

**Tommaso Mauri**  
**Giacomo Bellani**  
**Giuseppe Foti**  
**Giacomo Grasselli**  
**Antonio Pesenti**

## Successful use of neurally adjusted ventilatory assist in a patient with extremely low respiratory system compliance undergoing ECMO

Accepted: 30 July 2010  
Published online: 16 September 2010  
© Copyright jointly held by Springer and  
ESICM 2010

Dear Editor,  
Implementing lung protective ventilation while assuring acceptable arterial oxygenation and carbon dioxide removal in patients affected by severe acute respiratory failure and very low respiratory system compliance is a major challenge [1].

Extracorporeal membrane oxygenation (ECMO) may guarantee viable blood gases [2] and allow the use of assisted rather than controlled mechanical ventilation. This, in turn, will achieve improved respiratory muscle function, better gas exchange and decreased sedation level [3].

Pressure support ventilation (PSV), however, may be difficult to implement in patients with very low compliance [3]. In these patients, peak inspiratory flow is reached rapidly and the expiratory phase of the ventilator may start while the patient is still inspiring. This mechanism may lead to patient discomfort, asynchrony and possibly desaturation and barotrauma. We reasoned that neurally adjusted ventilatory assist (NAVA) may overcome this limitation as expiratory cycling time is based on diaphragmatic electrical activity

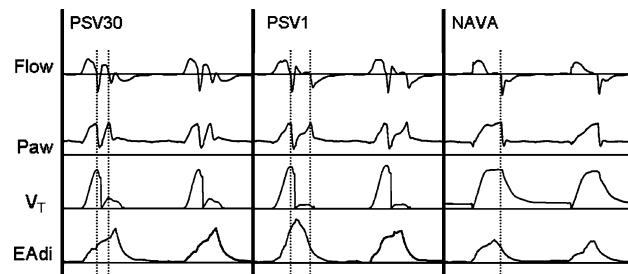
(EAdi) and should adapt better to the patient's own respiratory pattern [4].

A 58-year-old male patient (predicted body weight 90 kg) developed acute respiratory failure from exacerbation of idiopathic pulmonary fibrosis [5]. He was severely hypoxaemic and hypercapnic. Therefore ECMO was initiated while the patient was undergoing noninvasive ventilation. After 7 days, his respiratory function further declined and he was intubated and mechanically ventilated. On ECMO day 14, the patient was on controlled ventilation with tidal volume ( $V_T$ ) 2 ml/kg, respiratory rate (RR) 15 bpm, positive end expiratory pressure (PEEP) 10 cmH<sub>2</sub>O and inspired oxygen fraction (FiO<sub>2</sub>) 100%. ECMO blood flow was 2.3 l/min with 5 l/min of pure oxygen sweep gas flow. Arterial haemoglobin saturation (SatO<sub>2</sub>) was 75% and compliance was 7 ml/cmH<sub>2</sub>O. We tested each of the assisted ventilatory strategy listed below for 30 min, leaving PEEP, FiO<sub>2</sub> and ECMO settings unchanged, with the following observations:

1. PSV30 (PSV 12 cmH<sub>2</sub>O above PEEP, expiration cycling time 30% of flow peak value):  $V_T$  was 2 ml/kg and RR 22 bpm; patient inspiration overlapped machine expiration and this constantly triggered a second breath ("double hit breaths") (Fig. 1, PSV30);

global asynchrony index (AI) [6] was  $\geq 10\%$ ; patient respiratory drive was high ( $p0.1 = 3.3$  cmH<sub>2</sub>O, peak EAdi = 12  $\mu$ V); SatO<sub>2</sub> decreased to 73%.

2. PSV1 (PSV 12 cmH<sub>2</sub>O and cycling at 1%):  $V_T$  was 1.9 ml/kg and RR 22 bpm; reduction of the cycling-off criterion (until the latest allowed by the ventilator) did not improve synchrony and the inspiratory phase of the ventilator was still shorter than the patient's own, yielding double-hit breaths (Fig. 1, PSV1); AI remained  $\geq 10\%$ ,  $p0.1$  increased to 3.7 cmH<sub>2</sub>O and peak EAdi to 13  $\mu$ V; SatO<sub>2</sub> remained stable at 73%.
3. NAVA (gain 2 cmH<sub>2</sub>O/ $\mu$ V, to achieve the same peak airway pressure as in PSV; limit of airway pressure set on ventilator 35 cmH<sub>2</sub>O):  $V_T$  ranged between 1.9 and 2.1 ml/kg and RR was 23 bpm; airway pressure increased following the shape of the patient's EAdi, then reached a plateau and inspiratory flow zeroed, until EAdi had decreased to 70% of its peak value (i.e. NAVA cycling-off criterion, Fig. 1). AI lowered to  $< 10\%$ , respiratory drive decreased ( $p0.1 = 0.3$  cmH<sub>2</sub>O, peak EAdi = 5.9  $\mu$ V) and SatO<sub>2</sub> increased to 77%. Interestingly, airway pressure waveforms in our patient (Fig. 1,



**Fig. 1** Airway flow (Flow), airway pressure (Paw), tidal volume (VT) and diaphragm electrical activity (EAdi) of a patient with extremely low compliance of the respiratory system during pressure support ventilation with expiration cycling set at 30% of peak inspiratory flow (PSV30), pressure support ventilation with expiration cycling set at 1% of peak inspiratory flow (PSV1) and neurally adjusted ventilatory assist with gain 2 cmH<sub>2</sub>O/ $\mu$ V (NAVA). The dashed lines represent the starting of the expiratory phase of the ventilator

NAVA) differed from those found in previous studies on NAVA [7] where pressure increased and decreased without reaching a plateau. Usually, a plateau during NAVA is seen when the airway pressure limit is reached, but this was not the case in our patient as the limit was 2–8 cmH<sub>2</sub>O higher than the actual plateau pressures. Airway pressure in our patient instead reached a plateau probably because of his/her slow rate of decrease in EAdi from the peak value. In the presence of early flow termination due to low compliance, the slow rate of decrease in EAdi yielded a longer time between peak pressure and cycling-off, during which airway flow was null and airway pressure was held at a plateau.

By maintaining the above-mentioned NAVA settings, we managed the patient for several days without relevant asynchrony issues.

In conclusion, we report here the successful application of NAVA to maintaining assisted ventilation in a patient with extremely low

respiratory system compliance undergoing ECMO.

## References

1. Terragni PP, Rosboch G, Tealdi A, Corno E, Menaldo E, Davini O, Gandini G, Herrmann P, Mascia L, Quintel M, Slutsky AS, Gattinoni L, Ranieri VM (2007) Tidal hyperinflation during low tidal volume ventilation in acute respiratory distress syndrome. *Am J Respir Crit Care Med* 175:160–166
  2. Brogan TV, Thiagarajan RR, Rycus PT, Bartlett RH, Bratton SL (2009) Extracorporeal membrane oxygenation in adults with severe respiratory failure: a multi-center database. *Intensive Care Med* 35:2105–2114
  3. Cerda M, Foti G, Marcora B, Gili M, Giacomini M, Sparacino ME, Pesenti A (2000) Pressure support ventilation in patients with acute lung injury. *Crit Care Med* 28:1269–1275
  4. Brander L, Sinderby C, Lecomte F, Leong-Poi H, Bell D, Beck J, Tsoporis JN, Vaschetto R, Schultz MJ, Parker TG, Villar J, Zhang H, Slutsky AS (2009) Neurally adjusted ventilatory assist decreases ventilator-induced lung injury and non-pulmonary organ dysfunction in rabbits with acute lung injury. *Intensive Care Med* 35:1979–1989
  5. Porte A, Stoeckel ME, Mantz JM, Tempe JD, Jaeger A, Batzeneschlager A (1978) Acute interstitial pulmonary fibrosis. Comparative light and electron microscopic study of 19 cases. Pathogenic and therapeutic implications. *Intensive Care Med* 4:181–191
  6. Vignaux L, Vargas F, Roesseler J, Tassaux D, Thille AW, Kossowsky MP, Brochard L, Jolliet P (2009) Patient-ventilator asynchrony during non-invasive ventilation for acute respiratory failure: a multicenter study. *Intensive Care Med* 35:840–846
  7. Spahija J, de Marchie M, Albert M, Bellemare P, Delisle S, Beck J, Sinderby C (2010) Patient-ventilator interaction during pressure support ventilation and neurally adjusted ventilatory assist. *Crit Care Med* 38:518–526
- T. Mauri · G. Bellani · A. Pesenti  
Department of Experimental Medicine,  
University of Milan-Bicocca, Monza, Italy
- T. Mauri · G. Bellani · G. Foti ·  
G. Grasselli · A. Pesenti (✉)  
Department of Perioperative Medicine and  
Intensive Care, San Gerardo Hospital, Via  
Pergolesi 33, 20052 Monza, Italy  
e-mail: antonio.pesenti@unimib.it  
Tel.: +39-039-2333291  
Fax: +39-039-2332297