# Neurally Adjusted Ventilatory Assist: Insufficient Evidence of Broad Clinical Outcomes

Apnea is traditionally defined as complete cessation of air flow for 10 seconds or greater. Central apnea may be associated with pathophysiologic changes, including stroke, brainstem lesion, encephalitis, and congestive heart failure.[1] For patients with an artificial airway who are being supported with mechanical ventilation, apnea may be associated with oversedation and with hypocapnia during sleep, particularly during non-rapid-eye-movement sleep.[2,3] Small (2–5 mm Hg), transient (2–3 breath) hypocapnic episodes preceding central apnea during non-rapid-eye-movement sleep have been associated with increases in pressure support tidal volume.[2,3]

Ideally, apnea that occurs during mechanical ventilation in the context of an ICU is controlled by appropriate sedation management (eg, Richmond Agitation Sedation Scale score of 0, or Ramsay Sedation Scale score of 2) and by timely liberation of the patient from the ventilator for those patients who are physiologically ready to do so. Authors have suggested that the evolution of faster microprocessors and improved ventilator technology have the potential to improve control of breathing and reduce the frequency of central apneas.[3-5] Neurally adjusted ventilatory assist (NAVA) represents one such improvement that uses signals from the diaphragm to facilitate synchrony of patient-ventilator interactions.[4-6]

NAVA measures electrical activity of the diaphragm (EAdi) during inhalation, using an esophageal catheter.[4] The EAdi signals are filtered and amplified, and can be used to accomplish triggering and cycling of assisted or supported breaths, as well as modulation of airway pressure.[4-8] A growing body of research concludes that NAVA is associated with less timing (trigger and cycle) and flow asynchrony than conventionally applied mechanical ventilation.[6,8]

Delisle et al, in this issue of RESPIRATORY CARE, report results of their study examining any association between the occurrence of central apneas during weaning and the application of NAVA or pressure support ventilation.[9] NAVA triggering was applied when the EAdi reached 0.5 $\mu$V, and the EAdi signal was measured every 16 ms, both of which are consistent with the application of NAVA in other literature.[6,8]

Delisle et al have identified a statistically significant increase in tidal volume variability when supported breaths were flow assisted by NAVA, a finding that is consistent with the conclusions of other recent publications.[6,10] The authors suggest that the greater tidal volume variability measured during NAVA may be associated with the elimination of all central apneas during non-rapid-eye-movement sleep in their study.

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I commend the authors on their use of a prospective, randomized, controlled trial, the gold standard of experimental research methods. In addition, the research design integrated a crossover method with attention to minimizing residual effects. The statistically significant effect on tidal volume variation is especially noteworthy given the small sample size of 14.

While some authors are suggesting that NAVA has the potential to reduce the incidence of complications of mechanical ventilation, and decrease number of ventilator days, ICU stay, and hospital stay, little supportive evidence is available.[3-6] Considering a current healthcare context that is increasingly concerned with safety, clinically important patient outcomes, and cost containment, I believe that the use of NAVA has not yet been adequately justified in the literature. What is the clinical importance of this study; the "So what?" if you will. Until well designed studies provide evidence suggesting reduced morbidity, mortality, stay, number of ventilator days, or other desirable clinical outcomes, clinical managers are unlikely to invest in the required software and hardware upgrades necessary to implement NAVA.

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The author has disclosed no conflicts of interest.

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