Neonatal Respiratory Distress Syndrome and Its Treatment with Artificial Surfactant [1]

By: Mandile, Olivia

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Neonatal respiratory distress syndrome [4], previously called hyaline membrane disease, is a respiratory disease affecting premature newborns. Neonatal respiratory distress syndrome [4] involves shallow breathing, pauses between breaths that last a few seconds, or apnea, and a bluish tinge to the infant’s skin. The syndrome occurs when microscopic sacs called alveoli in infant lungs do not produce surfactant, a liquid that coats the inside of the lungs and helps them inflate during breathing. Respiratory distress syndrome is the leading cause of death among premature infants and, in rare cases, it can affect full-term infants. Physicians can administer artificial, animal-derived surfactant to treat respiratory distress syndrome [4]. As of 2017, the treatment has decreased the mortality rate of respiratory distress syndrome [4] from almost one hundred percent to less than ten percent.

Respiratory distress syndrome affects premature infants who are born before their lungs have developed enough to support breathing outside the uterus [5]. At full-term birth, an infant’s lungs contain millions of microscopic sacs called alveoli. In the alveoli, cells called Type II alveolar cells produce a liquid called surfactant. Surfactant is necessary for breathing. The lungs of premature infants, however, have not developed enough alveoli or Type II alveolar cells to produce the amount of surfactant needed to breathe properly. While respiratory distress syndrome usually affects premature infants, in rare cases the syndrome can also affect full-term infants. When respiratory distress syndrome [4] affects full-term infants, it is the result of a genetic condition, rather than underdeveloped lungs.

In the 1920s, physiologist Kurt von Neergaard hypothesized that there was some sort of liquid in human lungs that allowed them to open during respiration. However, Neergaard did not find what that liquid was. In the 1950s physician John Clements observed surfactant, the liquid that Neergaard hypothesized coated the inside of the lungs. In 1959, researchers Mary Ellen Avery and Jeremiah Mead at Harvard University in Cambridge, Massachusetts, reported the absence of surfactant in the lungs of infants who died from hyaline membrane disease, later called respiratory distress syndrome [4]. Avery and Mead hypothesized that the lack of surfactant was responsible for what was then called hyaline membrane disease. Once researchers established the existence and relevance of surfactant, they began researching the chemical makeup of the compound. Researchers found that surfactant is made up of fats and proteins, like the structure of a cell membrane.

Avery and Mead’s hypothesis that the lack of surfactant in premature infants causes neonatal respiratory distress syndrome [4] proved correct. Neonatal respiratory distress syndrome [4] primarily affects premature infants, whose lungs are not developed enough to provide enough oxygen to their brain and other organs. While developing in the uterus [5], a fetus’s lungs are unnecessary as a breathing organ because the pregnant woman provides oxygen to the fetus [6] through the umbilical cord [7] connecting them. However, lungs must be able to function immediately after birth to provide the newly independent fetus [6] with oxygen. Lung development extends through both the embryological and fetal stages of development, from week three gestation [8] until delivery. Initially, fetal lungs develop as a small bud from the endoderm [9], the innermost layer of cells in the developing embryo. The process occurs within the first few weeks of pregnancy [10]. From the bud, the trachea, or the windpipe that transfers air to and from the lungs, develops. The trachea branches into two bronchi, one for each lung. By the fourteenth week of development, seventy percent of the airway has developed.

At around twenty-six weeks gestation [8], microscopic balloon-like sacs called primitive alveoli develop in the fetal lungs. Those primitive alveoli continue to develop after birth and throughout childhood, and they eventually become mature alveoli. Alveoli are the sites at which gases are exchanged between the lungs and the bloodstream. During gas exchange, a person draws oxygen into their lungs, where the alveoli diffuse that oxygen into the person’s bloodstream. Simultaneously, carbon dioxide diffuses out of the bloodstream into the alveoli, where the carbon dioxide is exhaled through the airway. At full-term birth, each one of an infant’s lungs will contain about one hundred million alveoli. Throughout the first thirty-six months of life, alveoli continue to develop. Fully developed lungs contain approximately three hundred million alveoli each.

While the primitive alveoli are developing, cells called Type II alveolar cells begin to form in the developing lungs. The main role of those cells is to secrete surfactant. Surfactant plays two roles in the lungs. First, surfactant decreases the pressure of the atmosphere on alveoli. During inhalation, the alveoli expand, and during exhalation, they collapse inwards. Without surfactant, the inner tissue of the alveoli sticks together during exhalation, causing the alveoli to collapse and preventing gas exchange. If gas exchange does not occur at the alveoli, oxygen will not reach the bloodstream or tissues throughout the body, including the brain and other organs. The second role surfactant plays in the lungs is that it increases the compliance, or expandability, of the lungs. Lungs expand during respiration as they take in oxygen-rich air from the atmosphere. Without that expandability, the lungs do not take in enough oxygen. Infants born before thirty-five weeks gestation [8] are at risk for respiratory distress syndrome [4] because their Type II alveolar cells are not producing enough surfactant for sufficient breathing.

When a premature infant whose lungs are not producing enough surfactant is born, those lungs are unable to provide the infant with
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- The Embryo Project at Arizona State University, 1711 South Rural Road, Tempe Arizona 85287, United States

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