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CORRESPONDENCE

Driving airway pressure: should we use a static measure to describe a dynamic phenomenon?

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Initial correspondence from Drs. Santini, Votta and Protti

Dear Editor,

Driving airway pressure (ΔP) is the increase in airway pressure due to tidal volume inflation during mechanical ventilation. It is measured as the difference between end-inspiratory pause (plateau, P_{plat}) and end-expiratory (PEEP) airway pressure.

There is no consensus on how to measure P_{plat}. Mezidi and colleagues [1] highlighted how P_{plat}, and thus ΔP, depends on the duration of end-inspiratory pause. To obtain appropriate (i.e. truly *static*) measures, pauses as long as 2 s, compared to 0.5 s, are required. However, mechanical ventilation is a *dynamic* process that usually does not comprise such prolonged pauses.

As long as ΔP is defined as P_{plat}—PEEP, the longer the pause, the more reliable the measurement [1]. But if ΔP is meant to reflect the maximal increase in alveolar pressure during ongoing ventilation, then a prolonged pause will probably underestimate it. In fact, after an end-inspiratory pause, airway pressure initially drops to $P1$ while airflow drops to zero. Thereafter, it further declines to P_{plat} (Fig. 1) mainly because of gas redistribution and lung tissue stress relaxation [2, 3]. $P1$, but not P_{plat}, includes this latter airway pressure gradient that "artificially" dissipates during prolonged end-inspiratory pauses.

We thus wonder whether $P1$ should replace P_{plat} to better estimate the effective (i.e. *dynamic*) driving airway pressure during cyclic tidal inflation. If that is the case, then even end-inspiratory pauses as short as 0.5 s will be too long. Of note, $P1$ could be readily measured by modern ventilators, as the airway pressure when airflow becomes zero.

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Reply from Drs. Mezidi and Guérin

We thank Drs Santini, Votta and Protti for their comments on our letter about the impact of end-inspiratory plateau pressure duration on driving pressure (ΔP) value. They were wondering if driving pressure should be calculated as the difference between $P1$ (airway pressure at first zero flow) and PEEP.

$P1$ to plateau pressure decay results from two mechanisms: (1) visco-elastic properties of the lung and chest wall tissues, and (2) pendelluft phenomenon [4, 5]. Since the static inspiratory equilibrium is reached at the time of plateau pressure, it turns out that, stricto sensu, it is only ΔP computed with plateau pressure that really reflects the elastic properties of the lung. On the other hand, the heterogeneous ARDS lung is characterized by time constant inequalities. Hence, it turns out that, stricto sensu, it is only ΔP computed with $P1$ that reflects the overall elastic load and also the viscoelastic properties of lung and chest wall tissues.

Nonetheless, we performed a supplementary analysis of our dataset and computed ΔP with $P1$ ("dynamic" ΔP) and PEEP set on the ventilator, and compared it with the Amato ΔP definition. Bland–Altman analysis revealed that, at low PEEP, "dynamic" ΔP was 1.8 CI 95% (−1.6 to 5.3) cmH_2O higher than Amato ΔP and both were highly correlated (R^2 0.97; P 10–13). At high PEEP, these values were 1.6 CI 95% (−2.1 to 5.2) cmH_2O (R^2 0.95; $P < 10–7$). Therefore, we are not sure that "dynamic" ΔP brings additional information as compared to the Amato ΔP definition.

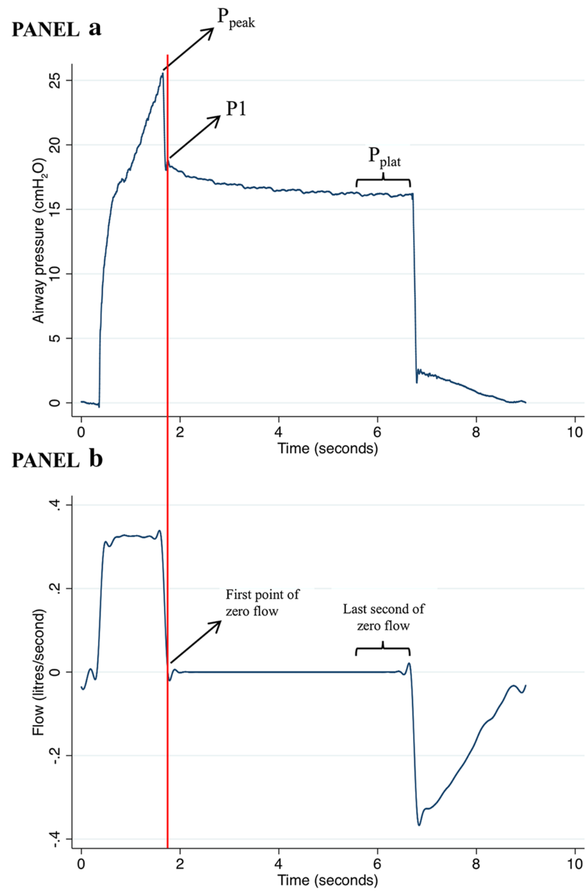

Fig. 1 How to measure *P1*. Representative airway pressure–time (**a**) and corresponding flow-time (**b**) traces during an end-inspiratory pause. *P1* is the point on the pressure–time trace corresponding to the first zero flow point (*red line*) at the beginning of the end-inspiratory pause

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Compliance with ethical standards

Conflicts of interest
All authors declare that they have no conflicts of interest.

Ethical approval
This article does not contain any studies with human participants or animals performed by any of the authors.

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