Individual response on patient's effort and respiratory mechanics to variations in pressure support.

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Background: In pressure support ventilation (PSV), a brief inspiratory occlusion maneuver may unveil the inspiratory effort generated by the patient.[1] In a retrospective study, we reported that, in PSV, the difference between Plateau pressure (Pplat) - measured by the estimation of the inspiratory effort using the pressure muscle index (PMI) - and the positive end-expiratory pressure (PEEP), i.e. driving pressure (ΔP), is associated with outcome in patients with acute respiratory distress syndrome (ARDS).[2] The ongoing prospective ICEBERG STUDY (NCT05203536) aims at validating these findings. However, a systematic assessment of the response of the patient's effort and respiratory mechanics across different levels of PS is lacking.

Aim: The aim of this physiologic study is to assess how patient's effort and respiratory mechanics vary across different levels of ventilatory assistance in PSV.

Materials and methods: We performed a single-center observational prospective study in adult patients with diagnosis of acute hypoxemic respiratory failure, intubated or tracheostomized, undergoing PSV. We cross-randomized patients to four steps of PS level, each one lasting 10 minutes, above (i.e. +3 and +6 cmH2O) or below (i.e. -3 and -6 cmH2O) the clinically set PS level. At the end of each step an inspiratory hold and an expiratory occlusion maneuver were performed to calculate respiratory mechanics parameters such as Pplat, ΔP, respiratory system static compliance (C_{RS}), PMI and P0.1. Electrical activity of both the diaphragm (Costmar) and the intercostal inspiratory muscles (Intercost) was continuously monitored via surface electromyography (sEMG). Differences between continuous variables were tested across PS levels using one-way ANOVA.

Results: 18 patients on PSV were enrolled. Results are presented as means ± standard deviation and one-way ANOVA significance level (P). sEMG results are shown as percentage of the signal measured at clinical PS (%clin). Ppeak (from 9.7 ± 3.8 to 21.7 ± 3.8 cmH_2O, P<0.001) Pplat (from 16.7 ± 2.9 to 20.5 ± 3.3 cmH_2O, P<0.001), ΔP (from 8.6 ± 2.3 to 12.5 ± 2.3 cmH_2O, P<0.001) and tidal volume per predicted bodyweight (VT/PBW, from 6.9 ± 1.5 to 10.0 ± 2.6 mL/kg_{PBW}, P<0.001) increased linearly from -6 to +6 cmH_2O of PS. Inversely, PMI decreased (from 6.4 ± 3.2 to -1.2 ± 2.3 cmH_2O, P<0.001) together with P0.1 (from 2.5 ± 1.2 to 0.8 ± 0.8 cmH_2O, P<0.001), Costmar (from 191 ± 78 to 70 ± 27 %clin, P<0.001) and Intercost (from 150 ± 51 to 74 ± 38 %clin, P<0.001). C_{RS} did not significantly vary through the steps (P=0.119).

Conclusion: The higher the PS the higher the ΔP driven by the increase in VT/PBW, while PMI decreased and C_{RS} did not significantly change (**Fig.1**). Accordingly, sEMG signals gradually decreased when PS was increased. The decreased in ΔP was not mirrored by the decrease of PS assistance because of the increasing inspiratory effort at lower levels of PS as estimated by PMI (**Fig.1A**). When PS was increased over the clinical level, VT/PBW exceeded the threshold of 8 mL/kg_{PBW} **(Fig.1B)**. Further studies may provide information on the potential risk of high tidal volume ventilation at high PS.

References:

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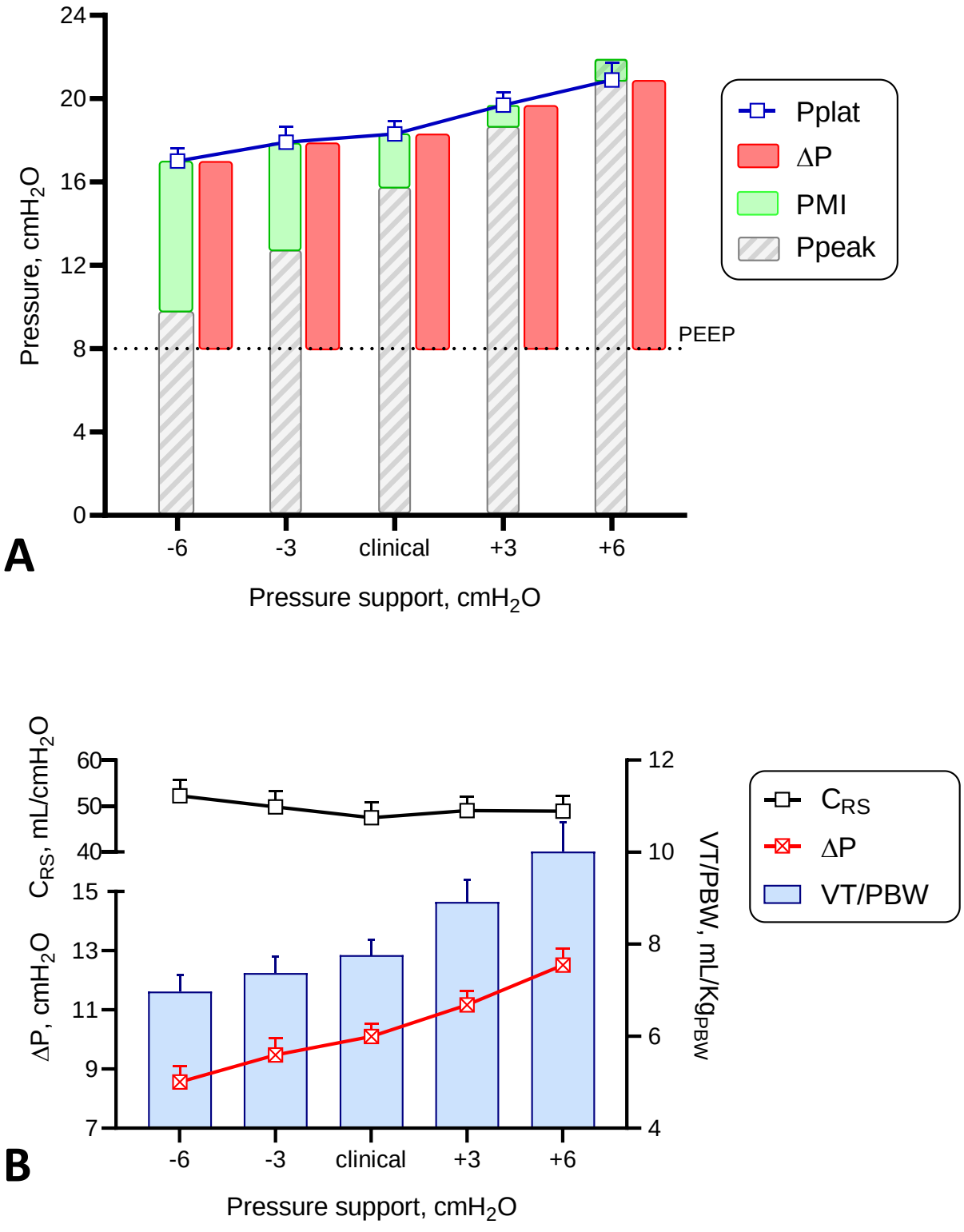

Figure 1. Data are shown as mean and SEM. Pplat, plateau pressure; ΔP, driving pressure; PMI, pressure muscle index; Ppeak, peak airway pressure; PEEP, positive end-expiratory pressure; $C_{RS,}$ respiratory system compliance; VT/PBW, tidal volume per kilograms of predicted bodyweight.