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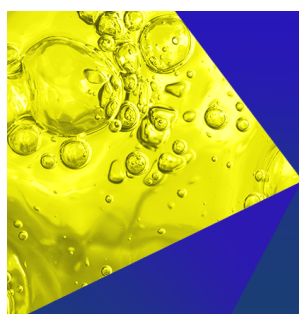
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# Impact of temporary common carotid artery strangulation on hemodynamics during surgical clipping of middle cerebral bifurcation aneurysms

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## ABSTRACT

Intracranial aneurysms (IAs) involve weakening and abnormal ballooning of blood vessel walls. To safely perform surgical clipping on IAs, reducing aneurysmal pressure is crucial. While “temporary clipping” has conventionally been a fundamental technique in aneurysm-clipping surgery, it presents several challenges. In response, we suggested and investigated a new method termed “common carotid artery (CCA) temporary strangulation,” which involves the temporary strangulation of the ipsilateral common carotid artery (CCA) to decrease pressure in the middle cerebral artery (MCA) aneurysms. This study utilized computational fluid dynamics simulations with SimVascular, incorporating large-scale vascular geometries extending from the aorta to the circle of Willis. The boundary conditions were established using a pulsatile inlet flow rate and an outlet condition based on the resistance–capacitance–resistance Windkessel model, with CCA strangulation modeled at 0%, 75%, 85%, and 95%. Our results indicate that CCA strangulation significantly reduces ipsilateral MCA aneurysmal pressure and blood flow in the second segment of the MCA (M2). Specifically, at 95% CCA strangulation, the mean aneurysmal pressure decreased by 50%, accompanied by a 50% reduction in blood flow. Furthermore, our analysis indicates that the anterior communicating artery plays a critical role in modulating changes in aneurysmal pressure. A regression model that used the reduction in M2 segment velocity as a predictor for aneurysmal pressure achieved a high coefficient of determination, suggesting its reliability and clinical applicability. These findings suggest that CCA temporary strangulation could considerably enhance surgical safety by effectively managing intra-aneurysmal pressure, providing a viable tool for patient-specific surgical strategies.

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## I. INTRODUCTION

Intracranial aneurysms (IAs) are pathological dilations that occur in the arteries within the brain.<sup>1</sup> They develop because of the weakening of arterial walls, which become unable to withstand the hemodynamic pressure.<sup>2</sup> Autopsy studies indicate that up to 5% of the population may have undetected brain aneurysms.<sup>3,4</sup> In addition, ruptured aneurysms are responsible for approximately 85% of nontraumatic subarachnoid hemorrhages (SAHs).<sup>5,6</sup> An SAH is a severe and life-threatening form of stroke, with a case fatality rate of approximately 50% following an aneurysmal subarachnoid hemorrhage (aSAH).<sup>5,7</sup>

IAs can occur in various regions of the cerebral vasculature. The most common sites include the anterior communicating artery

(Acom, 30%), the bifurcation of the middle cerebral artery (MCA, 20%), and the posterior communicating artery (Pcom, 25%).<sup>8</sup>

Two primary treatment modalities are available for IAs: surgical (or microsurgical) clipping and endovascular coiling. Clipping is an invasive technique requiring a craniotomy, during which a clip is placed across the aneurysm’s neck to stop blood inflow into the dilated vessel. In contrast, coiling is a minimally invasive approach in which a catheter is inserted through a peripheral artery into the cerebral circulation, and a coil is subsequently placed into the aneurysm, inducing clotting and occluding the aneurysm.<sup>9</sup>

Microsurgical clipping is often the preferred treatment for MCA bifurcation aneurysms because of their specific location within the vascular tree and the frequent occurrence of large-neck morphologies in

these aneurysms.<sup>10</sup> This method has demonstrated a lower risk of long-term recurrence and a reduced likelihood of retreatment compared to coiling.<sup>11</sup> However, one significant risk associated with microsurgical clipping is a potential aneurysm rupture during the procedure, induced by elevated intra-aneurysmal pressure. This is a reflection of the fact that numerous medical studies<sup>12–14</sup> have identified high blood pressure as a major risk factor for intracranial aneurysm ruptures and aSAH.

Until now, temporary vessel occlusion of the parent artery, termed “temporary clipping,” has been a critical technique used in aneurysm-clipping surgery.<sup>15,16</sup> This method aims to reduce the pressure within the aneurysm, thus minimizing the risk of rupture during surgical intervention. However, temporary clipping poses multiple challenges.<sup>17–19</sup> For example, Akyuz *et al.*<sup>18</sup> reported that temporary clipping may adversely affect cognitive functions and frontal lobe operations. Moreover, Araki *et al.*<sup>17</sup> noted that temporary clipping exceeding 20 min could lead to postoperative hyperperfusion, which highlights the risks associated with prolonged application. Temporary occlusion narrows the safe window for surgical manipulation and poses several risks, especially in patients with large aneurysms, complicating the location and exposure of the clipping site. Repeated clippings can induce atherosclerotic changes in the parent vessel, vasospasms, or vessel injury.

To address these limitations, in this study, we examined an alternative method: reducing aneurysmal pressure by temporarily strangling the common carotid artery (CCA) on the ipsilateral side of the IAs to clip the aneurysm’s neck. This technique potentially simplifies surgical procedures (particularly for sizable aneurysms at complex sites such as the MCA bifurcation), extends the safe operational period, and eases the management of premature ruptures.

The aim of this study was to assess the efficacy of the “CCA temporary strangulation” technique in decreasing aneurysmal pressure and to evaluate its impact on cerebral blood flow within the circle of Willis (CoW). To accomplish this, we quantitatively evaluated hemodynamic alterations in the ipsilateral MCA (iMCA) bifurcation aneurysm under different degrees of CCA strangulation using computational fluid dynamics (CFD). We tested four strangulation ratios (0%, 75%, 85%, and 95%) under two scenarios: variations in the anatomy of three patients and different CoW configurations. The CoW configurations were categorized into four types: a complete CoW, absence of the ipsilateral Pcom (iPcom), absence of the Acom, and absence of both the iPcom and Acom.

Patient-specific three-dimensional (3D) models spanning from the aorta to the CoW were utilized for large-scale simulations, which facilitated the accurate analysis and predictions of the changes in the aneurysmal pressure and blood flow. Additionally, we developed a regression model to predict the aneurysmal pressure based on velocity decreases in the second segment of the MCA (M2), a clinically measurable parameter critical for making surgical decisions. These results demonstrate the potential of CCA temporary strangulation for enhancing surgical safety via the effective control of the intra-aneurysmal pressure, as the predictive models provide actionable insights that are helpful for planning individualized treatments.

## II. METHODS

### A. Patient-specific, 3D hemodynamics

Patient-specific computed tomography angiography (CTA) data from a previous database study<sup>20</sup> were sourced from the Institutional

Review Board (IRB)-approved Screening Technology and Outcome Project in Stroke (STOP-Stroke) database. The selected images covered most of the upper-chest arterial domain, extending from the aorta to the CoW. All images were acquired by following protocols approved by the IRB, as outlined in Smith *et al.*<sup>20</sup> We selected three patient models identical to those used in Kang *et al.*,<sup>21</sup> and we modified these models to align with our specific research objectives.

### B. Modeling procedure

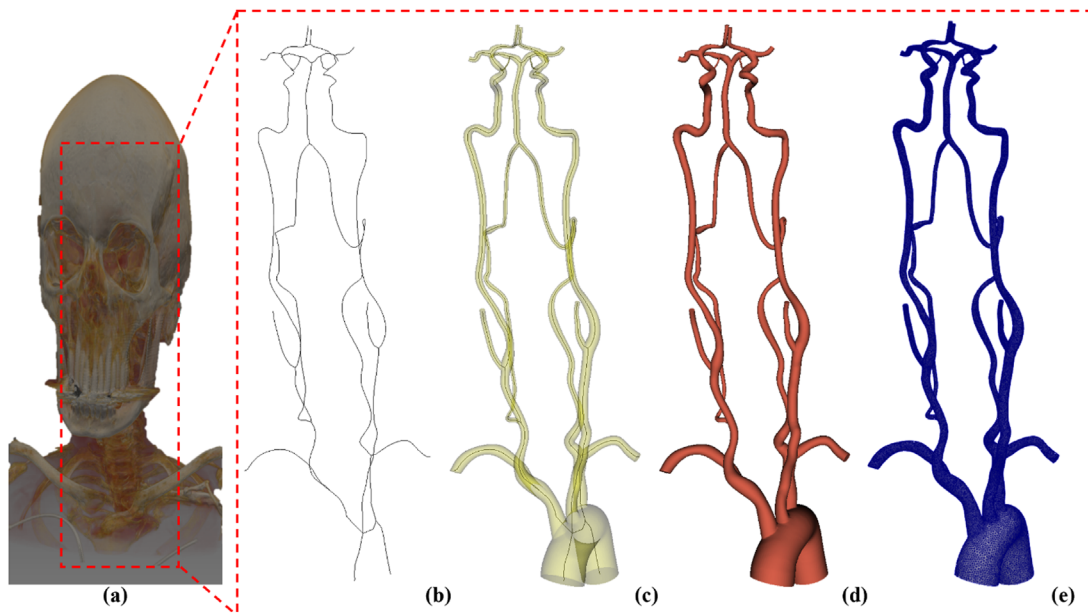
The 3D computational model was manually reconstructed from CTA images using the open-source hemodynamics simulation package SimVascular.<sup>22</sup> Figure 1 shows the modeling procedure used in this study. Initially, a series of two-dimensional slice images were compiled to create 3D volume-rendered images, identifying vessels and locating the cerebral arteries using volume visualization techniques, as shown in Fig. 1(a). A path was then established through the visualized volume to connect the midpoint of each blood vessel, as shown in Fig. 1(b). Next, segmentation was performed based on the established path, as shown in Fig. 1(c). Finally, the model was finalized by lofting the segmentation and smoothing out sharp areas (e.g., artery bifurcations), as shown in Fig. 1(d).

The normal CoW without M2 arteries and an MCA aneurysm, as described in a previous study,<sup>21</sup> served as the reference model. This reference model included all major arteries from the aorta to the CoW, encompassing the internal carotid artery (ICA), MCA, anterior cerebral artery (ACA), and posterior cerebral artery (PCA), among others. Figure 2 shows the modified model, in which M2 arteries and an MCA bifurcation aneurysm were added to the reference model of patient number one (P01). M2 arteries were added bilaterally to the MCA, and an aneurysm was created at its bifurcation (right side, ipsilateral). Similar models (P02 and P03) were created using the same procedure. Initially, the aneurysm was modeled with a diameter of approximately 6 mm, following the findings of Maslehaty *et al.*<sup>23</sup> However, given that the risk of rupture increases significantly when the size of the aneurysm exceeds 10 mm, and the aspect ratio (AR) is greater than 1.6,<sup>24</sup> the diameter was adjusted to approximately 13 mm, with an AR of approximately 2.3, simulating the typical size of a large aneurysm (10–25 mm)<sup>25,26</sup> and representing a feasible value for analysis. To validate the aneurysm modeling, we compared the ratios of the dome height (H), dome diameter (D), and neck width (N) to reference values reported by Parlea *et al.*<sup>27</sup> The ratios for our models were within the reported reference ranges:

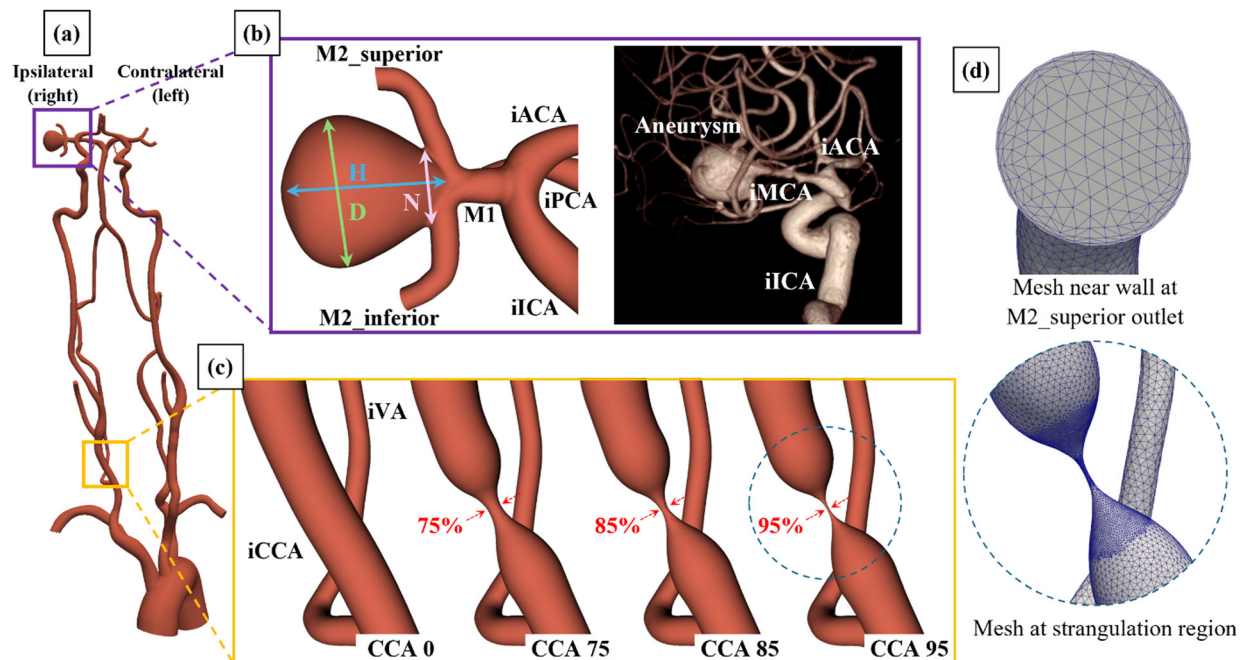
- D/H (Ref:  $1.11 \pm 0.29$ ) → P01: 0.93, P02: 0.98, P03: 0.97
- D/N (Ref:  $1.91 \pm 0.86$ ) → P01: 2.11, P02: 2.12, P03: 2.32
- H/N (Ref:  $1.86 \pm 0.86$ ) → P01: 2.27, P02: 2.17, P03: 2.39

These comparisons confirmed that the geometric characteristics of our aneurysm models were consistent with previously established data.

As shown in Fig. 3, strangulation was applied to the CCA using a vessel loop.<sup>28</sup> Using the carotid bifurcation area or the proximal ICA as the strangulation site was not ideal, as these regions are anatomically deeper. During the surgical approach, additional time for dissection is required owing to the surrounding structures (e.g., veins and nerves), increasing both the procedural complexity and risk of tissue damage. Furthermore, the closer the approach is to the aorta, the deeper the CCA is located, rendering dissection even more challenging.



**FIG. 1.** Modeling procedure: (a) Volume-rendered image of CTA and volume visualization. (b) Path generation. (c) Segmentation and lofting along the centerline. (d) Modeling. (e) Finite-element mesh generation.



**FIG. 2.** (a) Schematic of simulation model (P01, CCA 0). (b) Aneurysm modeling and actual computed tomography image of right MCA bifurcation aneurysm. The aneurysm was modeled using a relatively large size with the following dimensions: D: 12.95 mm, H: 13.9 mm, and N: 6.13 mm. In the MCA, the M1 segment starts at the bifurcation of the ICA, and the M2 segment begins at the bifurcation of the M1 segment. (c) Location of the CCA strangulation and the process of reducing the diameter of the CCA by 0% (normal), 75%, 85%, and 95%. (d) Mesh near the wall of the MCA, M2 superior outlet, and mesh in the 95% strangulation region. (Abbreviations – ICA: internal carotid artery; ilCA: ipsilateral internal carotid artery; iACA: ipsilateral anterior cerebral artery; iPCA: ipsilateral posterior cerebral artery; iCCA: ipsilateral common carotid artery; iVA: ipsilateral vertebral artery; M1: first segment of the MCA; M2: second segment of the MCA).

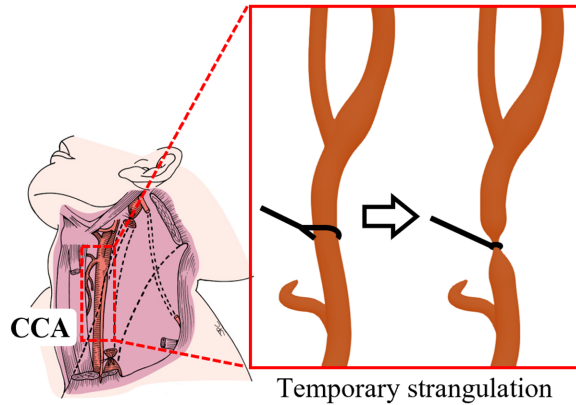


FIG. 3. Schematic representation of CCA temporary strangulation using a vessel loop.

Considering the superficial location of the CCA in the neck<sup>29</sup> and its shortest average distance to the skin,<sup>30</sup> the optimal strangulation site was determined to be 2 cm below the bifurcation of the CCA.

The reduction in the CCA diameter was calculated using the North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria,<sup>31</sup> which is expressed as

$$\text{NASCET} = 1 - \frac{D_s}{D_n} \times 100(\%), \quad (1)$$

where  $D_n$  represents the normal distal diameter of the artery and  $D_s$  denotes the diameter at the point of strangulation. Although precisely determining the extent of strangulation in actual surgery is clinically challenging, the simulation included scenarios of 0%, 75%, 85%, and 95% strangulation, as shown in Fig. 2(c). For patient 1 (P01), when 95% strangulation was applied, the smallest segmented area of the CCA measured  $0.05 \text{ mm}^2$ . For patients 2 (P02) and 3 (P03), the smallest areas were  $0.15$  and  $0.1 \text{ mm}^2$ , respectively (both also under 95% strangulation). These results demonstrated that the simulation was capable of modeling multiple scenarios from normal conditions to near-complete occlusion with 95% strangulation.

### C. Numerical scheme

For the computational analysis, the governing equations we used were the Newtonian, incompressible, and 3D Navier–Stokes equations. Although blood is inherently a non-Newtonian fluid, modeling it as Newtonian and incompressible in the context of large and cerebral arteries, which our model contains, is appropriate.<sup>32,33</sup> Previous studies have indicated that non-Newtonian properties are critically important for small arteries (radius  $< 0.1 \text{ mm}$ ).<sup>34</sup> However, because most arteries in our model are within the medium-sized range (2–6 mm) or larger, a Newtonian approximation is reasonable.<sup>35–37</sup> The viscosity and density were set to  $0.04 \text{ g}\cdot\text{s}^{-1}\cdot\text{cm}^{-1}$  and  $1.06 \text{ g}\cdot\text{cm}^{-3}$ , respectively, which approximated the corresponding values of actual human blood.<sup>38,39</sup> Despite the heterogeneous composition of blood (comprising red blood cells, plasma, platelets, and white blood cells), it was assumed to be homogeneous for simplicity, and the vessel walls were modeled as rigid. Given that our model spanned the aorta-to-cerebral vasculature, the rigid wall assumption was adopted to manage the computational

cost. This assumption was widely used in previous studies for both large-scale hemodynamic analyses and IAS simulations.<sup>21,35–37,40–42</sup>

Under the assumptions of three-dimensionality, incompressibility, Newtonian fluid dynamics, and rigid walls, the Navier–Stokes equations are expressed as

$$\nabla \cdot \mathbf{u} = 0, \quad (2)$$

$$\rho_f \left\{ \frac{\partial \mathbf{u}}{\partial t} + (\mathbf{u} \cdot \nabla) \mathbf{u} \right\} = -\nabla p + \nabla \tau + \rho_f \mathbf{g}, \quad (3)$$

$$\tau = 2\mu_f D(\mathbf{u}) = \mu_f (\nabla \mathbf{u} + \nabla \mathbf{u}^T), \quad (4)$$

where Eqs. (2)–(4) represent the continuity equation, the Navier–Stokes equation, and the constitutive stress–strain law, respectively;  $\mathbf{u}$ ,  $p$ ,  $\rho_f$ , and  $\mu_f$  denote the velocity fields, pressure fields, density of blood, and viscosity of blood, respectively;  $\mathbf{g}$  is the acceleration due to gravity; and  $\tau$  is the shear stress. To solve for the velocity and pressure fields, a stabilized finite-element method was employed, utilizing the streamline upwind Petrov–Galerkin (SUPG) and pressure-stabilizing Petrov–Galerkin (PSPG) formulations.<sup>43,44</sup> These stabilization techniques are critical for achieving numerical stability and accuracy in solving the continuity, mass, and momentum equations, as detailed in previous studies.<sup>45–47</sup> The formulations were implemented within SimVascular, an open-source computational package,<sup>22,48</sup> and the accuracy of which has been validated for patient-specific boundary conditions.<sup>36,49,50</sup>

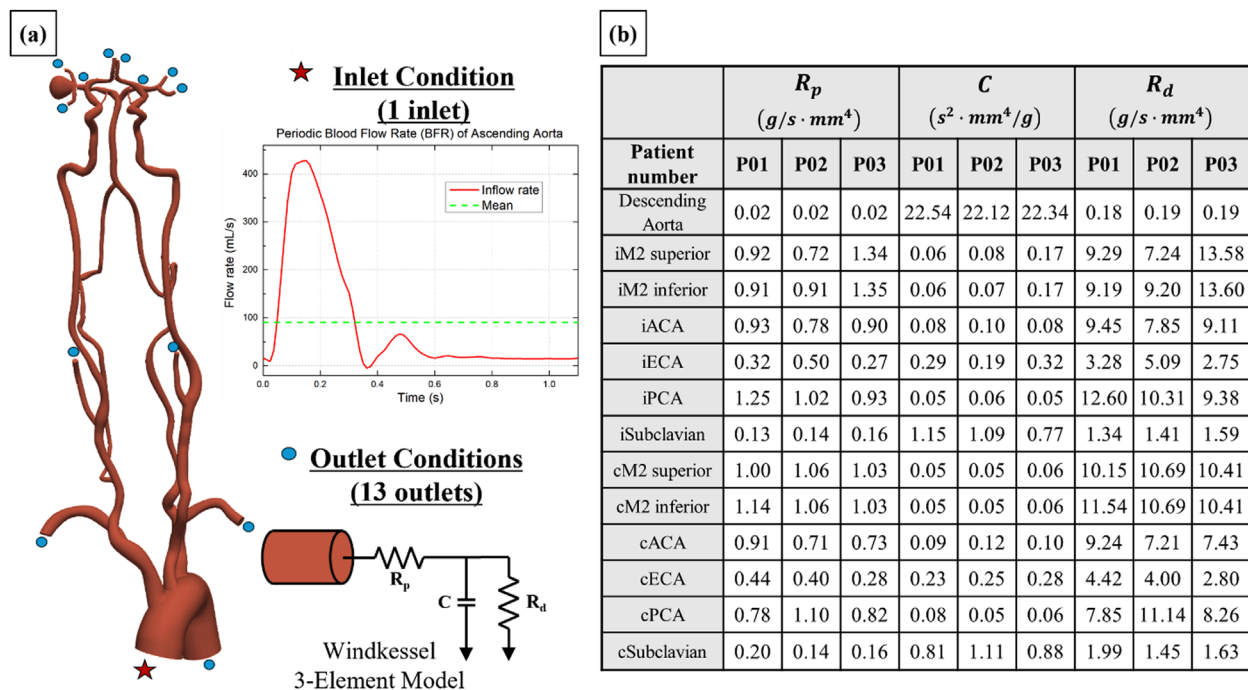
### D. Boundary conditions

The inlet condition utilized a pulsatile flow rate in the upper ascending aorta, as performed in a previous study.<sup>51</sup> For the inflow condition, a prescribed velocity profile was used, offering options such as a plug, parabolic, or Womersley profile. The plug-type velocity profile was determined to be the most appropriate assumption for modeling the flow in the ascending aorta. Considering the limited boundary-layer development, entry flow effects, and vessel geometry, the plug-type profile provides the most physiologically accurate and simplified representation of blood flow in this region.<sup>52–54</sup> The duration of the pulsatile flow was set to 1.1 s.

For the outlet boundary conditions, a Windkessel resistance–capacitance–resistance (RCR) model was adopted. This model comprises two resistances—the proximal resistance ( $R_p$ ) and the distal resistance ( $R_d$ )—and a single capacitance ( $C$ ), effectively representing the downstream vascular impedance. The same inlet flow conditions were consistently applied across all three patients, and the outlet RCR parameters were adjusted according to each patient’s specific pressure and flow rates. The RCR values for all three patients were calculated and are detailed in the table shown in Fig. 4(b). The calculations used the formulas

$$R_i = \frac{\sum \sqrt{A_i^k}}{\sqrt{A_i^k}} \times R_{tot}, \quad C_i = \frac{A_i}{\sum A_i} \times C_{tot}, \quad (5)$$

where  $R_i$  and  $C_i$  represent the resistance and capacitance of each outlet, respectively;  $A_i$  represents the outlet area; and  $k$  is Murray’s law coefficient.<sup>55</sup> The value of  $k$  can vary depending on the physiological conditions and vascular location; therefore, we set  $k = 3$  to account for the outlet conditions in small-diameter vessels such as the cerebral arteries.<sup>56,57</sup> The total resistance ( $R_{tot}$ ) and total capacitance ( $C_{tot}$ ) were defined as  $R_{tot} = \text{MAP}/Q$  and  $C_{tot} = \Delta Q(t)/\Delta P(t)$ , respectively,



**FIG. 4.** Overview of the boundary conditions and patient-specific RCR values. (a) Graph illustrating the periodic blood flow rate used for the inlet pulsatile flow condition applied at the ascending aorta for all three patients, with the Windkessel RCR model applied at 13 outlets. (b) Table listing the RCR values calculated for all three patients, which were adjusted based on individual pressure and flow rates. (Abbreviations –  $R_p$ : proximal resistance;  $C$ : capacitance;  $R_d$ : distal resistance; ECA: external carotid artery; ACA: anterior cerebral artery; PCA: posterior cerebral artery; i: ipsilateral; c: contralateral).

where  $MAP$  is the mean arterial pressure,  $Q$  is the mean volumetric flow, and  $P(t)$  is the time-dependent pressure. After calculating the total resistance for each outlet, the ratio  $\alpha$  was used to distribute the proximal resistance ( $R_p$ ) and distal resistance ( $R_d$ ) according to  $R_t = R_p + R_d$ , where  $R_d = \alpha \times R_t$ . The value of  $\alpha$  was set to a constant value of 0.91 (Ref. 58) across a total of 13 outlets.

These boundary conditions effectively captured the pressure–flow relationship at each outlet by accounting for the characteristics of the downstream vascular beds. Impedance and Windkessel-type boundary conditions were implicitly coupled to the 3D computational domain by prescribing pressure in a weak form, as detailed in a prior study.<sup>59</sup>

Simulations were conducted over six cardiac cycles with a time step of 0.001 s until the results converged, and the final cardiac cycle was used for the analysis. The time step was chosen to ensure that the Courant–Friedrichs–Lewy (CFL) number remained below 1, and previous studies<sup>21,36,37</sup> have demonstrated the validity of this time step and cycle count. Computations were performed on a workstation equipped with an AMD 3970X CPU (3.69 GHz, 32 cores) and 128 GB of RAM, and the runtime for each case was approximately five days.

### E. Grid independence test

For these simulations, each model was discretized into a computational mesh composed of approximately  $7.5\text{--}9.0 \times 10^6$  linear tetrahedral elements, with element sizes ranging from 0.36 to 0.52 mm. This mesh design was based on methodologies similar to those used by Kang *et al.*<sup>36</sup> and Mukherjee *et al.*<sup>35</sup> The inclusion of the MCA M2 segment and the IA necessitated a minor increase in grid density in these

specific areas. The meshes were generated using the TetGen software tool, which is integrated within the SimVascular package. Boundary-layer meshing was applied near vessel walls to ensure fine, high-quality results.

To assess grid independence, the CCA 95 case of P01, which exhibited the highest Reynolds number and the most dynamic flow conditions, was selected as the test model. We analyzed three different grid resolutions:  $7 \times 10^6$ ,  $9 \times 10^6$ , and  $13 \times 10^6$  elements. This evaluation focused on two key hemodynamic markers: mean arterial pressure (MAP) within the MCA bifurcation aneurysm and peak systolic velocity (PSV) across the entire model. The results indicated that the differences in MAP and PSV between the  $9 \times 10^6$  and  $13 \times 10^6$ -element grids were less than 1%. Therefore, the  $9 \times 10^6$ -element grid was chosen for further simulations as it provided an optimal balance between computational accuracy and cost.

### F. Validation

In this study, a three-step process was implemented to validate the simulation results. Initially, the blood flow rates (mL/min) in the ICA and vertebral artery were measured at their midpoints for the normal cases of the three patients, and these results were compared to the flow ranges reported in previous studies that measured actual patient data.<sup>60–62</sup> As shown in Fig. 5(a), while variations existed among the reported ranges because of differences in vascular morphology and cardiac output across patients, our simulation data generally aligned with these reported ranges, demonstrating consistency with prior studies.

Second, we assessed the flow rates at the outlets of the main cerebral arteries—the ACA, MCA, and PCA—within the CoW. As shown in Fig. 5(b), the simulation results were consistent with the flow ranges reported in a previous study.<sup>63</sup> A minor discrepancy was noted in the contralateral PCA (cPCA) flow rate, with an error margin of less than 10%. This variation was considered acceptable, given the inter-patient differences in the flow rate and pressure.

Finally, the CCA strangulation condition was validated by comparing the MAP above the ipsilateral ICA (iICA) before and after CCA strangulation. Although prior studies<sup>64,65</sup> have primarily examined CCA occlusion rather than strangulation, the 95% strangulation condition in this study closely resembled full occlusion. The results for the 95% CCA strangulation condition were compared to those for CCA occlusion from previous studies. As shown in Fig. 5(c), the simulation results were well within the expected ranges documented in the literature.

G. Simulation cases

This study included 24 simulation cases, as summarized in Fig. 6. Twelve cases were designed to investigate inter-patient differences in

the response to various degrees of CCA strangulation. The remaining twelve cases examined the impact of CoW variations under similar conditions, including scenarios without the Acom, without the iPcom, and without either.

P01 was used to investigate the effects of CoW variations, focusing specifically on the presence or absence of communicating arteries. The impact of the contralateral Pcom (cPcom) was not considered, as it had a negligible effect on the changes in the ipsilateral cerebral blood flow following ipsilateral CCA (iCCA) strangulation.

Each simulation case was characterized by a combination of the CCA condition and the degree of strangulation (%). The baseline (normal) case was labeled as “CCA 0.” Cases with 75%, 85%, and 95% CCA strangulation were labeled as “CCA 75,” “CCA 85,” and “CCA 95,” respectively.

III. RESULTS

A. Variation of velocity

1. iICA and contralateral ICA (cICA)

Figures 7(a) and 7(b) show the changes in the blood flow velocity for four cases under systolic conditions for P03. Under

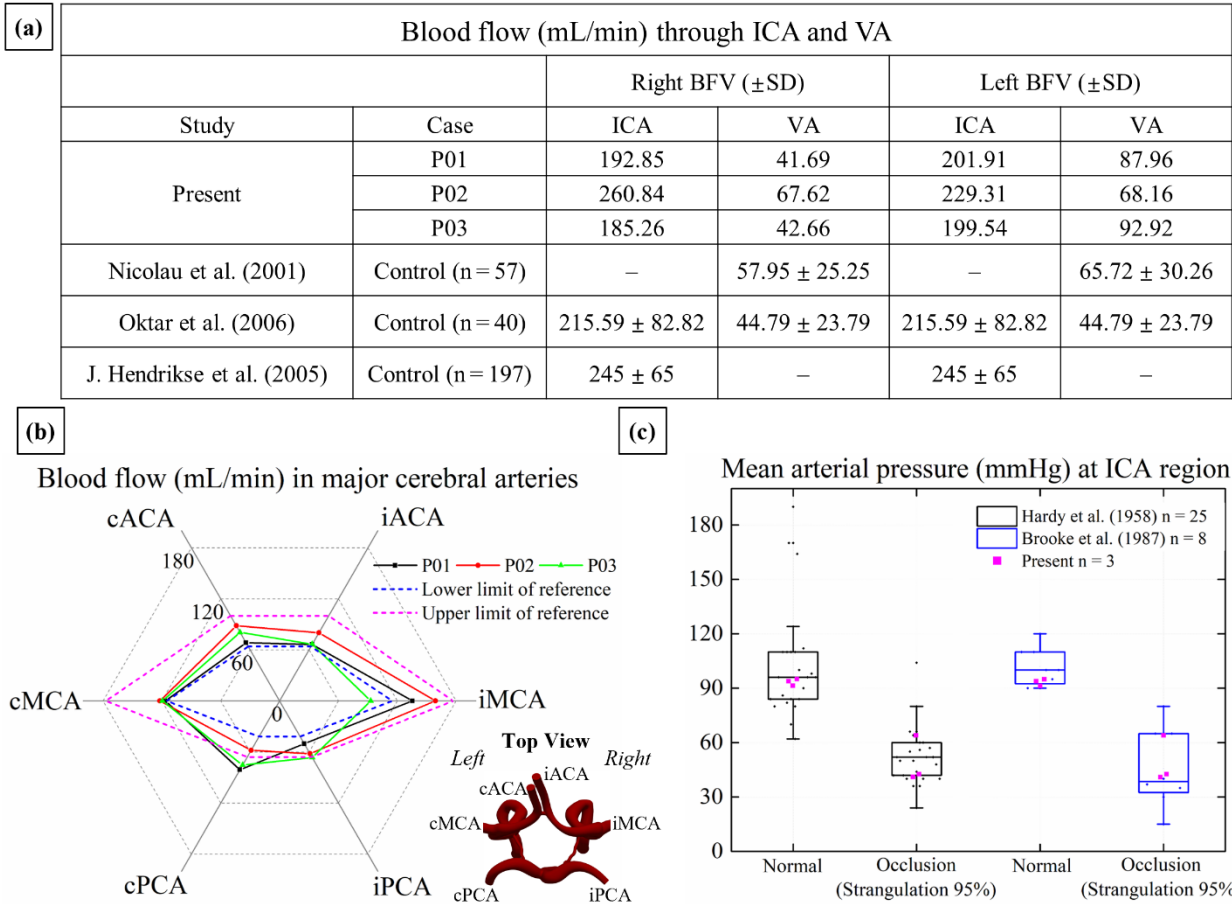
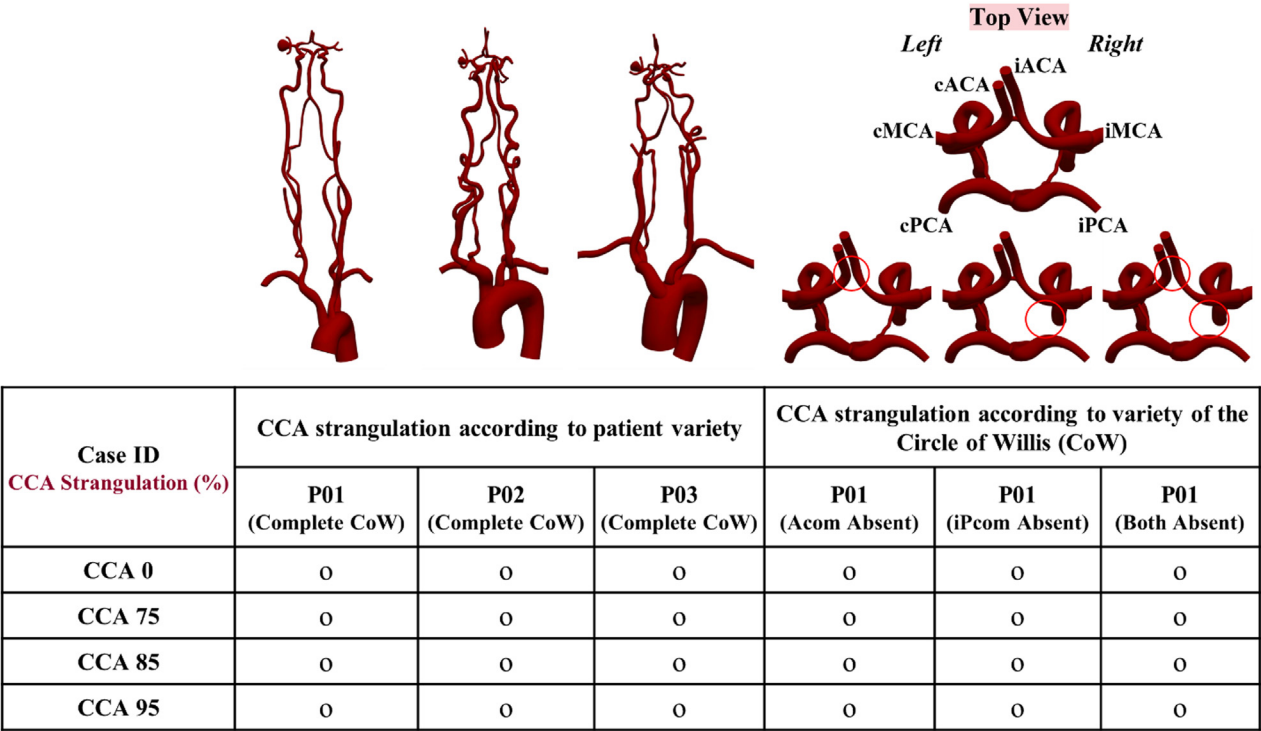


FIG. 5. (a) Comparison between the actual blood flow volume through the ICA and VA, experimental data from previous studies, and the simulation data from this study. (b) Radar chart showing a comparison between the blood flow ranges in the major cerebral arteries from previous studies and the simulation results from this study. (c) Comparison between the mean arterial pressure values in the ICA during CCA strangulation from this study and the mean arterial pressure values in the ICA when the CCA was occluded from previous studies. (Abbreviations – BFV: blood flow volume).



**FIG. 6.** Overview of the 24 simulation cases; 12 cases compared the inter-patient differences and 12 cases analyzed the CoW variations, both in response to various CCA strangulation ratios. The CoW variations were categorized into four configurations: complete CoW, absence of Acom, absence of the iPcom, and absence of both Acom and iPcom. P01 was used as the reference case for the CoW variations, and the presence or absence of communicating arteries was the sole variable. (Abbreviations – CoW: circle of Willis; CCA: common carotid artery; iACA: ipsilateral anterior cerebral artery; cACA: contralateral anterior cerebral artery; iMCA: ipsilateral middle cerebral artery; cMCA: contralateral middle cerebral artery; iPCA: ipsilateral posterior cerebral artery; cPCA: contralateral posterior cerebral artery; Acom: anterior communicating artery; iPcom: ipsilateral posterior communicating artery).

progressive strangulation of the CCA, the blood flow to the iICA significantly decreased, which was accompanied by a significant increase in the blood flow through the cICA. In the case of 95% CCA strangulation (CCA 95), the blood flow from the cICA to the iICA increased through the communicating artery, leading to a backflow in the iICA. As shown in Fig. 7(c), higher strangulation rates were correlated with a reduced blood flow volume (BFV) entering the iICA. In the normal case, an average BFV of 207.6 L/min was recorded, whereas for 95% CCA strangulation, a reverse flow of 83.5 L/min was recorded. Comparatively, the BFV in the communicating artery during this period increased from an average of 13.5 L/min in the normal case to 153.2 L/min for 95% CCA strangulation. These results indicate that as the CCA strangulation intensified, the blood flow to the iICA decreased, and the flow through the cICA increased, thus causing an elevated flow from the cICA to the iICA via the communicating artery, which induced backflow in the iICA.

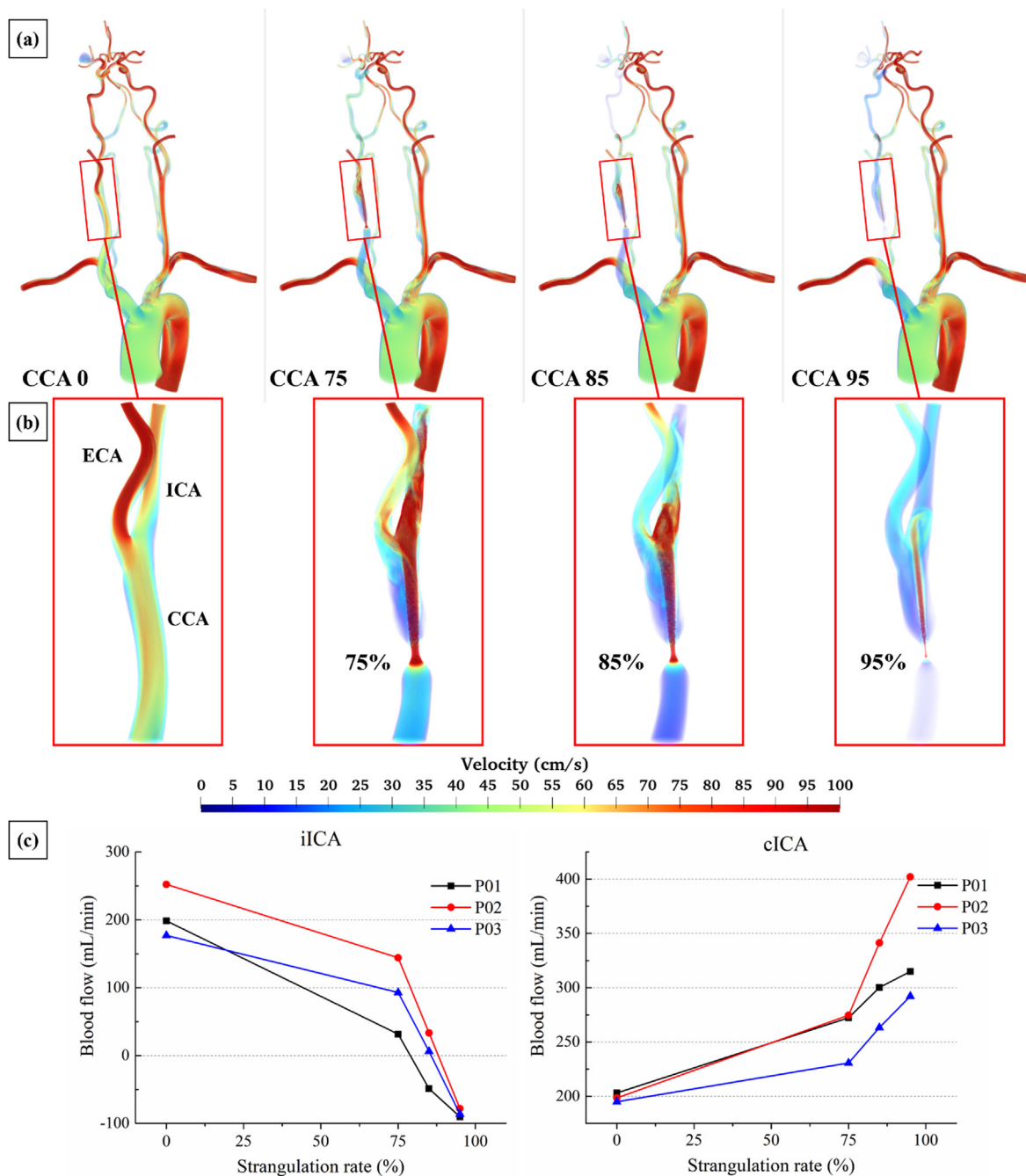
The occurrence of backflow varied among the patients. For P02 and P03, backflow was observed for 95% CCA strangulation, whereas for P01, this phenomenon occurred for 85% strangulation. This variability highlights the impact that anatomical differences in vessel shape, length, and diameter have on the degree of strangulation required to induce backflow.

2. Communicating artery

This subsection explores the causes behind the observed changes in the blood flow in the bilateral ICAs. Figure 8 shows the time-dependent changes in the blood flow through three key communicating arteries—the Acom, iPcom, and cPcom—generated using the P01 model.

As shown in Fig. 8(a), progressive CCA strangulation increased the flow from the contralateral to the ipsilateral side via the Acom, resulting in a significantly elevated flow within the Acom. Additionally, although the iPcom exhibited a minimal flow in the normal case (CCA 0), its flow rate increased substantially for the 75%, 85%, and 95% CCA strangulations. In the normal case, streamlines directed the flow from the iICA to the ipsilateral PCA (iPCA). However, as the strangulation intensified, the flow originated from the basilar artery (BA) and was directed toward the iICA, indicating a reversal of the flow direction through the iPcom. The cPcom exhibited a minimal variation in the flow rate, suggesting that iCCA strangulation had a minimal effect on the cPcom flow.

These observations are quantitatively presented in Fig. 8(b). For the P01 model, the average flow rate of the Acom increased sharply from 0.20 to 2.21 ml/s, while the iPcom rose from 0.040 to 0.99 ml/s. Similar trends were observed for the other patients. For P02, the Acom

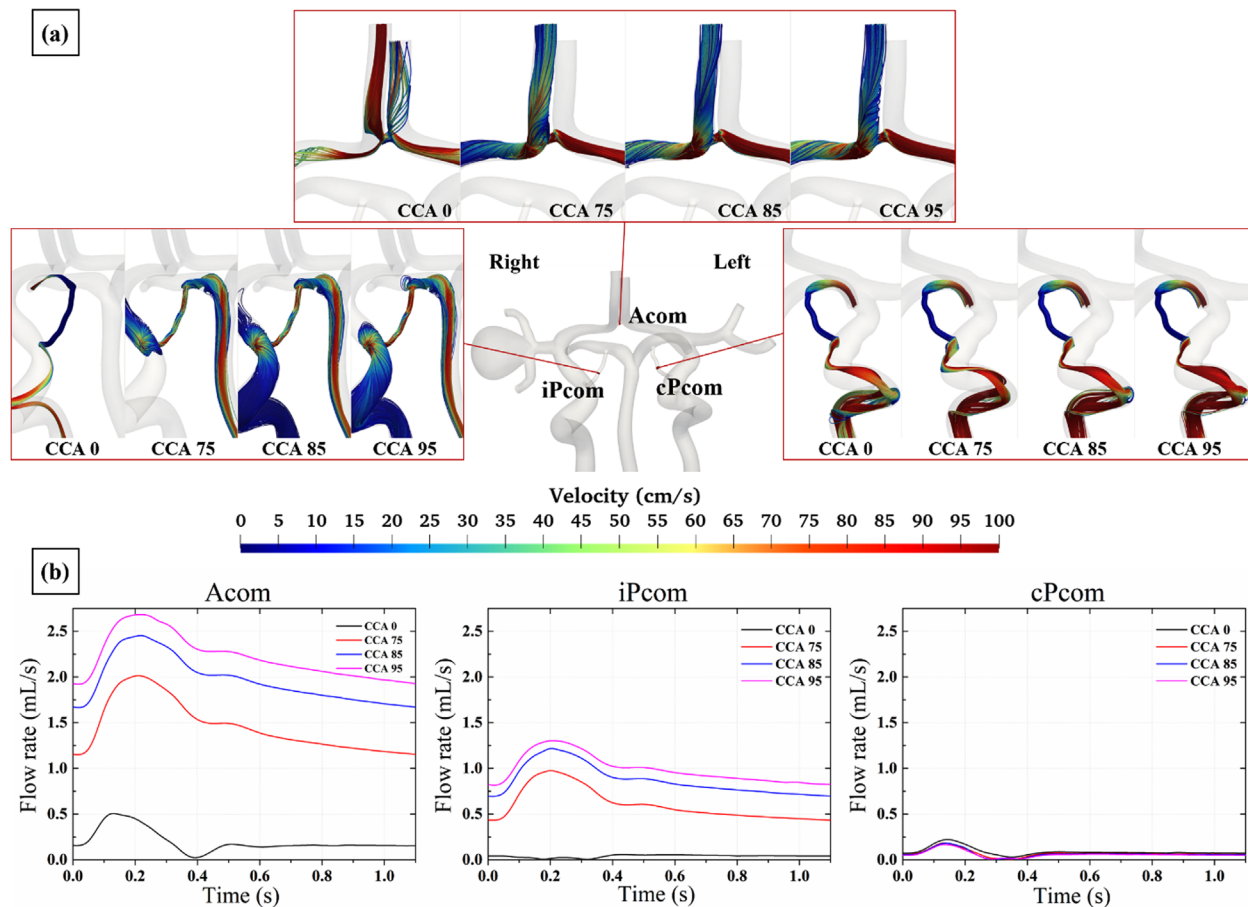


**FIG. 7.** (a) 3D volumetric velocity contour at systole for the P03 model for CCA strangulations of 75%, 85%, and 95%. (b) Blood flow velocity field at systole around the CCA strangulation region. (c) Changes in the blood flow volume through the bilateral ICA following CCA strangulation for the three patient models.

flow increased from 0.31 to 3.78 ml/s, and the iPcom flow increased from 0.012 to 0.63 ml/s. For P03, the Acom flow increased from 0.21 to 2.62 ml/s, and the iPcom flow increased from 0.04 to 0.84 ml/s. The average flow rate through the cPcom remained nearly unchanged in all cases.

## B. Recirculation at aneurysm

As the CCA was progressively strangulated, a shift in the direction of recirculation within the aneurysm was observed. Figure 9(a) shows the specific blood flow velocity in the x-direction expressed as a 3D volumetric velocity, highlighting that recirculation inside the



**FIG. 8.** (a) Systolic velocity streamlines in each communicating artery of the CoW in the P01 model, including the Acom and Pcom on both sides. (b) Changes in blood flow over time for the four CCA strangulations and in each communicating artery in the P01 model. (Abbreviations – CoW: circle of Willis; iPcom: ipsilateral posterior communicating artery; cPcom: contralateral posterior communicating artery).

aneurysm changes direction as blood flow through the Acom increases.

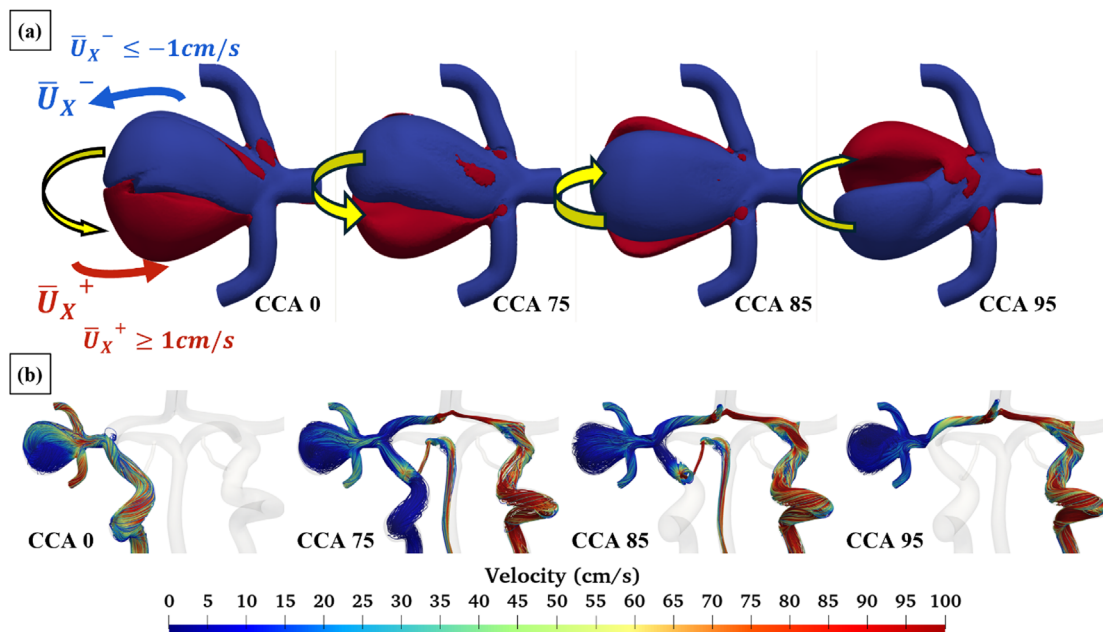
The underlying cause of this directional change in recirculation is depicted in Fig. 9(b), which shows streamlines emanating from the MCA region. A notable pattern emerges: the source of blood flow to the iMCA evolves as CCA strangulation progresses. In the baseline case (CCA 0), blood flow to the MCA is predominantly supplied by the iICA. However, at CCA 75, blood flow is drawn from three sources: the iICA, the contralateral side via the Acom, and the BA through the iPcom. At CCA 85, the iMCA is no longer supplied by the iICA and instead receives blood flow from two sources: the BA via the iPcom and the contralateral ICA via the Acom. By CCA 95, the iMCA relies entirely on blood flow from the contralateral side via the Acom, as the contralateral ICA flow increases substantially.

These findings reveal that the source of blood supply to the aneurysm transitions as CCA strangulation progresses, resulting in corresponding changes in the direction of recirculation within the aneurysm.

### C. Aneurysmal pressure

This study primarily focused on analyzing the pressure within the MCA bifurcation aneurysm. Because directly measuring the intra-aneurysmal pressure during surgery is challenging, CFD was used to estimate the pressure changes associated with CCA strangulation. Additionally, owing to the difficulty of precisely determining the percentage reduction of the CCA during clinical surgery, the relationship between the measurable blood flow velocity in the clinically assessable M2 segment and the aneurysmal pressure was investigated.

Figure 10 shows the systolic, mean, and diastolic pressures within the aneurysm for the four CCA strangulation levels for all patients. In each case, the aneurysmal pressure decreased as the CCA strangulation increased, although the degree to which the pressure was reduced varied among the patients. These differences were caused by the variations in the vessel sizes and shapes as well as the amount of blood supplied to the aneurysm, which depended on the presence or diameter of the communicating arteries. The variability of the trends in the aneurysmal pressure is further illustrated in Fig. 11, which also highlights the

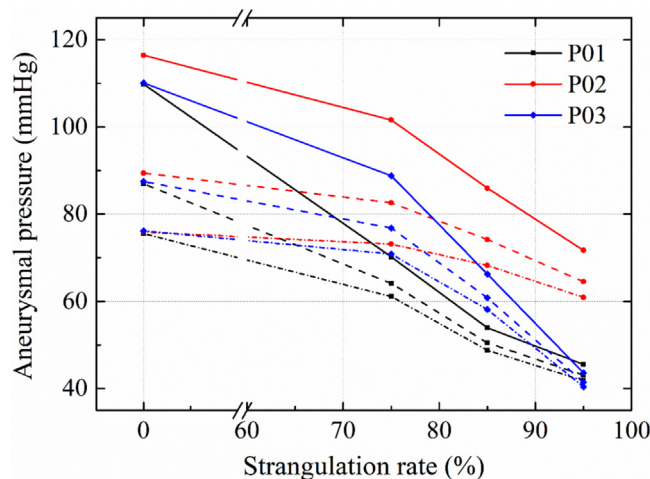


**FIG. 9.** (a) Changes in x-direction 3D volumetric velocity according to CCA strangulation inside the MCA bifurcation aneurysm of the P01 model. (b) Streamline at systole emitted from the MCA under varying CCA strangulation rates in the P01 model.

shape of the CoW, the aneurysmal pressure relative to the M2 mean velocity, and the blood flow in the major cerebral arteries for each patient.

### 1. Patient variations

Figure 11(b) shows the changes in aneurysmal pressure relative to the clinically measurable mean velocity in the M2 segment for each patient. Because the rate of CCA strangulation is difficult to directly



**FIG. 10.** Aneurysmal pressure changes, including systolic, mean, and diastolic pressures, within the MCA bifurcation aneurysm following CCA strangulation in various patients.

measure during surgery, the M2 mean velocity, which can be assessed using Doppler ultrasound,<sup>66,67</sup> was used as a practical indicator.

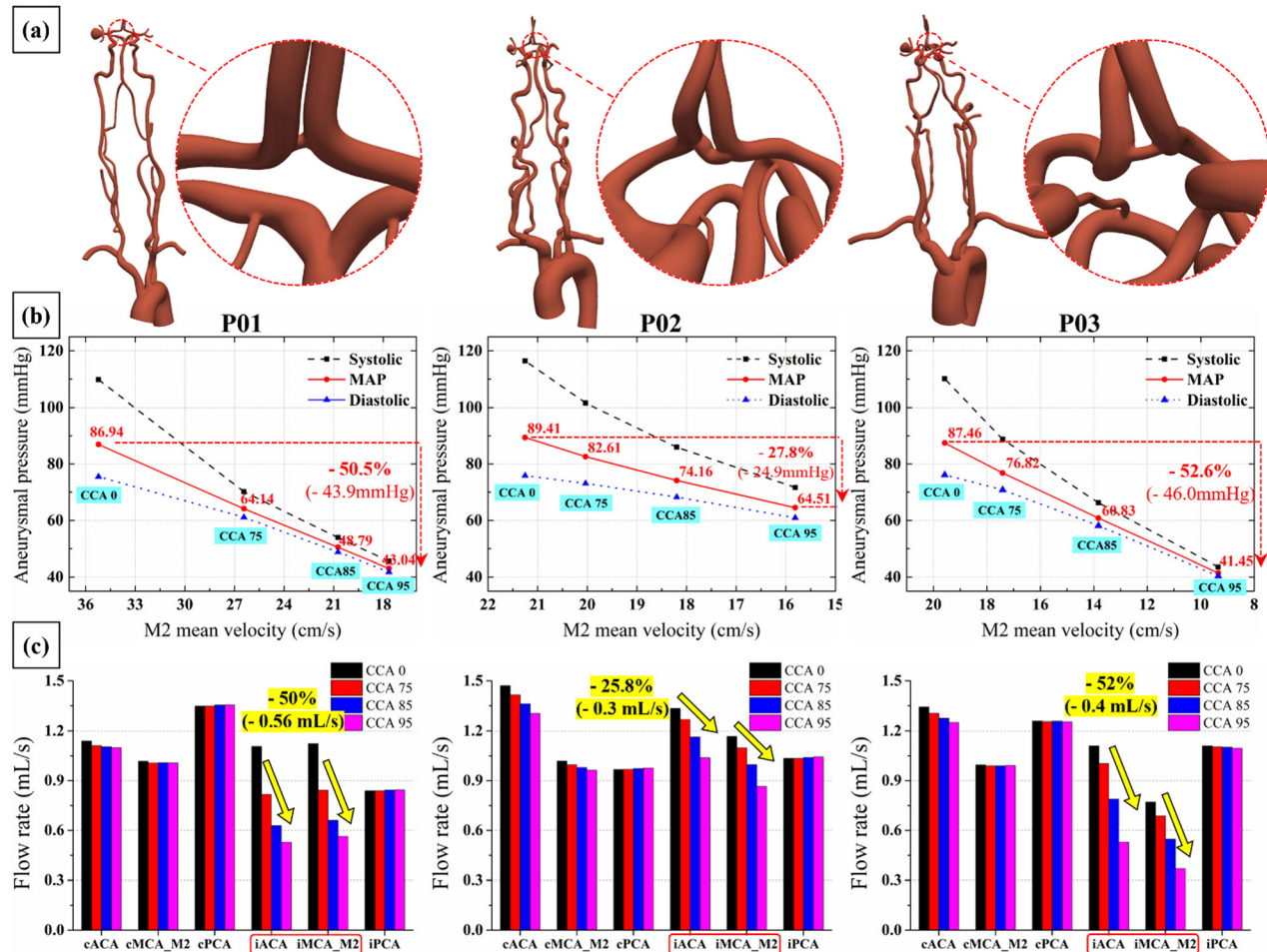
For patient P01, the 95% CCA strangulation reduced the MAP from 86 to 43 mm Hg, representing a 50% decrease. In contrast, P02 exhibited a less significant reduction, with only a 27% decrease in the average pressure, from 89 to 64 mm Hg.

This difference can be attributed to variations in the diameter of the communicating arteries, as shown in Fig. 11(a). For P02, the Acom was wider than for P01. Despite considerable strangulation of the iCCA, the larger Acom allowed significant blood flow from the contralateral side, ensuring more blood flow to the ipsilateral aneurysm. For P03, which had a narrower communicating artery similar to that of P01, the MAP decreased from 87 to 41 mm Hg (a decrease in 53%) under 95% CCA strangulation.

These findings suggest that the size and shape of the communicating arteries significantly influenced the aneurysmal pressure. Patients with larger Acom vessels required more extensive CCA strangulation to achieve a substantial reduction in pressure, whereas those with smaller Acom vessels exhibited significant pressure decreases with less CCA strangulation.

Figure 11(c) shows the blood flow in the major cerebral arteries of the CoW for each patient under the various CCA strangulation rates. The iCCA strangulation had no effect on the blood flow in the contralateral MCA M2, cPCA, or iPCA. However, it significantly affected the blood flow in the ipsilateral ACA (iACA) and ipsilateral MCA M2 (iMCA M2). This indicates that iCCA strangulation primarily impacted the blood flow in the iACA and iMCA but not in the PCA.

The observed changes in the blood flow to the major cerebral arteries aligned with the aneurysmal pressure trends. For patient P01, the iMCA M2 blood flow decreased by approximately 50%, from 1.12



**FIG. 11.** (a) Enlarged images of the Acom geometries for the three patient models. The Acom areas for patients P01, P02, and P03 were 0.95, 2.8, and 1.0 mm<sup>2</sup>, respectively. (b) Changes in the systolic, mean, and diastolic pressures within the aneurysm as a function of the M2 mean blood flow velocity for each patient. (c) Changes in the blood flow at the outlet of each patient's main cerebral artery (ACA, MCA, and PCA) for various CCA strangulation rates.

to 0.56 ml/s, under 95% CCA strangulation. For P02, the iMCA M2 blood flow decreased by 25.8%, from 1.17 to 0.87 ml/s, owing to the larger Acom, which facilitated significant contralateral blood flow to the iMCA, reducing the impact of CCA strangulation. For P03, the blood flow decreased by 52%, from 0.77 to 0.37 ml/s, under the same conditions.

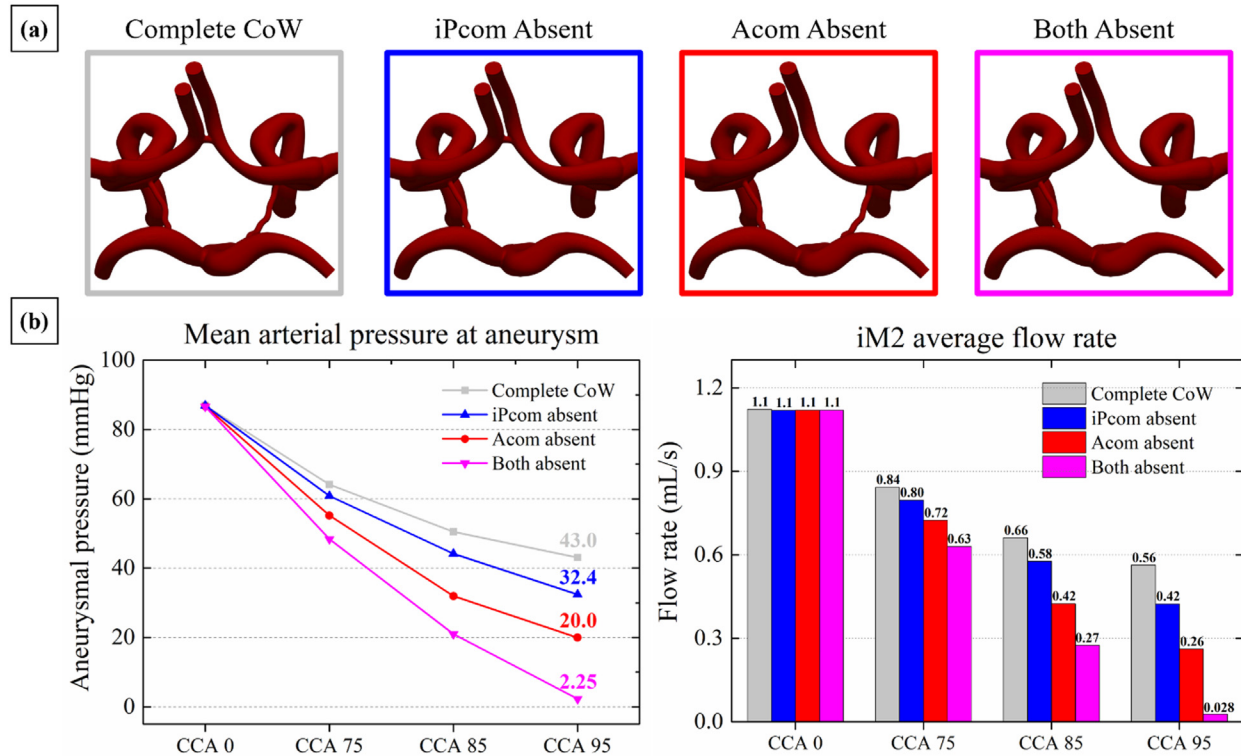
These results confirmed that the changes in the blood flow to the major cerebral arteries owing to iCCA strangulation were strongly influenced by the size of the communicating arteries in each patient.

## 2. CoW variations

This subsection examines the changes in the aneurysmal pressure and the M2 blood flow based on the presence or absence of communicating arteries for patient P01. The CoW is a critical collateral network that maintains cerebral blood flow when the primary arterial supply is compromised.<sup>37</sup> Prior studies have indicated that the CoW is incomplete in approximately 50%–90% of adults, and that the number of

missing segments is correlated with intolerance to arterial cross-clamping.<sup>68</sup> Furthermore, variations such as a hypoplastic Acom, an absence of Acom, and a bilateral absence of Pcom occur in 25.8%, 10.8%, and 22.5% of cases, respectively.<sup>69</sup> Given that not all individuals possess a complete CoW, we examined the impact of variability in the CoW on the aneurysmal pressure and blood flow. Additionally, previous studies have shown that CoW variations influence the formation and rupturing of IAs.<sup>70–73</sup> Therefore, our study systematically analyzed the effects of different CoW configurations by varying the presence of communicating arteries to assess their hemodynamic impact.

For P01, the model initially featured a complete CoW. To assess the effects of CoW variability, specific communicating artery segments were selectively removed in various scenarios. As shown in Fig. 12(a), the baseline case was labeled “Complete CoW,” while the variations were categorized as “iPcom absent” (absence of the ipsilateral Pcom), “Acom absent” (absence of the Acom), and “Both absent” (absence of both the ipsilateral Pcom and Acom). A scenario without the cPcom was excluded, as its absence did not significantly affect the major



**FIG. 12.** (a) Morphological classification according to the presence or absence of the communicating artery. From left to right—"Complete CoW": Acom and both Pcom are present; "iPcom absent": ipsilateral Pcom is absent; "Acom absent": Acom is absent; "Both absent": both Acom and ipsilateral Pcom are absent, with only the contralateral Pcom present. (b) Average intra-aneurysm pressure and average ipsilateral M2 (iM2) outlet flow rate for each CoW variability case and for various CCA strangulation rates (%).

arteries during iCCA strangulation. Figure 12(b) shows the trends in the aneurysmal pressure and changes in the M2 mean flow rate for each CoW variation under increasing iCCA strangulation rates.

The results indicate a progressive decrease in the aneurysmal pressure in the following order: "Complete CoW," "iPcom absent," "Acom absent," and "Both absent." The specific values for the 95% iCCA strangulation were as follows:

- "Complete CoW": aneurysmal pressure decreased by 50% (mean pressure decreased from 86 to 43 mm Hg).
- "iPcom absent": aneurysmal pressure decreased by 63% (mean pressure decreased from 86 to 32 mm Hg).
- "Acom absent": aneurysmal pressure decreased by 77% (mean pressure decreased from 86 to 20 mm Hg).
- "Both absent": aneurysmal pressure decreased by 97% (mean pressure decreased from 86 to 2.2 mm Hg).

In the "Both absent" scenario, where the only blood supply to the iMCA was through the iCCA, both the blood flow rate and aneurysmal pressure approached zero under 95% iCCA strangulation. Additionally, in the "Acom absent" scenario, the greater reduction in the aneurysmal pressure compared to that in the "iPcom absent" scenario highlights the critical role of blood flow through the Acom in sustaining pressure within the MCA bifurcation aneurysm. This finding indicates that the absence or hypoplasia of the Acom can significantly lower the aneurysmal pressure, reducing the need for extensive iCCA strangulation during surgery.

Thus, cases with a complete CoW require substantial iCCA strangulation to achieve significant reductions in the aneurysmal pressure. For scenarios in which the Acom or all communicating arteries are absent, even minimal iCCA strangulation is sufficient to substantially lower the aneurysmal pressure, potentially enhancing the safety and feasibility of aneurysm-clipping surgery.

#### IV. DISCUSSION

##### A. Regression model

To broaden the applicability of our findings, we developed a regression model to estimate aneurysmal pressure across a diverse patient population. A high coefficient of determination ( $R^2$ ) would indicate the model's reliability in predicting aneurysmal pressure across all patient data.

Figure 13 shows a regression model that quantifies systolic, mean, and diastolic aneurysmal pressures as a function of CCA strangulation rates. The model, constructed using computational analysis limited to strangulation rates of 0%, 75%, 85%, and 95%, employed a non-linear exponential fit. This approach was chosen over a polynomial fit to prevent the erroneous implication of increased aneurysmal pressure between 0% and 75% CCA strangulation. This non-linear method resulted in high  $R^2$  values for P01 and P03 (0.89 for both) but a notably lower  $R^2$  value for P02 (0.54). The lower  $R^2$  for P02, which includes all pressure data from diastolic to systolic, is influenced by the wide communicating artery that lessens the impact of CCA strangulation on aneurysmal pressure, thus creating a significant diastolic-systolic

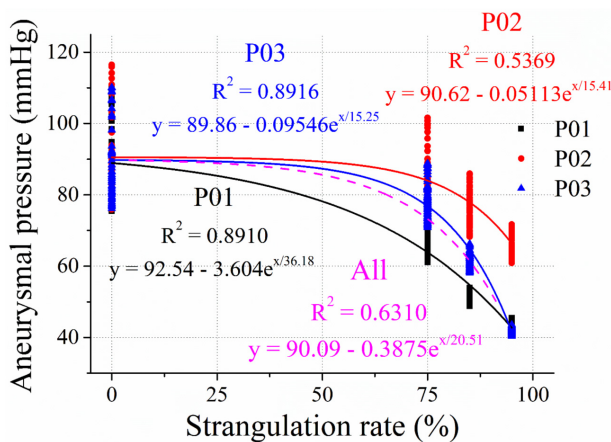


FIG. 13. Regression model based on different patients according to CCA strangulation.

pressure difference. Thus, the overall  $R^2$  for the regression model across all three patients was approximately 0.63. Although an  $R^2$  of 0.5 is generally considered acceptable for clinical applications,<sup>74</sup> the lower value in this instance may diminish the model's predictive precision in specific scenarios.

To enhance prediction accuracy, an alternative regression model was constructed based on clinically measurable M2 velocity rather than CCA strangulation rates. For instance, in P01, a 95% CCA strangulation resulted in an approximately 50% decrease in both aneurysmal pressure and M2 velocity. Similar patterns were noted in other patient cases, leading to the development of a regression model that predicts aneurysmal pressure reduction based on the rate of decrease in M2 velocity. Figure 14 shows this regression model, which achieved an  $R^2$  of approximately 0.99, demonstrating a robust linear relationship.

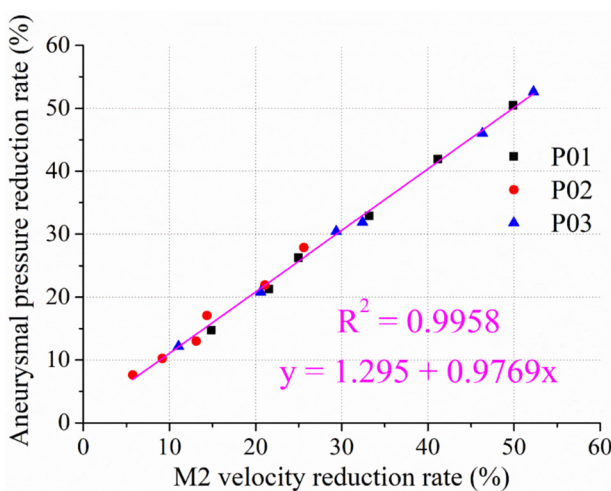


FIG. 14. Regression model based on aneurysmal pressure reduction rate according to M2 segment velocity reduction rate for each patient.

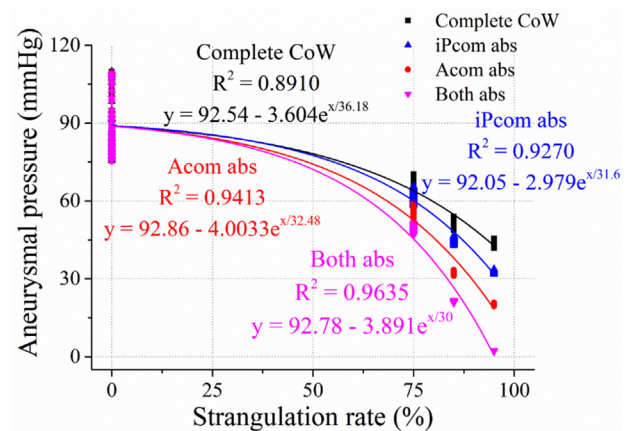


FIG. 15. Regression model based on changes in the circle of Willis according to CCA strangulation.

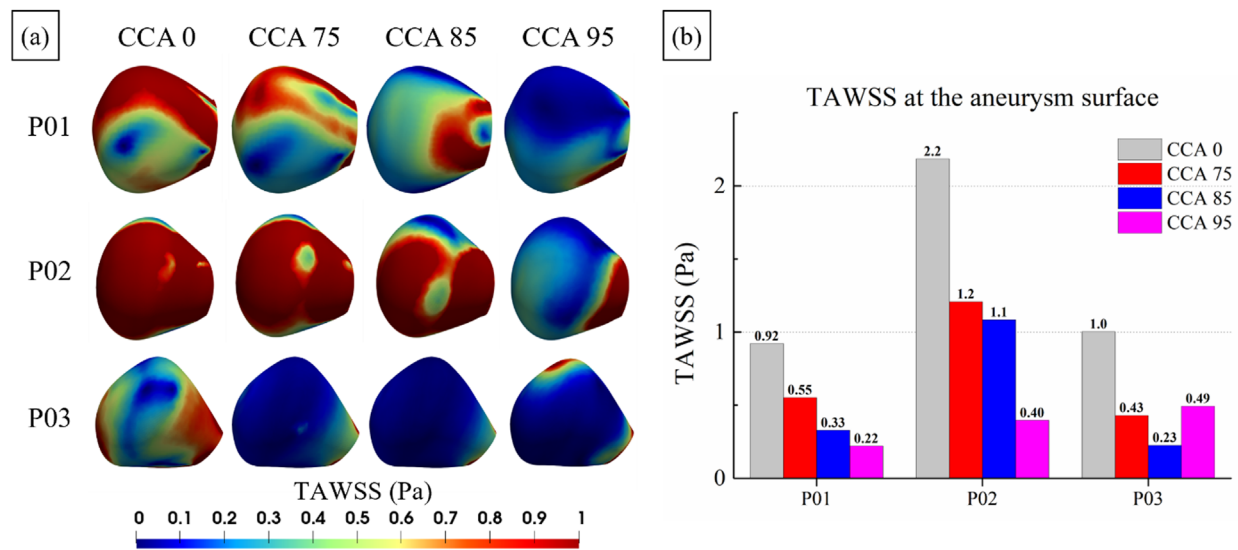
Given that M2 velocity is clinically measurable, this model provides a significant advantage in surgical settings. By using a parameter observable during surgery, neurosurgeons can estimate changes in aneurysmal pressure in real-time, thereby enhancing surgical decision-making and improving the safety and precision of aneurysm clipping procedures. The ability to measure M2 velocity and its rate of decrease allows neurosurgeons to reliably predict the relative decrease in aneurysmal pressure, even when the CCA strangulation rate is difficult to assess directly. This model is expected to improve the reliability of aneurysmal pressure predictions across diverse patient populations.

Figure 15 shows a regression model that considers changes in the CoW configuration resulting from CCA strangulation. The  $R^2$  values for different CoW variations were relatively high, increasing in the following order: Complete CoW (0.89), iPcom absent (0.93), Acom absent (0.94), and Both absent (0.96). This progression aligns with the findings in Sec. III C 2, where the greatest reductions in aneurysmal pressure were observed in the Both absent scenario. The increased  $R^2$  for cases with missing communicating arteries reflects the narrower diastolic-systolic pressure differences. Notably, the absence of the Acom led to a more pronounced reduction in the pressure gap compared to the absence of the iPcom, resulting in a higher  $R^2$  value.

## B. Aneurysmal pressure criterion

According to existing medical literature, elevated blood pressure is a significant risk factor for aneurysm rupture. Sanchez *et al.*<sup>12</sup> reported that every 10 mm Hg increase in systolic blood pressure (SBP) raises the risk of rupture and Karhunen *et al.*<sup>13</sup> found that high blood pressure is strongly correlated with an increased risk of IAs and aSAH. Based on these findings, the primary objective of CCA strangulation in our study was to lower the intra-aneurysmal pressure, thereby increasing the softness of the aneurysm and facilitating safer manipulation, dissection, and clipping during surgery.

However, an excessive reduction in the aneurysmal pressure during clipping surgery is also undesirable. Thongrong *et al.*<sup>75</sup> suggested that to prevent delayed ischemic neurologic deficits (DNID),<sup>76,77</sup> the intraoperative blood pressure should not fall below the following



**FIG. 16.** TAWSS on the surfaces of aneurysms. (a) Contour of TAWSS on aneurysmal surfaces of the three patients under varying CCA strangulation rates. (b) Graph showing the surface-averaged TAWSS for the three patients relative to CCA strangulation rates. (Abbreviations – TAWSS: time-averaged wall shear stress).

thresholds: SBP  $\geq 95$  mm Hg; diastolic blood pressure (DBP)  $\geq 50$  mm Hg; and MAP  $\geq 61.7$  mm Hg.

Comparing these thresholds to our model, the patient-specific limits for CCA strangulation for the MAP were determined as follows:

- P01: up to 75% CCA strangulation is permissible.
- P02: up to 95% CCA strangulation is permissible.
- P03: up to approximately 85% CCA strangulation is permissible.

In terms of the CoW variation:

- The “Complete CoW” and “iPcom absent” cases allowed for CCA strangulation up to 75% while maintaining safe MAP levels.
- The “Acom absent” and “Both absent” cases showed significant reductions in the aneurysmal pressure even with 75% CCA strangulation. However, from a DNID risk perspective, further reduction may pose clinical risks.

These findings suggest that adjusting the degree of CCA strangulation based on the patient-specific anatomy and CoW configuration is essential for maintaining optimal hemodynamic conditions. Lowering the aneurysmal pressure is crucial for preventing ruptures, but an excessive reduction in pressure may increase the risk of DNID. Thus, an individualized approach is necessary to balance protecting the patient from an aneurysm and minimizing their exposure to ischemic risk during clipping surgery.

### C. Wall shear stress analysis

Wall shear stress (WSS) is a critical parameter in hemodynamics, as it significantly influences aneurysm behavior.<sup>78</sup> A low WSS and a high oscillatory shear index are associated with an inflammatory cell-mediated pathway, which may contribute to the growth and rupture of large, atherosclerotic aneurysms. Conversely, a high WSS coupled with a positive WSS gradient can initiate a mural cell-mediated pathway, potentially leading to the growth and rupture of small or secondary

bleb aneurysms.<sup>79</sup> Therefore, managing the WSS within optimal ranges is crucial.

In this study, we examined the time-averaged wall shear stress (TAWSS) on the surfaces of aneurysms. For all patients, the average TAWSS on the aneurysm surface decreased progressively as the CCA strangulation rate increased, as shown in Fig. 16. On average, the TAWSS for the three patients decreased by approximately 73%, from 1.37 Pa in the normal case (CCA 0) to 0.37 Pa at 95% CCA strangulation. Prior studies<sup>79</sup> have indicated that a low WSS is associated with an increased risk of aneurysm rupture. However, additional studies<sup>79–81</sup> suggest that a high WSS may also contribute to the growth and rupturing of aneurysms. This contradiction indicates the necessity for ongoing research and discussions to fully elucidate the relationships between the WSS and aneurysm behavior. Accordingly, these findings indicate that the application of CCA strangulation should be managed with caution, considering its substantial impact on aneurysm surfaces, as reflected by changes in the WSS.

### D. Limitations

This study has several limitations that warrant consideration. First, cerebral autoregulation,<sup>82,83</sup> a critical physiological mechanism by which blood vessels adjust to ensure consistent cerebral blood flow, was not incorporated into our analysis. The exclusion of this factor may have marginally compromised the accuracy of our predictions. However, in large arterial domains spanning from the aorta to the CoW, such as those in our models, the collateral flow through the communicating arteries has been shown by previous studies<sup>21,50,84</sup> to exhibit behavior similar to that of autoregulated models.<sup>83,85</sup> Large-scale domains covering both long proximal and distal segments from the regions of interest are considered to have bulk autoregulatory effects comparable to those of vascular models incorporating autoregulation. Another limitation to our study is that our analysis focused exclusively on the major cerebral vessels and excluded collateral

circulations through smaller arteries and capillaries, which are critical for maintaining cerebral perfusion.<sup>86</sup> Although this simplification is unlikely to have a major impact, it may have slightly influenced the overall hemodynamic outcomes because the contributions of these smaller vessels were not taken into account.

Second, although this study involved numerous simulation cases, the number of patients included in this study was limited to three, which may restrict the generalizability of our findings. While these patient-specific models allowed for a detailed hemodynamic analysis, inter-patient anatomical and physiological variations could have influenced the observed trends. Therefore, conclusions drawn from this study should be interpreted with caution. Future research should expand the dataset to include a more diverse patient population, incorporating different anatomical configurations and pathological conditions to assess the robustness of the proposed methodology. However, constructing patient-specific models with a complete CoW that extends from the aorta remains computationally expensive, and optimizing the simulation efficiency for such models will be necessary for large-scale studies.

Third, to simplify the computational model, several assumptions were made. The blood was modeled as an incompressible, Newtonian fluid, and the vessel walls were considered rigid, neglecting the potential impacts of vessel elasticity on the hemodynamics. Although the rigid wall assumption optimized the efficiency of the computations, it may have reduced the accuracy of the WSS analysis.<sup>42,87</sup> Future studies aiming for more accurate analyses of IAs and CCA strangulation could benefit from utilizing non-Newtonian fluid models and fluid–structure interaction methods,<sup>88,89</sup> which take into account the elasticity of the vessel walls.

Fourth, validation based on experimental data remains challenging, as direct *in vivo* measurements of the intra-aneurysmal pressure during CCA strangulation are difficult. Even though we compared our simulation results to values reported in previous studies, patient-specific validation via clinical or experimental studies is necessary to confirm the accuracy of our findings. Future work could involve patient-specific computational analyses that incorporate patient-derived hemodynamic parameters, vessel wall properties, and real-time Doppler ultrasound measurements to enhance the reliability of the model. Additionally, simulations that incorporate aneurysm clipping as well as comparisons to clinical data from cases in which clipping was performed could further validate the proposed method in a surgical setting.

## V. CONCLUSIONS

This study introduces an approach for predicting intra-aneurysmal pressure under conditions of CCA strangulation using large-scale 3D CFD modeling that spans from the aorta to the CoW. Through detailed hemodynamic analysis, we quantitatively assessed aneurysmal pressure and the distribution of cerebral blood flow in major cerebral arteries—parameters that are typically challenging to measure clinically. These insights are critical in understanding the hemodynamic impacts of CCA strangulation on cerebral vasculature and highlight its potential utility in safely managing intra-aneurysmal pressure during neurosurgical procedures.

Comparative analyses among patients revealed that those with a larger Acom experienced a less significant reduction in aneurysmal pressure during CCA strangulation. Further analysis of CoW variations indicated that the Acom plays a more influential role in modulating intra-aneurysmal pressure compared to other communicating arteries. These results emphasize the significance of patient-specific

anatomical variations in influencing the hemodynamic outcomes of surgical interventions. For instance, neurosurgeons might need to increase the degree of iCCA strangulation for patients with a larger Acom, as these patients typically exhibit a smaller reduction in intra-aneurysmal pressure.

Moreover, by correlating the rates of reduction in aneurysmal pressure with changes in M2 velocity, we developed a linear regression model that demonstrates a high coefficient of determination. This model provides a reliable tool for predicting changes in aneurysmal pressure based on clinically measurable changes in M2 velocity, facilitating accurate assessments even when CCA strangulation rates are not directly measurable. By integrating computational modeling with clinically measurable parameters, this study provides practical insights that can enhance the safety and precision of aneurysm management in neurosurgical practice.

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## AUTHOR DECLARATIONS

### Conflict of Interest

The authors have no conflicts to disclose.

## Author Contributions

**Sangwon Ryu:** Data curation (lead); Formal analysis (lead); Investigation (lead); Methodology (lead); Software (lead); Validation (lead); Visualization (lead); Writing – original draft (lead). **Thi Thanh Giang Le:** Formal analysis (supporting); Writing – original draft (supporting); Writing – review & editing (equal). **Kyoungmin Jang:** Conceptualization (supporting); Formal analysis (supporting); Investigation (supporting). **Taek Kyun Nam:** Conceptualization (equal); Formal analysis (equal); Investigation (equal); Supervision (equal). **Jaiyoung Ryu:** Conceptualization (equal); Funding acquisition (equal); Investigation (equal); Supervision (equal); Writing – review & editing (equal).

## DATA AVAILABILITY

The data that support the findings of this study are available within the article.

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