Medtronic provides the following synopsis of a clinical publication involving PAV™**+ software.

**TITLE**  “Driving pressure during assisted mechanical ventilation: Is it controlled by patient brain?”

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**INTRODUCTION**

When patients are ventilated with conventional mechanical ventilation, their lungs are subjected to the degree of stretch (ΔP or VT) that is set by the caregiver and applied by the ventilator. Conventional mechanical ventilation strategies that employ sedation and analgesia to diminish respiratory drive (commonly called passive ventilation) and limit tidal volume (VT) delivery according to ideal body weight are a recognized means of avoiding ventilator-induced lung injury (VILI). However, Amato’s (2015) re-analysis of individual data from more than 3500 ARDS patients who had been enrolled in previous randomized control studies revealed that for patients who are ventilated passively, controlling driving pressure [ΔP; static end-inspiratory plateau pressure (Pplat) minus PEEP, or VT to respiratory system compliance (Crs) ratio] so that VT is normalized to functional lung size rather than to ideal body weight is more strongly associated with survival than is controlling VT alone. In other words, for these patients, a reduction in VT increases survival when associated with a decrease in ΔP.

When patients are ventilated with patient-directed, proportional forms of mechanical ventilation (proportional assist ventilation [PAV™**+] and neurally adjusted ventilatory assist [NAVA]) sedation is limited so that the patient’s respiratory drive remains intact and neither ΔP or VT are set by the caregiver. Instead, the patient’s intrinsic reflexes and instantaneous muscle activity control ventilation.

Georgopoulos et al. hypothesized that when patients are allowed to direct spontaneous breathing (as is the case with PAV™**+ and NAVA), their own feedback mechanisms may naturally act to restrict ΔP but not necessarily restrict VT. In the current study, they sought to compare the ΔP applied during passive controlled mechanical ventilation (CMV) using the currently accepted lung protective strategy (The ARDSnet, 2000; Malhotra, 2007) with the ΔP measured when the same patients are ventilated with the patient-directed, proportional form of mechanical ventilation called PAV™**+.

**PURPOSE OF THE STUDY**

The authors hypothesized that “the patients’ control of breathing system is adept at protecting the lungs by preventing high ΔP using appropriate feedback mechanisms, while not unnecessarily restricting tidal volume when this has no protective value” (Georgopoulos et al., 2016 pg. 70). They sought to determine which variable, VT or ΔP, is controlled by spontaneously breathing, critically ill patients when ventilated with PAV™**+ rather than with passive CMV.

**METHODS**

- Re-analyzed data from 108 patients who had been ventilated passively > 36 h with VC or PC CMV then switched to PAV™**+ (Puritan-Bennett™ 840 ventilator) with PEEP and FIO2 values set similarly to those during CMV and PAV™**+ assist settings adjusted using pre-defined written algorithms.

- Averaged the VT, Crs, and ΔP collected during the initial 8-h PAV™**+ period and compared those values to the corresponding values obtained during CMV within 8-h before switching to PAV™**+.

**RESULTS**
During lung protective passive CMV, VT was tightly controlled and as a result ΔP was less than 15 cmH2O in the majority of the patients.

When patients were switched to spontaneous breathing with PAV™*, ΔP (but not VT) was tightly controlled by the patients.

During PAV™*, the average ΔP did not differ from ΔP used during CMV [10.2 cmH2O (8.1–12.4) vs. 10.7 cmH2O (9.0–12.9), respectively].

VT and Crs were higher with PAV™*+ than during CMV.

CONCLUSION

The main findings of this study were:

1. When critically ill patients were switched from conventional passive, lung protective mechanical ventilation to PAV™*+ assisted spontaneous ventilation which permits them to direct their own depth of breathing, they controlled their driving pressure as well as it had been controlled by the passive, lung protective strategy but they did not constrain VT to the narrow limits used during passive ventilation.

2. The majority of measurements showed that when the lung protective strategy resulted in high driving pressure (ΔP ≥ 15 cmH2O), switching to PAV™*+ resulted patients spontaneously reducing driving pressure.

3. When switched to PAV™*+, patients were able to increase VT without compromising driving pressure because respiratory system compliance increased as a result in the change of ventilation mode.

REFERENCES


**THIS CONCLUDES THE CLINICAL SYNOPSIS OF THIS PUBLICATION**