REVISION 1

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

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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. Luminal area closely matched 2D Cine-MRI measurements with a mean error of less than 4.6% across the cardiac cycle, while physiological pressure and flow distributions were simulated to within 3.3% of patient-specific targets. Moderate agreement with 4D-Flow MRI velocity data was observed. Despite lower peak velocity, an equivalent rigid-wall simulation predicted a mean Time-Averaged Wall Shear Stress (TAWSS) 13% higher than the compliant simulation. The agreement observed between compliant simulation results and MRI data is testament to the accuracy and efficiency of this MRI-based simulation technique.

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

1. Introduction

Computational Fluid Dynamics (CFD) and phase-contrast Magnetic Resonance Imaging (PC-MRI) techniques such as Four-Dimensional Flow MRI (4DMR) facilitate the analysis of arterial haemodynamics, providing valuable insights to support clinical decision-making. Due to limitations in spatio-temporal resolution, 4DMR cannot accurately capture small-scale flow features such as the fluid boundary layer. It hence cannot accurately estimate clinically relevant indices such as Wall Shear Stress (WSS) that are implicated in the onset and development of various cardiovascular diseases (Castagna et al., 2021; Mazzi et al., 2020; Miyazaki et al., 2017; Piatti et al., 2017; Zimmermann et al., 2018). Informing CFD simulations with medical imaging data can facilitate both high resolution and patient-specific accuracy, yielding higher-quality haemodynamic data than any individual modality could provide. However, simulation accuracy depends on the choice of modelling assumptions, such as vessel wall compliance.

Modelling wall compliance is fraught with difficulties, so a rigid-wall assumption is commonly used. Unfortunately, this assumption has been shown to significantly affect the accuracy of haemodynamic metrics such as WSS (Lantz et al., 2011; Miyazaki et al., 2017; Qiao et al., 2019). Fluid-Structure Interaction (FSI), a technique that couples the structural dynamics of the vessel wall with the flow solution, is traditionally used to simulate compliance. However, the complex, inhomogeneous material properties of the aortic wall cannot be directly measured *in-vivo*. Instead, a constant literature value of Young's Modulus (E) is often assumed, which fails to accurately capture vessel wall movement throughout the aorta (He et al., 2021; Ryzhakov et al.,

26 2019; Saitta et al., 2019; Tang et al., 2020).

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

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

51 This study presents a novel MBM-based methodology to construct patient-
 52 specific, compliant simulations of a healthy thoracic aorta using a unique MRI
 53 data fusion process. MRA and Cine-MRI are used to segment the aorta and
 54 calibrate region-specific wall movement, while 4DMR and non-invasive pres-
 55 sure measurements inform patient-specific boundary conditions. Compared
 56 with FSI, the proposed CFD methodology can provide accurate results in sig-
 57 nificantly less time, using data from a single MRI acquisition session. Thus,
 58 this technique could facilitate shorter clinical decision-making timescales and
 59 reduce clinical resource requirements.

60 **2. Methods**

61 *2.1. Clinical Data*

62 Three MRI sequences of the thoracic aorta of a healthy volunteer were
 63 acquired using a Philips Achieva 3.0 TX multi-source MRI scanner at Beijing
 64 Institute of Technology, Beijing, China (Philips Medical Systems, Holland).
 65 MRA images of the thoracic aorta at diastole were acquired with a resolution
 66 of $0.48 \times 0.48 \times 1 \text{ mm}^3$. Cine-MRI images were acquired as transverse planes
 67 with a resolution of $1.25 \times 1.25 \times 4 \text{ mm}^3$ and a timestep of 38 ms. Finally,
 68 sagittal 4DMR images were acquired at 24 points across the cardiac cycle
 69 with a voxel size of $2.5 \times 2.5 \times 2.8 \text{ mm}^3$ and a single velocity encoding
 70 (VENC) of 1 m/s. A heart rate of 68 beats per minute and systolic and
 71 diastolic brachial blood pressures (P_{sb} , P_{db}) of 117 mmHg and 72 mmHg were
 72 measured using a sphygmomanometer after MRI acquisition. This work was
 73 ethically approved by the Institutional Review Board of the Chinese PLA
 74 General Hospital (S201703601).

75 2.2. Geometry and Meshing

76 [Figure 1 about here.]

77 The simulation workflow is shown schematically in Figure 1. A patient-
 78 specific luminal geometry was reconstructed by manual segmentation of MRA
 79 images using Mimics (Materialize NV, Leuven, Belgium) (Figure 1a). This
 80 diastolic segmentation was bounded by an inlet at the ascending aorta (AA)
 81 and an outlet at the abdominal aorta (AbAo). Proximal sections of the supra-
 82 aortic branches, the Brachiocephalic Trunk (BT), Left Common Carotid
 83 (LCC), and Left Subclavian Artery (LSA), were included, with outlets at
 84 the terminus of each. As MRA could not resolve the intercostal arteries,
 85 they were not included in the segmentation. As such, flow loss through these
 86 branches, measured as 5-10% of the stroke volume (SV), was not modelled.

87 Cross-sectional area measurements in the segmented proximal aorta were
 88 approximately 30% smaller than the Cine-MRI areas at diastole in the same
 89 locations. This is likely due to separation-induced flow stagnation in this
 90 region of the MRA images, which can reduce image contrast and erroneously
 91 indicate the presence of wall tissue (Henningsson et al., 2020). Fusing MRA
 92 and Cine-MRI, selected regions of the segmented aorta were dilated by up
 93 to 2 mm in the surface-normal direction using Simpleware ScanIP (Synopsis
 94 Inc., CA, USA) to match the diastolic (minimum) cross-sectional area mea-
 95 surements from Cine-MRI whilst preserving the morphology of the lumen.
 96 The segmented geometry was used to construct a tetrahedral computational
 97 mesh with 525k elements using Ansys Fluent 20.0 (Ansys Inc., PA, USA)
 98 (Figure 1b). Details on prismatic layer settings are provided in Appendix A.
 99 Mesh refinement was determined using a mesh independence study described

100 in Appendix B.

101 2.3. Boundary Conditions

102 The choice of inlet and outlet boundary conditions are critical in ac-
 103 curately simulating patient-specific physiological flow and pressure distri-
 104 butions. The patient-specific inlet flow rate waveform was extracted from
 105 4DMR data at the ascending aorta using GTFlow (GyroTools LLC., Zurich,
 106 Switzerland). Using spline interpolation in MATLAB (MathWorks Inc., Nat-
 107 ick, Massachusetts, USA), a smooth waveform with 1 ms increments, $Q_{SI}(t)$,
 108 was derived from these measurements and used to apply a uniform inlet
 109 velocity profile (IVP) $v_{inlet}(t) = Q_{SI}(t)/A_{inlet}$ (Figure 1c). Compared to a
 110 patient-specific, three-component (3D) IVP extracted from 4DMR, the choice
 111 of a uniform IVP is likely to impact the accuracy of velocity and WSS dis-
 112 tributions in the ascending aorta and aortic arch. It is expected to have
 113 a lesser impact on flow in the descending aorta (DA) according to Armour
 114 et al. (2021); Pirola et al. (2018), though the uniform IVP may under-predict
 115 flow helicity and radial velocity, particularly during late systolic deceleration
 116 (Morbiducci et al., 2013; Youssefi et al., 2017). Applying a non-uniform
 117 measured IVP may lead to improved velocity agreement between CFD and
 118 4DMR; however, doing so is non-trivial. Adding this step would not affect
 119 any other stages of the proposed compliance modelling methodology, so a
 120 uniform IVP was deemed suitable for this study. The inlet was fixed in place
 121 and not permitted to dilate across the cardiac cycle.

122 Three-element Windkessel (WK3) outlet pressure boundary conditions
 123 were applied at each CFD outlet to simulate the effects of the peripheral
 124 vascular system and minimise the incidence of non-physical pressure wave

125 reflections. Each WK3 is an electrical analogue consisting of a proximal
126 resistance R_p , a distal resistance R_d and a capacitance C_{WK3} , whose values
127 must be carefully calibrated to the specific patient.

128 WK3 calibration began by determining target values of aortic systolic and
129 diastolic pressure (P_{sa} , P_{da}) and mean flow rates at each outlet (Figure 1d).
130 Diastolic pressure remains relatively constant throughout the arterial tree,
131 so P_{da} was set to P_{db} . P_{sa} was derived from P_{sb} using $P_{sa} \approx 0.83P_{sb} + 0.15P_{db}$
132 (Westerhof et al., 2010). The target mean flow rate at each outlet was then
133 derived from 4DMR data. As one voxel accounted for 10-30% of the cross-
134 sectional area of each supra-aortic branch, measurements of flow rate at these
135 locations incurred high uncertainties. Instead, flow rate waveforms were first
136 extracted at four planes in the DA (DA1 - DA4 in Fig. 1). The total mean
137 flow rate through the supra-aortic branches, $\bar{Q}_{SA}$, was then calculated as

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

138 and $\bar{Q}_{SA}$ was split proportionally between BT, LCC and LSA based on their
139 diastolic cross-sectional areas.

140 Next, a single-WK3 analogue of the full arterial system (Figure 1e) was
141 used to determine the total arterial compliance, C^{tot} , with a method de-
142 scribed by Les et al. (2010). Using $Q_{SI}(t)$ at the inlet of the WK3, R_p^{tot} , R_d^{tot} ,
143 and C^{tot} were adjusted iteratively until target values of P_{sa} and P_{da} were
144 observed at the inlet.

145 A full 0D lumped parameter model of the aorta was then constructed
146 (Figure 1f). In the full 0D model, the CFD domain was represented by
147 nine discrete RLC units, each containing a resistor (R^i), an inductor (L^i)

148 and a capacitor (C_V^i) to simulate fluid pressure loss, inertance and volume
149 compliance due to wall movement, respectively. The inlet flow rate waveform
150 $Q_{SI}(t)$ was applied at the inlet, and a WK3 was connected to each outlet,
151 each with parameters R_p^i , R_d^i , and C_{WK3}^i .

152 To compute C_{WK3}^i , the peripheral compliance $C^{per} = C^{tot} - \Sigma C_V^i$ was
153 divided between each outlet proportionally to the mean flow rate of each
154 supra-aortic branch, $\bar{Q}^i$:

$$C_{WK3}^i = \frac{\bar{Q}^i}{\bar{Q}_{SI}} \cdot C^{per} \quad (2)$$

155 The total resistance of each outlet was calculated using:

$$R_{tot}^i = R_p^i + R_d^i = \frac{\bar{P}}{\bar{Q}^i} \quad (3)$$

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

$$R_p^i = \frac{\rho \cdot PWV^i}{A_{branch}^i} \quad (4)$$

158 where A_{branch}^i is the cross-sectional area of the branch outlet.

159 For each RLC unit, R^i were computed using the total pressure loss
160 through each segment from a steady-state CFD simulation at the mean inlet
161 flow rate. L^i were calculated using an expression for large arteries of length
162 l^i and cross-sectional area A^i :

$$L^i = (4/3)\rho l^i \cdot A^{i-1} \quad (5)$$

163 where ρ is the density of blood (Westerhof et al., 2010). The volume compli-
164 ances C_V^i were calculated as

$$C_V^i = D^i \cdot V^i \quad (6)$$

165 where V^i is the volume of the segment. D^i is the local area distensibility
166 which is calculated from Cine-MRI measurements of cross-sectional area at
167 each segment as

$$D^i = \frac{\Delta A^i}{A_{min}^i} \cdot \frac{1}{P_{pulse}} \quad (7)$$

168 where ΔA^i is the maximum cross-sectional area change across the cardiac
169 cycle and A_{min}^i is the diastolic area. Because the supra-aortic branches were
170 not resolved by Cine-MRI, their respective area distensibility values were
171 calculated using an empirical relationship from Reymond et al. (2009):

$$D_{branch}^i = \rho^{-1} \cdot PWV^i^{-2} \quad (8)$$

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

173 The full 0D model results in a system of ordinary differential equations
174 which were solved numerically using 20-sim (ControllabProducts B.V., En-
175 sschede, The Netherlands) by backward differentiation. R_p^i values were ma-
176 nipulated from their initial estimate until target values of P_{sa} and P_{da} were
177 observed at the inlet. The final WK3 parameters are shown in Table 1.

178 [Table 1 about here.]

179 2.4. Wall Compliance

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

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

184 where K_n is the local stiffness coefficient, p_n is the fluid pressure, and p_{ext} is
 185 the pressure exerted from the external side of the aortic wall, set as P_{da} . K_n
 186 is calculated (Figure 1g) using:

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

187 where A_n is the cross-sectional area of the lumen at node n . $C_A^i = D^i \cdot A_{min}^i$
 188 is the area compliance in the region of node n , where D^i values are obtained
 189 from equation 7. As the inlet and outlets are fixed in place and do not dilate,
 190 the faces bordering them were given an artificially high stiffness to prevent
 191 excessive cell distortion. Finally, five smoothing operations were performed
 192 to eliminate the discontinuities in stiffness between each aortic region.

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

198 2.5. CFD Simulation

199 The transient, three-dimensional Navier-Stokes equations were solved nu-
 200 merically using the finite-volume solver Ansys CFX 20.0 (Figure 1h). Blood
 201 was modelled as an incompressible non-Newtonian fluid using the Carreau-
 202 Yasuda viscosity model with empirical constants from Gijzen et al. (1999)
 203 and a density of 1056 kg/m^3 . Using the Reynolds number (Re) definitions
 204 for pulsatile cardiovascular flow from Peacock et al. (1998), a nominal shear
 205 rate defined by Cagney and Balabani (2019), and the peak velocity from
 206 4DMR, the peak Re_p of 4157 was observed to exceed the critical Re_c of 3505,

207 indicating the onset of turbulence. The $k-\omega$ Shear Stress Transport (SST)
 208 Reynolds-Averaged formulation of the Navier-Stokes equations was employed
 209 to model turbulence due to its ability to predict the onset and amount of flow
 210 separation under adverse pressure gradients (Lantz et al., 2011). A low turbu-
 211 lence intensity of 1% was applied at the inlet and outlets, which was found
 212 to most accurately represent healthy aortic flow by Kousera et al. (2013).
 213 Timesteps of 1 ms were solved using the implicit, second-order backward-
 214 Euler method. A root-mean-square residual value of 10^{-5} was achieved for
 215 all equations within each timestep during the final cycle.

216 Simulations were run until periodic conditions were achieved, defined as
 217 less than 1% change in systolic and diastolic pressures between cycles, re-
 218 quiring five cycles in the compliant simulation (CS) and three in the rigid
 219 simulation (RS). The RS and CS required 2.5 and 9.2 hours per cycle on 8
 220 Intel Xeon cores, respectively. Based on a direct comparison by (Bonfanti
 221 et al., 2018) on the same workstation, an equivalent FSI simulation would
 222 require approximately double the simulation time as the MBM simulations
 223 (≈ 18 h per cycle).

224 3. Results

225 Inlet pressure and mean outlet flow rates were compared with target
 226 values to assess whether the WK3 BCs yielded physiological pressure and
 227 flow conditions. Targets for P_{sys} , P_{dia} and $\bar{Q}_i$ were achieved within 3.3% for
 228 both RS and CS (Table 2).

229 [Table 2 about here.]

230 Simulated volume flow rate across the cardiac cycle was compared against
 231 4DMR at DA1-DA4 (Figure 2). As intercostal flow loss was not simulated,
 232 every 4DMR point on each analysis plane (DA*) was linearly scaled by
 233 $\bar{Q}_{CFD,DA^*}/\bar{Q}_{4DMR,DA^*}$ such that the SV in CFD and 4DMR were equal on
 234 plane DA*. This scaling facilitates a more direct comparison of the flow rate
 235 waveforms, even though measured flow loss is small (5-10%) (Bäumler et al.,
 236 2020). Raw 4DMR measurements are also shown as a band encapsulating
 237 ± 22 ml of SV, representing the mean uncertainty for single-VENC 4DMR
 238 data (Kroeger et al., 2021). The peak flow rate is slightly over-predicted
 239 by the CS, an effect which is more pronounced in the proximal DA, and is
 240 under-predicted by the RS.

241 [Figure 2 about here.]

242 Simulated luminal cross-sectional area changes across the cardiac cycle
 243 were compared with Cine-MRI measurements at the arch and DA1-DA4
 244 (Figure 3). In addition to the measured diastolic area from Cine-MRI, the
 245 discrepancy in peak systolic area change between CFD and Cine-MRI and
 246 the mean error at each point on the waveform are indicated on each plot.
 247 Excellent agreement was observed throughout the DA, with less than 3%
 248 peak error and 2% mean error at all planes. Maximum and minimum areas
 249 matched closely at the arch, with less than 0.5% peak error. However, with
 250 an in-plane resolution of only 4 mm x 1.25 mm, Cine-MRI data at the arch
 251 appears noisy, and CFD and MRI waveforms do not appear as closely aligned,
 252 resulting in a 4.6% mean error. In addition, CFD yielded a faster rate of
 253 area expansion during the acceleration phase, an earlier peak, and a more
 254 pronounced secondary peak at end-systole compared to Cine-MRI.

255 [Figure 3 about here.]

256 Velocity magnitude contours from RS and CS were compared with 4DMR
257 data at peak systole (T1, Figure 4) and late systole, as flow is decelerating
258 (T2, Figure 5). At T2, CFD data was extracted such that the flow rate
259 was equal to 4DMR at each plane. Note that CFD analysis planes were
260 chosen to match the angle and location of 4DMR planes and thus were not
261 perpendicular to the centreline.

262 [Figure 4 about here.]

263 [Figure 5 about here.]

264 A moderate qualitative agreement in velocity distribution is observed
265 between CFD and 4DMR. At the arch, MRI shows that flow is more concen-
266 trated to the left side than CFD. At T1, regions of high velocity in the DA
267 correspond closely between MRI and CFD, particularly at DA0 and DA1.
268 At T2, CFD predicts a high-velocity region that is concentrated towards the
269 posterior wall, whereas in MRI, it progressively rotates from the anterior to
270 the left posterior wall along the DA.

271 Peak velocity tends to increase along the DA in both CFD and 4DMR.
272 It is almost universally under-predicted by CFD, by 0-28% at T1 and 10-
273 28% at T2. The agreement is closest at T1 in the proximal aorta, where the
274 compliant peak flow rate is over-predicted. Flow rates are precisely matched
275 between CFD and MRI at each plane at T2, where under-prediction in peak
276 velocity is universal. The reasons for the observed under-prediction will be
277 discussed in Section 4.

278 Using the definitions from Gallo et al. (2012), Time Averaged Wall Shear
279 Stress (TAWSS) and Oscillatory Shear Index (OSI) contours from the CS are
280 shown alongside contours of difference between CS and RS in Figure 6, with
281 simulated velocity streamlines to assist in their interpretation.

282 [Figure 6 about here.]

283 High TAWSS values (above 5 Pa, according to Lantz et al. (2012); Peng
284 et al. (2019)) are observed in the ascending aorta near the inlet, within
285 LCC and LSA, and at their bifurcation, in agreement with other studies
286 (Boccadifuoco et al., 2018; Lantz et al., 2011). These regions are exposed
287 to high-velocity flow near the wall (Figure 6a). Regions of high OSI are
288 observed in the DA where disturbed flow develops during diastole, and the
289 WSS vector becomes highly misaligned with its average (Figure 6d). OSI
290 was low within the supra-aortic branches, though isolated regions of high
291 OSI are seen at their bifurcations and in the ascending aorta. This effect has
292 been attributed to low backflow in the branches and the pulsatile separation
293 and recirculation at their bifurcations (Lantz et al., 2011).

294 Although the distribution of TAWSS and OSI were qualitatively similar
295 in both RS and CS, the rigid case exhibited substantially higher TAWSS
296 with a 13% higher mean, a 16.4% higher maximum, and a 19.3% higher
297 minimum value than the compliant case. These differences are concentrated
298 in the proximal aorta (Figure 6c). As WSS indices have been identified as
299 markers for disease progression, this has implications for the prognostic value
300 of rigid-wall simulations that will be further discussed in Section 4.

301 4. Discussion

302 We have presented an efficient methodology to model patient-specific ves-
 303 sel compliance in CFD simulations, adapting an existing MBM workflow
 304 (Bonfanti et al., 2017) to rely solely on MRI data as input. Using a novel
 305 MRI data fusion process to segment a healthy aorta, we validated MBM re-
 306 sults against 4DMR for the first time. By comparing compliant and rigid
 307 simulations, we have also provided further evidence that rigid simulations
 308 may not accurately reproduce physiologically accurate velocity and WSS
 309 distributions when wall movement is significant.

310 In this study, simulated luminal area change matched Cine-MRI measure-
 311 ments closely throughout the aorta, with a mean error of less than 2% in the
 312 DA and 4.6% in the arch. In comparison, recent FSI studies observe errors
 313 of 10-20% or more using a uniform Young’s Modulus, requiring numerous
 314 costly FSI simulations to determine the most appropriate value (Boccardi-
 315 fuoco et al., 2018; Bäumlér et al., 2020). Our method achieves much higher
 316 accuracy in wall movement throughout the aorta with substantially lower
 317 computational cost than FSI and without the need for numerous calibration
 318 runs. This efficiency would be of clear benefit in a clinical setting. Small dis-
 319 crepancies in the shape of the waveforms may relate to differences in pressure
 320 wave transmission, and errors in Cine-MRI data due to spatial and temporal
 321 resolution.

322 Simulated volume flow rate waveforms agree well with 4DMR (Fig. 2).
 323 Despite equal SV, the CS and RS slightly over- and under-predict the peak
 324 flow rate, respectively. The CS models the accumulation and subsequent ejection
 325 of flow from each aortic segment as it expands and contracts under the

326 same pulse pressure as the RS, resulting in a narrower, higher peak. Although
327 compliance acts to delay the peak in flow rate, it was predicted slightly ear-
328 lier than 4DMR, possibly indicating an under-prediction in compliance of
329 the aortic arch and supra-aortic branches as longitudinal compliance of the
330 proximal aorta is not accounted for in the presented methodology. Indeed,
331 Pagoulatou et al. (2021) observed under-predictions in proximal aortic dis-
332 tensibility of 20-62% when longitudinal deformation was neglected.

333 4DMR is known to under-predict peak velocity due to cycle averaging
334 errors (Markl et al., 2012). Therefore, it seems counter-intuitive that veloc-
335 ity in the DA would be under-predicted by CFD, as we observe in Figs. 4
336 and 5. However, 4DMR would systematically under-predict peak velocity
337 throughout the aorta, leading to erroneously low flow rate measurements ev-
338 erywhere, including the AA where we extracted our inlet flow rate waveform.
339 Therefore, this effect would be implicitly accounted for by our inlet BC and
340 should not contribute substantially to velocity under-prediction in CFD. As
341 the choice of IVP has not been shown to affect peak velocity in a healthy
342 DA, we do not believe that a 3D IVP would improve the velocity agreement
343 in the DA (Youssefi et al., 2017). It also seems counter-intuitive that peak
344 velocity increases along the DA when 5-10% of flow is lost to the intercostals.
345 However, the luminal area decreases by about 20% from DA1 to DA4, leading
346 to a progressive increase in peak velocity.

347 We believe that the under-prediction in velocity is predominantly caused
348 by partial volume effects and poor signal-to-noise ratio of 4DMR in the near-
349 wall region, which contribute to an erroneously thick boundary layer of $\approx$
350 5 mm, exceeding the expected thickness of $\approx$ 1mm that CFD accurately

captures (see 4). Excessive boundary layer thickness would lead to a non-physiological concentration of flow in the centre of the lumen. Therefore, *at equal flow rate* on a given plane, we would expect CFD to yield a lower peak velocity than 4DMR as the total flow is distributed across a larger area. We indeed observe this at T1 at DA3 and DA4, where peak flow rate and area are well-matched, but peak velocity is under-predicted by the simulations. This effect may be obscured by the over-estimation in peak flow rate in the compliant proximal aorta at T1, where peak velocity agrees very closely. Peak velocity is also under-predicted throughout the aorta at T2, where simulation data was extracted to exactly match the 4DMR flow rate at each plane. In the RS, this effect is obscured by the smaller cross-sectional area, which acts to increase peak velocity. Lumen areas may also be over or under-predicted as they were calibrated with Cine-MRI measurements with an associated uncertainty of $\pm 6\%$ in the DA. Any error in the area would compound or mitigate the impact of the thick boundary layer in 4DMR and might lead to the varying degree of velocity under-prediction observed between planes (10-28%).

We observed a moderate qualitative agreement in velocity distribution between CFD and 4DMR. However, at the arch, flow is biased to the left (outer) side in 4DMR, which CFD did not capture, which may be improved with a 3D IVP as this has been shown to improve accuracy in the proximal aorta Pirola et al. (2018); Armour et al. (2021). In the DA, the agreement is good at T1 but less favourable at T2, where simulated high-velocity regions in the DA are similar in shape but rotated clockwise by about 90 degrees compared to 4DMR. At T2, Simulations predict a high-velocity region con-

376 centrated towards the posterior wall, while 4DMR shows a concentration on
377 the left-side wall that rotates along the DA, indicating flow helicity. As dis-
378 cussed in Section 2, a 3D IVP may better predict this helical flow in the DA
379 at T2, which we will investigate in a future study.

380 The presence of intercostal arteries *in-vivo* may disturb the flow near
381 each bifurcation and affect the velocity distribution throughout the DA in a
382 way that is not simulated. 4DMR imaging errors can be substantial and are
383 also likely to influence the agreement between CFD and 4DMR (Montalba
384 et al., 2018; Ebel et al., 2019; Puiseux et al., 2019; Bock et al., 2019; Demir
385 et al., 2021; Kroeger et al., 2021; Casciaro et al., 2021; Rose et al., 2016).
386 The impact of turbulence modelling, outlet boundary conditions, rheological
387 assumptions, aortic morphology and movement across the cardiac cycle may
388 also affect agreement.

389 Without accurate resolution of the boundary layer or the location of the
390 wall in 4DMR, WSS indices extracted from these images will incur significant
391 uncertainties (Miyazaki et al., 2017; Piatti et al., 2017; Zimmermann et al.,
392 2018). Despite predicting a lower peak flow rate and velocity than the CS,
393 the mean TAWSS in the RS was 13% higher, providing further evidence that
394 rigid-wall simulations may substantially over-predict WSS if wall movement
395 is significant, even under identical, patient-specific and physiological pressure
396 and flow conditions. The influence of WSS on the onset and progression of
397 a multitude of cardiovascular diseases has been widely demonstrated (Mazzi
398 et al., 2020). For example, specific WSS distribution characteristics have
399 been identified as markers of aneurysm development (Chung and Cebal,
400 2015), and regions of low WSS have been associated with numerous adverse

401 effects, including endothelial dysfunction and the formation of atherosclerotic
402 regions (Wee et al., 2018). Due to the strong prognostic value of WSS, our
403 results highlight the importance of compliance modelling.

404 There are some limitations to the presented method. First, the MBM
405 cannot handle large deformations due to an associated deterioration of mesh
406 quality. Wall movement can be in the order of 10mm in pathologies such
407 as TBAD (Bäumler et al., 2020; Yang et al., 2014), so further work aims
408 to improve the robustness of this technique to large deformations. Second,
409 our methodology considers wall movement in the surface-normal direction, so
410 any longitudinal compliance or bulk movement of the aorta is not modelled.
411 Additionally, the effects of surrounding tissues are not considered, and the
412 wall is assumed to exhibit linear elastic behaviour. These assumptions may
413 contribute to differences in velocity contours between 4DMR and CFD in
414 the proximal aorta, where the movement of the aorta away from its diastolic
415 centreline is most significant. These effects may also impact pressure wave
416 transmission and contribute to the minor discrepancies in flow rate and lu-
417 minal cross-sectional area curves between CFD and MRI. The limitations of
418 the uniform IVP have been discussed in detail and will be investigated in
419 further work. Finally, the MRI data used to inform and validate the sim-
420 ulations is subject to various errors, as discussed. Without high-resolution
421 experimental data (e.g. Particle Image Velocimetry), we cannot provide a
422 systematic evaluation of these imaging errors.

423 This study represents a significant methodological advance in its ability to
424 accurately reconstruct patient-specific compliant aortic haemodynamics cost-
425 effectively using MRI data alone. Our method exhibits numerous advantages

over rigid-wall simulations and FSI simulations and could facilitate improved patient safety whilst minimising healthcare resources and reducing clinical decision-making timescales. Furthermore, our technique could be generalised to other types of cardiovascular flows and aortic diseases whose morphological features can be captured accurately with MRI.

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Conflict of Interest

The authors declare no conflicts of interest.

Appendices

Appendix A Prismatic Layers

In all meshes used for this study, ten prismatic layers with a first-layer thickness corresponding to a y^+ of 1 were used to ensure that the first cell height lay within the viscous sublayer of the turbulent boundary layer. The

total thickness of the prismatic layers exceeded the expected boundary layer thickness, δ , of 1.0 mm, estimated as $\delta = \sqrt{\nu/\Omega}$, where ν is the kinematic viscosity, and Ω is the cycle frequency (Pier and Schmid, 2017).

Appendix B Mesh Independence

Three successively refined meshes were used to perform a rigid-wall transient simulation with identical WK3 boundary conditions. Mesh element count approximately doubled between successive refinements, and the total prismatic layer thickness never fell below the expected boundary layer thickness of 1.0mm. Simulations were initialised using a previously converged simulation, and two further cycles were run. Less than 1% change in systolic and diastolic pressures were observed between these two cycles for each mesh. Key metrics from the final cycle of each of the three simulations are shown in Table 3.

[Table 3 about here.]

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

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Figure 1: Flowchart of the simulation methodology including schematic diagrams of the system WK3 (e), the 0D domain (f) and the 3D CFD domain (h). Steps (a) through (h) are referred to throughout Section 2, where they are each described in detail.

Figure 2: Volume flow rate comparison between CS, RS and 4DMR at four planes in the DA (see Figure 4 for locations). 4DMR results are shown as a band with a ± 22 ml uncertainty in SV, found to be the mean uncertainty for single-VENC 4DMR measurements of flow rate by Kroeger et al. (2021). 4DMR results are also shown as discrete measurements, with the SV scaled to match CFD due to the lack of 4DMR mass conservation.

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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*

		BT	LCC	LSA	AbAo
Compliant	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
Rigid	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. The 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	Mesh Medium	Fine	% change M/C F/M	
Node count	68465	186202	418977	63.2	55.6
Element count	199076	525274	1199742	62.1	56.2
Mean 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
Mean velocity magnitude @ T1	0.67	0.64	0.62	-4.75	-3.56
Maximum velocity magnitude @ 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