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A Short Note on Low Birth Weight Infant and Breathing Morbidity

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Description

Infant thermal instability during admission to a Newborn Intensive Care Unit (NICU) has been connected to respiratory morbidity; however, the relation between continued thermal instability and respiratory morbidity is unknown.

On 12 VLBW newborns, a longitudinal data analysis was performed. Supplemental oxygen demand (FiO_2) or planned diuretic dosage at 36 weeks post-menstrual age was used to determine the risk of chronic respiratory morbidity. Desaturations ($SpO_2$90%), bradycardia with desaturations (HR100 and SpO_2 90%), apnea, increased FiO_2 need, or increased respiratory support were all used to measure acute respiratory morbidity. The connections between body temperature and respiratory morbidity across the first 14 days of life were investigated using multi-level, mixed-effects models and regression analysis.

For optimal growth and development, VLBW newborns require a neutral thermal environment. As a result, further research is needed to determine whether the significant associations between hypothermia and symptoms of acute respiratory morbidity are causal relationships that could be mitigated with clinical practise changes, or if these are concurrent symptoms that cluster during episodes of physiological instability.

Each year, around 50,000 newborns with Very Low Birth Weight (VLBW) (less than 1500 g) are born in the United States. Bronchopulmonary dysplasia (BPD) is the most prevalent consequence of extremely preterm delivery, affecting almost half of these babies. BPD is caused by the halting of normal lung development after a preterm delivery. Large, sparse alveoli and dysmorphic pulmonary vasculature define it, resulting in a reduction in the surface area accessible for gas exchange. Although advancements in newborn care have lowered the occurrence of severe BPD and its accompanying mortality, the incidence of BPD has increased as the survival rate of extremely preterm infants (less than 1000 grammes) has improved. Many risk factors have been related to the development of BPD, however only a small percentage of newborns with these risk factors develop the condition, complicating disease prevention and therapy.

The development of BPD in VLBW newborns has been linked to hypothermia and hyperthermia in the early hours of life. While the optimal body temperature for these babies is unknown, temperatures between 35.5 and 38.2°C have been linked to an increased risk of BPD. The VLBW newborn is unable to sustain euthermic body temperatures on his or her own due to immaturity of the thermoregulatory system. Thermal instability in these newborns is caused by autonomic dysfunction as well as epidermal immaturity. Normally, neonates rely on nonshivering thermogenesis (i.e., heat production *via* sympathetic stimulation of brown adipose tissue) to keep warm during periods of hypothermia; however, NST is ineffective in VLBW infants due to a lack of essential elements for brown fat metabolism (e.g., thermogenin and 5'-monodeiodinase) so oxygen is metabolised to keep warm.

VLBW newborns are unable to accommodate for an increase in oxygen demand due to their immaturity of the pulmonary system, therefore if hypothermia persists, hypoxemia and acidosis might occur. Desaturations (SpO_2 90%) or bradycardia (HR 100) accompanying desaturations (B/D) might be signs of hypoxemia. Desaturations have been linked to long-term morbidity and death in extremely preterm newborns, including chronic lung illness.

The inflammatory process that is highly related with the development of BPD can be stimulated and/or sustained by reactive oxygen species (ROS), a product of supplementary FiO_2, and positive pressure from RS, which can result in pulmonary epithelial injury.

Finally, when VLBW infants grow and develop, the optimal temperature range for minimising morbidity risk should be identified. If a real causative association between longitudinal temperature instability and CRMR is discovered, as well as an optimal body temperature range for reducing baby risk, intervention studies and therapeutic recommendations to minimise infant risk can be produced.