LETTER

Diaphragm myoclonus-induced autotriggering during neurally adjusted ventilatory assist

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Dear Editor,

Neurally adjusted ventilatory assist (NAVA) is activated by the electromyographic activity of the diaphragm (Edi) and delivers pressure-regulated respiratory support proportionally tailored to the patient's neural effort. As compared to pressure support ventilation (PSV) and flow-based triggering systems, NAVA lowers trigger delay, limits the risk of over-assistance and improves patient–ventilator interaction [1, 2]. Little is known about how NAVA interacts with epileptic seizures.

An 85-year-old male patient with myasthenia gravis and chronic obstructive pulmonary disease was admitted to our intensive care unit due to viral meningoencephalitis with seizures and progressive neurological decay that led to intubation. Seizures were initially generalized and, after treatment (valproic acid 1.6 g/24 h and levetiracetam 3 g/24 h), became partial, mostly involving the right side of the body.

Early after intubation, the patient developed inhalation pneumonia, with lower left pulmonary infiltrates on the chest X-ray. Arterial blood gases showed PaO₂/FiO₂ 323 mmHg, PaCO₂ 39 mmHg. His plateau pressure during PSV with PEEP 5 cmH₂O was 15 cmH₂O, with tidal volume 430 mL (predicted body weight: 68 kg) and respiratory system compliance 43 mL/cmH₂O [3].

To improve patient–ventilator interaction, a catheter for diaphragm electrical activity (Edi) was positioned, Edi signal validated according to current recommendations

[1], and NAVA initiated (NAVA gain 1 cmH₂O/μV, EdiTrigger 0.5 μV, PEEP 5 cmH₂O).

Episodes of partial epileptic seizure were confirmed by continuous electroencephalogram monitoring and were accompanied by clinical myoclonus of the right cheek, mouth, arm and forearm; during these events, the Edi tracing showed an uncontrolled activation of diaphragm, consisting of repetitive, brief, involuntary neural stimuli (Fig. 1, panel A). Edi spikes occurred at a frequency of 2–4 Hz, thereby configuring a phenomenon known as diaphragm flutter or Leeuwenhoek disease, which has been reported to cause respiratory distress in nonintubated patients [4–6].

The electric signal of myoclonic contraction was captured by the Edi-catheter, activating the Edi-based inspiratory trigger, although this was increased up to 2 μV (minimum sensitivity provided by the manufacturer): this generated significant ventilator autotriggering, leading to a flurry of respiratory acts with low tidal volumes, occasionally followed by large tidal volumes induced by proportional support of huge Edi swings.

During myoclonus, Edi spikes were accompanied by a high level of tonic Edi. Previous authors hypothesized that a tonic diaphragmatic activity could limit aeration loss during lung injury [7]; in our case, it may have represented a consequence of diaphragm depolarization occurring at high frequency.

After propofol administration, the crises were resolved, returning to an efficient ventilatory pattern (Fig. 1, panel b).

After myoclonus resolution, NAVA was switched to PSV. Flow trigger was set at 2 L/s and no autotrigger was detected despite new episodes of myoclonus, likely

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Fig. 1 Pressure, flow at airway opening and inflated volume were recorded by the ventilator (Servo-U, Maquet, Rastatt, Germany) at a sample rate of 100 Hz; EEG of left fronto-parietal cortex (Fp1 C3 electrode) was acquired at a sample rate of 256 Hz. All the signals were synchronized and analyzed with a dedicated software (ICU Lab Analyzer, Kleistek, Bari, Italy). Manuscript figures were prepared with GraphPad Prism (GraphPad Software, V.7, La Jolla, CA, USA). Panel A: NAVA ventilation and EEG monitoring (Fp1 C3 electrode): the diaphragm is depolarized during a partial crisis by several fast, inefficient and abnormal fans, that trigger the ventilator, yielding ventilator assistance proportional to Edi swings. Tidal volume was essentially low due to scarce patient–ventilator interaction, but could reach 1 L (approximately 15 mL/kg of predicted body weight) as the proportional assistance corresponded to patient's effective breath (black arrow). Panel B: The administration of 80 mg propofol resolved the crisis. In the middle of the sequence (black arrow), ventilator waveforms normalize, generating optimal neuro-ventilator coupling. After the crisis, each diaphragm trigger is followed by a ventilator breath, yielding optimal patient–ventilator synchrony, which is typical of NAVA

due to the patient's own low neuromuscular coupling (i.e., ratio of the effort-induced decay in airway pressure to the corresponding maximal Edi during an end-expiratory occlusion, $0.3 \text{ cmH}_2\text{O}/\mu\text{V}$), which limited the capability of the myoclonus to activate the flow-based trigger despite a high neural stimulus.

These tracings show that diaphragm myoclonus may occur in critically ill patients with seizures, can be easily diagnosed by Edi monitoring, can yield significant patient–ventilator asynchronies and, eventually, high tidal volumes, also when a proportional ventilatory mode is used.

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Compliance with ethical standards

Conflicts of interest

DLG has received payments for travel expenses by Maquet, Getinge and Air Liquide. MA has received payments for Board participation from Maquet, Air Liquide and Chiesi. All the other authors declare that no conflict of interest exists regarding the material discussed in the manuscript.

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