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Case Report

Pulmonary Artery Perfusion as a Rescue Strategy for Differential Hypoxemia During Partial Cardiopulmonary Bypass in Thoracoabdominal Aortic Replacement

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Thoracoabdominal aortic replacement requires complex anesthetic management, including one-lung ventilation (OLV) and partial cardiopulmonary bypass (CPB). During OLV, partial CPB is typically established with right atrial drainage via a femoral venous cannula and femoral arterial perfusion while the aorta is cross-clamped above and below the aneurysm. Under these conditions, the coronary and supra-aortic branches are perfused primarily by native cardiac output, making adequate pulmonary oxygenation essential. However, maintaining adequate oxygenation during OLV may occasionally be challenging.

To the authors' knowledge, the use of additional pulmonary artery (PA) perfusion as part of a triple-cannulation strategy for refractory differential hypoxemia during partial CPB has rarely been reported. This report describes a case in which PA perfusion was implemented as a rescue strategy after conventional interventions failed to maintain adequate upper-body oxygenation.

Case Report

A 66-year-old woman (height, 155 cm; weight, 63.7 kg; body mass index, 26.5 kg/m²) presented with acute lower back pain and was diagnosed with a ruptured thoracoabdominal aortic aneurysm requiring emergency surgery. On admission, she was alert with a blood pressure of 152/77 mmHg, heart rate of 90 beats/min, respiratory rate of 21 breaths/min, peripheral oxygen saturation (SpO₂) of 90% on room air, and body temperature of 36.9°C. Wheezing was noted on auscultation. The patient's medical history was notable only for hypertension.

Electrocardiography demonstrated atrial fibrillation without ischemic changes. Chest radiography showed cardiomegaly (cardiothoracic ratio, 68%), diffuse bilateral pulmonary opacities, and bilateral pleural effusions (Fig 1, A). Contrast-enhanced computed tomography revealed a contained rupture of a Crawford extent III thoracoabdominal aortic aneurysm with a mediastinal hematoma. An approximately 5-cm rupture of the aneurysm wall was confirmed intraoperatively.

Transthoracic echocardiography showed preserved left ventricular function (ejection fraction, 65%); mild tricuspid, mitral, and pulmonary regurgitation; and an estimated mean PA pressure of 40 mmHg, without evidence of an atrial septal defect (ASD). Intraoperative transesophageal echocardiography (TEE), however, identified a previously undiagnosed

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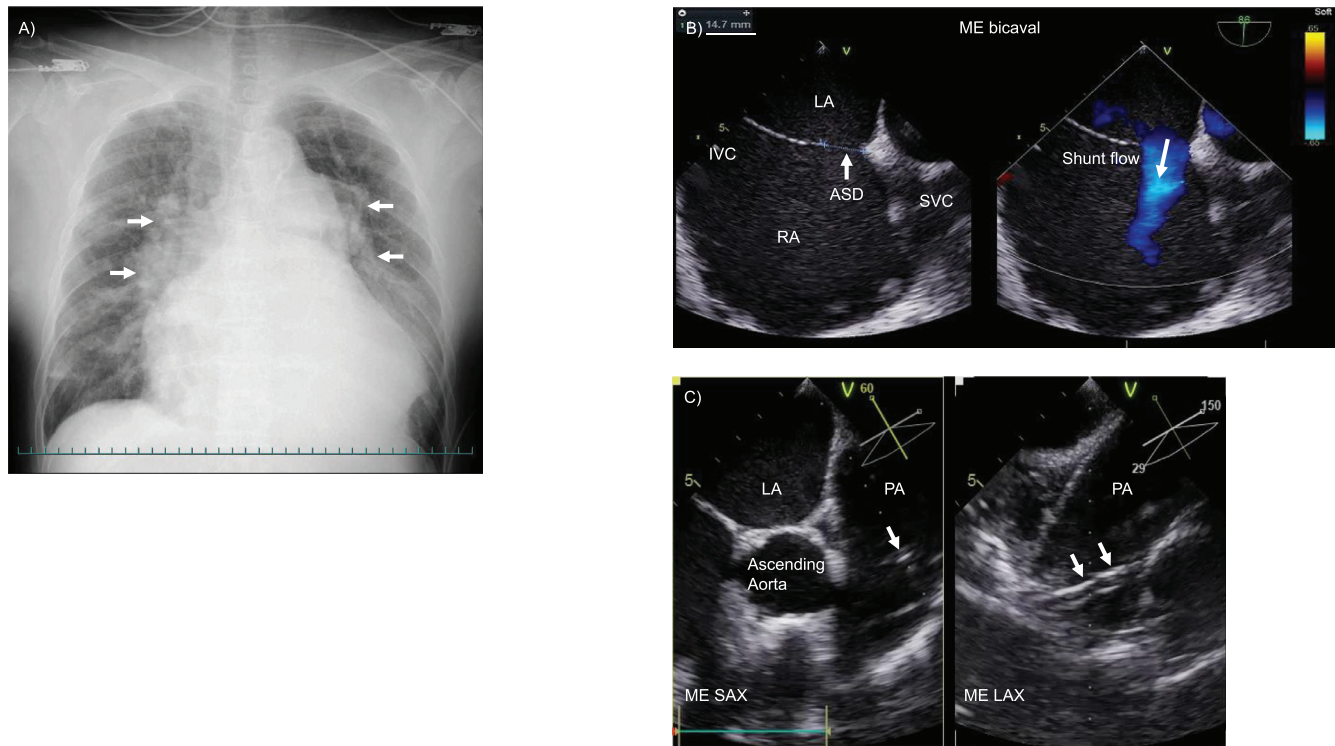


Fig 1. (A) Chest radiograph demonstrating cardiomegaly, diffuse bilateral pulmonary opacities (arrows), and bilateral pleural effusions. (B) Transesophageal echocardiography (TEE), mid-esophageal (ME) bicaval view, demonstrating a 14.7-mm secundum-type atrial septal defect (ASD): 2-dimensional image showing ASD diameter measurement (left) and color Doppler imaging demonstrating left-to-right shunt flow (arrow, right). (C) A 15-Fr cannula (arrows) inserted into the main pulmonary artery (PA), visualized on intraoperative TEE. The probe angle was adjusted to optimize visualization of the PA cannula; therefore, the long-axis (LAX) image differs slightly from the conventional ascending aortic long-axis view. IVC, inferior vena cava; LA, left atrium; RA, right atrium; SAX, short-axis; SVC, superior vena cava; V, velocity.

14.7-mm secundum-type ASD with left-to-right shunting on color Doppler imaging (Fig 1, B).

Arterial blood gas analysis on room air before supplemental oxygen demonstrated pH of 7.48, partial pressure of oxygen in arterial blood of 65 mmHg, partial pressure of carbon dioxide in arterial blood of 40 mmHg, and a base excess of 4.2 mmol/L. The B-type natriuretic peptide concentration was 313 pg/mL.

Standard American Society of Anesthesiologists monitoring was supplemented with bispectral index monitoring (BIS Complete Monitoring System; Covidien, Mansfield, MA), bilateral cerebral near-infrared spectroscopy (NIRS) (NIRO-200NX; Hamamatsu Photonics, Hamamatsu, Japan), TEE, and invasive arterial pressure monitoring via the left radial and right dorsalis pedis arteries. A PreSep central venous oximetry catheter and FloTrac system (Edwards Lifesciences, Irvine, CA) were used for continuous monitoring of central venous oxygen saturation (ScvO₂) and cardiac output, respectively.

General anesthesia was induced with propofol, remifentanyl, and rocuronium and was maintained with total intravenous anesthesia using propofol, remifentanyl, and ketamine. The trachea was intubated with a 35-Fr left-sided double-lumen tube, and correct positioning was confirmed by fiberoptic bronchoscopy. A standard adult multiplane TEE probe was carefully inserted using a gentle blind technique. After induction, the patient was placed in the right lateral decubitus position with slight anterior rotation of the torso and pelvis to facilitate left femoral cannulation for CPB.

Right-sided OLV was initiated after left thoracotomy and laparotomy to assess oxygenation during OLV. Pressure-controlled ventilation was applied with a fraction of inspired oxygen (F_IO₂) of 1.0, peak airway pressure of 25 to 30 cmH₂O, positive end-expiratory pressure of 7 cmH₂O, and respiratory rate of 12 breaths/min. The measured tidal volume was approximately 3 to 4 mL/kg based on actual body weight, consistent with reduced respiratory system compliance. However, because SpO₂ rapidly decreased to the 80% range, OLV was temporarily discontinued, and two-lung ventilation was resumed. Fiberoptic bronchoscopy confirmed correct positioning of the double-lumen tube, and recruitment maneuvers were performed. Ventilator settings, including positive end-expiratory pressure, were adjusted to improve oxygenation. When OLV was resumed, continuous positive airway pressure was not applied to the nonventilated lung because maintaining surgical exposure was prioritized during this emergency procedure. As further conventional interventions would have required prolonged interruption of surgery, the anesthesiologist proposed additional PA perfusion. Following discussion with the surgeon and perfusionist, the team proceeded with triple-cannulation partial CPB (right atrial drainage, femoral arterial perfusion, and additional PA perfusion) to support oxygenation during OLV.

A 23-Fr multistage venous cannula was advanced from the left femoral vein into the right atrium under TEE and fluoroscopic guidance, and a 17-Fr arterial cannula was inserted into

the left femoral artery. Heparin was administered to maintain an activated clotting time > 400 seconds. Partial CPB was initiated at a flow of 3.0 L/min, and OLV was resumed. Before aortic cross-clamping, the coronary and supra-aortic branches were perfused by a mixture of native cardiac output and oxygenated blood from the femoral arterial cannula at the watershed (Fig 2, A). While upper-body oxygenation remained acceptable, a 15-Fr cannula was inserted into the main PA through the left thoracotomy under direct vision (Fig 1, C), thereby completing PA cannulation before differential hypoxemia developed. All cannulas were manufactured by Maquet (Rastatt, Germany).

After the proximal aortic cross-clamp was applied to the descending thoracic aorta and the distal cross-clamp was placed between the origins of the superior mesenteric artery (SMA) and the renal arteries, selective antegrade perfusion of the celiac artery and SMA was initiated at a total flow of 0.35 L/min. Consequently, the coronary and supra-aortic branches were perfused exclusively by poorly oxygenated native cardiac output (Fig 2, B), and SpO₂ decreased to the 80% range.

PA perfusion was initiated (Fig. 2, C) at a fixed flow rate of 1.2 L/min, while the main CPB flow was maintained at 3.0 L/min, including the PA perfusion flow, using a

centrifugal pump. The PA perfusion line originated from a Y branch of the main arterial line of the CPB circuit and was driven by a roller pump incorporated into the authors' institutional selective cerebral perfusion circuit, which also supplied visceral perfusion (Fig 2, D). PA perfusion pressure was maintained at 80 to 100 mmHg, whereas the main arterial line pressure remained approximately 130 mmHg. Both the PA and femoral perfusates were maintained at mild hypothermia (32°C–34°C), and the lowest core temperature during CPB was 32.7°C. Oxygenation, systemic hemodynamics, venous reservoir volume, and TEE assessment of cardiac filling were continuously monitored throughout PA perfusion to guide volume management.

Repeated color Doppler TEE demonstrated persistent left-to-right shunting across the ASD without any apparent change in shunt direction or flow pattern. Following initiation of PA perfusion, SpO₂ improved and upper-body oxygenation remained acceptable, as reflected by ScvO₂, NIRS, and arterial blood gas analysis (Table 1).

The aneurysm was replaced with a prosthetic graft extending from the proximal descending thoracic aorta to just proximal to the origin of the SMA. The patient was weaned from CPB, and the surgery was completed uneventfully. Postoperative transfusion was administered for anticipated postoperative

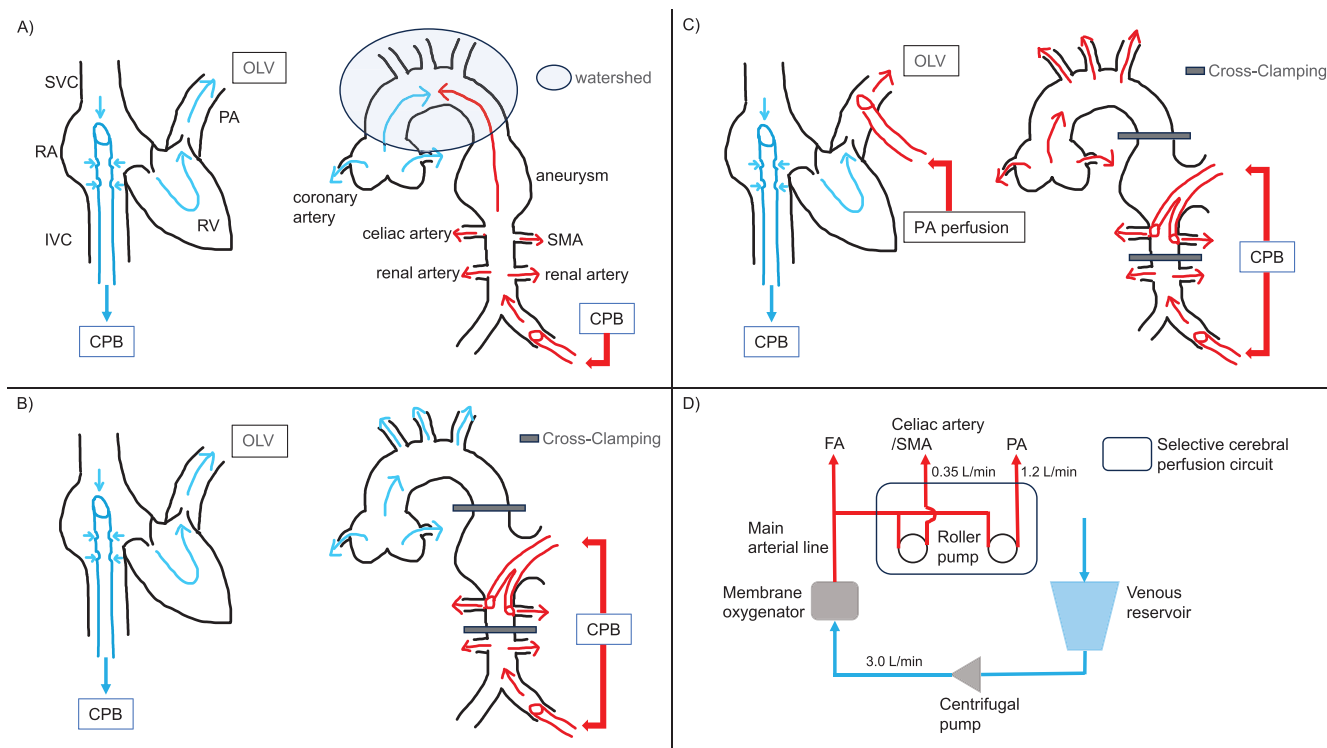


Figure 2. (A) Before aortic cross-clamping, the coronary and supra-aortic branches received a mixture of oxygenated blood from femoral partial cardiopulmonary bypass (CPB) and native cardiac output, with a watershed (mixing point) between the two perfusion sources. (B) After aortic cross-clamping, the watershed disappeared, and the coronary and supra-aortic branches were perfused exclusively by poorly oxygenated native cardiac output, resulting in differential upper-body hypoxemia. (C) Pulmonary artery (PA) perfusion delivered oxygenated blood through the pulmonary circulation, restoring oxygen delivery to the coronary and supra-aortic branches. (D) Schematic of perfusion system. The main CPB circuit, driven by a centrifugal pump, was Y-branched to a roller pump-driven selective cerebral perfusion circuit. The branch supplied PA, celiac artery, and superior mesenteric artery (SMA) perfusion, while the main CPB flow was maintained through the femoral arterial cannula. Red arrows represent oxygenated blood flow from the CPB circuit, whereas blue arrows represent poorly oxygenated native blood flow. Throughout one-lung ventilation (OLV), ventilation was performed with a fraction of inspired oxygen (F_IO₂) of 1.0. During CPB, the membrane oxygenator F_IO₂ was maintained at 0.7 to 0.8. FA, femoral artery; IVC, inferior vena cava; RA, right atrium; RV, right ventricle; SVC, superior vena cava.

Table 1
 Perioperative Changes in Oxygenation, Cerebral Oxygenation, Hemodynamic Parameters, CPB Settings, and Ventilator Settings During OLV and PA Perfusion

Time Point	SpO ₂ , %	SevO ₂ , %	NIRS		PaO ₂ , mmHg	SaO ₂ , %	Sampling Site	CO, L/min	Hb, g/dL	PA: Flow, L/min/ Pressure, mmHg	CPB: Flow, L/min/ Pressure, mmHg	CPB F _i O ₂	Ventilator Settings: Mode/F _i O ₂ /PIP/ PEEP, cmH ₂ O/RR, breaths/min
			L	R									
Baseline (two-lung ventilation)	98	74	56	59	132	100	L RA	3.4	11.6	*	*	—	PCV/1.0/25/7/12
OLV initiation	87	75	52	56	—†	—†	*	3.4	—†	*	*	—	PCV (OLV)/1.0/25/7/12
Before PA perfusion	89	81	59	60	—†	—†	*	3.3	—†	*	3.0/130	0.7	PCV (OLV)/1.0/30/7/12
After PA perfusion	99	93	65	68	371	100	L RA	3.6	10.6	1.2/80-100	3.0/130	0.8	PCV (OLV)/1.0/30/7/12
After aortic declamping	98	87	64	68	162	99.7	L RA	3.1	12.9	1.2/80-100	3.0/130	0.8	PCV (OLV)/1.0/30/7/12

NOTE. Arterial blood gas values were obtained from the left radial artery. CPB F_iO₂ indicates the oxygen fraction delivered to the membrane oxygenator.

Abbreviations: CO, cardiac output; CPB, cardiopulmonary bypass; F_iO₂, fraction of inspired oxygen; Hb, hemoglobin; L, left; OLV, one-lung ventilation; NIRS, near-infrared spectroscopy; PA, pulmonary artery; PaO₂, partial pressure of oxygen in arterial blood; PCV, pressure-controlled ventilation; PEEP, positive end-expiratory pressure; PIP, peak inspiratory pressure; R, right; RA, radial artery; RR, respiratory rate; SaO₂, arterial oxygen saturation; SevO₂, central venous oxygen saturation; SpO₂, peripheral oxygen saturation.

* Not applicable.

† Not measured.

blood loss; however, the patient remained hemodynamically stable, and vasoactive agents were discontinued on postoperative day (POD) 1. The patient was extubated on POD 3 after recovery of consciousness and adequate respiratory function, completed the institutional postoperative rehabilitation program, and was discharged on POD 16 without neurologic complications. The incidentally identified ASD was managed conservatively, with elective outpatient evaluation planned.

Discussion

Thoracoabdominal aortic aneurysm surgery commonly involves OLV and partial CPB or left heart bypass, both of which depend on adequate native pulmonary oxygenation. During OLV, a rapid decline in partial pressure of oxygen in arterial blood predicts persistent intraoperative hypoxemia¹ and may increase the risk of adverse neurologic outcomes.² Therefore, prompt restoration of adequate oxygen delivery may be particularly important for the heart and brain because of their high oxygen demand.^{3,4}

The North-South phenomenon, also known as the Harlequin phenomenon, is a form of differential hypoxemia in which the upper body is perfused by poorly oxygenated native cardiac output while the lower body receives oxygenated blood from peripheral circulatory support. Although most commonly described during venoarterial (VA) extracorporeal membrane oxygenation (ECMO), a similar physiological mechanism may occur during partial femoral CPB.^{5,6} The location of the mixing point between retrograde oxygenated blood from the femoral arterial cannula and antegrade native cardiac output determines which circulation receives oxygenated blood. Poorly oxygenated native blood preferentially perfuses the coronary and supra-aortic circulations as the watershed shifts distal to the supra-aortic vessels with recovery of native cardiac function.

In the present case, descending thoracic aortic cross-clamping (Fig 2, B) likely prevented effective mixing of retrograde CPB flow and native cardiac output, such that the coronary and supra-aortic circulations depended predominantly on native cardiac output despite femoral arterial perfusion, resulting in differential hypoxemia.

To address differential hypoxemia, additional superior vena cava cannulation is commonly used during VA ECMO.⁷ Conversion to venoarterial-venous ECMO has been described as another strategy to improve upper-body oxygenation by augmenting oxygen delivery through the pulmonary circulation. The present approach shares this physiological objective by improving oxygen delivery to the coronary and supra-aortic circulations while maintaining systemic circulatory support. Unlike venoarterial-venous ECMO, this approach delivered oxygenated blood directly into the PA during partial CPB rather than returning it to the right atrium or central venous circulation. A practical advantage of this approach is that PA cannulation can be readily performed because the main PA is already exposed through the left thoracotomy.

This case suggests that management during femoral partial CPB should include assessment of upper-body oxygenation and differential arterial perfusion. Continuous monitoring of SpO₂, ScvO₂, and NIRS, together with upper- and lower-extremity arterial pressure monitoring, may facilitate early recognition of differential hypoxemia and guide appropriate management.

The previously undiagnosed ASD may have influenced baseline oxygenation and pulmonary hemodynamics, although the preoperative hypoxemia was more likely related to heart failure–associated pulmonary edema and bilateral pleural effusions. During OLV, an ASD may contribute to hypoxemia if right-sided pressures and pulmonary vascular resistance increase, resulting in right-to-left shunting.⁸ Intraoperative color Doppler TEE demonstrated persistent left-to-right shunting both after the onset of OLV and after initiation of PA perfusion, with no apparent change in shunt direction or flow pattern.

Potential complications of PA perfusion include PA injury and pulmonary edema. Previous reports have suggested that both perfusion flow and duration may contribute to bleeding complications.⁹ In this case, PA perfusion was performed at a flow rate of 1.2 L/min for 103 minutes, with the PA perfusion line pressure maintained at 80 to 100 mmHg under continuous monitoring, and no pulmonary complications were observed.

This report has several limitations. A comprehensive preoperative pulmonary evaluation was not feasible because of the emergent nature of the procedure. Although the risk of hypoxemia during OLV was recognized, PA perfusion was not planned preoperatively; rather, it was selected intraoperatively as a rescue strategy after conventional measures failed.

Because this report describes a single case without a controlled comparison, the contribution of PA perfusion to the observed improvement in oxygenation cannot be determined with certainty. The influence of the previously undiagnosed ASD and other physiological factors, including pulmonary vascular resistance and CPB flow distribution, also remains uncertain.

Alternative rescue strategies, such as superior vena cava cannulation, axillary arterial cannulation, or conversion to full CPB, should be considered when appropriate. In this patient, PA perfusion was selected because the main PA had already been surgically exposed. Nevertheless, this case suggests that PA perfusion may represent a feasible rescue strategy for refractory differential hypoxemia during thoracoabdominal aortic surgery with femoral partial CPB. Further physiological and clinical studies are needed to define appropriate patient selection, the optimal PA perfusion flow, the safety of PA perfusion, and the role of this strategy relative to other treatment strategies.

Consent for publication

Written informed consent for the publication of this case report and the accompanying images was obtained from the patient.

Declaration of generative AI and AI-assisted technologies in the manuscript preparation process

During the preparation of this work, the authors used ChatGPT (Open AI) for English translation and language editing. The authors reviewed and edited the output as necessary and take full responsibility for the content of the manuscript.

Declaration of competing interest

The authors declare no competing interests.

CRediT authorship contribution statement

Dai Tanahira: Writing – original draft. **Yukiko Okamura:** Writing – review & editing. **Kohei Tomita:** Writing – review & editing. **Naoyuki Hirata:** Writing – review & editing.

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