Quantification of Visceral Perfusion and Impact of Femoral Cannulation in Vitro Model of Aortic Dissection

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This certifies that the Doctoral Dissertation of Woon Heo is approved.

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ACKNOWLEDGEMENTS

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ABSTRACT

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We aimed to simulate blood flow at the aortic dissection with an in-vitro vascular model and assess the impact of the cannulation method on visceral perfusion. An aortic dissection model with an acrylic aortic wall and silicone intimal flap was developed to study visceral perfusion under various cannulation conditions. The primary tear was placed in the proximal descending aorta and the re-entry site in the left common iliac artery. A cardiovascular pump was used to reproduce a pulsatile aortic flow rate as well as axillary and femoral cannulation flow rates of 3-7 L/min. Hemodynamics were analyzed by using four-dimensional flow magnetic resonance imaging. Axillary cannulation was found to collapse the true lumen at the celiac and superior mesentery arteries while combined axillary and femoral cannulation did not change the size of the true lumen. Combined axillary and femoral cannulation resulted in a larger visceral flow than did axillary cannulation alone. When axillary and femoral cannulation were used, the amount of visceral flow increased by 125% at 3 L/min, by 89% at 4 L/min, by 67% at 5 L/min, by 98% at 6 L/min, by 101% at 7 L/min, respectively, compared to those with the axillary cannulation only. Our model was useful to understanding the hemodynamics in aortic dissection. We confirmed that the intimal-flap motion can partially block blood flow to the celiac and superior mesenteric arteries, and that additional femoral cannulation can increase visceral perfusion.

Key words : aortic dissection, perfusion, hemodynamics

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I. INTRODUCTION

Aortic dissection is a disease characterized by tearing of the aortic intimal layer, increasing the risk of aortic rupture and malperfusion to the heart, brain, viscera, and lower extremities. Malperfusion patterns differ depending on the location of the intimal tear and the extent of dissection, and in severe malperfusion, injury to the affected organ can have a major impact on the patient's prognosis [1-4].

Visceral malperfusion is a severe complication in patients with aortic dissection [4]. This may occur in both acute type I aortic dissection (AIAD) and acute type III aortic dissection (AIIIAD). In AIAD, there are two types of intraoperative visceral malperfusion that may occur during cardiopulmonary bypass (CPB). One is the persistence of preoperative visceral malperfusion; the other is new visceral malperfusion that occurs after the initiation of CPB. Understanding the hemodynamics of visceral malperfusion in AIAD may improve outcomes by allowing optimization of the organ-protection strategy.

Many cardiovascular centers favor right-axillary-artery and central cannulation, based on the belief that the antegrade perfusion strategy prevents cerebral malperfusion [5-7]. Moreover, Numata et al. reported a stroke-prevention effect of axillary cannulation (AC) for aortic aneurysm, as determined with computational fluid dynamics [8]. However, femoral cannulation is also a safe and feasible

technique, and in hemodynamically unstable situations, femoral cannulation is generally faster to perform than AC [9]. In AIIIAD, visceral malperfusion is treated with endovascular fenestration or stent-graft insertion to restore blood flow to the true lumen.

We hypothesized that femoral-artery cannulation in the initial phase of CPB or in veno-arterial extracorporeal membrane oxygenation (VA-ECMO) would improve visceral perfusion via restored blood flow in the true lumen in patients with visceral malperfusion due to acute aortic dissection. We used an in-vitro cardiovascular model and four-dimensional (4D) flow magnetic resonance imaging (MRI) to perform experiments with the aim to analyze the impact of additional femoral cannulation on visceral malperfusion in aortic dissection.

II. MATERIALS AND METHODS

In-vitro aortic-dissection model

An idealized geometry of AIIIAD was constructed by using computer-aided design software (SpaceClaim v.2021 R1, Ansys, Inc., Canonsburg, PA, USA). The diameters of the aorta and all the arteries were designed in proportion to an ascending aortic diameter of 30 mm [10, 11]. The in-vitro model was made using an acrylic aortic wall and silicone intimal flap. The silicone intimal flap was made of silicone rubber (Shore 20A, Trando 3D Medical Technology Co., Ltd., Ningbo, China) and its thickness was uniform, at 1 mm. The primary tear was placed in the proximal descending aorta and re-entry in the left common iliac artery (CIA_L; Fig. 1). The detailed values for the geometry are provided in the supporting information (Fig. S1 and Table S1).

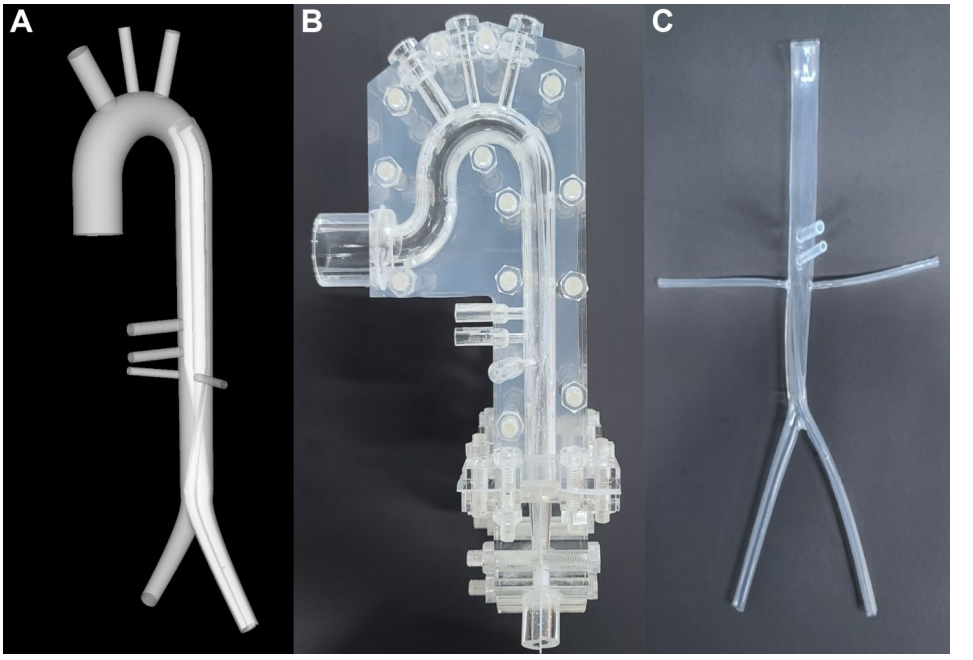

Figure 1. Idealized model of aortic dissection, the idealized geometry (A), the acrylic aortic wall (B), the silicone flap (C).

In-vitro flow experiment

The cardiovascular flow-mimicking circuit was equipped with a piston pump (PC40, Thomson Industries, Inc., Radford, VA, USA) and a centrifugal pump (universal 3400, EHEIM GmbH & Co. KG, Deizisau, Germany) (Fig. 2). The outer diameter and thickness of the ascending aorta tube were 45 mm and 3 mm, respectively, and the BT and CIA_R tube were 23 mm and 2 mm, respectively, and those of the outlet tubes of reservoir were 30 mm and 3 mm, respectively. For MRI experiments, all tubes were at least 7 m long. The working fluid was a mixture of water and glycerol in a 60:40 mass ratio, mimicking blood. All outlet arteries were open to the reservoir to ensure the same outlet pressure throughout.

Pulsatile flow experiment

Pulsatile flow was tested to obtain the baseline characteristics of the blood flow through the aortic dissection. A previously developed cardiovascular pump was used to replicate physiological aortic flow [12]. The cardiac output and the peak flow rate of the pulsatile flow was 4.2 L/min and 19.78 L/min, respectively, at a heart rate of 60 beats/min and a stroke volume of 70 mL/beat.

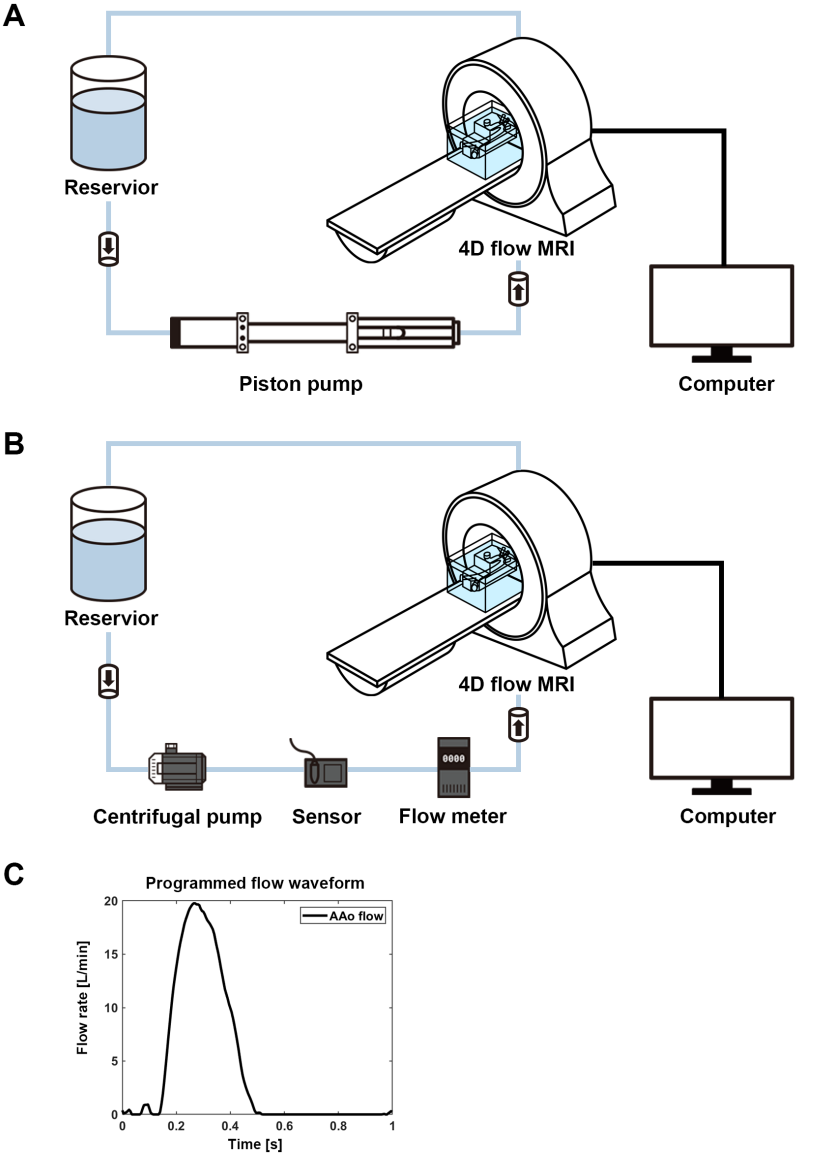

Figure 2. Schematics of the 4D flow MRI experiments in the pulsatile flow (A),

in the AC and AFC (B), the flow rate waveform in the pulsatile (C).

Cannulation experiment

In the cannulation experiments, the flow rate was maintained at a constant level with the centrifugal pump and was controlled by opening and closing the valve. The flow rate was monitored with an electromagnetic flow meter (VN20, Wintech Process Co. Ltd., Gyeonggi-do, Korea). Two different cannulation conditions were tested: AC and axillary and femoral cannulation (AFC). For AC, a steady flow rate of 3-7 L/min was applied to the brachiocephalic trunk (BT). AFC was tested with the same flow rate, but the total cannulation flow was divided between the BT and the right common iliac artery (CIA_R; Fig. 3).

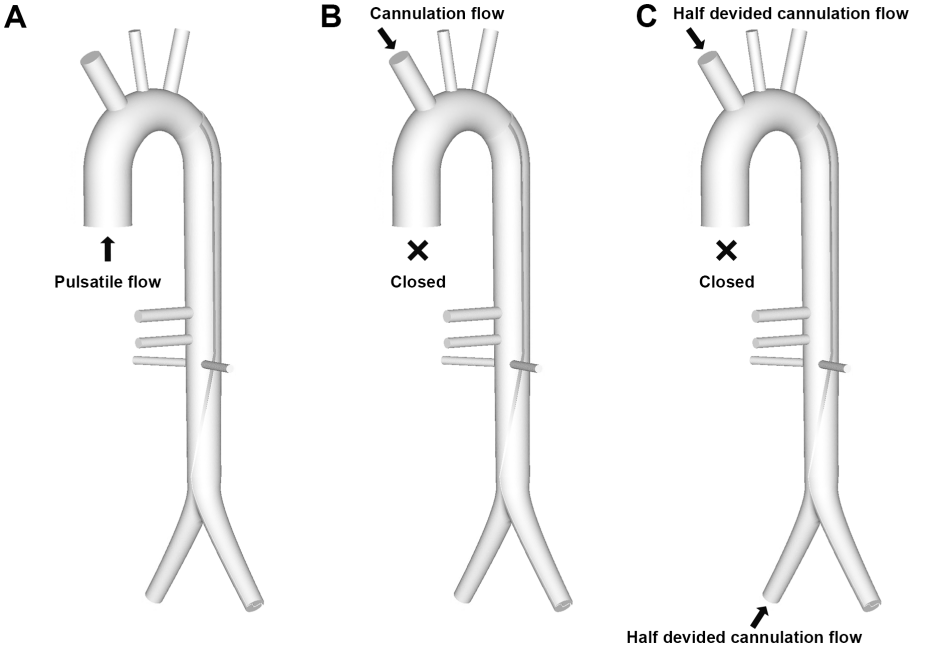

Figure 3. Inflow conditions of 4D flow MRI experiments in the pulsatile flow (A), in the AC (B), in the AFC (C).

Four-dimensional flow magnetic resonance imaging measurement

4D flow MRI was performed using a 3T-MRI machine (Skyra, Siemens AG,

Munich, Germany). The 4D flow MRI sequence (Work In Progress; WIP) was acquired as follows for all experiments: scanner software version = VE11E, field of view = 176 × 352 × 56 mm^3, matrix = 88 × 176 × 28, voxel size = 2 × 2 × 2 mm^3, and flip angle = 7°, effective temporal resolution = 60 bpm and 25 cardiac phase (40 ms), coil = 16 channel coil, cardiac gating = retrospective electrocardiogram gated, and respiratory compensation = navigator respiratory gating. For pulsatile flow, further parameters were as follows: echo time (TE) = 2.43 ms, repetition time (TR) = 9.95 ms, velocity encoding (Venc) = 250 cm/s, and gated 25 time frames. For AC, further parameters were as follows: TE = 2.43-2.50 ms, TR = 10.18-11.10 ms, Venc = 100-200 cm/s, and 1 time frame with steady state. For AFC, further parameters were as follows: TE = 2.43-2.50 ms, TR = 5.16-5.63 ms, Venc = 90-170 cm/s, and 1 time frame with steady state.

Image processing and analysis

4D flow MRI images were converted from the Dicom format to the Nifti format via MATLAB (v.R2020a, The MathWorks, Inc., Natick, MA, USA). Magnitude and velocity images were imported into ITK-SNAP software (v.3.8.0, University of Utah, Salt Lake City, UT, USA) to segment the intimal flap and fluid domain into 3D medical images (Fig. S2). In order to calculate and visualize the flow, we used the Ensight data visualization tool (v.2021 R1, Ansys Inc.). The flow rate was calculated by positioning 10 slices at 2-mm intervals perpendicular to each artery, integrating the normal directional velocity of each slice with respect to the area. The flow rate of each artery was reported as a mean ± standard deviation value. The calculation of the renal flow was excluded because the renal diameter was smaller than the MRI resolution. We measured the true lumen of the flap in the center line of the celiac artery (CA) and superior mesenteric artery (SMA), and normalized it to the true lumen area before deformation.

III. RESULTS

Pulsatile flow characteristics

The velocity contour revealed that the cardiovascular pump replicated the pulsatile flow with a peak velocity of ~1.0 m/s (Fig. S3). The magnitude image indicated that the intimal flap fluttered during the cardiac cycle. The size of the true lumen of the intimal flap at the CA and SMA levels was reduced to half of its length before deformation at around 0.5 s, corresponding to the end-systole period. The area of the true lumen at the CA and SMA level decreased by 76 and 74%, respectively (Fig. 4). Mean flow rates through the BT, left common carotid artery, left subclavian artery, CA, SMA, CIA_L, and CIA_R were 1.61, 0.34, 0.63, 0.24, 0.15, 0.17, and 0.87 L/min, respectively (Fig. S4).

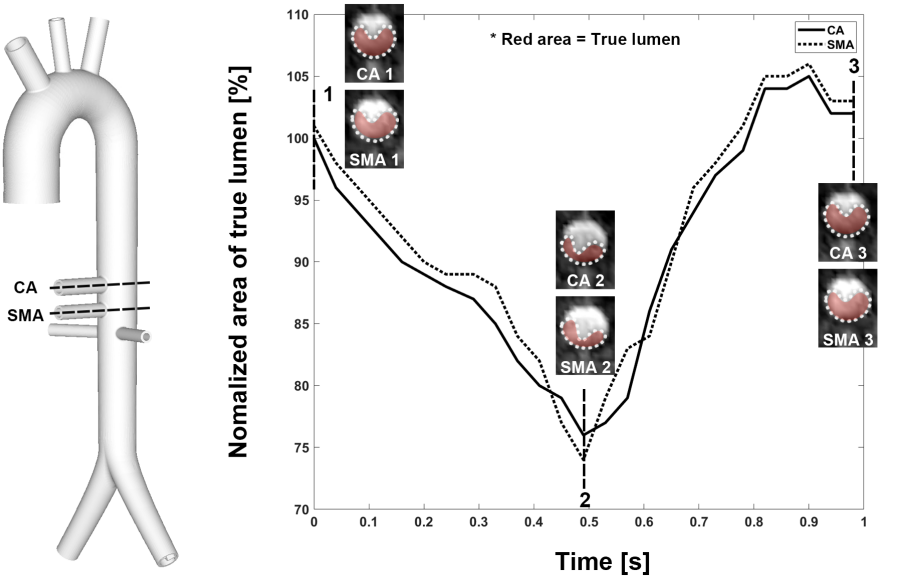

Figure 4. Normalized area and the flap deformation of the celiac and superior mesenteric artery in the pulsatile.

Deformation of the intimal flap

As AC flow increased, the size of the true lumen was reduced as the intimal flap collapsed toward the CA and SMA. With AFC, an increase in the cannulation flow had little deformation of the intimal flap. With AC, the area of the true lumen

of the intimal flap at the CA level decreased from 87 to 72%, respectively, and at the level of the SMA from 75 to 71%, respectively. With AFC, the area of the true lumen of the intimal flap at the level of the CA changed from 99 to 103%, respectively, and at the level of the SMA 99 to 104%, respectively (Fig. 5).

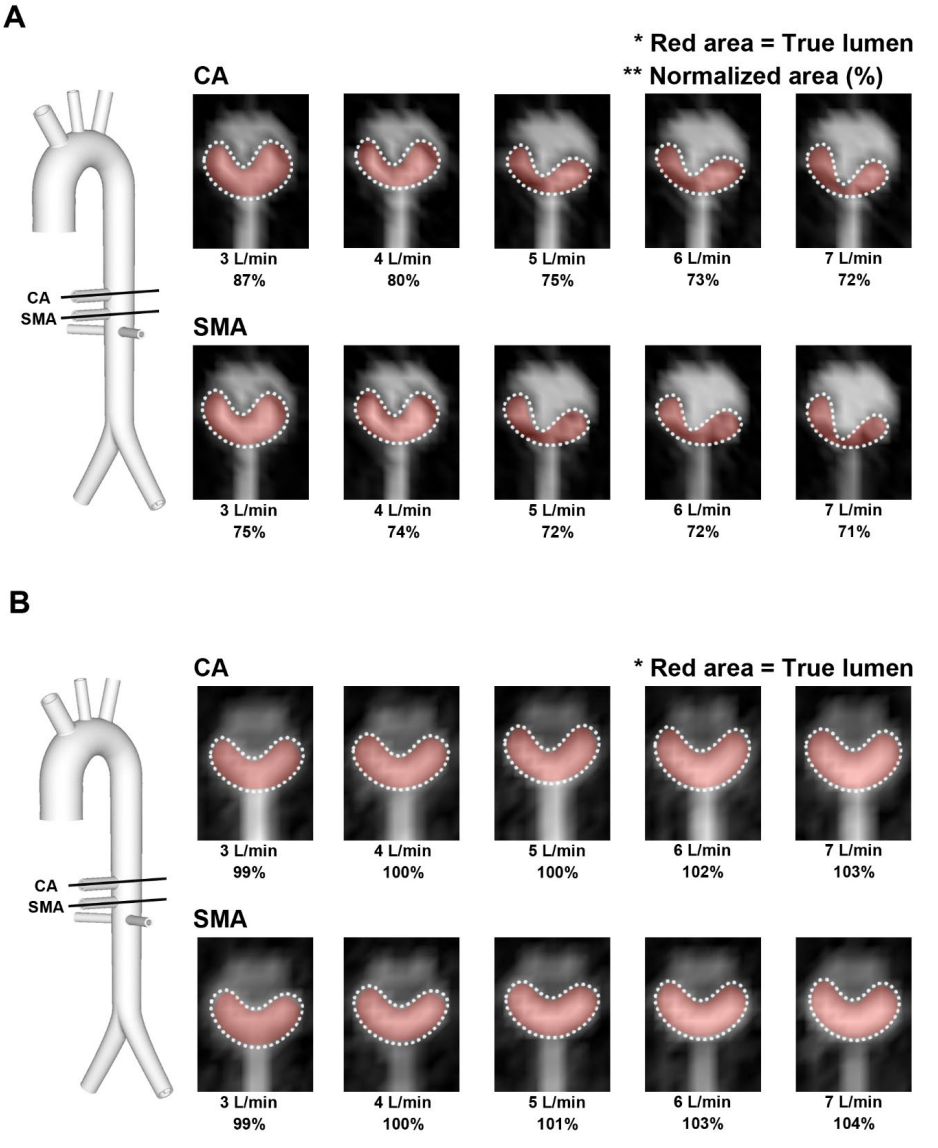

Figure 5. The flap deformation and normalized area of the true lumen at the celiac and superior mesenteric artery level in the AC (A), in the AFC (B)

Effect of cannulation on the visceral perfusion

AFC resulted in a larger visceral flow than did AC. When AFC were conducted, the amount of visceral flow increased by 125% at 3 L/min, by 89% at 4 L/min, by 67% at 5 L/min, by 98% at 6 L/min, by 101% at 7 L/min, respectively, compared to AC (Fig. 6 and Fig. S5).

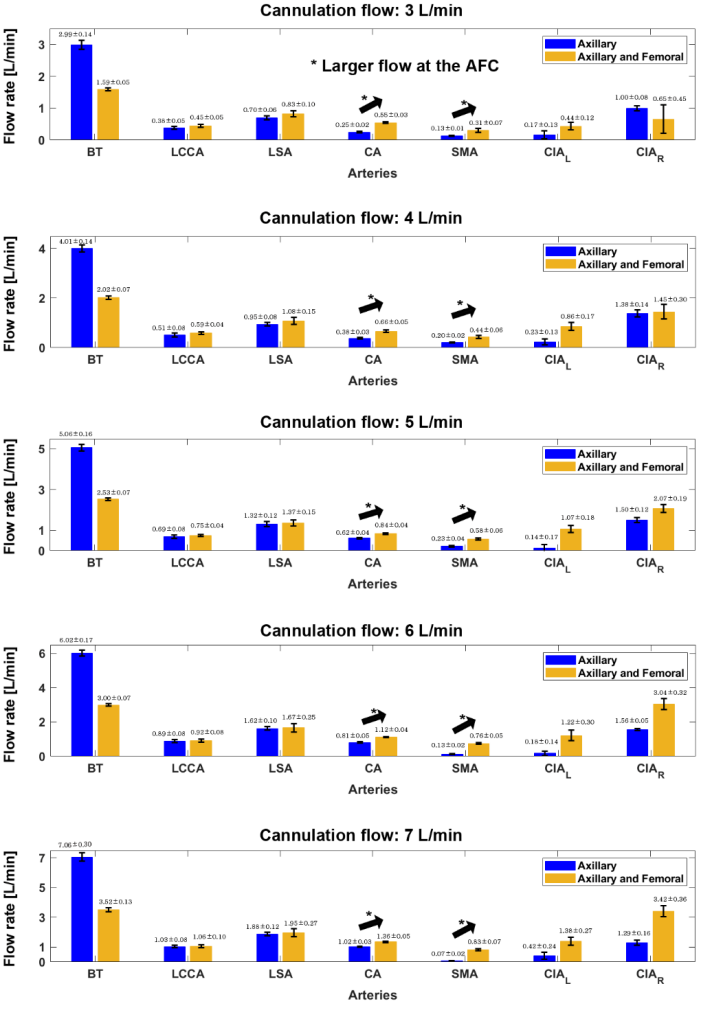

Figure 6. Flow of entire arteries in the AC and AFC

IV. DISCUSSION

Collapse of true lumen and visceral malperfusion

In this study, we hypothesized the AC can collapse the true lumen and cause visceral malperfusion due to the flexible intimal flap at the aortic dissection. The main findings of the study were that (i) blood flow at the aortic dissection and the impact of the cannulation method can be simulated with an in-vitro vascular model; (ii) AC collapsed the true lumen at the CA and SMA, while the use of AFC extended the size of true lumen compared to the use of AC alone; and (iii) visceral blood flow was greater with AFC than with AC alone.

Flow mismatch through the true and false lumen causes a pressure difference between the two lumens. As the flow through the true lumen is larger than that through the false lumen, the larger pressure drop through the true lumen causes a smaller pressure in the true lumen than that in the false lumen. As a result, the pressure difference between true and false lumens causes the intimal flap to collapse into the CA and SMA [13]. According to the fluid-dynamics in the pipe flow [14], this collapse of the true lumen occurs more frequently when the flow through the true lumen is larger, the size of the true lumen is smaller, and the flow mismatch between the true and false lumen is greater. In this study, we demonstrated that dividing a total cannulation flow in to axillary and femoral cannulation efficiently reduced the flow mismatch and resolved the visceral malperfusion.

Malperfusion in aortic dissection

Aortic dissection almost invariably causes malperfusion. Sievers et al. recently introduced an aortic-dissection classification system, incorporating the extent of malperfusion, dissection type, and tear location [15]. Malperfusion may lead to myocardial, cerebral, spinal, visceral, renal, and or peripheral ischemia, causing various symptoms and yielding differing laboratory results depending on its severity; therefore, malperfusion is not a homogeneous entity and the prognosis

depends on its severity (2, 4, 16-18). Cho et al. reported malperfusion syndrome was not a risk factor for surgical repair for AIAD [16]. However, visceral malperfusion with organ failure is a catastrophic event. Some authors reported a mortality rate of aortic dissection with visceral malperfusion was over 60% [2]. Yang et al. emphasized the importance of distinguishing between static and dynamic occlusion and between simple ischemia and necrosis; they also noted that the prognosis is much worse when symptoms such as hematochezia and a serum lactate concentration > 6 mmol/L are present [4].

Treatment options for visceral malperfusion in aortic dissection

The rapid restoration of blood flow into the true lumen and obliteration of the false lumen is the main principle for treating malperfusion in aortic dissection. In AIAD, resection of the primary tear and grafting of the ascending aorta or aortic arch can prevent aortic rupture and resolve dynamic visceral malperfusion. For residual tears in the descending thoracic aorta, the frozen elephant trunk procedure could be a suitable additional treatment for central repair of AIAD. However, in the case of severe visceral malperfusion with organ failure, various authors have reported prioritizing treatment of the visceral malperfusion, either surgically [19] or endovascularly [15], above central repair. Surgeons may be prone to prioritize central repair by default, but it is important to weigh the risk of aortic rupture to that posed by visceral malperfusion.

In complicated cases of AIIIAD, the endovascular approach is well established. Visceral and peripheral malperfusion can be treated with intimal fenestration and stent-graft insertion [17, 18, 20, 21]. In that case, usually the patient had a big primary intimal tear in the proximal descending aorta and small or no visible re-entry in the distal aorta, as determined with computed tomography. Patients who present with paraplegia or monoplegia because of spinal-cord or lumbosacral ischemia are also treated with an endovascular approach [22, 23]. However, fenestration is not always feasible, especially in lower-volume centers. To minimize ischemic

injury, especially in spinal malperfusion, the procedure should be performed as quickly as possible. In terms of procedure time and ischemic injury, there may be a role for VA-ECMO with additional spinal protection including anticoagulation and cerebrospinal fluid drainage. Acute aortic dissection is often listed as an absolute contraindication for VA-ECMO [24, 25]. However, VA-ECMO support may be a suitable option in some cases [26, 27].

Femoral cannulation during cardiopulmonary bypass for aortic dissection repair

Organ protection is one of the primary goals of aortic AIAD repair. For myocardial and cerebral protection, the optimal arterial cannulation site and degree of hypothermia have been well established [28-30]. For visceral protection, Song et al. reported the effects of intermittent femoral perfusion in dissection repair [3]. Generally, during the initial cooling phase of AIAD repair, AC is sufficient for cerebral and visceral protection. However, when there is a thin, fluctuating intimal flap in the descending thoracic aorta, AC may aggravate visceral malperfusion. In this in-vitro study, we confirmed that intimal-flap fluctuation can impede the flow to the CA and SMA via AC than pulsatile condition, and that additional femoral cannulation can increase visceral perfusion. However, aortic dissection may progress due to retrograde flow from the femoral cannulation and there is also a risk of embolic cerebral infarction. For these reasons, in our center, during AIAD repair, we try to reduce embolic risk by inducing a mixing zone in descending thoracic aorta by starting CPB first through AC and then starting femoral flow later. Progression of dissection is dangerous when it progresses to the ascending aorta, it does not seem to need much attention during AIAD repair.

Utility of the in-vitro visceral-malperfusion model and future prospects

With the in-vitro malperfusion model, it is possible to recreate intimal tears in various locations and to quantitatively measure their effects on the change in

blood flow. It can also be used to analyze the hemodynamic change according to the location of endovascular fenestration and stent-graft insertion. We demonstrated that the in-vitro experiment can be used to perform fluid-dynamic analysis by taking into account the fluid–structure interaction, and the simulator is useful in predicting the hemodynamic changes caused by aortic dissection.

Limitations

First, this was not an in-vivo experiment; however, it is challenging to perform 4D flow MRI scans in patients with acute aortic dissection. In-vitro experiments are sufficient for hemodynamic analysis, considering these practical limitations. Second, because the aortic wall is a rigid model made of acrylic, it does not have the properties of an actual aortic wall. However, it is designed to better simulate the motion of the intimal flap made of silicone. And intimal-flap motion is imperfect, and the intimal-tear geometry also differs from those in patients. However, we confirmed that the experimental model simulates visceral malperfusion, and demonstrated that such malperfusion may be resolved by additional cannulation. Development of an experimental model in which intimal-flap motion is more natural is required, as well as additional research on additional femoral cannulation in resolving visceral malperfusion.

V. CONCLUSION

Via in-vitro experiments and 4D Flow MRI flow analysis, we confirmed that intimal-flap motion can partially block blood flow to the CA and SMA. As the total flow increased with AC, the intimal flap moved closer to the CA and SMA, which indicates that AC-derived flow may induce visceral malperfusion. In our in-vitro experiment, we also confirmed that additional femoral cannulation can increase visceral perfusion. Thus, AFC may increase visceral perfusion during the initial phase of CPB.

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APPENDICES

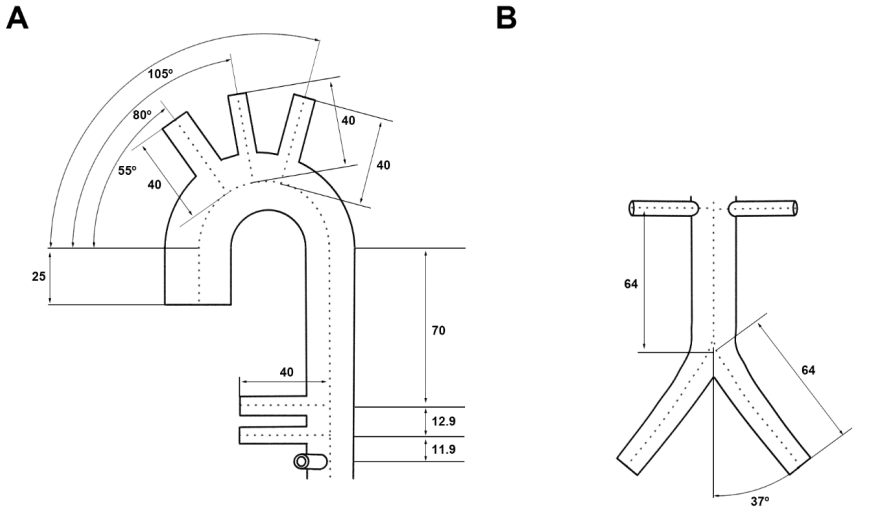

Figure S1. Drawing of idealized aortic dissection model (mm). A: upper of the aorta; B: lower of the aorta

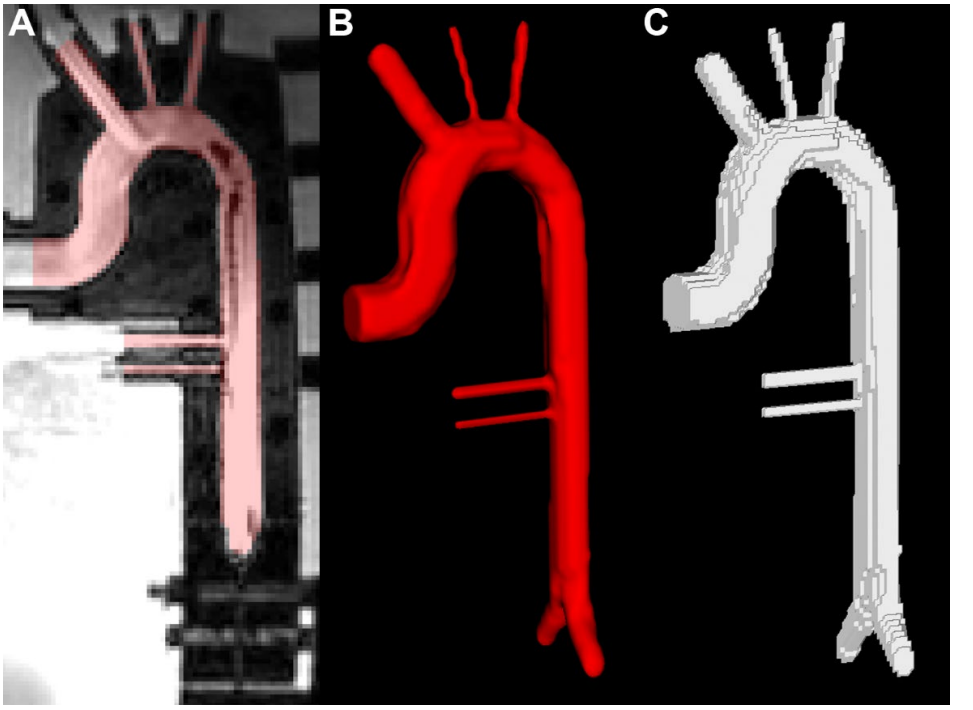

Figure S2. Process of 4D flow MRI to 3-dimensional geometry, the image to geometry (A-B), the geometry to visualization (C).

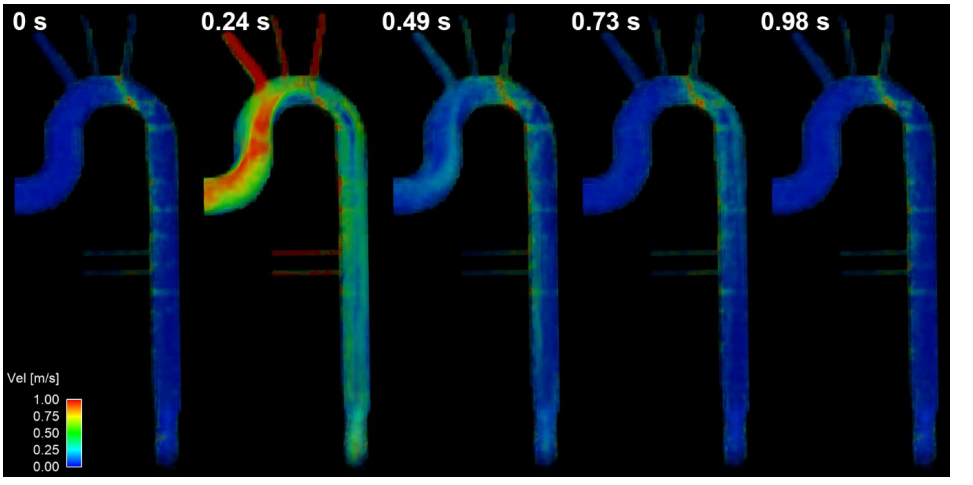

Figure S3. Velocity contour of the pulsatile

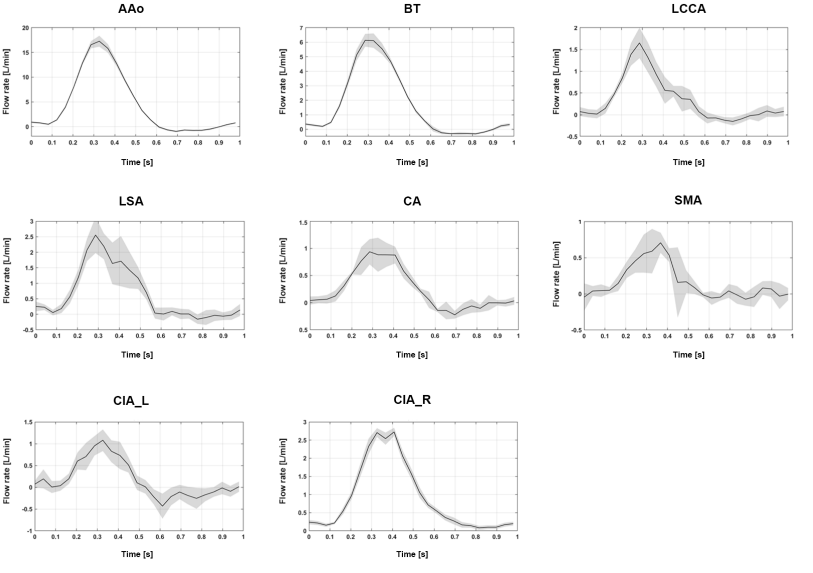

Figure S4. Flow of entire arteries in the pulsatile flow

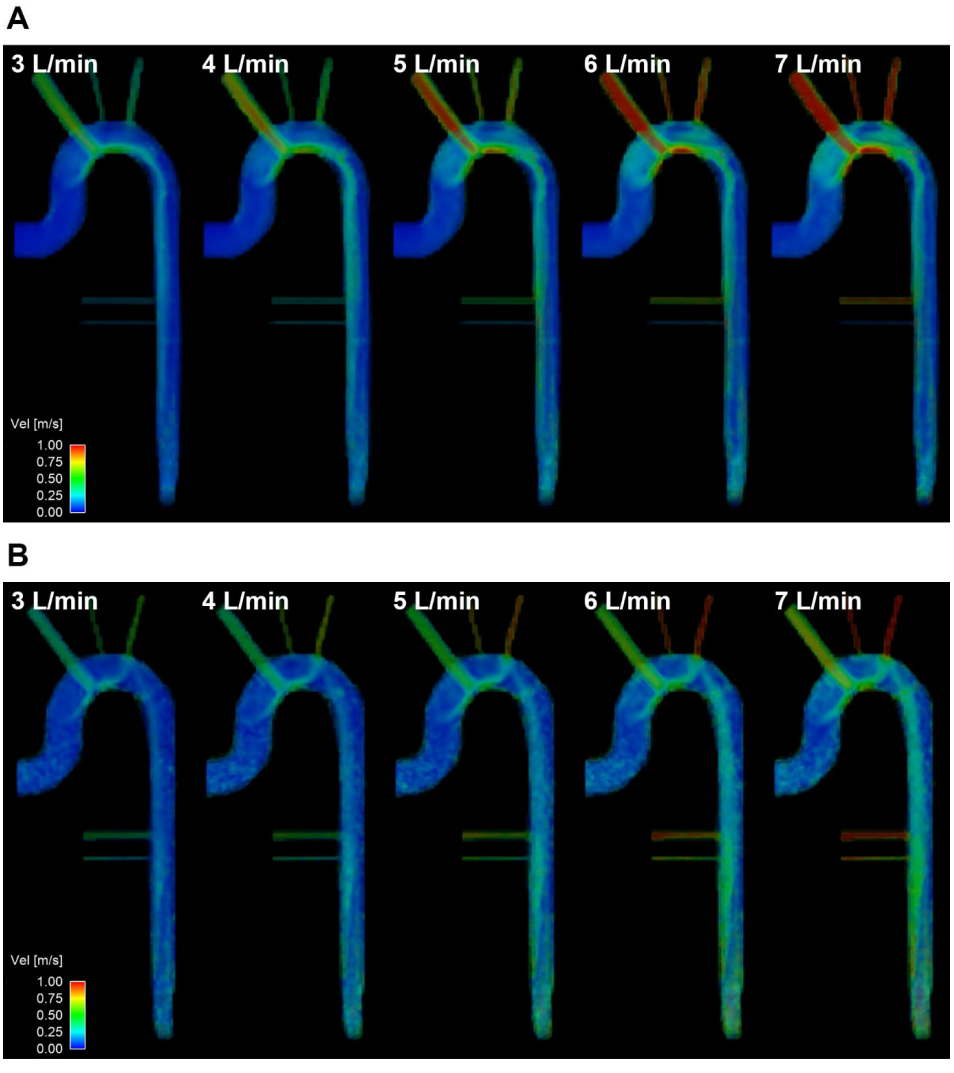

Figure S5. Velocity contour of the AC (A), of the AFC (B)

Table S1. Outer diameter and wall thickness of arteries.

Artery	Outer diameter [mm]	Wall thickness [mm]
Ascending aorta	29.1	2.8
Brachiocephalic truck	13.2	1.2
Left common carotid artery	8.2	1.2
Left subclavian artery	9.86	1.2
Celiac artery	8.4	1.2
Superior mesenteric artery	7.4	1.2
Renal artery	6.4	1.2
Abdominal aorta	19.6	1.9
Common iliac artery	12	1.5

ABSTRACT(IN KOREAN)

복부 장기 관류장애를 동반한 급성 제 1형 대동맥 박리증에서 이중 동맥 도관 삽관의 효과: 전산 유체 역학 연구

<지도교수 송석원>

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허 운

혈관 실험 모델을 사용하여 대동맥 박리 시 혈류를 시뮬레이션하고 복부 장기 관류에 대한 동맥 도관 삽입 방법의 영향을 평가하는 것을 목표로 하였음. 아크릴 대동맥 벽과, 실리콘으로 혈관 내막 박리를 구현한 모델을 제작하였고, 여러 조건에서 복부 장기 관류를 분석하기 위해 고안되었음. 일차 내막 천공은 하행 대동맥의 근위부에, 이차 내막 천공은 좌측 총장골 동맥에 위치시켰음. 심혈관 펌프를 사용하여 3-7 L/min 의 박동성 혈류, 액와 및 대퇴 동맥 도관 혈류를 재현하였고, 4D flow MRI를 이용하여 혈역학을 분석하였음. 액와 도관 삽입은 복강 및 상장간막 동맥 부위에서 진성 내강을 협착시켰지만, 액와 및 대퇴 동맥 도관을 함께 삽입하였을때는 진성 내강의 크기 변화를 보이지 않았음. 액와 및 대퇴 동맥 도관의 동시 삽입은, 액와 동맥 도관만을 사용했을 때와 비교하여 복부 장기 관류량의 증가를 보였음. 액와 동맥 도관 단일 사용과 비교하여, 액와 및 대퇴 동맥 도관 삽입 시 복부 장기 관류량은 3L/min에서 125%, 4L/min에서 89%, 5 L/min에서 67%, 6 L/min에서 98%, 7 L/min에서 101% 만큼 증가함을 확인하였음. 본 실험 모델이 대동맥 박리의 혈역학을 이해하는데 유용함을 확인함. 본 실험으로 박리된 내막의 움직임이 복강 및 상장간막 동맥의 혈류를 부분적으로 차단할 수 있으며, 추가 대퇴 동맥 도관 삽입이 복부 장기 관류를 증가 시킬 수 있음을 확인하였음.

핵심되는 말 : 대동맥 박리, 관류, 혈역학