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Exercise Catheterization for Hemodynamic Evaluation of Adults with Coarctation of the Aorta

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ABSTRACT

Background: Coarctation of the aorta (CoA) is associated with a generalized arteriopathy and long-term complications despite repair. Data on invasive exercise hemodynamics in this population are lacking. Accordingly, we reviewed adults with CoA undergoing exercise catheterization to assess 1. hemodynamic profile; 2. feasibility for assessment of CoA severity. **Methods:** Twenty patients undergoing exercise cardiac catheterization (12 arm adduction and 8 supine cycle ergometry) at a quaternary care center between 2004 and 2021 were identified. Resting and exercise hemodynamic data were abstracted from the procedure logs. **Results:** Mean age was 43.6 ± 12.0 years. Eleven patients (55%) had resting pulmonary arterial wedge pressure (PAWP) >15 mmHg; among those undergoing arterial catheterization, left ventricular end-diastolic pressure was >15 mmHg in 63%. Eleven patients (55%) had pulmonary hypertension: 7 (35%) combined and 4 (20%) isolated post-capillary. At peak exercise, ΔPAWP/Δcardiac output (CO) ≥2 and Δmean pulmonary artery pressure/ΔCO ≥3 mmHg/l/min were found in 7 (78%) and 6 (67%) patients, respectively; the composite of exercise PAWP ≥25 mmHg or ΔPAWP/ΔCO >2 was seen in 12 (86%). CoA peak-to-peak gradients at baseline (n = 14) and during exercise (n = 9) were 12 (3–16) and 16 mmHg (9–28), respectively. Only 2 patients had an increase in CoA gradient to >20 mmHg with exercise. **Conclusions:** Diastolic dysfunction and pulmonary hypertension were highly prevalent, with exercise unmasking abnormal diastolic and pulmonary vascular reserve in some individuals. Most patients failed to show significant increases in CoA peak-to-peak gradients with exercise. Further studies are warranted to establish the best diagnostic method for CoA severity assessment.

KEYWORDS

Coarctation of the aorta; exercise heart catheterization; diastolic dysfunction; hypertension

1 Introduction

Coarctation of the aorta (CoA) is associated with generalized arteriopathy that persists despite successful treatment/repair, and carries reduced survival [1–6]. The management of adults with CoA mostly relates to long-term complications following repair in childhood, although late diagnosis/intervention may occur [7,8]. Intrinsic aortic wall abnormalities and chronic pressure overload from mechanical obstruction result in impaired vascular compliance and endothelial dysfunction, which contribute to the development of

arterial hypertension, premature coronary artery disease, cerebrovascular disease, and aortic aneurysms [9–11]. Ultimately, a maladaptive response of the left ventricle (LV) occurs, with myocyte hypertrophy and replacement fibrosis [12], alterations in myocardial viscoelastic properties, and, ultimately, diastolic and systolic dysfunction [13,14].

The need for re-intervention of repaired CoA has been reported in approximately 30% [3] but the definition of re-coarctation has varied in the literature. In addition, the challenges in hemodynamic assessment of CoA are well-known. This is reflected in the American College of Cardiology and the European Society of Cardiology guidelines for management of adult congenital heart disease, where indication for intervention is based on a combination of complications (e.g., hypertension), as well as hemodynamic and anatomic parameters [15,16]. Studies aimed at improving the diagnosis of CoA and re-coarctation have analyzed myocardial and hemodynamic response to exercise by non-invasive methods [17–20]. However, data on exercise invasive hemodynamic evaluation of pulmonary, systemic and ventricular pressures are lacking. Accordingly, we reviewed our experience of adults with CoA undergoing invasive rest and exercise hemodynamic evaluation. Our goals were: 1) to assess the hemodynamic profile at rest and under invasive exercise stress testing, 2) to analyze feasibility of this method for evaluation of CoA severity.

2 Methods

Adults with native and repaired CoA undergoing exercise cardiac catheterization at a quaternary care center between January 2004 and November 2021 were retrospectively identified using an electronical search tool. The study was approved by the Mayo Clinic Institutional Review Board (Mayo Clinic Adult Congenital Heart Disease Registry, IRB#20-007695) and only patients providing research authorization were included in the study.

Cardiac catheterization was performed in a fasting state under light sedation. Exercise was performed via supine cycle ergometry or arm adduction with 4-pound weights, as previously described [21]. Invasive blood pressure measurements in the ascending and descending aorta at baseline and maximal exercise were recorded to assess peak-to-peak gradient across the CoA at rest and its response to exercise. At baseline, LV diastolic dysfunction was defined as an end-diastolic pressure (LVEDP) >15 mmHg. Pulmonary arterial wedge pressure (PAWP) or direct left atrial pressure >15 mmHg was defined as elevated filling pressures. During exercise, a mean PAWP ≥25 mmHg or pulmonary artery pressure (mPAP) ≥40 mmHg were considered abnormal [22]. Precapillary pulmonary hypertension (PH) corresponded to resting mPAP ≥25 mmHg, PAWP ≤15 mmHg and pulmonary vascular resistance >3 Wood units, while those with post-capillary PH were subdivided into isolated postcapillary PH (defined as mPAP ≥25 mmHg, PAWP >15 mmHg and pulmonary vascular resistance ≤3 Wood units) and combined PH (mPAP ≥25 mmHg, PAWP >15 mmHg and pulmonary vascular resistance >3 Wood units) [23]. Pulmonary vascular resistance was calculated as (mPAP–PAWP or left atrial pressure)/pulmonary flow. Reported pressure measurements correspond to computer-generated averages over ≥5 consecutive cardiac cycles recorded during spontaneous breathing.

Pulmonary and systemic flows were calculated by the direct Fick method; alternatively, the indirect Fick or thermodilution methods were used. Cardiac output (CO) response to exercise was calculated as [ΔCO/(ΔVO2*0.006)] * 100; [24] a value ≥80% was defined as preserved CO reserve. ΔPAWP/ΔCO and ΔmPAP/ΔCO were assessed as measures of LV diastolic dysfunction and pulmonary vascular reserve; cut-off values of ≥2 and ≥3 mmHg/l/min, respectively, were defined as abnormal [22].

Continuous variables are presented as mean ± standard deviation or median (interquartile range) and nominal variables as counts (%). Comparisons between resting and exercise data were performed using paired Wilcoxon rank-sum test (non-parametric continuous variables). Statistical analysis was conducted with JMP for SAS V.14.1.0; p values < 0.05 were considered statistically significant.

3 Results

The final cohort consisted of 20 patients with only 1 individual having unrepaired CoA. Demographic and clinical data are presented in Table 1. Mean age was 43.6 ± 12.0 years. All CoA were juxtaductal. Among those with repaired CoA, 18 (95%) had primary surgical repair while 1 (5%) had primary percutaneous intervention, with the median age at repair being 3 years (0–11). Fourteen patients (70%) required subsequent interventions [12 surgical (60%), 2 (10%) percutaneous] for re-coarctation. Associated congenital defects included aortic arch hypoplasia (10, 63%), bicuspid aortic valve (10, 53%), mitral valve anomalies (9, 45%), aortic valve/subaortic stenosis (4, 20%), atrial and ventricular septal defects (3, 15% each), and pulmonary valvular and supravalvular stenosis (1, 5%). Two patients (10%) had Turner syndrome. Coronary artery disease with >50% vessel obstruction was diagnosed in 4 patients (24%) by invasive angiography or cardiac computed tomography.

Table 1: Demographic and clinical features

Variables	n	
Demographics and comorbidities		
Sex, male (%)	20	10 (50%)
Age at diagnosis, years	20	3.5 (0–12.3)
Hypertension (%)	20	17 (85%)
Dyslipidemia (%)	20	10 (50%)
Diabetes mellitus (%)	20	0
Body mass index, kg/m^2	20	31.2 ± 6.5
Obstructive sleep apnea (%)	20	6 (30%)
Clinical diagnosis of heart failure (%)	20	12 (60%)
Coronary artery disease (stenosis >50%) (%)	17	4 (24%)
Prior atrial arrhythmias (%)	20	10 (50%)
Prior ventricular arrhythmias (%)	20	2 (10%)
CIED (%) *	20	3 (15%)
Chronic kidney disease (%)	20	3 (15%)
Medications at time of cardiac catheterization		
Loop diuretics (%)	20	5 (25%)
Thiazides (%)	20	4 (20%)
Pulmonary vasomodulator therapy (%)	20	2 (10%)
Beta blockers (%)	20	12 (60%)
ACEI/ARB (%)	20	11 (55%)
Aldosterone receptor antagonists (%)	20	4 (20%)
Calcium channel blockers (%)	20	3 (15%)
Hydralazine/nitrates (%)	20	2 (10%)

Notes: ACEI = angiotensin converting enzyme inhibitor; ARB = angiotensin receptor blocker; CIED = cardiac implantable electronic device.
*One patient had a pacemaker; two patients had an implantable cardioverter defibrillator.

Four patients (20%) were in New York Heart Association functional class III–IV, while 4 were asymptomatic. Nine patients had pre-procedural outpatient upper and lower limb blood pressure

measured; median systolic pressure gradient was 25 (15–31) mmHg. Detailed information of pre-procedural imaging is depicted in Table 2. Echocardiography was performed a median of 6 days (1–31) before catheterization. LV ejection fraction was >50% in all but 2 patients. Two patients had a small residual atrial septal defect (10%) and 1 (5%) had a small ventricular level shunt. An aortic or mitral valve prosthesis was present in 4 patients (20%) each (coexistent in 1). Ascending to descending aorta peak and mean gradients were 19 (13–30) and 13 (7–18) mmHg, respectively. The mean ratio of diameters between the aortic isthmus and the descending thoracic aorta at the diaphragm measured by cross-sectional imaging was 0.8 ± 0.2.

Table 2: Pre-procedural imaging data

Variables	n	
Echocardiography		
Left ventricle ejection fraction, %	20	60.8 ± 9.7
Left ventricular mass index, g/m^2	17	97 (82–128)
≥Moderate right ventricular enlargement, %	20	3 (15%)
≥Moderate right ventricular systolic dysfunction, %	20	1 (5%)
Right ventricular systolic pressure, mmHg	18	38 (30–47)
Mitral annulus medial e' velocity, m/s	15	0.07 ± 0.02
Medial E/e' ratio	12	12 (9–14)
Mean mitral valve gradient, mmHg	9	5 (3–7)
≥Moderate mitral regurgitation, %	20	2 (10%)
Mitral valve prosthesis, %	20	4 (20%)
≥Moderate tricuspid regurgitation, %	20	4 (20%)
≥Moderate aortic valve stenosis, %	20	5 (25%)
Mean aortic valve gradient, mmHg	15	7 (4–25)
≥Moderate aortic valve regurgitation, %	20	0
Aortic valve prosthesis, %	20	4 (20%)
Ascending to descending aorta mean gradient, mmHg	17	13 (7–18)
Cross-sectional imaging[⌟]		
Time from catheterization, days	16	26 (3–400)
Distal aortic arch diameter, mm	15	21 ± 3
Aortic isthmus/narrowest site diameter, mm	16	16 ± 4
Distal descending aorta diameter (diaphragm), mm	16	20 ± 4
Aortic isthmus ratio	16	0.8 ± 0.2

Note: [⌟] Cardiac computed tomography or cardiac magnetic resonance.

3.1 Resting and Exercise Invasive Hemodynamics

Detailed information about rest and exercise catheterization is shown in Table 3. Arterial catheterization was performed in 17 patients (85%) while venous catheterization was performed in all. The type of exercise was arm adduction in 12 cases (60%) and supine cycle ergometry in 8 (40%). Procedure indication was clinical deterioration in 9 patients (45%), need for additional hemodynamic evaluation following

echocardiographic assessment in 8 (40%), preoperative evaluation for non-coarctation cardiac surgery in 2 (10%), and exercise-induced hypertension in 1 (5%).

Table 3: Cardiac catheterization measurements

Variables	n		n	
	Baseline catheterization		**Exercise catheterization***	
LV systolic pressure, mmHg	16	135 (123–162)	7	168 (142–187)
LV end-diastolic pressure, mmHg	16	20 (14–28)	7	25 (15–26)
Systolic arterial pressure AAo, mmHg	17	129 ± 19	16	159 (132–169)
Diastolic arterial pressure AAo, mmHg	17	70 ± 8	16	80 (73–93)
Mean arterial pressure AAo, mmHg	17	93 ± 12	16	111 (100–128)
Systolic arterial pressure DAo, mmHg	14	112 (104–134)	9	121 (114–150)
Diastolic arterial pressure DAo, mmHg	14	69 (62–79)	9	82 (69–96)
Mean arterial pressure DAo, mmHg	14	87 (81–102)	9	103 (91–118)
AAo to DAo peak-to-peak gradient, mmHg	14	12 (3–16)	9	16 (9–28)
Right atrial pressure, mmHg	20	7 (5–11)	6	18 (10–23)
Pulmonary artery mean pressure, mmHg	20	27 (21–33)	19	43 (32–68)
PAWP/left atrial pressure, mmHg	20	16 ± 5	18	29 ± 12
Cardiac index, l/min/m^2	20	2.5 ± 0.6	9	4.1 (3.1–7.1)
Systemic vascular resistance, dyn·s·cm^{-5}	17	1425 (1065–1886)	4	1059 (854–1324)
Pulmonary vascular resistance, WU	20	1.9 (1.4–3.9)	9	1.5 (0.9–3.8)
Cardiac output response, %	-	-	9	82 (72–116)

Notes: AAo = ascending aorta; DAo = descending aorta; dyn·s·cm^{-5} = dynes per second per $centimeter^{-5}$; LV = left ventricular; PAWP = pulmonary arterial wedge pressure; WU = Wood units.
*Data correspond to peak exercise.

At rest, 10 patients (63%) had LVEDP >15 mmHg and 11 (55%) had PAWP >15 mmHg. Resting PH was present in 11 patients (55%): 7 (35%) combined and 4 (20%) isolated post-capillary. At peak exercise, 11 (61%) had PAWP ≥25 mmHg with 2 of them having normal resting PAWP. Thirteen individuals (68%) had an exercise mPAP ≥40 mmHg. Of 9 patients with CO measurement during exercise, normal cardiac output response was present in 6 (67%). ΔPAWP/ΔCO ≥2 and ΔmPAP/ΔCO ≥3 was found in 7 (78%) and 6 (67%), respectively. The composite variable of PAWP ≥25 mmHg or ΔPAWP/ΔCO >2 was present in 12 patients (86%) at peak exercise.

3.2 Assessment of Coarctation of the Aorta during Exercise

Peak-to-peak CoA gradient was measured at rest in 14 patients and during maximal exercise in 9. Comparison between rest and exercise peak-to-peak CoA gradients did not show statistically significant differences (12 [3–16] *vs.* 16 [9–28] mmHg, $p = 0.4$). All patients had resting peak-to-peak CoA gradients ≤ 20 mmHg. Two patients developed a gradient >20 mmHg with exercise (from 13 to 36 and from 20 to 64 mmHg); both undergoing cycle ergometer exercise. One patient had both arm exercise and dobutamine stress catheterization (maximal dose 10 mcg/kg/min); peak-to-peak gradient with exercise across the CoA was 17 mmHg with arm exercise and 36 mmHg with dobutamine.

3.3 Follow-Up

Following catheterization, 7 patients (35%) underwent subsequent intervention due to re-coarctation. Their hemodynamic profile is presented in Table S1. Surgical repair with interposition grafting or extraanatomic bypass graft was performed in 2 patients each (13%); the remaining 3 cases were managed percutaneously (balloon angioplasty in 1 and coarctation stenting in 2). One additional patient underwent repair of coarctation site pseudoaneurysm. Four of the 7 patients with re-coarctation repair had post-intervention follow-up >6 months. Although improvement or stabilization of blood pressure control was seen early post-operatively, at mid-term all 4 patients had worsening hypertension with 3 needing intensification of their antihypertensive regimen. Of the 3 remaining patients in whom only early evaluation (<6 months post-intervention) was available, improvement in blood pressure control was observed in 2, while the remaining experienced no change in blood pressure readings.

4 Discussion

To the best of our knowledge, this is the first study to assess rest and exercise invasive hemodynamics in adults with CoA. The main findings of the study are: (1) most patients demonstrated evidence of LV diastolic dysfunction/elevation in left filling pressures, particularly during exercise; (2) similarly, in this cohort, the prevalence of PH was high, with exercise unmasking underlying abnormal pulmonary vascular reserve; (3) most patients failed to show significant increases in CoA peak-to-peak gradients with arm exercise, and despite CoA re-intervention, multidrug antihypertensive treatment was still required.

Lifelong follow-up of patients with repaired CoA is mandatory due to long-term comorbidities and common need for re-intervention [4,25]. CoA represents the most frequent cause of hypertension in congenital heart disease, and it is associated with premature coronary artery disease and decreased life expectancy [1,3,12]. Symptomatic heart failure with preserved ejection fraction due to diastolic dysfunction with increased LV filling pressures is a common complication, especially in older patients [17,26]. Diastolic dysfunction has been well-reported in CoA by non-invasive methods, and results from a complex interplay of ventriculo-arterial uncoupling, intrinsic arteriopathy, LV hypertrophy, and additional congenital or acquired cardiovascular conditions [6,13,14]. Non-invasive exercise testing is recommended by the guidelines to assess exercise-induced hypertension and gradient response across the CoA and may also unmask abnormal myocardial reserve [15–18].

We present herein invasive hemodynamic evaluation in CoA, which is gold standard for diagnosis of diastolic dysfunction and PH. Our findings confirm previous non-invasive observations, demonstrating abnormal pulmonary vascular reserve and hemodynamic response to exercise in this population. In our cohort, LV diastolic dysfunction was highly prevalent and >50% had elevated PAWP at rest. Furthermore, exercise data showed high prevalence of impaired diastolic reserve and abnormal left heart compliance as shown by the composite of PAWP ≥25 mmHg or ΔPAWP/ΔCO >2 in 86%. These findings are of clinical importance, particularly in the evaluation of symptomatic patients late after CoA repair or those with noninvasive evidence of elevated pulmonary pressures. In addition, they provide further pathophysiologic basis for non-invasive observation of abnormal left atrial function [27] and the high burden of atrial arrythmias in this population [28].

Noteworthy, all patients with resting PH had concomitant elevated PAWP. These findings provide hemodynamic support to classifying patients with CoA and elevated pulmonary pressures as group II PH based on their anatomic substrate and expected pathophysiology [29]. Some individuals showed resting combined PH, consistent with pulmonary vascular remodeling, and evidence of reduced pulmonary vascular reserve during exercise was also common. Our group has recently reported on the prevalence of right heart dysfunction and PH in adults with CoA assessed by echocardiography and their association with increased risk for death and heart failure; similar findings have also been reported by others [30,31]. The findings of the present study do not only corroborate these noninvasive observations but underscore

that the subpulmonary ventricle and the pulmonary vasculature should not be neglected in this population, despite CoA initially being a "left-sided" pathology [32].

Despite still being the ultimate diagnostic test, the invasive assessment of hemodynamics of native CoA and especially re-coarctation remains a clinical challenge. Gradients can be highly variable according to structural (presence of collateral vessels, for example) or physiologic (degree of sedation or need for general anesthesia) substrates. To mitigate this, some authors have studied the hemodynamic response to inotropes during cardiac catheterization. A recently published study in a younger CoA population (mean age at catheterization 27.3 ± 13.2 years) analyzed the use of epinephrine for gradient provocation prior to CoA intervention. In patients with low baseline CoA gradient but high gradients (>20 mmHg) after epinephrine administration, percutaneous intervention resulted in significant decrease in hypertension prevalence at mid-term follow-up [33]. In contrast, the subgroup without provocable gradient and therefore no CoA intervention, had worsening hypertension during follow-up, raising the question as to whether this was due to diagnostic failure of epinephrine stress testing, or rather to concomitant less modifiable factors. Studies have reported the use of isoproterenol [34,35] and dobutamine [36] as provocative maneuvers for assessment of CoA hemodynamics. The limitations of inotropic drugs for mimicking exercise are well-known and have been widely recognized in studies focused on stress testing for ischemic heart disease. Interestingly, in our cohort, 1 patient had both arm exercise and dobutamine stress catheterization, showing higher gradients during drug challenge. This patient did undergo subsequent intervention based on these findings without a long-term improvement in blood pressure control.

Invasive exercise evaluation might therefore represent a more physiologic test, better compared to noninvasive stress tests currently recommended by the guidelines. Most of our patients failed to show significant increases in blood pressure and CoA peak-to-peak gradients with exercise. Pressure gradient across an anatomic stenosis is determined by cross-sectional area and flow across the stenosis. One could argue that arm exercise could have been suboptimal for gradient provocation as flow augmentation in the descending aorta could have been insufficient with this form of exercise [20]. However, the fact that a high proportion of patients did have significant increases in intracardiac and pulmonary pressures at peak exercise with this type of exercise would argue against that, suggesting the lack of gradient provocation across the CoA to be indeed related to true absence of significant aortic obstruction. To support that hypothesis, of the 7 patients who underwent intervention after cardiac catheterization for "significant re-coarctation" by combination of anatomic, hemodynamic and clinical factors, all 4 with available long-term follow-up post-intervention had persistent hypertension. These patients might represent a more complex subgroup with severe diffuse arteriopathy, which limit the potential yield of CoA intervention [2,9,12,37].

4.1 Future Directions

The optimal method for the hemodynamic evaluation of re-coarctation remains to be elucidated and further studies are warranted to establish whether exercise and pharmacologic stress protocols at the time of catheterization could be used to unmask CoA severity. It is possible that using these provocative maneuvers, patients with unrepaired CoA and particularly re-coarctation could be categorized into 2 groups: those with true severe CoA who may benefit from invasive intervention [11,25], *vs.* those with a more complex phenotype but lacking major degrees of obstruction, where aggressive medical management, rather than CoA repair, may be of greater benefit [3,4,10].

Lastly, it should be noted that the only 2 patients with significant increase in CoA peak-to-peak gradients underwent exercise via supine cycle ergometer instead of arm adduction, highlighting the need to further investigate whether the diagnostic accuracy of these 2 types of exercise for CoA severity assessment is comparable. In addition, the "expected" gradients during exercise in those with and without anatomical obstruction need to be defined, as the cut-off of 20 mmHg used for resting gradients might not be applicable.

4.2 Limitations

We acknowledge the small sample size and retrospective nature of the study. Patients referred for cardiac catheterization in current practice are typically more complex and this is reflected in the demographics of our cohort; thus, our results may not be fully applicable to all asymptomatic patients post-coarctation repair. For PH definition, we intentionally chose a mPAP cut-off value of ≥25 mmHg since our aim was to be more specific than sensitive. Peak-to-peak CoA gradient was not available in all cases; however, it was assessed in every case where there was concern of significant coarctation/re-coarctation. The type of exercise was chosen at the operator's discretion, resulting in inherently heterogeneous results. Nevertheless, to the best of our knowledge, this is the first report of rest and exercise invasive hemodynamics in adults with CoA.

5 Conclusions

CoA is associated with significant vascular and myocardial disease at young ages. Prevalence of diastolic dysfunction and PH in our cohort was high, and exercise unmasked underlying abnormal diastolic and pulmonary vascular reserve. Most patients failed to show significant increases in CoA peak-to-peak gradients with exercise; further studies are warranted to establish the best diagnostic method to assess severity of CoA and select those patients who might benefit from CoA intervention.

Authorship: The authors confirm contribution to the paper as follows: study conception and design: WRM, IMM; data collection: WRM, IMM; analysis and interpretation of results: WRM, IMM, CCJ; draft manuscript preparation: WRM, IMM. All authors reviewed the results and approved the final version of the manuscript.

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Table S1: Hemodynamic profile of patients undergoing aortic coarctation re-intervention after the index procedure

Baseline catheterization	Patient 1		Patient 2		Patient 3		Patient 4		Patient 5		Patient 6		Patient 7	
	Rest	Exercise Arm	Rest	Exercise Arm	Rest	Exercise Arm	Rest	Exercise Cycle	Rest	Exercise Cycle	Rest	Exercise Cycle	Rest	Exercise Arm
LV systolic pressure, mmHg	114	-	145	149	124	-	166	-	125	-	-	-	108	132
LV end-diastolic pressure, mmHg	19	-	60	15	13	-	31	-	20	-	-	-	10	12
Systolic arterial pressure AAo, mmHg	112	169	123	123	121	133	154	183	130	167	117	126	105	136
Diastolic arterial pressure AAo, mmHg	66	107	67	72	69	74	82	91	65	70	71	75	54	72
Mean arterial pressure AAo, mmHg	86	134	91	95	92	104	111	130	85	109	90	97	73	98
Systolic arterial pressure DAo, mmHg	97	152	112	-	105	121	141	147	110	103	100	110	89	117
Diastolic arterial pressure DAo, mmHg	65	105	62	-	60	66	94	100	60	59	72	77	53	71
Mean arterial pressure DAo, mmHg	81	128	86	-	81	90	115	121	80	79	86	94	69	92
AAo to DAo peak-to-peak gradient, mmHg	15	17	11	-	16	12	13	36	20	64	17	16	16	19
Pulmonary artery mean pressure, mmHg	26	41	25	32	22	-	22	49	17	26	31	75	12	20
Mean PAWP, mmHg	16	31	14	19	14	-	12	39	11	20	22	55	5	8
Cardiac index, l/min/m^2	2.7	-	3.8	-	3.4	-	1.7	3.6	2.8	7.9	2.1	4.2	2.3	-
Pulmonary vascular resistance, WU	1.7	-	1.7	-	1.2	-	3.1	1.5	0.9	0.3	2.0	2.2	1.3	-

Note: AAo = ascending aorta; DAo = descending aorta; LV = left ventricular; PAWP = pulmonary arterial wedge pressure; WU = Wood units.