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Use of Inhaled Iloprost in a Case of Pulmonary Hypertension during Pediatric Congenital Heart Surgery

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IMPAIRED endothelium-dependent vasodilatation is present in children with high pulmonary flow and pressure which might be exacerbated by cardiopulmonary bypass (CPB).[1,2] It has been reported that an increased pulmonary vascular resistance, either directly or as a surrogate of the systemic inflammatory response after cardiopulmonary bypass, has a significant effect on the postoperative recovery of infants after cardiac operations.[3] Iloprost is the stable carbacyclin derivative of prostaglandin I_2. The use of aerosolized prostaglandin I_2 has shown to be safe in healthy lambs with regard to coagulation parameters, hemodynamics, and pulmonary toxicity.[4,5] Inhaled iloprost has been used as a diagnostic tool to assess the vasodilator capacity of the pulmonary vascular bed in children with congenital heart disease and elevated pulmonary vascular resistance, as well as intensive care unit treatment of pulmonary hypertension in a small series of children after cardiac surgery.[6] In adults, inhaled iloprost has been successfully used to control pulmonary hypertension after CPB.[7] However, no data are available about the intraoperative use of inhaled iloprost in infants younger than 1 yr with pulmonary hypertension undergoing cardiac surgery.

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Case Report

A 6-month-old infant girl, weighing 3.66 kg, was scheduled for atrial and ventricular septal closure. The preoperative medical history included gestational age of 29 weeks at birth, trisomy 21, and bronchopulmonary dysplasia. Preoperative cardiac catheterization revealed an unrestrictive ostium secundum type atrial septum defect and an unrestrictive perimembranous ventricular septal defect, resulting in pulmonary hypertension with a pulmonary-to-systemic perfusion ratio (Qp/Qs) of 1.4 and a pulmonary-to-systemic vascular resistance ratio (Rp/Rs) of 0.6. The preanesthetic medication consisted of aldactone, hydrochlorothiazide, digoxin, and antibiotics. In the operating room, general anesthesia was induced with fentanyl followed by pancuronium bromide and was maintained with fentanyl (total dose, 82 $\mu g \times kg^{-1}$), isoflurane (maximum end-tidal concentration 0.4 vol%), and midazolam (total dose, 0.4 mg $\times kg^{-1}$) after starting CPB. CPB was performed using nonpulsatile flow (2.4 $l \times min^{-1} \times m^{-2}$) with a membrane oxygenator in moderate hypothermia (rectal temperature > 33°C). To maintain full CPB flow at acceptable systemic pressures, the α-adrenergic antagonist urapidil (total dose, 1.0 mg $\times kg^{-1}$) was administered to keep the mean systemic blood pressure below 40 mmHg. Cold crystalloid cardioplegia (Bretschneider [histidine tryptophane ketoglutarate] solution, 110 ml) was given before clamping the aorta. The aortic clamping time was 65 min. During reperfusion of the heart, a loading dose of milrinone (50 $\mu g \times kg^{-1}$ over 60 min) followed by a continuous infusion of 0.5 $\mu g \times kg^{-1} \times min^{-1}$was started. After a total CPB time of 112 min, weaning off CPB was successful at the first attempt. Inhaled iloprost (2.5 $\mu g \times kg^{-1}$ over 20 min) was administered after weaning off CPB, because the mean pulmonary artery pressure/mean systemic blood pressure ratio (Pp/Ps) was increased to 0.72 and arterial oxygen saturation was 76%, despite hyperventilation ($Paco_2$, 30–35 mmHg) with an inspired oxygen fraction of 1.0. Iloprost was prepared from a vial of Ilomedin 50 i.v.® (Schering AG, Berlin, Germany) containing iloprost 50 μg/2.5 ml and was diluted with isotonic saline to obtain a concentration of iloprost 2 μg/ml. For inhalation, 4.5 ml of iloprost 2 μg/ml were administered using an ultrasonic nebulizer. Inhaled iloprost decreased the Pp/Ps to 0.59 and increased the oxygen saturation to 90%. The hemodynamic parameters and oxygen saturation readings are summarized in table 1. The patient was transferred with stable hemodynamic parameters to the pediatric intensive care unit. However, 120 min after terminating

Table 1. Changes in Hemodynamic Parameters and Arterial Oxygen Saturation

	Before Il	End of Il	60 min after Il	120 min after Il
Heart rate, beats/min	137	143	142	162
Systemic blood pressure, mmHg	73/58/45	73/54/41	72/54/42	62/43/35
Pulmonary artery pressure, mmHg	59/42/28	46/34/25	43/32/22	47/35/26
Pp/Ps	0.72	0.63	0.59	0.81
Arterial oxygen saturation, %	76	90	90	89

Il = inhaled iloprost; Pp/Ps = mean pulmonary artery pressure/mean systemic blood pressure ratio.

inhalational therapy with iloprost, the Pp/Ps increased again to 0.81. The postoperative course was complicated by recurrent pulmonary hypertensive crises during recovery from anesthesia that required prolonged sedation, relaxation, and nonselective pulmonary vasodilators despite the application of inhaled nitric oxide (iNO). We speculate that this may be because of a higher sympathetic activation during recovery from anesthesia and/or a minor response to iNO. Inhaled iloprost, however, has not been used during mechanical ventilation in the pediatric intensive care unit. The patient was ventilated for 6 postoperative days and was discharged to the referring hospital on the seventh postoperative day.

Discussion

This case report demonstrates that a single dose of inhaled iloprost (2.5 $\mu g \times kg^{-1}$ over 20 min) may be used to decrease Pp/Ps and to improve oxygen saturation in an infant after weaning off CPB; 120 min later the Pp/Ps returned to baseline. A documented hemodynamic effect for 1 to 2 h has previously been described.[8] The effective dose of inhaled iloprost in infants is not clear and seems to be dependent on the clinical setting. From previous applications, we speculate that a lower dose of inhaled iloprost is not very effective in infants after weaning off CPB, who were already hyperventilated with 100% oxygen. In accordance with Rimensberger *et al.*,[6] we observed no decrease in systemic blood pressure even though we used a fivefold higher dose. This may be explained by our clinical setting (*i.e.,* immediately after weaning off CPB; intraoperative use of the systemic vasodilators urapidil and milrinone). Theoretically, different characteristics of the aerosol spray may result in different intrapulmonary drug depletion characteristics, which could explain the lack of spillover into systemic circulation. However, we used a tested ultrasonic nebulizer (Optineb®; Nebu-Tec, Elsenfeld, Germany) that provided an aerosol with a mass median aerodynamic diameter of the droplets of 3.4 μm.

Although iNO is widely used to decrease pulmonary vascular resistance in infants undergoing cardiac surgery, the effects of iNO vary among patients and cumbersome devices are necessary to administer iNO safely.[9,10] Furthermore, rebound phenomena have been described with iNO withdrawal, bearing the risk of life-threatening pulmonary hypertensive crisis (*e.g.*, during transportation to the intensive care unit).[11] Inhaled iloprost may, therefore, be an alternative for selective pulmonary vasodilation in infants undergoing cardiac surgery because it is effective, easy to use, and long-acting. Furthermore, from an economic point of view inhaled iloprost may be attractive because iNO became very expensive after approval by the Food and Drug Administration.

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Intraoperative Management of Severe Pulmonary Hypertension during Cardiac Surgery with Inhaled Iloprost

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PULMONARY hypertension is an important risk factor for the development of acute right heart failure after cardiac surgery.[1,2] Even with early and adequate therapy, right ventricular (RV) failure is associated with increased morbidity and mortality.[1,3] We report the case of a patient with severe pulmonary hypertension related to aortic valve stenosis and mitral valve insufficiency who underwent combined bivalvular surgery and coronary artery bypass grafting. Pulmonary vascular resistance (PVR) was effectively decreased after the administration of inhaled iloprost before cardiopulmonary bypass (CPB) and during weaning from CPB. RV failure could be avoided and the perioperative course was uneventful.

Case Report

A 78-yr-old female patient (height, 1.75 m; weight, 74 kg) presented with a history of syncope and congestive heart failure. Cardiac catheterization revealed severe aortic valve stenosis (aortic valve area, 0.49 cm^2; mean pressure gradient 58 mmHg), mitral valve insufficiency (degree II), critical stenosis of the left main coronary artery, impaired left ventricular function, and hypokinesia of the anterior and apical left inferior wall. Furthermore, severe pulmonary hypertension was diagnosed (pulmonary artery pressure, 80/30 mmHg; mean pulmonary artery pressure, 65 mmHg; pulmonary artery occlusion pressure, 45 mmHg).

After the induction of anesthesia with sufentanil and midazolam, anesthesia was maintained with isoflurane and sufentanil. Hemodynamic monitoring consisted of arterial, central venous, and pulmonary artery catheterization. Hemodynamic parameters are presented in table 1. In addition, transesophageal echocardiography (Omniplane II T6210 probe; Sonos 5500, Philips Medical Systems, Best, The Netherlands) was performed intraoperatively. Before CPB, transesophageal echocardiography confirmed the diagnoses obtained by cardiac catheterization and revealed severe RV dysfunction. Detailed echocardiographic data are listed in table 2.

After the induction of anesthesia, nitroglycerin was administered intravenously to decrease PVR; however, the nitroglycerin was not effective (table 1). After sternotomy, PVR increased, probably because of increased RV preload caused by the reduction in intrathoracic pressure. Therefore, we administered 12.5 μg aerosolized iloprost (Ilomedin®; Schering Deutschland GmbH, Berlin, Germany) over 15 min *via* a commercially available nebulizer (Aeroneb® Pro; Aerogen Inc., Mountain View, CA) connected to the inspiratory limb of the ventilator circuit. The administration of iloprost significantly decreased pulmonary artery pressure and PVR and was accompanied by an increase in cardiac output. CPB was performed using moderate hypothermia (30°C), and cardioplegic arrest was instituted with 2 l of crystalloid cardioplegia. The patient underwent aortic valve replacement, mitral valve repair, and aorto-coronary bypass grafting to the left anterior descending and circumflex arteries. The duration of ischemia was 140 min. After 80 min of reperfusion, 12.5 μg inhaled iloprost were again administered over 15 min. Weaning from CPB was completed after a reperfusion time of 97 min. Moderate doses of vasoactive agents were administered to achieve adequate hemodynamic parameters. Transesophageal echocardiography showed an improvement in RV-function parameters after CPB: the RV-fractional area change increased from 18% (pre-CPB) to 38% (post-CPB). The patient was transferred to the intensive care unit, and endotracheal extubation was performed 13 h postoperatively.

Discussion

Impaired RV function is associated with a poor outcome in the surgical and nonsurgical settings.[1,4] The mortality of patients with combined arterial hypotension and severe RV dysfunction after CPB (defined as RV-fractional area change $< 35\%$) can reach 86%.[3]

Adequate treatment of RV failure consists of different strategies. The main goal is to decrease RV afterload by using vasodilating agents. The use of intravenously applied vasodilators is limited, as they are not selective to the pulmonary circulation and often cause arterial hypotension. Therefore, the administration of selective pulmonary vasodilators such as inhaled nitric oxide and prostacyclin may be beneficial.[5,6] Inhaled prostacyclin seems to be the more favorable agent because of its lack of toxicity, ease of application, and reduced costs.[5] Iloprost is the stable carbacyclin derivative of prostacyclin and can be administered intermittently, as the hemodynamic effects of a single dose are sustained for approximately 60–120 min.[7] Although the plasma half-life time of intravenously administered iloprost is known (20–30 min), no pharmacokinetic data are available concerning the plasma half-life time and the bioavailability after administration of inhaled iloprost.[8]

Similar to inhaled prostacyclin, inhaled iloprost causes a more pronounced increase in cardiac output and a

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Table 1. Hemodynamic Data

		Pre-CPB			Post-CPB†	
	Preoperative*	After Anesthesia Induction	Chest Open	After 12.5 μg Inhaled Iloprost	Chest Open	Chest Closed
MAP, mmHg	100	78	72	62	66	57
CVP, mmHg	10	15	11	8	13	13
MPAP, mmHg	65	43	42	21	33	29
PAOP, mmHg	45	30	23	14	17	14
TPG, mmHg	20	13	19	7	15	15
HR, min^{-1}	80	60	77	66	87	83
CO, l/min	3.5	2.5	2.5	5.2	5.7	5.0
SV, ml	32	41	32	78	65	60
SVR, dyne · s · cm^{-5}	2,057	2,016	1,952	830	743	704
PVR, dyne · s · cm^{-5}	457	416	608	107	224	240
PVR/SVR ratio	0.22	0.21	0.31	0.13	0.30	0.34
Epinephrine, μg · kg^{-1} · min^{-1}					0.07	0.07
Norepinephrine, μg · kg^{-1} · min^{-1}					0.05	0.05
Nitroglycerin, μg · kg^{-1} · min^{-1}			1	1		
Milrinone, μg · kg^{-1} · min^{-1}					0.5	0.25

* Data obtained by cardiac catheterization. † After 12.5 μg of inhaled iloprost.

CO = cardiac output; CPB = cardiopulmonary bypass; CVP = central venous pressure; HR = heart rate; MAP = mean arterial pressure; MPAP = mean pulmonary artery pressure; PAOP = pulmonary artery occlusion pressure; PVR = pulmonary vascular resistance; SV = stroke volume; SVR = systemic vascular resistance; TPG = transpulmonary gradient (MAP − PAOP).

greater degree of PVR-reduction when compared with inhaled nitric oxide.[7] Inhaled iloprost has been successfully used in the long-term therapy of pulmonary hypertension and in the testing of pulmonary vascular responsiveness.[9,10] To our knowledge, only three reports are available concerning the use of inhaled iloprost during cardiac surgery, two of them in patients awaiting or having undergone heart transplantation.[11–13]

In the present case, we used inhaled iloprost as part of a stepwise approach to prevent RV failure in a patient with severe pulmonary hypertension undergoing combined valve surgery and coronary artery bypass grafting.

Table 2. Intraoperative Changes for Hemodynamic Data Obtained by Transesophageal Echocardiography

	Pre-CPB*	Post-CPB†
LV-EDA, cm^2	32.3	27.0
LV-ESA, cm^2	24.0	21.6
LV-FAC, %	25.70	20
LVVD, ml	118	86.5
LVVS, ml	63.3	60.7
LVEF, %	46.36	29.83
LVIDD, cm	5.00	5.26
LVIDS, cm	3.01	3.74
FS, %	39.80	28.90
RV-EDA, cm^2	18.7	7.74
RV-ESA, cm^2	15.3	4.83
RV-FAC, %	18.18	37.60

Mid-esophageal four-chamber view and the short axis of transgastric view were evaluated.

* Closed chest, before administration of iloprost. † Closed chest, after administration of 12.5 μg inhaled iloprost.

CPB = cardiopulmonary bypass; EDA = end-diastolic area; EF = ejection fraction (determined by "Simpson's rule"); ESA = end-systolic area; FAC = fractional area change; FS = fractional shortening; IDD = end-diastolic inner diameter; IDS = end-systolic inner diameter; LV = left ventricular; RV = right ventricular; VD = end-diastolic volume; VS = end-systolic volume.

Administration of inhaled iloprost before CPB showed that the substance acted as an effective pulmonary vasodilator in our patient. Despite a concomitant decrease in mean arterial pressure and systemic vascular resistance (SVR), iloprost led to a more pronounced reduction of pulmonary artery pressure and PVR, so that the PVR/SVR ratio was remarkably decreased before CPB. During reperfusion, iloprost was again administered. PVR and pulmonary artery pressure were significantly decreased when compared with the preoperative values. However, the PVR/SVR ratio was increased after CPB, which can be attributed to an increase of PVR due to CPB-induced pulmonary vascular injury and to a decrease in SVR. Reduction of SVR after CPB is a well-known phenomenon mainly caused by hemodilution and activation of inflammatory mechanisms by extracorporal circulation. The additional use of milrinone contributed to the decrease in SVR.

We used inhaled iloprost during weaning from CPB as an integral part of the therapy and not as a rescue medication. This is in contrast to other case reports, in which inhaled nitric oxide, prostacyclin, or iloprost were used after RV failure had already occurred.[14,15] The most effective dose and the best time for the administration of iloprost are still unknown. We used a dose of iloprost that is within the range described in the literature,[7,11] and we administered the second dose before starting the weaning from CPB. Thus, an effective RV unloading could be expected in the immediate post-CPB period. RV failure with the need for an excessive dosage of catecholamines or even for reinstitution of CPB could be avoided. Despite the use of positive inotropic substances and surgical correction of valvular disease, echocardiographic parameters indicated a significant impair-

ment of left ventricular function after CPB, most probably caused by severe myocardial stunning. Thus, it seems unlikely that improvement of RV function was caused solely by the surgical procedure.

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