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## Improvement of Extreme Hypoxemia during End-stage Congenital Heart Disease Using Nasal Nitric Oxide

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NASAL nitric oxide in association with oxygen may constitute a noninvasive therapeutic approach to severe hypoxemia secondary to cardiac right-to-left shunting. We describe a young woman with end-stage congenital heart disease in whom this technique was used.

### Case Report

A 30-yr-old woman with congenital complete atrioventricular canal was admitted to the intensive care unit for acute respiratory distress.

Recently, worsening exercise intolerance because of right ventricular failure resulted in her candidacy for heart-lung transplantation. Shortness of breath developed subsequently necessitating home oxygen therapy and, 5 days later, the patient was referred to the hospital.

At admission, respiratory distress and peripheral cyanosis were extreme despite nasal oxygen (7 l/min). Heart rate was 100 beats/min and radial arterial blood pressure was 100/60 mmHg. Respiratory rate was 32 breaths/min. Physical examination showed signs of predominantly right-sided congestive heart failure. The electrocardiogram showed a sinus rhythm with a right bundle branch block and multiple ventricular premature beats. The initial arterial blood sample while receiving nasal oxygen indicated an oxygen tension ( $P_{aO_2}$ ) of only 22 mmHg, with a measured hemoglobin saturation ( $S_{aO_2}$ ) of 29%. Arterial carbon dioxide tension ( $P_{aCO_2}$ ) was 78 mmHg, arterial pH was 7.26, and bicarbonate concentration was 35 mm. Arterial lactate was 2.19 mm (normal, < 2.44). Blood cell count showed marked polycythemia ( $6.17 \times 10^6/\text{mm}^3$ ) with an elevated hemoglobin concentration (19.2 g/dl). Standard chest radiography showed massive enlargement of the cardiac silhouette. The lung texture was abnormal with diffuse interstitial infiltrates and some degree of alveolar infiltrates. A Doppler-echocardiographic study at the bedside showed dilated right heart chambers with severe hypokinesia of the right ventricle. Using continuous-wave Doppler, the

peak systolic pressure gradient between the right ventricle and atrium was estimated to be at least 75 mmHg.

We decided to avoid tracheal intubation and mechanical ventilation, which might have precipitated circulatory failure. Instead, we hypothesized that pulmonary artery pressure reduction might decrease the intracardiac shunt and improve arterial hemoglobin saturation. Therefore, we added nitric oxide (NO; 225 ppm; 0.5 l/min) to oxygen (7 l/min, representing an NO fraction of 15 ppm) through the same nasal catheter. Five minutes after initiation of NO inhalation, the patient was able to talk and alleged some relief from her respiratory distress. Systemic blood pressure was unchanged (100/67 mmHg), whereas heart rate was reduced to 80 beats/min. Peak pressure gradient was reduced to 60 mmHg, and this was associated with an increase in  $P_{aO_2}$  (from 22 to 41 mmHg) and  $S_{aO_2}$  (from 29 to 71%). Further clinical improvement was obtained with normovolemic hemodilution to reduce hemoglobin concentration to 14.0 g/dl and by suppressing the numerous premature ventricular contractions using oral amiodarone. Increases in NO fraction up to 30 or 60 ppm did not result in additional improvement.

The patient remained dependent on NO inhalation. Nitric oxide withdrawal tests were immediately followed by worsening of dyspnea, reduction in  $S_{aO_2}$ , and an increase in the Doppler-estimated systolic ventriculoatrial pressure gradient. On the fourth day after admission, the patient removed the nasal prongs and experienced profound hemoglobin desaturation followed by asystole. This was quickly reversed with mask oxygenation and cardiopulmonary resuscitation maneuvers. Except for this incident, we were able to obtain an adequate  $P_{aO_2}$  and a relatively stable hemodynamic status for 6 days, while awaiting an organ donor. Unfortunately, on day 6, the patient died of sudden ventricular tachycardia and cardiovascular collapse.

### Discussion

This observation emphasizes the potential usefulness of inhaled NO in a spontaneously breathing patient to improve extreme hypoxemia secondary to cardiac right-to-left shunting.

In the absence of surgical repair, natural evolution of congenital atrioventricular canal leads invariably to pulmonary hypertension followed by right ventricular failure. In our patient, hypoxia resulted from right-to-left

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## CASE REPORTS

shunting, and possibly some degree of alveolar ventilation-perfusion mismatch and low cardiac output. Because peripheral oxygen saturation remained very low with nasal oxygen alone, the adjunction of NO was attempted. Inhaled NO concentration was certainly less than 15 ppm, however, sufficient NO delivery was achieved to increase  $\text{SaO}_2$  (29% to 71%) and obtain rapid clinical improvement. The systolic pulmonary artery pressure was reduced by 20%, indicating a pulmonary vasodilation and a lesser driving pressure for the shunting flow.<sup>1,2</sup> Other patients in whom inhaled NO improved pulmonary hypertension and systemic oxygenation were newborns with right-to-left shunting related to persistent pulmonary hypertension<sup>3,4</sup> and adults with patent foramen ovale and pulmonary hypertension.<sup>5,6</sup>

Few cases of long-term nasal NO delivery in spontaneously breathing patients with pulmonary hypertension have been reported.<sup>7,8</sup> This simple approach is effective, but the nasal route seems not very reliable, as emphasized by the episode of cardiac arrest that occurred when NO delivery was discontinued suddenly. This technique should be considered in selected patients, although medical personnel need to be very watchful.

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## An Unexpected Arousal Effect of Etomidate in a Patient on High-dose Steroids

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CATATONIA presents with stupor, excitement, or alternating stupor and excitement<sup>1</sup> and covers a broad group of movement disorders sometimes seen in psychotic illness. It

is most often associated with schizophrenia but can also be found in connection with mania and depression.<sup>2</sup> Other conditions in which catatonia can be present include neurologic disorders, systemic metabolic disorders, and as a side effect of certain medications.<sup>1</sup>

Any two of the following signs manifests catatonia: motor immobility, excessive motor activity, negativism or mutism, peculiarities of voluntary movement, echolalia, or echopraxia.<sup>2</sup> Motor immobility may be manifested as a waxy flexibility. The excessive motor activity apparently is purposeless and is not influenced by external stimuli. There may be extreme negativism that is manifested by resistance to all instructions or the mainte-

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