

Florida Association of Neonatal Nurse Practitioners  
FANNP Symposium 2025  
East Carolina University

**TITLE of PRESENTATION:** Pharmacologic Approaches to the Management of Persistent Pulmonary Hypertension in the Newborn

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**CONFLICTS OF INTEREST:** None

**About the Author:**

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**URL to Keyword Search Meeting:** <https://tinyurl.com/PPHN-Keywords>

**Abstract**

**Introduction:** Persistent pulmonary hypertension of the newborn (PPHN) is a failed transition to extrauterine life causing increased pulmonary vascular resistance, reducing systemic oxygenation. PPHN is classified as maldevelopmental or secondary, with pulmonary vs. non-pulmonary causes. This case study shares the story of an infant with PPHN secondary to perinatal asphyxia.

**Case presentation:**

*History of present illness:* Caucasian, male infant born in a Level II NICU facility at 38 2/7 weeks gestation to a 23-year-old primigravida with adherence to prenatal care. Labor was induced due to cholestasis of pregnancy and complicated by a single, tight nuchal cord and fetal intolerance to labor, prompting Cesarean section. Apgars were 0 at 1 minute and 1 at 5 minutes of life. Resuscitation efforts at birth included positive pressure ventilation, and intubation. Cord arterial blood gas at birth showed severe metabolic acidosis (pH 6.26 CO<sub>2</sub> 15.3 mmHg HCO<sub>3</sub> 3.5 mEq/L base deficit (BD) -21). Thirty minutes post-resuscitation, acidosis continued with a pH

6.89 CO<sub>2</sub> 24 mmHg PaO<sub>2</sub> 164 mmHg HCO<sub>3</sub> 4.6 mEq/L BD 26.9. The infant's presentation was consistent with severe hypoxic-ischemic encephalopathy via Sarnat scoring, prompting passive cooling to begin while awaiting transport to a Level III NICU for therapeutic hypothermia. At one hour of life, refractory hypotension necessitated normal saline boluses and the initiation of a dopamine infusion. On the transport team's arrival, hypoxemia persisted despite 100% FiO<sub>2</sub> and corrected hemodynamics. Inhaled nitric oxide (iNO) therapy was started in effort to stabilize the patient.

*Hospital course:* The infant arrived at the Level III NICU at 4.5 hours old with an oxygenation index of 30, warranting continuation of iNO. Formal therapeutic hypothermia continued for 72 hours, followed by a 6-hour rewarming period. Dopamine infusion continued through day two. On day three, he warmed, weaned off hydrocortisone, and moved from invasive ventilation to CPAP. A room air transition occurred day four and he began enteral feedings. He continued to progress and discharge home on day of life 10 with pediatric cardiology and neurology follow-up appointments at one month post-discharge.

*Labs, Images, and Studies:* An ECHO on admission was significant for the following markers of PPHN: small-to-moderate PDA with left-to-right flow, PDA flow nearing systemic RV pressures, small atrial septic defect (ASD) vs PFO with left-to-right flow, TR jet pressure elevated at 40 mmHg, and TR jet septal motion. Day four, a post-warming MRI of the brain produced normal results.

### **Discussion:**

PPHN incidence averages 0.4-6.8 per 1000 live births in the United States, leading to continuous research to improve outcomes. Secondary PPHN accounts for 80-90% of all cases, and is a sequela of parenchymal lung disease, abnormal transition, perinatal asphyxia, sepsis, or lung hypoplasia. Inhaled nitric oxide is a potent and pulmonary-specific vasodilator that helps aid in correcting this morbidity with varying and growing costs in the United States.

Understanding the indications, mechanisms, and contraindications for iNO therapy is critical to limiting adverse effects and offsetting financial burden for patients and the healthcare system.

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