Use of neurally adjusted ventilatory assist (NAVA) in a patient with severe SARS-CoV-2 pneumonia: A case report

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J M Haynes. Use of neurally adjusted ventilatory assist (NAVA) in a patient with severe SARS-CoV-2 pneumonia: A case report. Can J Respir Ther 2021;57:90–92. doi: 10.29390/cjrt-2021-017.

Introduction: Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) pneumonia may necessitate intubation and prolonged mechanical ventilation. Early in the course of mechanical ventilation neuromuscular blocking agents may be used to allow synchronous lung protective ventilation. However, patients with SARS-CoV-2 pneumonia tend to have an intense respiratory drive resulting in patient-ventilator asynchrony when neuromuscular blocking agents are discontinued.

Case and Outcomes: A 75-year-old male was admitted to the hospital with SARS-CoV-2 pneumonia requiring invasive mechanical ventilation. By ventilator day 5 the neuromuscular blocking agent had been discontinued, and the patient was markedly asynchronous in the volume control mode despite receiving continuous intravenous sedatives. The ventilator mode was changed to the neurally adjusted ventilatory assist (NAVA) mode. Initially NAVA resulted in improved synchrony and reduced work of breathing. However, a few days later the patient’s tidal volume had fallen to <500 mL on NAVA despite increases in the NAVA level. It appeared that the inspiratory phase was prematurely terminating, and the expiratory threshold in NAVA is not adjustable. The ventilator mode was changed to pressure support resulting in an increased tidal volume and reduced respiratory frequency.

Conclusion: In patients with SARS-CoV-2 pneumonia and intense respiratory drive, the performance of NAVA may be variable. NAVA may result in hypopnea and tachypnea when compared with pressure support. An assessment of the impact of an adjustable expiratory threshold in NAVA is warranted.

Key Words: respiratory failure; mechanical ventilation; neurally adjusted ventilatory assist; COVID-19; SARS-CoV-2; case report

INTRODUCTION
Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) pneumonia may necessitate intubation and prolonged mechanical ventilation. Early in the course of mechanical ventilation neuromuscular blocking agents may be used to allow synchronous lung protective ventilation. However, patients with SARS-CoV-2 pneumonia tend to have an intense respiratory drive resulting in patient-ventilator asynchrony when neuromuscular blocking agents are discontinued. This case describes the use of the neurally adjusted ventilatory assist (NAVA) mode to manage a patient with SARS-CoV-2 pneumonia and marked patient-ventilator asynchrony.

Case description
This work did not require review committee approval because a single case report or case series does not constitute human subjects research requiring review and approval.

A 75-year-old male resident of a long-term care facility was admitted to the hospital after a 2-week history of fever, chills, and congested cough. The patient had chronic obstructive pulmonary disease and emphysema chronically requiring supplemental O₂ at 2 L/min via nasal cannula. A chest radiograph revealed bilateral infiltrates and a nasopharyngeal swab culture tested positive for SARS-CoV-2.

The patient was intubated on hospital day 2 due to increasing dyspnea and O₂ requirements. The ventilator settings immediately after intubation were as follows: volume control mode, set rate 18 b/min, tidal volume (Vₕ) 450 mL (6 mL/kg), positive end-expiratory pressure (PEEP) 5 cm H₂O, and FIO₂ 100% (Servo-i, Getinge, Maquet Critical Care AB, Solna Sweden). Blood gas data on these settings were: pH 7.35, PaCO₂ 39 mm Hg, PaO₂ 200 mm Hg, and O₂Hb 98%. The PaO₂/FIO₂ ratio was 200 compatible with mild to moderate acute respiratory distress syndrome (ARDS) according to the Berlin definition [1]. The initial static respiratory compliance was 61 mL/cm H₂O compatible with the proposed “L” phenotype of SARS-CoV-2 pneumonia [2]; however, the patient’s emphysema presumably contributed to a finding of “normal” compliance.

By ventilator day 5, chemical neuromuscular blockade had been discontinued. The patient’s level of sedation was –3 on the Richmond Agitation and Sedation Scale while receiving continuous intravenous infusions of fentanyl and propofol. Despite sedation the patient remained in respiratory distress on volume control ventilation. Flow delivery had been changed to the square waveform with “flow support”, which allows the patient to receive additional flow if the airway pressure falls 3 cm H₂O during the inspiratory phase of ventilation. An orogastric tube with embedded electromyogram electrodes (Edi catheter, Getinge, Maquet Critical Care AB, Solna Sweden) was inserted to monitor diaphragmatic activity and assess patient-ventilator synchrony. Proper positioning of the Edi catheter was confirmed using the Edi catheter positioning function. Despite the use of square waveform gas delivery with flow support, Figure 1 shows marked asynchrony with inspiratory pressure falling below PEEP during Vₕ delivery and pressure spikes at the termination of the inspiratory phase. The mean airway pressure was identical to the PEEP level, which would be expected during unassisted ventilation with continuous positive airway pressure. The negative deflections in airway pressure due to volume starvation may be injurious due to a Pendelluft effect [3, 4]. The electrical activity of the diaphragm (EAdi) was 25–30 μV,
FIGURE 1
Ventilator graphics from a patient with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) pneumonia receiving volume control ventilation. Airway pressures (yellow tracing) falling below PEEP during the inspiratory cycle. The electrical activity of the diaphragm (EAdi; white tracing) shown in the bottom graphic and is superimposed over the airway pressure tracing.

FIGURE 2
Ventilator graphics after the ventilator mode was changed to the neurally adjusted ventilatory assist (NAVA) mode.
suggesting elevated respiratory drive and work of breathing [5]. In Figure 1 the EAdi waveform is superimposed over the pressure waveform as a preview of what the pressure waveform might be when changed to the NAVA mode. However, this overlay can be misleading because a NAVA breath terminates at 70% of the EAdi peak, where the preview graphic overlays the entire EAdi cycle over the airway pressure graphic. The ventilator mode was changed to NAVA at 1 cm H₂O per EAdi μV. Figure 2 shows the ventilator graphics after the change to the NAVA mode. In NAVA the inspiratory airway pressure rose above PEEP, the Vₗ increased to >600 mL (8 mL/kg), with an increase in minute ventilation and decrease in EAdi. A few days later the patient's Vₗ had fallen to <300 mL on NAVA despite increases in the NAVA level. It appeared that the inspiratory phase was prematurely terminating, and the expiratory threshold in NAVA (70% of EAdi peak) is not adjustable. The ventilator mode was changed to pressure support with an expiratory flow sensitivity of 30%. The change to pressure support increased the Vₗ and reduced respiratory frequency.

Titrating sedation to respiratory effort instead of focusing on consciousness alone may improve synchrony. Objective measures of respiratory drive including EAdi and airway occlusion pressure (P′₀ₐ) may be helpful when titrating sedation [7]. Clinicians should also be prepared to change approaches according to the patient’s response to support and evolving pathophysiology.

**CONCLUSION**

In patients with SARS-CoV-2 pneumonia and intense respiratory drive, the performance of NAVA may be variable. NAVA may result in hypopnea and tachypnea when compared with pressure support. An assessment of the impact of an adjustable expiratory threshold in NAVA is warranted.

**DISCLOSURES**

**Competing interests**

The author is a paid consultant for Morgan Scientific Inc., a pulmonary function test manufacturer, which would not influence the content of this submission.

**Funding**

This study did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

**Ethical approval**

This work did not require review committee approval for experimental study on human participants and/or confirmation of clinical trial registration because a single case report or case series (three or fewer cases) does not constitute human subjects research requiring review and approval.

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