



CURRENT TREATMENT OF PERSISTENT PULMONARY HYPERTENSION OF THE NEWBORN *by Respiratory Care Student - Diana Jones*

Persistent pulmonary hypertension of the newborn is a cardiopulmonary disorder that is characterized by increased pulmonary vascular resistance and systemic arterial hypoxemia.

In PPHN, pulmonary artery pressures exceed systemic pressures, which results in a right to left shunt through the ductus arteriosus. This shunting causes the blood to bypass the lungs resulting in a decrease in oxygenation in the systemic circulation, which causes systemic arterial hypoxemia. There are many factors that can contribute to the development of PPHN including meconium aspiration, respiratory distress syndrome, pneumonia (esp. group B strep), hyaline membrane disease, congenital diaphragmatic hernia, and pulmonary vascular bed abnormalities. Infants usually present with cyanosis and respiratory distress with tachypnea but little to no retractions. The first step in treatment is usually supplemental oxygen followed by an echocardiograph to confirm the diagnosis of persistent pulmonary hypertension. After that different treatment options can include inhaled nitric oxide, high frequency ventilation, extra corporeal membrane oxygenation and conventional ventilation.

There are currently a handful of treatments available for persistent pulmonary hypertension of the newborn that include High Frequency Ventilation, Inhaled Nitric Oxide Therapy, Extracorporeal Membrane Oxygenation, and Surfactant Replacement. A few treatments that are on the horizon but still being researched are Magnesium Sulphate and Sildenafil administration.

Nitric Oxide is a gas that is inhaled and helps to relax and vasodilate vascular smooth muscle. This action improves blood flow to ventilated alveoli resulting in reduced pulmonary shunting, decreased pulmonary vascular resistance, decreased pulmonary artery pressure, reversing of hypoxic pulmonary vasoconstriction in an obstructed airway which improves V/Q mismatching and an increase/improvement in oxygenation.

High Frequency Ventilation is a type of ventilation that uses low pressure to deliver small tidal volumes at very high frequencies. There are three types of HFV, high frequency positive pressure ventilation (delivers 60-150 cycles/min), high frequency jet ventilation (delivers 240 to 660 cycles/min), and high frequency oscillatory ventilation (480-1800 cycles/min).

HFV helps to reduce barotrauma with low pressures, biotrauma and atelectrauma. With the constant expansion of alveoli, they are never overexpanded on inspiration, nor under-expanded

or collapsed on expiration. The alveoli are held open with low pressure all the time, which greatly improves oxygenation and allows for optimal recruitment of lung segments.

Extracorporeal Membrane Oxygenation is a therapy that takes circulating blood outside the body, completely bypassing the lungs for oxygenation, which allows diseased or injured lungs time to heal. By taking the blood from the lungs outside the body the cycle of pulmonary hypertension is stopped. This reduces the need for high pressures and high FIO₂, which will reduce barotrauma, biotrauma, atelectrauma and possibly even volutrauma. With the blood being oxygenated outside the body there is no more refractory hypoxemia, acidosis or vasoconstriction.

The oxygenation provided by the machine membrane allows for a perfect V/Q ratio, increased oxygenation and reduced shunt with absolutely no effect on the lungs whatsoever. The most common route for ECMO is a venoarterial bypass where blood leaves the body through the right internal jugular vein and enters the body through the right carotid artery. One disadvantage is that some patients may need vessel reconstruction.

Surfactant replacement uses an artificial surfactant product that lowers surface tension of the alveoli and helps improve lung compliance. It is normally given to premature infants, < 35 weeks, whose bodies have not had enough time to produce their own natural surfactant.

Magnesium Sulphate and Sildenafil have been proven to be selective pulmonary artery vasodilators that improve oxygenation and have been shown to significantly improve oxygen index and alveolar-arterial oxygen gradient (A-aDO₂).

I researched approximately 20-30 various articles and studies that had to do with persistent pulmonary hypertension of the newborn and it's treatments. The majority of articles I found said that nitric oxide is one of the best treatments for PPHN. The studies show that inhaled NO improves oxygenation and reduces the need for ECMO (extra corporeal membrane oxygenation). In one study a lower OI (oxygen index) and MAP (mean airway pressure) along with a significant response within the first hour of iNO treatment were shown to be favorable factors in response to iNO treatment (Hwang, 2004). Several of the studies looked at dosing of iNO to see if higher or lower amounts would improve/reduce oxygenation outcomes. It appears that lower doses <20ppm actually improve oxygenation

Diana Jones is an RC Student at Spoakane Community College in Spoakane, WA. Her paper was chosen from papers submitted to Focus for this issue. Ms. Jones will receive a \$100 gift certificate and a gratis registration to the 2009 Focus Conference. Her school's RC Program will also receive a \$100 donation. Students are encouraged to submit their papers for the Sep/Oct issue by August 24th. Papers should be 900 - 1250 words and should be submitted as MS Word files to Craig Baker at BakerCT78@yahoo.com.

The Magic Surgeon... Continued from page 58

go into some detail about the various subtleties of the respiratory care and ventilator management different types of defects.

One of the most important things the RT can remember when caring for these patients is related to the pulmonary vascular resistance (PVR). It is beyond the scope of this column to examine all the affects of respiratory care interventions on PVR. But a simple rule of thumb is that changes in pH, oxygenation, and CO₂ can affect PVR and much of what the RT does in the post operative period is an attempt to help manage the balance between pulmonary and systemic blood flow using changes in PVR. Pulse oximetry is an important tool in managing these patients.

Tidal volumes are typically kept in the 6-8 mL/kg range, although occasionally they are increased to 10 ml/kg, depending on disease. As an example, in post-operative bi-directional Glenn repairs some people prefer to use larger tidal volumes and lower rates to allow for longer expiratory times, while keeping the same minute ventilation. The rationale is that pressures created within the thorax through the airways can be transmitted to the alveolar capillaries, causing a mechanical impediment to blood flow through the capillary bed with the resultant increase in PVR. Thus, keeping inspiratory time short and expiratory time long minimizes the effect of this pressure transmission.

I have watched the close-up video of our cardiac surgeons repairing a complex atrioventricular canal defect. They were essentially creating septa and valves that were not there at birth. It is a long and complex procedure. The entire surgical field was about 3 inches across. This is what I think Arthur C. Clark meant when he referred to, "sufficiently advanced technology".

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Student Paper... Continued from page

tion and outcomes more so than any dose above 20ppm. While ECMO is probably the best treatment for PPHN due to the fact that it bypasses the lungs completely, allowing for healing time, it is very expensive and imposes higher risk factors than other forms of treatment. iNO is the much preferred method of treatment and is easily used in conjunction with other forms of therapy such as high frequency and conventional ventilation. However, studies also show that while iNO does reduce the need for ECMO it does not prevent it. Many infants who were placed on iNO therapy still ended up requiring ECMO at some point in time, once their PPHN had become severe. An OI greater than 20 and an AaO gradient more than 600 after 4 hours of iNO therapy could be indicative of an immediate need for ECMO (Fakioglu, 2005). But research did find that infants placed on iNO when they were in a mild to moderate state of PPHN did not go on to require ECMO, only infants who were placed on iNO late, with severe PPHN failed the iNO treatment. It's been found that iNO improves oxygenation but doesn't reduce the incidence of ECMO/death when initiated at an OI of 15 to 25 compared to greater than 25. Studies are also looking at Magnesium Sulphate and Sildenafil for treatment. Sildenafil

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has been found to be a selective pulmonary vasodilator when given orally, intravenously or in an aerosolized form with no significant effects. Magnesium sulphate has also been shown to significantly improve oxygen index and alveolar-arterial oxygen gradient (A-aDO₂) within the first 24 hours. There have not been very many studies done on these two forms of therapy but certainly the outcomes warrant further investigation. It is proposed that if these really do work as well as they appear to they will be of great benefit to developing countries who cannot afford the more common treatments such as nitric oxide, mechanical/high frequency ventilation and ECMO.

There are many exciting results being found in the recent studies for Persistent Pulmonary Hypertension of the Newborn that project a kind of hope towards the future for better patient outcomes. With ongoing research of nitric oxide and extracorporeal membrane oxygenation it will be even better understood and possibly even improved upon. And with the positive research results done on Sildenafil and Magnesium Sulphate there is the possibility and anticipation that these drugs could be a turning point in care for patients in developing countries who have limited resources and money for treatment.