
170 namics of the wall are not explicitly modelled. Instead, the magnitude of  
171 displacement of each mesh node at the aortic wall,  $\delta_n$ , is computed as a  
172 linear function of the local pressure in the surface-normal direction  $\mathbf{n}_n$ :

$$\delta_n = \frac{p_n - p_{ext}}{K_n} \mathbf{n}_n \quad (9)$$

173 where  $K_n$  is the stiffness coefficient,  $p_n$  is the fluid pressure, and  $p_{ext}$  is the  
174 pressure exerted from the external side of the aortic wall, set to the diastolic  
175 pressure  $P_{da}$ .  $K_n$  is calculated (Figure 1f) using:

$$K_n = \frac{2}{C_A^i} \sqrt{\pi A_n} \quad (10)$$

176 where  $A_n$  is the cross-sectional area of the lumen at node  $n$ .  $C_A^i = D^i \cdot A_{min}^i$   
177 is the area compliance in the region of node  $n$ , where  $D^i$  values are obtained  
178 from equation 4.

179 We also performed a simulation with rigid walls to assess the impact of  
180 compliance on parameters of interest. In the rigid 0D model, the capacitors  
181 in each *RLC* unit representing the CFD domain were removed, and identical  
182 pressure and flow rate values were targeted. This resulted in a separate set  
183 of WK3 parameters for each simulation (Table 1).

### 184 *2.5. CFD Simulation*

185 The transient, three-dimensional Navier-Stokes equations were solved nu-  
186 merically using the finite-volume solver Ansys CFX 20.0 (Figure 1g). Blood  
187 was modelled as an incompressible non-Newtonian fluid using the Carreau-  
188 Yasuda viscosity model with empirical constants from Gijsen et al. (1999)  
189 and a density of  $1056 \text{ kg/m}^3$ . Using the Reynolds number (*Re*) definitions  
190 for pulsatile cardiovascular flow from Peacock et al. (1998), a nominal shear  
191 rate defined by Cagney and Balabani (2019), and the peak velocity from  
192 4D-Flow MRI, the peak  $Re_p$  of 4157 was observed to exceed the critical  $Re_c$   
193 of 3505, indicating the onset of turbulence. The  $k-\omega$  Shear Stress Transport  
194 (SST) Reynolds-Averaged formulation of the Navier-Stokes equations was  
195 employed to model turbulence due to its ability to reasonably predict the  
196 onset and amount of flow separation under adverse pressure gradients (Lantz  
197 et al., 2011). A low turbulence intensity of 1% was applied at the inlet and  
198 outlets, which was found to most accurately represent healthy aortic flow  
199 by Kouser et al. (2013). Timesteps of 1 ms were solved using the implicit,  
200 second-order backward-Euler method. A root-mean-square residual value of  
201  $10^{-5}$  was achieved for all equations within each timestep. Simulations were  
202 run until periodic conditions were achieved, defined as less than 1% change  
203 in systolic and diastolic pressures between cycles, requiring five cycles in the

204 compliant simulation and three in the rigid simulation.

### 205 **3. Results**

206 Inlet pressure and mean outlet flow rates were compared to target val-  
207 ues to assess whether the WK3 BCs yielded physiological pressure and flow  
208 conditions. Targets for  $P_{sys}$ ,  $P_{dia}$  and  $\bar{Q}_i$  were achieved within 3.3% for both  
209 rigid and compliant simulations (Table 2).

210 [Table 2 about here.]

211 Volume flow rate from compliant and rigid simulations across the cardiac  
212 cycle were compared against 4D-Flow MRI measurements at DA1-DA4 (Fig-  
213 ure 2). As mass conservation is not observed between the arch and abdominal  
214 aorta in 4D-Flow MRI but is enforced in the simulations, 4D-Flow MRI data  
215 measurements were scaled at each cross-section to match the stroke volume  
216 (SV) from simulations following the approach of Bäumlér et al. (2020). Un-  
217 scaled 4D-Flow MRI measurements are also shown as a band encapsulating  
218  $\pm 22$ ml of stroke volume, representing the mean SV uncertainty for single-  
219 VENC 4D-Flow MRI data (Kroeger et al., 2021).

220 [Figure 2 about here.]

221 Simulated luminal cross-sectional area changes across the cardiac cycle  
222 were compared with 2D Cine-MR measurements at the arch and DA1-DA4  
223 (Figure 3). Close agreement was observed throughout. Minor discrepancies  
224 in the shape of the area waveforms between CFD and MRI include a faster  
225 rate of area expansion during the acceleration phase in CFD, an earlier peak,

226 and a more pronounced secondary peak at end-systole. Maximum and min-  
227 imum areas matched closely at the arch, though with an in-plane resolution  
228 of only 4 mm x 1.25 mm, MRI measurements appear noisy and CFD and  
229 MRI waveforms do not appear as closely aligned.

230 [Figure 3 about here.]

231 Velocity magnitude contours from compliant and rigid CFD simulations  
232 were compared with 4D-Flow MRI data at peak systole (T1, Figure 4) and  
233 during mid-deceleration (T2, Figure 5), showing excellent agreement. It  
234 should be noted that CFD analysis planes were chosen to match the angle  
235 and location of 4D-Flow MRI planes and thus were not perpendicular to the  
236 centreline. At T1, a slight enlargement of the separation zone in the rigid  
237 simulation is observed across the aortic arch due to its smaller cross-sectional  
238 area. The peak velocity at T1 is better predicted by the compliant than the  
239 rigid simulation. Peak velocity is slightly under-predicted from DA2 to DA4  
240 by both simulations, possibly due to the local effects of branching vessels  
241 that were not modelled. At T2, rigid and compliant simulations show similar  
242 distributions, though, in contrast to T1, slightly higher peak velocities are  
243 seen in the rigid simulation. High-velocity regions in both simulations are  
244 rotated by  $\approx 90^\circ$  compared with 4D-Flow MRI.

245 [Figure 4 about here.]

246 [Figure 5 about here.]

247 Using the definitions from Gallo et al. (2012), Time Averaged Wall Shear  
248 Stress (TAWSS) and Oscillatory Shear Index (OSI) contours from the com-

249 pliant simulation are shown alongside contours of difference between compli-  
250 ant and rigid simulations in Figure 6, with simulated velocity streamlines to  
251 assist in their interpretation.

252 [Figure 6 about here.]

253 High TAWSS values (above 5 Pa, according to Lantz et al. (2012); Peng  
254 et al. (2019)) are observed in the ascending aorta near the inlet, within  
255 LCC and LSA, and at their bifurcation, in agreement with other studies  
256 (Boccadifuoco et al., 2018; Lantz et al., 2011). These regions are exposed to  
257 high-velocity flow near the wall (Figure 6a). Regions of high OSI are observed  
258 in the descending aorta where disturbed flow develops during diastole, and  
259 the WSS vector becomes highly misaligned with its average (Figure 6d).  
260 OSI was low within the supra-aortic branches, though isolated regions of  
261 high OSI are seen at their bifurcations and in the ascending aorta. This has  
262 been attributed to low backflow in the branches and the pulsatile separation  
263 and recirculation at their bifurcations (Lantz et al., 2011). Although the  
264 distribution of TAWSS and OSI were qualitatively similar in both rigid and  
265 compliant simulations, the rigid case exhibited substantially higher TAWSS  
266 with a 13% higher mean, a 16.4% higher maximum, and a 19.3% higher  
267 minimum value than the compliant case. These differences are concentrated  
268 in the proximal aorta (Figure 6c). As WSS indices have been identified as  
269 markers for disease progression, this has implications for the prognostic value  
270 of rigid-wall simulations that will be further discussed in Section 4.

## 271 4. Discussion

272 We have presented a novel, efficient methodology to model patient-specific  
273 vessel compliance in CFD simulations using MRI data alone. Our MBM-  
274 based technique eliminates the need for explicit structural modelling and  
275 multiple simulations to tune structural properties, thus substantially reduc-  
276 ing simulation timescales compared with FSI. Furthermore, because patient-  
277 specific wall compliance is tuned locally at each region, high accuracy is  
278 achieved in wall movement throughout the aorta compared with FSI, where  
279 structural properties are typically assumed to be uniform. By comparing  
280 equivalent compliant and rigid simulations of a healthy aorta, we have pro-  
281 vided further evidence that rigid simulations may not accurately reproduce  
282 physiologically accurate velocity and WSS distributions when wall movement  
283 is significant.

284 Previous efforts to reconstruct the haemodynamics of a compliant healthy  
285 aorta from MRI data with FSI could not accurately characterise the move-  
286 ment of the aortic wall, underpredicting the extent of luminal area expansion  
287 despite multiple simulations with varying structural properties (Boccadifuoco  
288 et al., 2018). This effect may be of greater concern in simulations of patho-  
289 logical aortae, for example, in the case of TBAD. In a recent FSI study of  
290 TBAD, aortic compliance was captured accurately in some but not all regions  
291 due to the assumption of uniform  $E$  throughout the aorta (Bäumler et al.,  
292 2020). The image-based compliance tuning technique in the present study  
293 enabled patient-specific regional variations in compliance without the need  
294 for compliance tuning, resulting in excellent agreement with 2D Cine-MRI  
295 in luminal cross-sectional area change throughout the aorta.

296 Under the same pulse pressure, the compliant aorta produced a higher  
297 peak flow rate and a more negative flow rate at end-systole than the rigid  
298 simulation, resulting in favourable agreement with 4D-Flow MRI data in the  
299 compliant case. This effect is expected due to the accumulation and subse-  
300 quent ejection of flow from each aortic segment as it expands and contracts  
301 under pressure. Although compliance acts to delay the peak flow rate, the  
302 compliant simulation predicted a slightly earlier peak in flow rate than 4D-  
303 Flow MRI, mirroring the faster increase and earlier peak observed in area  
304 waveforms. This may indicate an under-prediction in compliance of the aortic  
305 arch and supra-aortic branches due to longitudinal compliance of the prox-  
306 imal aorta that is not modelled. Indeed, Pagoulatou et al. (2021) observed  
307 under-predictions in proximal aortic distensibility of 20-62% when longitudi-  
308 nal compliance was neglected. There is also uncertainty in the 2D Cine-MRI  
309 data due to spatial and temporal resolution, so smaller features of the area  
310 waveform may not be represented accurately by the imaging data.

311 Close agreement was observed between simulations and 4D-Flow MRI  
312 velocity contours, both in flow structure and peak velocity. However, simula-  
313 tions resolved the boundary layer with high resolution while MRI could not.  
314 The boundary layer thickness of  $\approx 1\text{mm}$  (see 4) is much smaller than the  
315 highest achievable resolution of 4D-Flow MRI in the large vessels, which typ-  
316 ically exceeds  $2\text{mm}$  (Fathi et al., 2020). As discussed, the compliant simula-  
317 tion achieved a higher peak flow rate. This resulted in better agreement with  
318 4D-Flow MRI than the rigid simulation, outweighing the velocity-lowering  
319 effect of increased cross-sectional area. Peak velocities were slightly higher  
320 and closer to 4D-Flow MRI measurements in the rigid simulation at T2. Re-



321 gions of high velocity in the compliant simulation were more dispersed at  
322 T2 than the rigid case, which lowered the peak velocity. Because area mea-  
323 surements agree particularly well between compliant CFD and MRI at T2  
324 ( $\approx 6.5s$ ), higher MRI velocities may result from the effects of un-modelled  
325 local branching vessels or bulk movement of the aorta away from its diastolic  
326 centreline. However, imaging errors in 4D-Flow MRI may also contribute.

327 Without accurate resolution of the boundary layer or the location of the  
328 wall in 4D-Flow MRI, WSS indices extracted from these images will incur  
329 substantial uncertainties (Miyazaki et al., 2017; Piatti et al., 2017; Zimmer-  
330 mann et al., 2018). Despite under-predicting peak velocity values, the mean  
331 TAWSS in the rigid simulation was 13% higher than the compliant simu-  
332 lation. This provides further evidence that under identical, patient-specific  
333 and physiological pressure and flow conditions, rigid simulations may sub-  
334 stantially over-predict WSS if wall movement is significant. The influence of  
335 WSS on the onset and progression of a multitude of cardiovascular diseases  
336 has been widely demonstrated (Mazzi et al., 2020). For example, specific  
337 WSS distribution characteristics have been identified as markers of aneurysm  
338 development (Chung and Cebal, 2015), and regions of low WSS have been  
339 associated with numerous adverse effects, including endothelial dysfunction  
340 and the formation of atherosclerotic regions (Wee et al., 2018). Due to the  
341 strong prognostic value of WSS, our results highlight the importance of com-  
342 pliance modelling.

343 There are some limitations to the presented method. The MBM cannot  
344 handle large deformations due to an associated deterioration of mesh quality.  
345 Wall movement can be in the order of 10mm in pathologies such as TBAD

346 (Bäumler et al., 2020; Yang et al., 2014), so further work aims to improve  
347 the robustness of this technique to large deformations. Our methodology  
348 considers wall movement in the surface-normal direction, so any longitudinal  
349 compliance or bulk movement of the aorta is not modelled. Additionally, the  
350 effects of surrounding tissues are not considered, and the wall is assumed to  
351 exhibit linear elastic behaviour. These assumptions may contribute to differ-  
352 ences in velocity contours between 4D-Flow MRI and CFD in the proximal  
353 aorta, where the movement of the aorta away from its diastolic centreline is  
354 most significant. These effects may also impact pressure wave transmission  
355 and contribute to the minor discrepancies in flow rate and luminal cross-  
356 sectional area curves between CFD and MRI. Despite numerous reports that  
357 the impact of the inlet velocity profile is negligible beyond the aortic arch,  
358 complex inlet flow may still have an impact further downstream and may con-  
359 tribute to discrepancies in velocity between CFD and 4D-Flow MRI through-  
360 out the aorta. We are currently extending this work to investigate the effects  
361 of inlet velocity conditions.

362 This study represents a significant methodological advance in its ability  
363 to accurately reconstruct patient-specific compliant aortic haemodynamics  
364 in a cost-efficient manner using MRI data alone. Our method exhibits nu-  
365 merous advantages over rigid-wall simulations and FSI simulations and could  
366 facilitate improved patient safety whilst minimising healthcare resources and  
367 reducing clinical decision-making timescales. Our technique could be gen-  
368 eralised to other types of cardiovascular flows and to aortic diseases whose  
369 morphological features can be accurately captured with MRI.

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376 performance computing cluster resources used to perform the simulations.

## 377 **Conflict of Interest**

378 The authors declare no conflicts of interest.

# 379 **Appendices**

## 380 **Appendix A Prismatic Layers**

381 In all meshes used for this study, ten prismatic layers with a first-layer  
382 thickness corresponding to a  $y^+$  of 1 were used to ensure that the first cell  
383 height lay within the viscous sublayer of the turbulent boundary layer. The  
384 total thickness of the prismatic layers exceeded the expected boundary layer  
385 thickness,  $\delta$ , of 1.0 mm, estimated as  $\delta = \sqrt{\nu/\Omega}$ , where  $\nu$  is the kinematic  
386 viscosity, and  $\Omega$  is the cycle frequency (Pier and Schmid, 2017).

## 387 **Appendix B Mesh Independence**

388 Three successively refined meshes were used to perform a rigid-wall tran-  
389 sient simulation with identical WK3 boundary conditions. Mesh element

390 count approximately doubled between successive refinements, and the total  
391 prismatic layer thickness never fell below the expected boundary layer thick-  
392 ness of 1.0mm. Simulations were initialised using a previously converged  
393 simulation, and two further cycles were run. Less than 1% change in systolic  
394 and diastolic pressures were observed between these two cycles for each mesh.  
395 Key metrics from the final cycle of each of the three simulations are shown  
396 in Table 3.

397 [Table 3 about here.]

398 Percentage differences between coarse and medium meshes were an av-  
399 erage of 3.4 times higher across all metrics than between medium and fine  
400 meshes. Differences between medium and fine meshes did not exceed 3.6%  
401 for all metrics, similar to acceptable differences noted in similar studies, so  
402 the medium mesh was used for all further analysis.

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6    *a) Velocity streamlines at peak systole, b) TAWSS contours from the compliant simulation, clipped at 5 Pa, c) contours of TAWSS difference between compliant and rigid simulations, d) velocity streamlines during diastole, e) OSI contours from the compliant simulation f) contours of OSI difference between compliant and rigid simulations. . . . . 35*

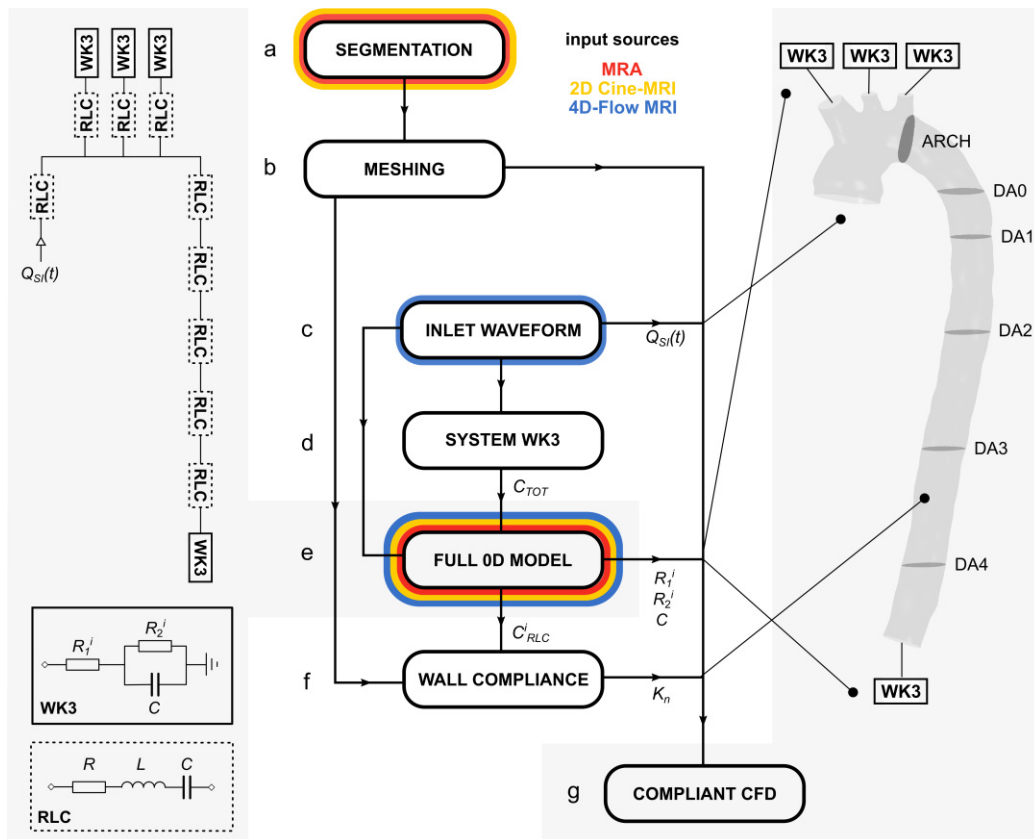


Figure 1: Flowchart of the simulation methodology including schematic diagrams of the 0D domain (e) and the 3D CFD domain (g). Steps (a) through (g) are referred to throughout Section 2, where they are each described in detail.

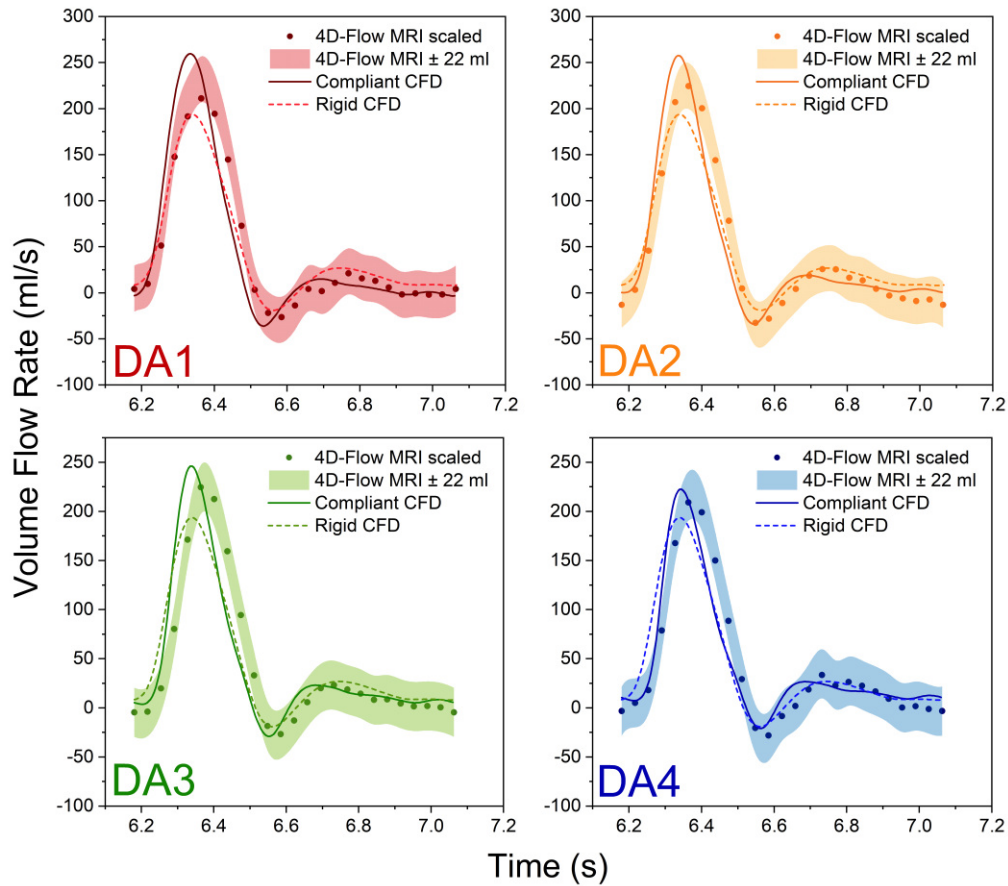


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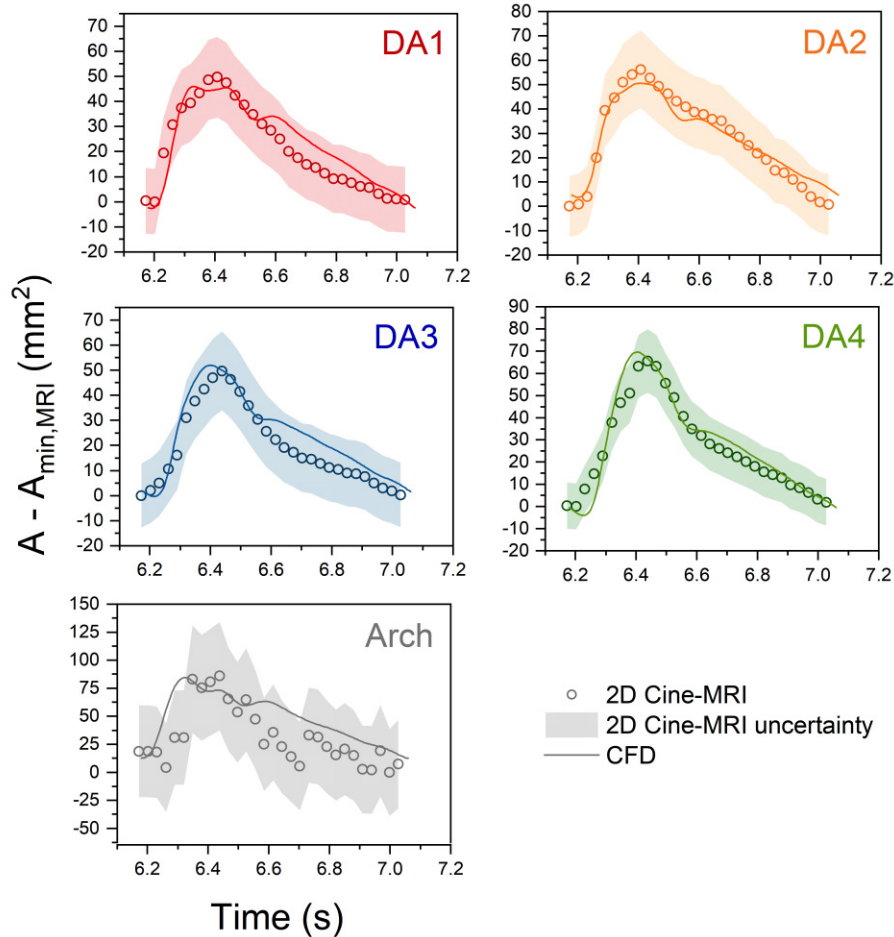


Figure 3: Absolute luminal cross-sectional area comparison between 2D Cine-MRI and compliant CFD at the arch and descending aorta across the cardiac cycle. A band encapsulating  $\pm 6\%$  error are indicated in the DA planes and  $\pm 12\%$  at the arch. These errors were calculated by manually selecting the smallest and largest areas that could reasonably be chosen at each plane in GTFlow. See Figure 4 for plane locations.



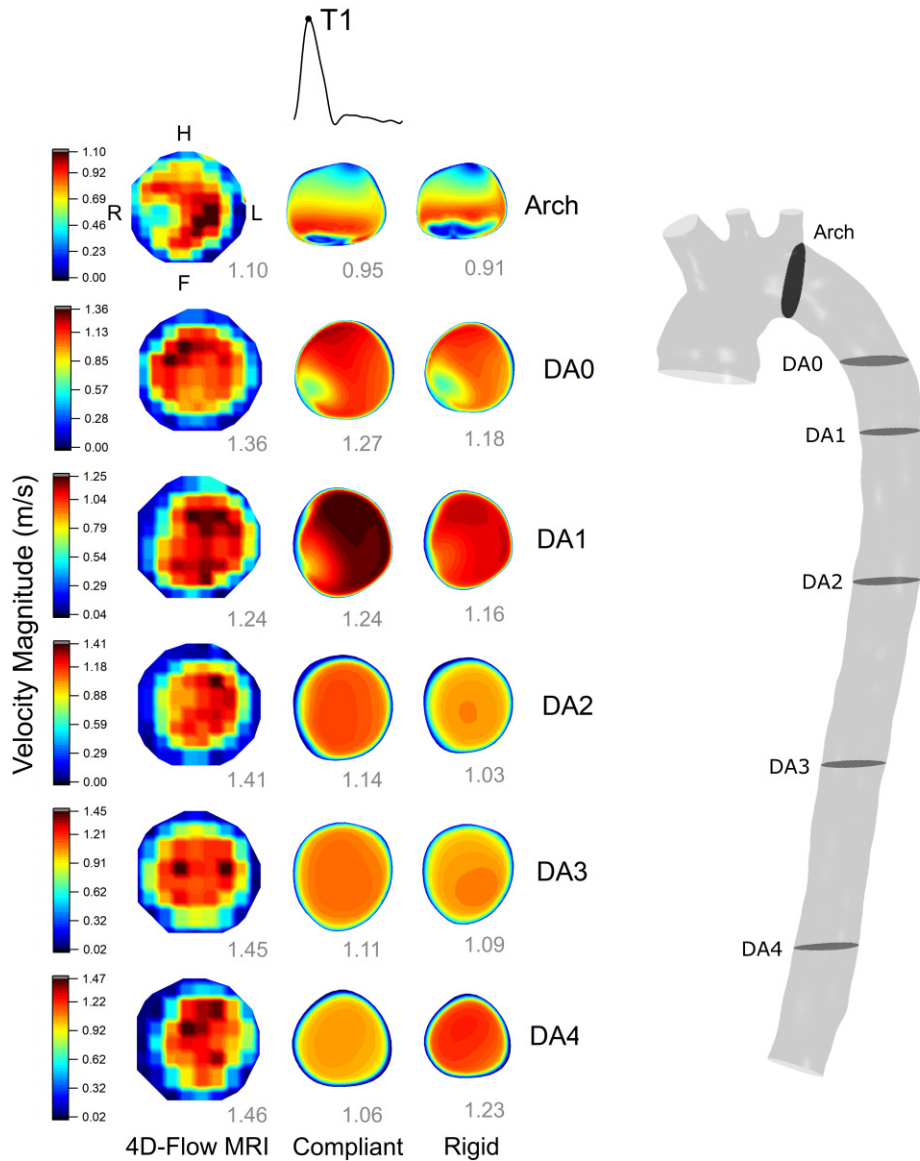


Figure 4: Velocity contour comparison between 4D-Flow MRI, compliant and rigid CFD simulations at peak systole throughout the aorta. Velocity contour ranges are set by the minimum and maximum values from 4D Flow-MRI, and the peak velocity magnitude at each CFD plane is indicated at the bottom right of each contour. The arch plane orientation is indicated, while the orientation of the DA planes are indicated in Figure 5

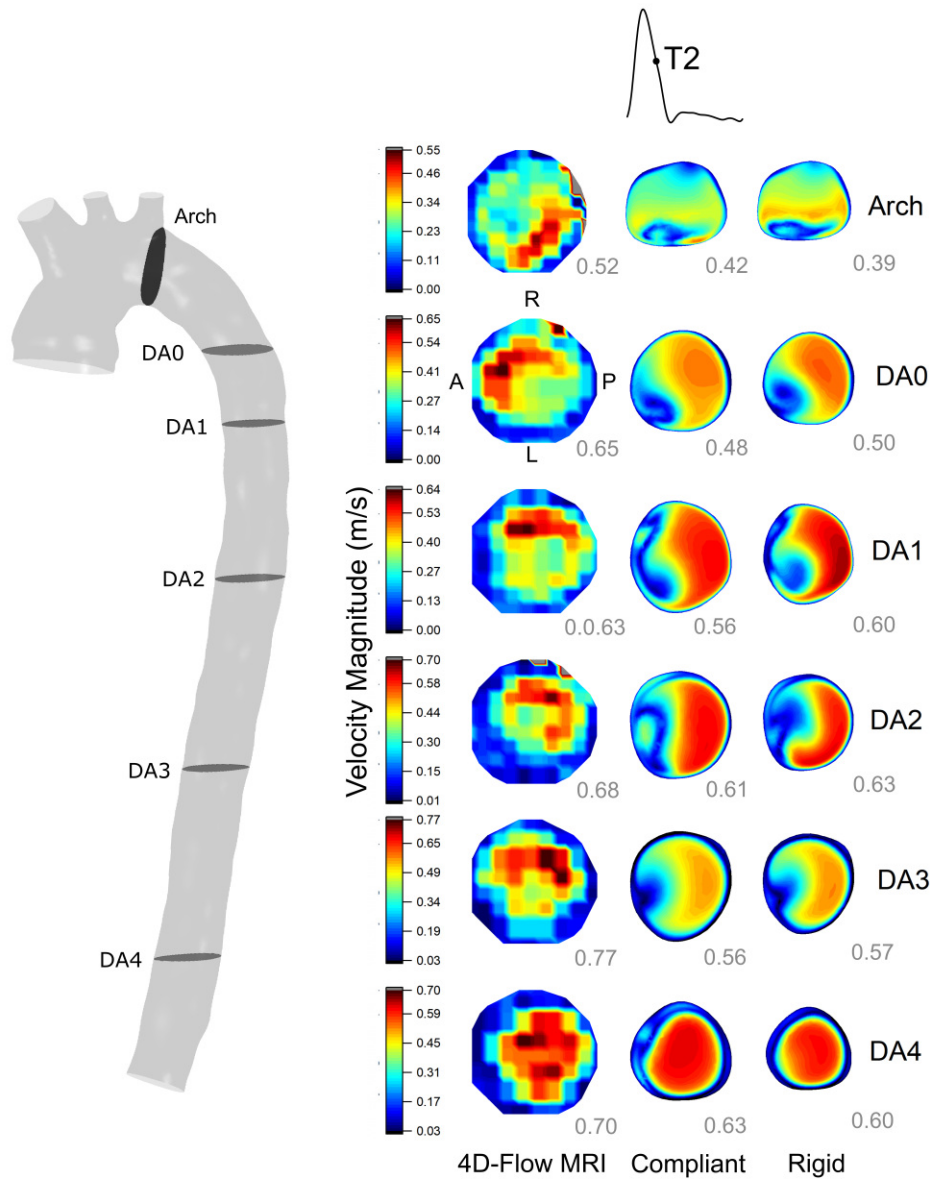


Figure 5: *Velocity contour comparison between 4D-Flow MRI, compliant and rigid CFD simulations at mid-deceleration throughout the aorta. Velocity contour ranges are set by the minimum and maximum values from 4D Flow-MRI, and the peak velocity magnitude at each CFD plane are indicated at the bottom right of each contour. The DA plane orientation is indicated, while the orientation of the arch planes are indicated on Figure 4*

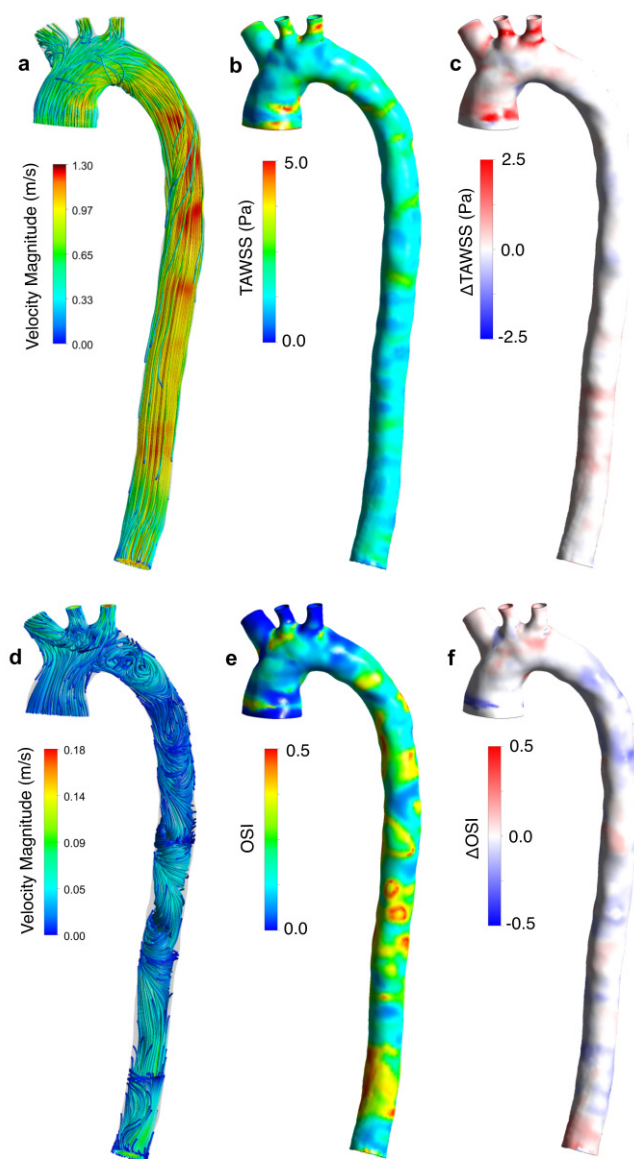


Figure 6: a) Velocity streamlines at peak systole, b) TAWSS contours from the compliant simulation, clipped at 5 Pa, c) contours of TAWSS difference between compliant and rigid simulations, d) velocity streamlines during diastole, e) OSI contours from the compliant simulation f) contours of OSI difference between compliant and rigid simulations.

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Table 1: *Patient-specific three-element Windkessel parameters for compliant and rigid CFD simulations, determined using the 0D tuning process described in Section 2*

		<b>BT</b>	<b>LCC</b>	<b>LSA</b>	<b>AbAo</b>
<b>Compliant</b>	$R_1$ (mmHg/ml/s)	0.4872	0.9744	0.9179	0.1154
	$R_2$ (mmHg/ml/s)	4.2231	9.7188	6.3783	1.8281
	$C$ (ml/mmHg)	0.3818	0.1682	0.2465	0.5702
<b>Rigid</b>	$R_1$ (mmHg/ml/s)	0.1500	0.2500	0.2500	0.0750
	$R_2$ (mmHg/ml/s)	4.5600	10.4430	7.0460	1.8690
	$C$ (ml/mmHg)	0.3818	0.1682	0.2465	0.5702

Table 2: *Simulated compliant and rigid inlet systolic and diastolic pressure and mean outlet flow rates compared with target values. The percentage error between simulated and target values are indicated.*

Quantity	Measurement	Target	Rigid / % err	Compliant / % err
$P_{sys}$ (mmHg)	117	108	104.9 / 2.9%	105.1 / 2.7%
$P_{dia}$ (mmHg)	72	72	69.6 / 3.3%	71.7 / 0.4%
$\bar{Q}_{BT}$ (ml/s)	13.57	18.34	18.20 / 0.8%	17.99 / 1.9%
$\bar{Q}_{LCC}$ (ml/s)	6.17	8.11	8.02 / 1.1%	7.99 / 1.5%
$\bar{Q}_{LSA}$ (ml/s)	4.37	11.84	11.52 / 2.7%	11.51 / 2.8%
$\bar{Q}_{AbAo}$ (ml/s)	44.60	44.44	44.20 / 0.4%	44.73 / 0.9%

Table 3: *Table of key metrics from coarse, medium and fine (C, M, F) rigid-wall simulations. Final two columns show the percentage difference between medium/coarse and fine/medium meshes. T1 refers to peak systole. Velocity metrics are calculated across all cells in the domain, while pressure metrics are measured only at the wall surface.*

	Coarse	Medium	Fine	% diff: M/C	% diff: F/M
Node count	68465	186202	418977	63.2	55.6
Element count	199076	525274	1199742	62.1	56.2
Avg. pressure @ T1: wall	96.02	95.78	95.67	-0.25	-0.11
Max. pressure @ T1: wall	111.35	111.63	111.70	0.25	0.06
Avg. vel. mag @ T1	0.67	0.64	0.62	-4.75	-3.56
Max. vel. mag. @ T1	1.85	1.85	1.87	0.018	0.81
Mean TAWSS	1.94	1.90	1.91	-2.18	0.40
Max. TAWSS	9.62	11.06	10.78	12.97	-2.66
Mean. OSI	0.151	0.153	0.153	1.71	-0.32