

https://doi.org/10.5090/jcs.24.046 pISSN: 2765-1606 eISSN: 2765-1614 J Chest Surg. 2024;57(4):369-370



# Commentary: Concomitant Pulmonary Artery Angioplasty after Congenital Heart Defect Repair: Should We Consider Early Independent Surgery?

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ARTICLE INFO Received April 26, 2024 Accepted April 26, 2024

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Patients with congenital heart defects often present with branch pulmonary artery stenosis (BPAS) due to abnormal ductal tissue extension, postoperative damage, or changes in configuration from nearby structures [1]. Surgical pulmonary artery angioplasty is typically necessary after the failure of percutaneous balloon angioplasty and is usually performed concurrently with other procedures, such as pulmonary valve implantation. However, the optimal strategies for surgical angioplasty, including its timing and necessity, remain a matter of debate.

Son et al. [2] reported the benefits of surgical pulmonary artery angioplasty, noting increased lung perfusion in adolescents, particularly in those with normal hilar diameter who have focal BPAS. The authors suggested that relieving BPAS before complete microvascular maturation (under 21 years old) in adolescent patients could lead to improved pulmonary perfusion. However, this procedure mostly failed in adult patients. A hypoplastic pulmonary artery was identified as a negative factor for enhanced pulmonary perfusion, potentially due to poor peripheral pulmonary vasculature distal to the hilum.

These outcomes highlight the clinical significance of chronic BPAS on pulmonary development. Limited ipsilat-

eral pulmonary blood flow leads to reduced distensibility and increased vascular resistance. This diminished blood flow decreases pulmonary artery shear strength, thereby inhibiting the formation of new vessels. As a result, vascular structures remain underdeveloped, characterized by a smaller and shorter central pulmonary artery with disorganized side branches. Consequently, alveolar growth is impeded, and limited blood flow leads to reduced lung volume. Peripheral vasculatures become dilated with medial atrophy as a compensatory mechanism to decrease total pulmonary vascular resistance, which requires careful interpretation of angiographic data [1]. Additionally, post-stenotic dilatation of the pulmonary artery, categorized by the authors as type III, is likely caused by turbulent blood flow, indicating the severity and prolonged duration of the stenotic lesion. The study suggests that concomitant pulmonary artery angioplasty during adolescence is worth considering, as it may provide an opportunity for pulmonary artery development.

Another surgical option we should consider is "independent" pulmonary artery angioplasty. BPAS may be detected following the reconstruction of the right ventricular outflow tract using bioprosthetic materials. Increased pulmo-

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nary artery pressure in the proximal BPAS is a risk factor for right heart hypertrophy/dysfunction and structural valve deterioration. BPAS increases the shear strength at the flexion of the leaflet, which could lead to deterioration through leaflet tearing or calcification. Additionally, the leaflet surface experiences increased downstream tensile strength due to BPAS, further contributing to calcification [3-6]. Since valvular failure can result in right heart dysfunction with poor clinical outcomes, independent surgical pulmonary artery angioplasty should also be considered as an option, rather than waiting for other surgical issues to arise.

I would like to thank the authors for the important work presented in this article.

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### Author contributions

All the work was done by Won Young Lee.

#### Conflict of interest

No potential conflict of interest relevant to this article was reported.

### Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-forprofit sectors.

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