Physiologic Basis for Nasal Continuous Positive Airway Pressure, Heated and Humidified High-Flow Nasal Cannula, and Nasal Ventilation

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KEYWORDS

• Nasal CPAP • High-flow nasal cannula • Nasal ventilation

KEY POINTS

- Non-invasive support modalities utilize different applications and mechanisms but share similar physiologic mechanisms of support.
- All modes assist care givers in avoiding mechanical ventilation and the associated injuries to the lungs and airways.
- None of the modalities can achieve the ideal goal of being the right therapy for all patients across the age and disease spectrum treated in the newborn intensive care unit setting.

PHYSIOLOGY OF NORMAL BREATHING AND PATHOPHYSIOLOGY ENCOUNTERED IN NEONATAL MEDICINE

Introduction

Many readers of this issue of *Clinics in Perinatology* will have extreme familiarity and knowledge concerning spontaneous breathing physiology in newborns and infants. Although this is undoubtedly the case, it does seem appropriate to review some basic principles regarding neonatal spontaneous ventilation to better understand the variety of pathophysiologies that are presented to neonatal care practitioners and the application of noninvasive respiratory therapies.

Although the disease process themselves represent a heterogeneous group of unique physiologic challenges, the therapeutic interventions that are noninvasive support are broken into 3 large categories, each of which supports spontaneous ventilation during both phases of the respiratory cycle leading to an improvement in patient

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comfort and ventilation efficiency. The goal of these therapeutic interventions is to avoid invasive mechanical support. Over the past 50 years in newborn medicine, our understanding of these different support modalities has allowed us to pursue these strategies at younger gestational ages and smaller birth weights, with significantly more success. The goal of all of these noninvasive therapies is to avoid ventilator-induced lung injury and improve patient outcomes.[1–6]

Control of Respiration

Control of ventilation is a complex feedback system between the central nervous system and the lungs through the result of alveolar ventilation that leads to normal gas tensions and pH in a healthy state. In a disease state, insufficient oxygen or elevated carbon dioxide concentrations in the blood result in abnormal responses of the feedback loop leading to either inability to correct the abnormalities or cessation of breathing. The lowered oxygen concentration in the blood stimulates chemoreceptors in both the carotid and aortic bodies, while elevated concentrations of carbon dioxide similarly elevate the concentrations of carbon dioxide in the cerebral spinal fluid. These changes stimulate central chemoreceptors in the medullary respiratory center via signaling through the glossopharyngeal and vagus nerves. In response, the phrenic and intercostal nerves, through descending corticospinal tracts, stimulate more frequent breathing. The same stimuli lead to an increase in the amplitude of respiration, leading ultimately to an increase in tidal volume. These changes ultimately lead to an increase in minute ventilation with the overall trend of normalizing the partial pressure of carbon dioxide and oxygen with the secondary impact of balancing the pH of the blood.[7]

It is well understood that neonates have an abnormal response through these pathways to rising partial pressures of carbon dioxide and falling concentrations of oxygen. Most neonatologists focus on the relationship between rising and falling partial pressures of carbon dioxide in newborn breathing patterns. The more preterm an infant is born, the more likely the infant will have significant apnea.[8] This apnea is in large part related to the preterm infant's inability to respond with a normal linear response to rising partial pressures of carbon dioxide. One of the most common medicinal therapies available to the neonatologist, caffeine, directly targets this abnormal response, normalizing the slope closer to that of healthy preterm infants without apnea, or full-term infants.

Neonates also demonstrate a significant abnormality in the way in which they respond to hypoxemia. Although the initial response of neonates is to increase respiratory drive, and thus minute ventilation, this response is only temporary. After approximately 1 to 2 minutes, neonates have hypoxemic depression of the respiratory drive. This results in a return to their initial state or even depression of the respiratory activity. This paradoxic response may play an important role in the apnea observed in preterm infants.[9]

Finally, it is important to note the impact of nasopharyngeal airway patency and pulmonary stretch receptors, and the interplay they have on respiratory timing and maintenance of minute ventilation. Neonates who suffer apnea often experience obstruction of the upper airway. This obstruction may be related to disrupted control in the neonate's ability to maintain a patent upper airway in the face of a more compliant tissue. The combination of this and abnormal timing of pharyngeal muscle activation, as compared with the diaphragmatic contraction, may predispose the upper airway to collapse, leading to the observed obstructions. Pulmonary stretch receptors also play an important role in maintaining appropriate lung inflation and preventing overdistention. These may be an important mechanism through which the therapeutic approaches discussed here influence respiratory timing and apnea.[9]

Although clearly the medicinal approach with caffeine is the most common in treating apnea of prematurity, clinicians often use all of the available noninvasive devices