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Acute Hemodynamic Effects and Home Therapy Using a Novel Pulsed Nasal Nitric Oxide Delivery System in Children and Young Adults With Pulmonary Hypertension

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In 26 patients, we evaluated a novel pulsed nasal delivery system for nitric oxide (NO) that, in the short term, was as effective as continuous delivery for decreasing pulmonary artery pressure and pulmonary vascular resistance. In 2 patients, NO delivered in the home using this pulsing system was well tolerated for up to 2 years. The long-term safety, efficacy, and acceptability of NO delivered in the home remains to be studied. ©2003 by Excerpta Medica, Inc.

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Inhaled nitric oxide (NO) therapy improves gas exchange and lowers pulmonary vascular resistance in several clinical diseases, including primary pulmonary hypertension (PH) and congenital heart disease.[1–5] Most studies have demonstrated the utility of inhaled NO during treatment of PH using mechanical ventilation.[3–8] Investigators have begun to explore methods of delivering NO for use in the long-term treatment of patients with PH.[9–12] Nasal-inhaled NO may be useful for testing pulmonary vasoreactivity to determine operability in congenital heart disease or eligibility for calcium channel blockade therapy in established PH.[2–4,13–17] Furthermore, inhaled NO may play a role in the treatment of patients with PH, who are not intubated during short-term exacerbations,[18] or for long-term treatment in the hospital and at home. However, little is known about optimal treatment strategies that may be effective in decreasing PH when noninvasive delivery of inhaled NO is used.

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To determine whether inhaled NO has potential for the long-term treatment of patients with PH, we studied the acute hemodynamic effects of pulsed nasal delivery of low-dose NO gas in children and young adults with PH who underwent cardiac catheterization. To begin studying the potential role of long-term pulsed inhaled NO therapy in patients with PH, we tested the delivery system in 2 patients in the home for up to 2 years.

After we received institutional review board approval to conduct the study, 26 patients with PH who underwent routine catheterization for the diagnosis and treatment of PH were prospectively enrolled in the study after they gave informed consent and assent where appropriate. Patient characteristics are listed in Table 1. Patient hemodynamics were (1) measured in room air at baseline, (2) measured by continuous mask or pulsed nasal delivery in random fashion for a second baseline, and (3) measured again with the method not yet used. Hemodynamic measurements were performed after each 15-minute study period.

Mask NO was delivered using a standard resuscitation mask (Baxter, Deerfield, Illinois) connected to a T piece and large-bore corrugated tubing. Continuous flows of NO and oxygen were delivered through an INO Vent (Datex-Ohmeda, Andover, Massachusetts) at 20 ppm. Pulsed nasal NO was delivered through a split nasal cannula (model number 4703; Salter Labs, Arvin, California) using an INO pulse experimental system manufactured by Datex-Ohmeda. This system delivers a set volume of 100 ppm NO at the beginning of inspiration. Two devices were studied: adult and pediatric. The adult device delivers 15- to 60-ml NO/breath in increments of 5-ml NO/breath, whereas the

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TABLE 1 Baseline Characteristics of the Study Population

Patient	Age (yrs)	Sex	Diagnosis	PAP (mm Hg)	AOP (mm Hg)	Weight (kg)	Pulse NO (ml/breath)	EPO	CCB	Warfarin	Diuretics	Oxygen
1	2	F	PPH	62	66	8	10	0	0	0	+	+
2	6	M	PPH	39	80	16	15	+	0	+	0	+
3	6	M	PPH	28	69	23	20	+	0	+	0	0
4	8	M	PPH	45	80	40	40	0	0	0	0	+
5	10	F	PPH	46	87	49	40	0	+	0	+	0
6	10	M	PPH	59	84	41	40	0	0	0	+	0
7	11	F	PPH	71	90	58	60	0	+	0	+	+
8	12	M	PPH	75	78	32	30	0	+	0	0	+
9	12	F	PPH	25	62	36	40	+	+	+	0	0
10	12	F	PPH	62	76	40	40	0	+	+	0	0
11	14	M	PPH	51	93	60	60	0	+	+	0	0
12	14	F	PPH	66	84	48	45	+	0	+	0	0
13	15	M	PPH	30	82	45	50	0	+	+	0	+
14	4	M	MS/CoA	59	73	14	15	0	0	+	+	+
15	4	F	PDA	32	63	16	15	0	+	0	+	+
16	8	M	PDA*	67	69	23	25	0	0	0	0	+
17	8	M	Absent PA	45	83	25	25	0	0	0	0	+
18	8	M	ASD	64	78	22	25	0	0	0	0	0
19	15	F	VSD	113	83	39	40	0	0	0	0	0
20	24	F	VSD*	79	87	49	50	0	0	0	0	0
21	1	F	CDH	60	65	5	5	+	0	+	+	+
22	10	F	BPD	39	74	27	30	0	0	0	0	+
23	12	M	BPD	44	88	36	40	0	0	0	0	+
24	4	F	s/p HAPE	25	74	16	15	0	0	0	0	+
25	3	M	V-A shunt	63	70	12	10	0	0	0	0	+
26	23	F	Portal HTN	73	75	52	50	+	+	+	+	+

AOP = mean arterial pressure; ASD = atrial septal defect; BPD = bronchopulmonary dysplasia; CCB = calcium-channel blocker; CoA, coarctation of the aorta; CDH = congenital diaphragmatic hernia; EPO = epoprostenol; HAPE = high-altitude pulmonary edema; HTN = hypertension; MS = mitral stenosis; PDA = patent ductus arteriosus; PPH = primary pulmonary hypertension; PA = pulmonary artery; s/p = status post; VSD = ventricular septal defect; V-A = ventriculo-atrial
*Unrepaired congenital heart disease.

TABLE 2 Hemodynamic Findings During Nitric Oxide (NO) Administration by Mask and by Pulse Delivery*

Hemodynamic Finding	Baseline	NO Mask (20 ppm)	Baseline	NO Pulse
Pulmonary artery pressure (mm Hg)	53 ± 4	39 ± 4*	54 ± 4	41 ± 4*
Pulmonary capillary wedge pressure (mm Hg)	9 ± 1	9 ± 1	9 ± 1	9 ± 1
Cardiac index (l/min/m^2)	3.8 ± 0.3	3.8 ± 0.3	4.0 ± 0.3	3.9 ± 0.3
Pulmonary vascular resistance (U · m^2)	12.7 ± 1.9	8.8 ± 1.9*	13.6 ± 2.1	9.4 ± 1.9*
Mean systemic arterial pressure (mm Hg)	80 ± 2	81 ± 2	77 ± 2	83 ± 2
Mean right atrial pressure (mm Hg)	7 ± 1	7 ± 1	7 ± 1	7 ± 1
Systemic vascular resistance (U · m^2)	21.6 ± 1.7	23.3 ± 2.1	21.0 ± 1.9	22.5 ± 1.7
Ratio of pulmonary to systemic vascular resistance	0.58 ± 0.06	0.38 ± 0.06*	0.62 ± 0.06	0.41 ± 0.06*

*Values expressed as mean ± SEM.
†$p < 0.05$ versus baseline.

pediatric device delivers 3- to 10-ml NO/breath in increments of 1-ml NO/breath. Flow through the adult device is 10 L/min, and flow through the pediatric device is 2 L/min. The devices detect the following and sounds an alarm for: breath not detected, low batteries, low supply pressure, and system failure. If a breath is not detected, a pulse is delivered every 15 seconds for the adult device and every 10 seconds for the pediatric device. Pulsed inhaled NO volume was determined from estimations of alveolar volume, based on patient size, to deliver an estimated alveolar concentration of 20 ppm. The tidal volume (V_t) was estimated at 7 cm^3/kg and dead-space volume (V_{ds}) at 2.2 cm^3/kg. The delivered volume was derived from the formula:

$$\frac{(V_t - V_{ds}) \times \text{desired alveolar dose (20 ppm)}}{\text{tank concentration (100 ppm)}}$$

Oxygen flow rates were adjusted to maintain oxygen saturation similar to the room air saturation.

Statistical analysis was performed using Statview (Berkeley, California). One-way analysis of variance with repeated measures and Fisher's protected least-significant difference test were used to detect differences between variables. A p value <0.05 was considered statistically significant.

Patient demographics are shown in Table 1. Baseline values for arterial blood gases were pH = 7.40 ± 0.01; $PaCO_2$ = 35 ± 2 mm Hg; and PaO_2 = 73 ± 4

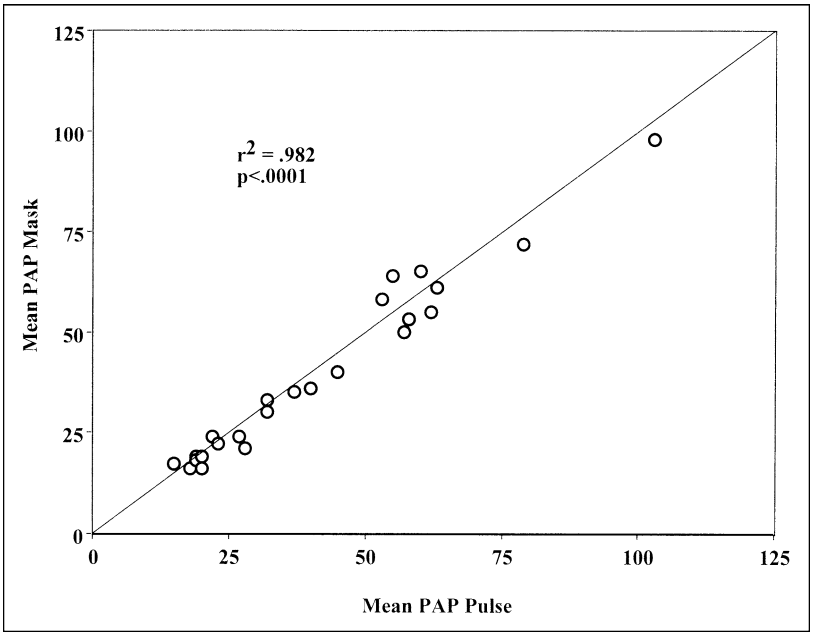

FIGURE 1. Correlation between the mean PAP during mask NO delivery and pulsed nasal NO delivery.

mm Hg. Arterial pH and $PaCO_2$ did not change during the study. Arterial PaO_2 increased to 102 ± 10 mm Hg during pulse inhaled NO delivery ($p < 0.05$) but was not different from mask delivery. Pulsed delivery of inhaled NO decreased mean pulmonary artery pressure (PAP), the pulmonary vascular resistance index, and the ratio of pulmonary to systemic arterial vascular resistance (Table 2).

No patient had a methemoglobin level of >3% during treatment with NO. All patients effectively triggered the NO pulsing device. There was good correlation between PAP during mask and pulsed delivery (Figure 1; $r^2 = 0.982$, $p < 0.0001$) and the percentage of change in PAP during NO delivery ($r^2 = 0.89$, $p < 0.01$). In 1 patient (patient 11), who had a transtracheal catheter used for oxygen delivery, the tracheal concentration of NO was measured using a chemiluminescence device during pulsed nasal NO delivery at 20, 40, and 60 ml/breath. In this patient, the tracheal NO concentration during pulsed delivery of inhaled NO ranged between 28 and 38 ppm (Figure 2).

Two patients were selected, on a compassionate-use basis, to receive inhaled NO therapy using a pulsed device in the home. After we received institutional review board approval and the 2 patients gave informed consent and assent where appropriate, the patients were hospitalized to assess their response to pulsed inhaled NO therapy and to teach the patients and their families how to use the delivery system. The patients were sent home with 3 delivery systems, 2 of which could be used as backup in case of device failure. These patients were followed up on a weekly basis by phone contact and clinic visits. Patient number 10 is a 13-year-old girl with familial PH. She had a large secundum atrial septal defect that was repaired at 9 years of age (Table 3). Epoprostenol was not available because of her country of residence, so she was treated with calcium channel blockers. Three years later her PH had progressed to systemic levels and was minimally reactive to inhaled NO. She was treated with inhaled NO by pulsed delivery for 7 months. She was stabilized off inhaled NO and was transitioned to bosentan, an oral endothelin receptor antagonist.[19] During the course of inhaled NO treatment, 2 delivery devices failed and were replaced without incident. She had no nosebleeds, syncope, or presyncope. Patient number 26 is a 26-year-old woman with PH related to portal hypertension. She presented at 18 years of age and was treated with calcium channel blockers because she refused epoprostenol (Table 3). Her PH progressed; 4 years later epoprostenol therapy was initiated (Figure 3). Transplantation was denied by several centers. She developed an ovarian dysgerminoma with associated suprasystemic PH. She underwent a graded balloon atrial septostomy for recalcitrant right-sided cardiac failure. During the course of inhaled NO treatment at home, 6 delivery devices failed and 3 episodes of severe nosebleed required cauterization. She had no episodes of syncope or presyncope.

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We report that acute delivery of pulsed nasal NO effectively lowers PAP and pulmonary vascular resistance in patients with PH. Pulsed nasal delivery of NO with small volumes of 100 ppm was as effective in lowering mean PAP as inhaled NO delivered at 20 ppm by continuous face mask delivery. In 2 patients, home use of inhaled NO stabilized PH and allowed the transition to bosentan in 1 child.

Several delivery systems, including continuous-flow and pulsed nasal delivery, have been described for NO delivery to the patient who is not intubated[9–12,18]. Pulsed nasal delivery of NO may be beneficial in lowering tank utilization, decreasing environmental exposure, decreasing potential toxicity, and, in theory, more effectively delivering NO than continuous delivery systems.[10] Distribution of inhaled NO throughout the lungs in conventional delivery systems may worsen gas exchange in certain disease states, such as chronic obstructive pulmonary disease.[20] We previously described the use of an oxygen delivery system using 800 ppm NO tanks and a blender system to deliver spikes of 20 ppm by nasal cannula. However, this system is complex and not portable. In this study, we found that a novel NO system, which delivers a set volume of 100 ppm NO via nasal cannula, was both portable and effective in decreasing PAP in patients with PH.

Although pulsed delivery of nasal NO decreased PAP in this study, several limitations of the current delivery system exist. The actual dose of NO in the alveoli using

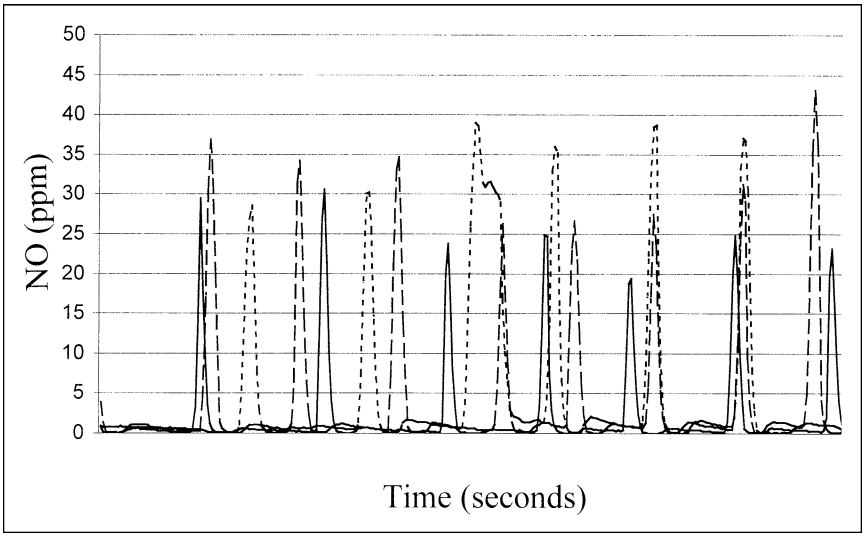

FIGURE 2. Tracheal concentration of NO (100 ppm) measured in the transtracheal catheter of 1 patient at 20 *(solid line)*, 40 *(long dashed line)*, and 60 *(short dashed line)* ml/breath. In this patient, note the breath-to-breath variability in NO concentration. Also note that the pulsewidth of the dose increases with dose volume because the flow rate of delivery is fixed.

TABLE 3 Cardiac Catheterization in Patients Receiving Nitric Oxide (NO) at Home

Patient	Age (yrs)	Diagnosis	Condition	PAP (mm Hg)	AOP (mm Hg)	PVRI (U · m^2)	PVR/SVR	Qp:Qs
10	9	ASD	Room air	51	70	8.6	0.48	1.4
			NO + oxygen	44	70	6.1	0.30	1.9
	12	S/P ASD, CCB	Room air	88	94	19.1	0.91	
			NO + oxygen	80	94	15.8	0.83	
	13	Home NO	Room air	62	76	16.9	0.79	
			Pulse NO	60	77	16.1	0.74	
26	18	Portal HTN	Room air	90	104	28.2	0.81	
			NO + oxygen	60	88	21.3	0.60	
	18	CCB	Room air	65	93	18.6	0.62	
			NO + oxygen	55	93	17.0	0.50	
	23	EPO/cancer	Oxygen	73	75	12.7	0.91	
			NO + oxygen	61	83	8.1	0.67	
	26	EPO/home NO, BAS	NO + oxygen	55	52	7.7	1.16	

BAS = graded balloon atrial septostomy; cancer = ovarian dysgerminoma; PVRI = pulmonary vascular resistance index; Qp = pulmonary blood flow; Qs = systemic blood flow; SVR = systemic vascular resistance; other abbreviations as in Table 1.

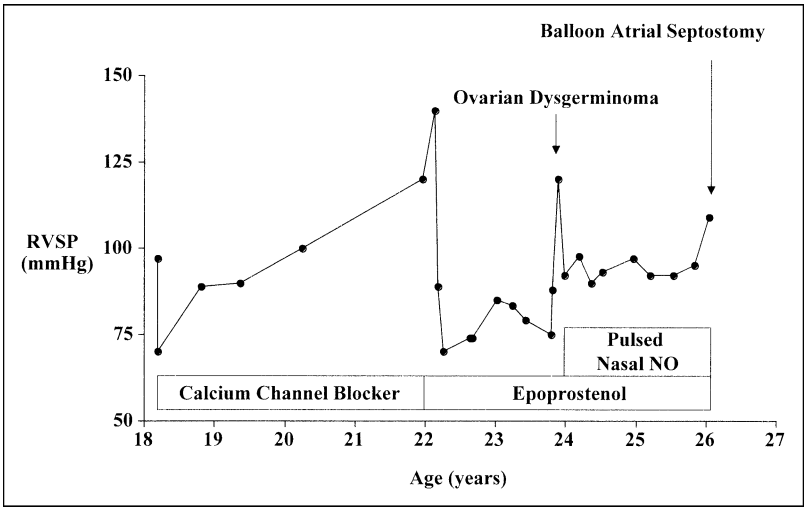

FIGURE 3. Right ventricular systolic pressure (RVSP) during treatment of a 26-year-old patient with PH related to portal hypertension.

the INO pulse system remains uncertain, but measurement of tracheal NO in 1 patient suggests that the lung concentration of NO approximates 20 ppm using approximately 1 ml/kg/breath of 100 ppm NO. The tracheal concentration of NO in this patient may be higher than the alveolar concentration because of metabolism of NO or dilution of NO in the distal airways. Both patients in the chronic setting experienced delivery device failures that were caused most commonly by kinking of the oxygen tubing and failure of the transducer circuits. The cannula tubing was changed to a more rigid type, thus resulting in fewer transducer failures. The utility of this device during the long-term requires further study because rebound PH is a serious risk of NO delivery[6,8,21] and should carefully be monitored. Patients receiving acute and long-term inhaled NO did not manifest acute rebound PH, but most patients in the home setting were receiving other medications, including epoprostenol.

We describe a novel pulsed nasal delivery system for NO that is as effective during the short-term in

lowering PAP and pulmonary vascular resistance as continuous delivery. In 2 patients, home-delivered NO using this pulsing system was well tolerated for up to 2 years. The long-term safety, efficacy, and acceptability of NO delivered in the home remains to be studied.

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Usefulness of Magnetic Resonance Imaging of Cardiac and Paracardiac Masses

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In this study, magnetic resonance imaging was compared with histology to evaluate the usefulness of magnetic resonance imaging to distinguish malignant from benign cardiac and paracardiac masses in 55 patients. Tumor location, tissue composition, and pericardial or pleural effusion all were identified as key predictors of lesion type. Observers were accurate in the prediction of lesion type (area under curve 0.88 and 0.92), and there was good interobserver variability (Cohen's $\kappa = 0.64$).

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Magnetic resonance imaging (MRI) is particularly suited for the assessment of cardiac masses. In addition to having a large field of view that allows imaging of the paracardiac space and the great vessels, MRI has value because of its multiplanar capability and its ability to address blood flow and cardiac function. In addition, electrocardiographic gating allows for freezing of heart motion and permits high-quality imaging of tumor morphology and composition.[1–6] The goal of this retrospective study was to test whether MRI allows an accurate and diagnostically useful distinction of malignant cardiac and paracardiac tumors in a large cohort of patients.

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Between 1988 and 2001, 216 consecutive patients were referred for a cardiac MRI study. Histology was available for 55 patients (23 men and 32 women). Histology was obtained intraoperatively in 23 patients and via transvenous biopsy in 32 patients. Thirty-three lesions were histologically classified as benign, and 22 were classified as malignant. The average age of the patients was 47.7 ± 23.4 years (range 1 to 84). Patients with malignant tumors (ages 44 ± 22.1 years;

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