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1 **Computational study on hemodynamic changes in patient-specific proximal neck**
2 **angulation of abdominal aortic aneurysm with time-varying velocity**

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51 **Abstract**

52 Aneurysms are considered as a critical cardiovascular disease worldwide when they rupture. The clinical
53 understanding of geometrical impact on the flow behaviour and biomechanics of abdominal aortic aneurysm
54 (AAA) is progressively developing. Proximal neck angulations of AAAs are believed to influence the
55 hemodynamic changes and wall shear stress (WSS) within AAAs. Our aim was to perform pulsatile simulations
56 using computational fluid dynamics (CFD) for patient-specific geometry to investigate the influence of severe
57 angular ($\geq 60^\circ$) neck on AAA's hemodynamic and wall shear stress. The patient's geometrical characteristics
58 were obtained from a computed tomography images database of AAA patients. The AAA geometry was
59 reconstructed using Mimics software. In computational method, blood was assumed Newtonian fluid and an
60 inlet varying velocity waveform in a cardiac cycle was assigned. The CFD study was performed with ANSYS
61 software. The results of flow behaviours indicated that the blood flow through severe bending of angular neck
62 leads to high turbulence and asymmetry of flows within the aneurysm sac resulting in blood recirculation. The
63 high wall shear stress (WSS) occurred near the AAA neck and on surface of aneurysm sac. This study explained
64 and showed flow behaviours and WSS progression within high angular neck AAA and risk prediction of
65 abdominal aorta rupture. We expect that the visualization of blood flow and hemodynamic changes resulted
66 from CFD simulation could be as an extra tool to assist clinicians during a decision making when estimation the
67 risks of interventional procedures.

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69 **Keywords:** Abdominal aortic aneurysm; Angulated neck; Computational fluid dynamics; Wall shear stress;
70 Hemodynamic; Computed tomography

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80 **Abbreviations**

81	3D	Three-dimensional
82	AAAs	Abdominal aortic aneurysms
83	CAD	Computer-aided design
84	CFD	Computational fluid dynamics
85	CT	Computed tomography
86	CVD	Cardiovascular disease
87	DICOM	Digital imaging and communications in medicine
88	EVAR	Endovascular aortic aneurysm repair
89	ILT	Intraluminal thrombus
90	MR	Magnetic resonance
91	ROI	Region of interest
92	STL	Stereolithography
93	UDF	User-defined function
94	WSS	Wall shear stress

95

96 **Introduction**

97 Cardiovascular disease (CVD) is one of the foremost common cause of global mortality rate [1]. In
98 2013, a report of Global Burden of Disease stated that 17.3 million cases of death caused by CVD globally,
99 which accounted approximately 31.5% of total deaths [1, 2]. One of most prevalent cardiovascular diseases is
100 abdominal aortic aneurysm (AAA) [3]. Abdominal aortic aneurysm is defined as a dilatation of the artery that
101 located below the renal arteries[4, 5], with at least a diameter of 30 mm or about 1.5 times the normal size of
102 aorta [6]. Abdominal aortic aneurysms are often diagnosed through the presence of intraluminal thrombus
103 deposition and are linked to the degradation of the connective tissue in the arterial wall, which made up of cell
104 debris and fibrinous blood clots [7]. Abdominal aortic aneurysms are formed due to several mechanisms,
105 including inflammation of immune responses and aortic wall degradation, which are affected by molecular
106 genetics [8, 9]. During the aneurysm formation, a complex blood flow environment and altered wall shear stress
107 distribution are induced. Moreover, AAA is considered life-threatening health condition, which can require
108 urgent surgical intervention [7]. Continuous AAA expansion leads to the decline of aortic wall strength, in
109 which case the wall becomes susceptible to collapse or eventual aortic rupture [10]. Current clinical

110 recommendations are the following; when the AAA diameters reach 55 mm in men and 50 mm in women, with
111 a development rate of 8.0 mm/year, then surgical intervention is necessary [5, 10].

112 Currently, AAA intervention approaches include open surgery, endovascular aneurysm repair and
113 endovascular aneurysm sealing and are based on the diameter size of the aneurysm sac with a follow up
114 routines [11–15]. However, the aneurysm diameter is still a poor indicator of rupture since some reported
115 aneurysms with larger diameter remain intact, while aneurysms of a smaller size have been reported to rupture
116 [5, 10, 16]. Thus, AAA rupture is ranked as the 13th leading cause of mortality in the US alone with
117 approximately 15,000 patients every year, and reports of more than 8,000 cases of death in the UK [7, 17].
118 Furthermore, a ruptured AAA is considered a fatal surgical emergency which has a mortality rate of 90% [18].
119 The numerous studies conducted on the prediction of rupture and its risks, have proposed several possible AAA
120 rupture factors including asymmetry flow index, maximum aneurysm diameter, age, aortic wall stiffness,
121 mechanical stress, aneurysm growth rate, intraluminal thrombus ratio, smoking, hypertension and high
122 cholesterol [8, 11, 19–21]. However, morphologies such as aortic neck angulation related to adverse events and
123 outcome after endovascular aneurysm repair (EVAR) [22] have often been overlooked.

124 Generally, magnetic resonance (MR) and a computer tomography (CT) can be used to obtain the
125 anatomy of cardiovascular structures [23, 24]. The resulting images of vasculature are valuable to generate
126 numerical models which can be used to predict mechanical behavior under these conditions. Thus,
127 Computational fluid dynamics (CFD) has been used for cardiovascular research, including flow analysis and
128 calculation of wall shear stress [25–29]. For the study of AAAs, CFD has been implemented in the applications
129 of idealized or patient-specific geometries to assist in predicting the rupture risks [30, 31]. Several studies
130 suggested that a rupture site may be linked with the wall stress, itself dependent on geometric characteristics
131 including surface curvature and the asymmetry of aneurysms [12, 32–37]. There is the potential to address the
132 paucity of research into the influence of neck angulation on AAA disease progression and AAA risk of rupture,
133 through the use of a numerical model.

134 The aim of this present study was to use a three-dimensional finite volume method for CFD simulation
135 to determine the impact of severe proximal aneurysm neck angulation on the blood flow in AAAs and wall
136 shear stress (WSS) based on a patient-specific AAA geometry.

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140 **Materials and Methods**

141

142 **Image acquisition**

143 The three-dimensional (3D) vasculature was based on CT images of a single patient with the AAA
144 fully analysed. These images were acquired from radiology department under the approval of Faculty of
145 Medicine Ethics Committee, Prince of Songkla University with number (REC.61-010-25-2). The CT images
146 were obtained in a DICOM format by AQUILION PRIME (Toshiba, Japan) with single slices, rows and
147 columns of 512 x 512 pixels, a slice thickness of 3 mm and mean pixel spacing of 0.669 x 0.669. Table 1
148 presents patient's demographic information including aneurysm length, aneurysm diameter, infrarenal neck
149 length and angle at the proximal neck.

150

151 **Three-Dimensional Model Reconstruction**

152 The Three-dimensional (3D) smoothed model was generated from DICOM files by using the
153 commercial medical imaging software Mimics v18.0 (Materialise, Belgium). Mimics was used to convert the
154 acquired CT images into a patient specific 3D CAD model. The region of interest (ROI) was segmented by
155 applying grayscale-based thresholding tools. The DICOM images were cropped from the position of the infra-
156 renal aorta towards the bifurcation of common iliac arteries. The artery branches such as parietal and visceral
157 arteries were excluded from the reconstruction to reduce the complexity of the geometry. Owen et al. showed
158 that the error associated with the exclusion of small branches was smaller than the effect of the simple
159 simulation set up [31]. Examples of the thresholding and segmentation processes are shown in Fig. 1(a) and
160 1(b). Finally, the 3D smoothed geometry was generated and exported as a binary 'STL (stereolithography)'
161 format as shown in Fig. 1(c). The proximal neck angulation of patient's specific model was measured by using
162 Mimics, with measurement provided in Table 1.

163

164 **Meshing**

165 The geometry was meshed by using the Octree method in ANSYS ICEM v16.2 (ANSYS Inc., USA)
166 for tetrahedral meshing. An inflation at the wall boundary was implemented with five prism layers. The height
167 of first layer was set to 0.1 mm, and next layers grow with a size ratio of 1.2. Quality and smoothing checks
168 were repeatedly performed to ensure a satisfactory mesh. A grid-size independency study was performed using a
169 $\pm 2.5\%$ for peak velocity as the key criterion. The final selected mesh has 2,077,498 elements.

170

171

172 **Boundary conditions and material properties**

173 A finite volume method was implemented to solve the Navier-Stokes and continuity equations of the
174 fluid motion under transient conditions in ANSYS FLUENT v16.2 (ANSYS Inc., USA) solver. Blood flow was
175 assumed to be homogeneous, incompressible, and blood was modelled as a Newtonian fluid. These assumptions
176 are adequate in larger arteries with a constant dynamic viscosity and blood density of 0.0035 Pa·s and 1,060
177 kg/m³, respectively [38, 39]. Furthermore, an assumption of flow in aorta with > 0.5 mm diameter as a
178 Newtonian is acceptable since the viscosity of blood is comparatively constant at the high shear rates (100/s),
179 and this case is typically found in abdominal aortas [40, 41]. For the fluid domain, the flow of blood in vessels
180 and arteries are pulsatile [42]. Thus, a user-defined function (UDF) for a pulsatile velocity profile was used at
181 the inlet for the whole cardiac pulse cycle with a velocity magnitude between 0 and 0.3 m/s as shown in Fig. 2.
182 The inlet velocity profile was adopted from Rissland *et al.*[10]. For the outlet boundary, a fully developed
183 outflow of a zero diffusion flux boundary condition was applied at the common iliac arteries [43]. A no-slip and
184 rigid conditions for the arterial walls were assumed.

185

186 **Simulation setup**

187 All CFD transient simulations to solve the Navier-Stokes equations were carried out using ANSYS
188 FLUENT v16.2 (ANSYS Inc.) under the shear stress transport k-omega (SST $k-\omega$) turbulence model with a
189 second order implicit method for transient formulations. The pressure-velocity coupling was set as SIMPLE
190 algorithm to solve the continuity equation under 2nd order upwind momentum for spatial discretization. The
191 convergence criteria for the normalized continuity and velocity residuals were 1×10^{-5} . A fixed time step of
192 0.01s was used and three cardiac cycles ($3 \times 0.94s$) = 2.82s or 282 time-steps were completed for each simulation.

193

194 **Results**

195 The unsteady results of flow patterns (velocity contours in cross-sectional areas and streamlines) and
196 WSS are presented at four different time points of a cardiac cycle indicated by the points in Fig. 2. These time-
197 frames are (a) peak systole $t = 0.25$ s, (b) early diastole $t = 0.55$ s, (c) mid-diastole $t = 0.70$ s, and (d) late
198 diastole $t = 0.94$ s. Table 2 presents the comparison of peak systolic velocity, early diastolic velocity, WSS and
199 vorticity location between our work and previous studies that were performed on patient-specific geometries for

200 healthy and diseased (AAA) abdominal aorta. Furthermore, additional data comparing a healthy artery and an
201 angulated AAA is provided, along with a comparison of a laminar model and a turbulence model, and included
202 as supplementary material.

203

204 **Flow patterns**

205 The velocity contours in regions of interest are presented in both horizontal and longitudinal cross-
206 sectional areas as shown in Fig. 3(a). Four horizontal cross-sectional slides (A, B, D and C) in Fig. 3(b), and one
207 longitudinal cross-sectional slide (E) extended from upper neck region towards the distal area of the sac as in
208 Fig. 3(c). The contours of velocity within the horizontal cross-sectional slides show that the magnitude of
209 velocity is significantly changed over the time. The maximum velocity at the peak systole clearly seems to be
210 higher by approximately 55% than other maximum velocities over the different time points in a cardiac cycle,
211 while velocity flow among the diastolic stages show similarity with slightly difference of only 4%. However, at
212 all four time-points of a cardiac cycle the maximum blood flow occurs near the inner wall of the aorta, but
213 cross-section of slide D views maximum blood flow near both inner and outer walls with local average velocity
214 (0.15 m/s). The flows within slides C and D tend to form a circular shape within the aortic sac that can cause a
215 high blood recirculation while maintaining a low velocity at the center of aorta with approximately 0.04 m/s.

216 Figure 3c emphasizes the velocity flow starting from upper the neck bending region towards the distal
217 sac of aneurysm represented by the square box for the ROI. At a peak systolic time of 0.25 s, the velocity of
218 flow entering the proximal neck of aneurysm towards the sac increased and led to an impingement of blood flow
219 on the outer wall of aorta, subsequently diminishing through diastolic phase. At a full cardiac cycle of 0.94 s,
220 high velocity flow is observed on both sides of the aneurysm sac which appears to coil up in this sectional view.
221 The streamlines of velocity flows are presented in Fig. 4. The swirling of instantaneous velocity streamlines was
222 acquired at different time points of a cardiac cycle as displayed in the ROI around the angular neck AAA. The
223 recirculation blood vortexes are easily recognizable in various patterns over time.

224

225 **WSS distribution**

226 The WSS distribution of four different time points of a cardiac cycle configuration (peak systole, early
227 diastole, mid diastole and late diastole) for high proximal neck angulation of AAA is depicted in Fig. 5. The
228 WSS distributions are illustrated in three different views. View 1 and 2 show the WSS distribution at the regions
229 of proximal aneurysm neck and view 3 illustrates the WSS distribution on the surface of aneurysm sac. We can

230 observe that the high WSS of 1.24 Pa occurs at the area of proximal neck due to the turbulent flow exhibited
231 within the region of angulation. The high bending, the severe tortuosity of aortic surface and asymmetric blood
232 flow seem to be possible indicators of WSS and aortic rupture. At the peak systole and a fully developed cardiac
233 cycle as in Fig. 6, high WSS regions are located at the areas below the angular neck and over the aneurysm sac
234 as indicated by the red arrows with average value of 0.94 Pa, while the locations of low WSS with the average
235 of 0.077 Pa are indicated by the black arrows. Furthermore, the values of WSS vary between the healthy subject
236 and AAA patients. In the AAA patients, WSS is lower than WSS in the healthy subjects as presented in Table 2.

237

238 **Discussion**

239 In this study, three-dimensional computational fluid dynamics simulations of a severe angulation neck
240 of patient-specific AAA has been used to assess time-dependent hemodynamic. The three-dimensional geometry
241 of angular neck AAA was reconstructed from computed tomography images. More specifically, the impact of
242 high angular neck AAA on blood flow and wall shear stress (WSS) were assessed for an angle ($> 60^\circ$);
243 particularly important due to the lack of studies in this area [22], where previous studies focused on smaller
244 proximal angles ($\leq 60^\circ$) or using idealized geometries [6, 17, 22, 43, 44].

245 Our study demonstrated the hemodynamic changes occur more pronounce at peak systole and
246 turbulence flow was generated at the neck throughout the aneurysm sac during a cardiac cycle. In this study, the
247 presence of a bending angle greater than 60° caused high flow turbulence and irregularities of blood-flow
248 streamlines. This indicates that WSS and their distribution will be altered, with potential impact on weakening
249 of arteries wall [43, 44].

250 The flow patterns at the systolic stage were observed to have complex and high velocity values within
251 the proximal neck, while these maximum velocity values seemed to be decreased at the early and mid-diastole
252 stages before it increased again at a complete cardiac cycle. As shown in Table 2, it is noted that the normal
253 aorta model has higher peak systolic velocity than the AAA model. Vortex formation can be observed in
254 geometries from AAA patients, whereas they cannot be observed in healthy aortas unless there is surface
255 curvature, where only minimal recirculation may occur. The impact of proximal angular neck on blood flow
256 within aneurysm sac was clearly showed to form a complex recirculation and flow impingement. This impact
257 demonstrated a clear difference between the flow in AAA with an angulated proximal neck and without an
258 angulated proximal neck. When the proximal neck is straight, the blood flows can be observed to follow laminar

259 flow (i.e. not cross over streamlines) within the aneurysm sac with a very small region of recirculation [45]. It
260 also showed that the velocity flow within the aorta was observed to have vorticity flow and recirculation
261 particularly through the aneurysm sac and aortic bifurcation, and these findings are consistent with idealized and
262 real geometries in prior studies [31, 43, 44, 46], but our study reveals more complex recirculation and vorticity
263 due to highly angulated neck and complexity of patient specific AAA geometry. Furthermore, a larger diameter
264 for ruptured AAAs was associated with greater recirculation flow whereas less recirculation was found in
265 smaller ruptured AAAs [47].

266 Several factors that influence the hemodynamic and the biomechanical conditions of arteries in
267 cardiovascular system. For instance, vascular geometry, elasticity of the wall, blood viscosity and pathological
268 conditions [48, 49]. Xenos et al.[17] conducted numerical simulations for 26 idealized geometries based on
269 patient-specific data by using Fluid-Structure Interaction (FSI) simulations to investigate the effect of proximal
270 necks ($40.10\pm 16.30^\circ$) in AAA. Correspondingly, Drewe et al. [6] studied similar range of neck angles in Xenos
271 et al.[17] for idealized geometries in order to observe the stresses and hemodynamics. Both studies reported that
272 peak WSS seems to be increased with the increase of proximal neck angles. However, the smaller angle of
273 necks in their studies predicted peak WSS in the middle region of aneurysm sac due to the less turbulence of
274 blood flow generated, while our findings with a larger neck showed WSS can be located more diffuse across
275 areas such as below the proximal neck, middle of sac as well as at the lower side of the aneurysm sac wall. It has
276 been reported that high WSS can promote endothelial injury, while low WSS can lead to inflammatory
277 infiltration [50]. Therefore, this study has predicted a link between the behavior of blood flow and the change of
278 WSS distribution. This correlation is consistent and demonstrates agreement with a previous study conducted by
279 Arzani and Shadden [51].

280 The SST $k-\omega$ model was used in this study [46, 52]. According to Banks et al.[53] who found that this
281 model was preferred for CFD turbulent flow simulations in arteries due to its better performances from other
282 turbulence models when comparing the simulation outcomes against the results of experimental data.
283 Furthermore, this turbulent model showed a good performance for the flow at boundary layers close to the wall,
284 without applying a function of wall enhancement [53]. Therefore, it was observed that SST $k-\omega$ model was the
285 most suitable method that provides better comparisons against the experimental results [54], it can be used for
286 transitional flows for low Reynolds number. In addition, both laminar and turbulence models for AAA
287 simulation show the formation of vortices within the aneurysm sac, which is similarly found in our study as
288 presented in Table 2.

289 It should be noted that only one patient with a severe angular neck was studied. Following our study,
290 we believe that it would be beneficial to increase the number of subjects to assess a wider range of proximal
291 angular necks. However, this study demonstrates the effect of geometrical features based on realistic time-
292 varying velocity waveform, which is can be considered for personalized healthcare. It is appropriate also to
293 mention that this study implemented outflow boundary conditions at outlets which used the same waveform at
294 the inlet of AAA section. This assumption is not expected to alter the overall findings as regards AAA neck
295 angle and altered hemodynamics. Furthermore, it is worthy to point out that a possible thrombus was not
296 included in this study. The presence of intraluminal thrombus (ILT) encourages the change of geometrical
297 features that can consequently influence the biomechanics of AAA [55]. However, ILT was not involved in a
298 scope of our study.

299

300 **Conclusions**

301 To summarize this work, the simulation concluded that the tortuosity of the aortic neck angulation
302 causes a downstream of blood flow to be a turbulent flow and leads a weakening of the aortic wall, resulting in
303 forming locations of high WSS. Thus, this study presented a comprehensive idea on the behavior of blood flow
304 in highly angulated abdominal aortic aneurysm necks and its influence on wall shear stress. Furthermore, we
305 recommend that more cases of patient-specific geometries are necessary to study the wider effect of angularity
306 of the proximal neck on blood flow and subsequent hemodynamic changes in abdominal aortic aneurysm sac
307 and aortic bifurcation.

308

309 **Compliance with Ethical Standards**

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316 **Conflict of interest** The authors declare that they have no conflict of interest.

317 **Ethical approval:** This article does not contain any studies with human participants or animals performed by
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320 25-2.

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Table legends

Table 1 Patient demographics and geometry dimensions.

Table 2 Comparison between present study and previous studies in terms of laminar and turbulence models for healthy abdominal aorta and abdominal aortic aneurysm (AAA) geometries.

Figure legends

Fig. 1 Overall geometry reconstructions process, (a) CT image for the whole aorta, (b) the thresholding mask for aorta in the axial, coronal and sagittal view, and (c) 3D geometry for AAA after reconstruction, inner and outer walls are indicated by arrows to represent both side of abdominal aorta.

Fig. 2 Velocity waveform profile imposed at the inlet. (a) peak systole at 0.25 s; (b) early diastole 0.55 s; (c) mid diastole 0.70 s; and (d) late diastole 0.94 s.

Fig. 3 Velocity contours at different horizontal cross-sectional areas for the angular neck AAA and aortic sac indicated by letters: (a) the four different locations in the geometry; (b) comparisons of the magnitude of the flow velocity at different time points in a cardiac cycle; (c) the vertical cross-sectional area of the model from proximal neck to lower region of sac.

Fig. 4 Flow streamline contours at four different time points in a cardiac cycle.

Fig. 5 WSS distribution on the angular neck and aneurysm sac regions at different time points in a cardiac cycle.

Fig. 6 WSS distribution for two time-points (0.25 s and 0.94 s) in a cardiac cycle. The high WSS regions are located with red arrows with average value of 0.94 Pa, while the locations of low WSS with the average of 0.077 Pa are indicated by the black arrows.