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Editorial

# Neurally Adjusted Ventilator Assist Mode

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## Editorial

Normal breathing patterns determined by the respiratory center are dynamic and synchronous. A signal from the respiratory center is transmitted via the phrenic nerve to the diaphragm leading to contraction and descent of the diaphragm initiating respiration. However, patients requiring respiratory support using positive pressure ventilation may have asynchronous breathing patterns. Issues such as need for increased ventilator support and duration, intracranial hypertension, fluctuations in blood pressure, increased need for sedation and/or muscle paralysis, development of edema, and unreliable assessment of neurologic status have been well documented. A newer technology, Neurally Adjusted Ventilator Assist Mode (NAVA), assists ventilation in proportion to the patient's demand improving the match between the patient's needs and the assistance delivered by the ventilator [1,2]. Introduced in the late 1990's, NAVA uses Electrical Activity Of The Diaphragm (EAdi) to trigger the respiratory cycle and deliver proportional assist on a breath-to-breath basis thus improving patient-ventilator synchrony. Invasive and noninvasive NAVA mode ventilators are available for all ages [3,4].

The EAdi signal, central to NAVA, is a vital sign reflecting the efficacy of the respiratory muscles, degree of respiratory demand, and the degree of respiratory center output. Expressed in microvolts, the EAdi signal is controlled by adjusting nerve fiber recruitment and respiratory rate. It is measured through a specialized nasogastric tube with eight bipolar electrodes acting as sensors and is positioned at the level of the crural diaphragm. It is inserted as any other nasogastric tube. Signals from each electrode pair are sent to the ventilator, amplified, digitized, and processed and electrical contamination from the heart, esophagus, and environment are filtered to give the highest possible signal-to-noise ratio. The EAdi signal is not influenced by changes in lung volume, body position, intra-abdominal pressure, postural and expiratory muscles, subcutaneous layers, Positive End-Expiratory Pressure (PEEP), nasogastric feeds, or oral feeding. Failure to detect the EAdi signal is usually related to failure of the respiratory center to deliver a signal (i.e. apnea of prematurity, central hypoventilation syndrome, over-assist, hyperventilation, brain injury, and sedation), anatomic reasons such as a diaphragmatic hernia, and/or peripheral nerve abnormalities such as phrenic nerve conduction failure, disease, or chemical paralysis. Catheter positioning is assessed on the ventilator screen by observing p-waves and QRS complexes in upper and lower leads.

Current ventilators initiate spontaneous breaths by detecting changes in flow rate or pressure. In NAVA mode, the electrodes in the EAdi catheter detect the changes in electrical activity of the phrenic nerve. This electrical activity is transmitted via the EAdi catheter to the ventilator resulting in ventilator assisted spontaneous breathing. The highest EAdi value of the waveform (EAdi peak) represents the neural Inspiratory effort and is responsible for the size and duration of the breath. The lowest EAdi (EAdi min) reflects the spontaneous tonic activity of the diaphragm. EAdi trigger (in $\mu$V) is the minimum increase in electrical activity from the previous trough that triggers the ventilator. NAVA level is a conversion factor that converts the EAdi signal into a proportional pressure (units of cm $H_2O/\mu V$.) Peak Inspiratory Pressure (PIP) equals the NAVA level X (EAdi peak- EAdi min) + PEEP. The diaphragm is completely unloaded at high NAVA levels. NAVA is continually adjusted based on the neural feedback from the respiratory center of the brain. At the breakpoint, respiratory muscle unloading is adequate and the EAdi signal decreases and PIP reaches a plateau. Clinical evaluation of EAdi support of ventilation is determined by clinical comfort and blood gases. Over-ventilation suppresses spontaneous respiration and decreases the signal while under-ventilation increases respirator drive and results in higher EAdi signaling.

Initial set-up, management, and trouble-shooting of invasive NAVA and noninvasive NAVA are described in the MAQUET brochures [5]. Patient safety is paramount – PIP limit is set to prevent excessively large breaths. Malpositions or failed EAdi signal results in activation of PSV and PCV is activated if PSV fails. The Hering Breuer reflex provides negative feedback to help terminate the EAdi signal terminating breath. Weaning begins with improvement in neuromuscular coupling and lower airway pressure suggesting improvement in clinical condition. For incubated patients, extubation to noninvasive NAVA is an option. CPAP/HF has been used if the patient is on noninvasive NAVA.

This proportional mode of ventilation results in adequate gas exchange, improves patient-ventilator interaction, averts the risk of over-assistance, and minimizes occurrence of asynchronies. In this mode, there are minimal changes in the breathing pattern with tidal volumes that usually do not exceed 6 cc/kg [6]. Economically favorable results have been shown for NAVA versus PSV [7]. Experience with this technology is still limited and best NAVA settings for patient's needs remain a major question [8].

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