Intensive Care Med (2018) 44:1532–1535
https://doi.org/10.1007/s00134-018-5045-8

WHAT'S NEW IN INTENSIVE CARE

The airway occlusion pressure ($P_{0.1}$) to monitor respiratory drive during mechanical ventilation: increasing awareness of a not-so-new problem

Irene Telias[1,2,3,4], Felipe Damiani[1,2,5] and Laurent Brochard[1,2*]

Importance of monitoring respiratory drive during mechanical ventilation

An inadequate respiratory drive under mechanical ventilation, either too high or too low, has recently been incriminated as a risk factor for both lung [1] and diaphragmatic injury [2]. Monitoring and controlling the drive to breathe might, therefore, be important for clinical practice. However, respiratory drive assessment has mostly been limited to research purposes, with few techniques available at the bedside [3]. A simple non-invasive measure, the airway occlusion pressure ($P_{0.1}$), i.e. the pressure developed in the occluded airway 100 ms after the onset of inspiration (Fig. 1), was first described 40 years ago. Currently, nearly all modern ventilators provide a means of measuring $P_{0.1}$. Despite having a better understanding of the importance of the respiratory drive during mechanical ventilation, no recommendations exist about its use.

Original description and rationale

In healthy subjects, Whitelaw et al. [4] performed random, short end-expiratory occlusions through a special circuit during both resting and CO_2 rebreathing. They found that the decrease in airway pressure (P_{aw}) during the first 100 ms (i.e. 0.1 s) of an occluded breath was relatively constant, consistent for each patient in each condition, and correlated better with end-tidal CO_2 than minute ventilation. They named this new parameter airway occlusion pressure or $P_{0.1}$ (Fig. 1).

Several characteristics make $P_{0.1}$ a good measure of respiratory center output. There is no conscious or unconscious reaction to the mechanical load during the first milliseconds of an unexpected occlusion. Since it starts from end-expiratory lung volume, any drop in P_{aw} is independent of the recoil pressure of the lung or thorax. Because the flow is interrupted, $P_{0.1}$ is independent of resistance, and there is no change in lung volume that could induce inhibitory reflexes or modify the force–velocity relationship. Finally, there is a good correlation between $P_{0.1}$ and inspiratory effort measured either by the work of breathing (WOB) or the pressure–time product [5, 6]. Importantly, $P_{0.1}$ is still reliable during respiratory muscle weakness if spontaneous breathing is preserved [7].

Range of values

In healthy subjects, $P_{0.1}$ varies between 0.5 and 1.5 cmH_2O [3]. In stable, non-intubated patients with COPD, $P_{0.1}$ varies between 2.5 and 5.0 cmH_2O [3]. Ranges of $P_{0.1}$ from 3.0 to 6.0 cmH_2O have been reported in patients with ARDS under mechanical ventilation, and from 1.0 to as high as 13 cmH_2O during weaning.

Sources of errors and potential pitfalls

There is a significant breath-to-breath variability of $P_{0.1}$, and an average of 3–4 values of $P_{0.1}$ in one patient in one clinical condition should be obtained to represent a reliable index of respiratory drive [8]. In patients with intrinsic positive end-expiratory pressure (PEEPi), there is a delay between the onset of inspiratory effort and the drop

*Correspondence: BrochardL@smh.ca
[2] Keenan Research Centre, Li Ka Shing Knowledge Institute, St. Michael's Hospital, 209 Victoria Street (Room 408), Toronto, ON M5B 1T8, Canada
Full author information is available at the end of the article

Springer

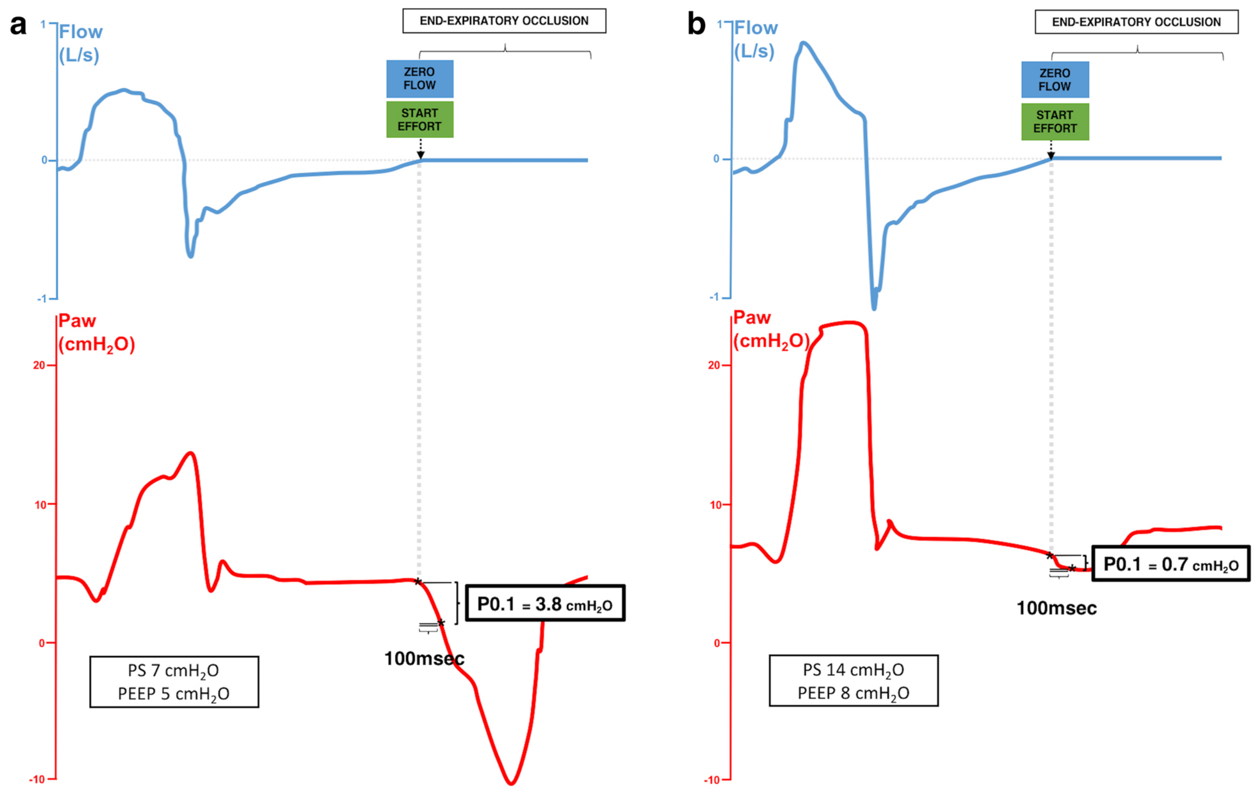

Fig. 1 Measurement of airway occlusion pressure ($P_{0.1}$). Airway pressure (P_{aw} in red) and flow (in blue) during an un-occluded breath and a breath during an end-expiratory occlusion of two different patients under assisted mechanical ventilation with two very different levels of drive. $P_{0.1}$ is measured from the P_{aw} tracing as the drop in airway pressure during the first 100 ms of the breath against an occluded airway. Patient corresponding to tracing (**a**) has a lower ventilatory support and higher respiratory drive than patient corresponding to tracing (**b**). *PS* pressure support level, *PEEP* positive end-expiratory pressure level

in airway pressure during an end-expiratory occlusion. $P_{0.1}$ measured at the mouth in non-intubated patients can underestimate respiratory drive [9]. However, Conti et al. [10] proved that measurement of $P_{0.1}$ from the drop in P_{aw}, since flow reaches zero during the triggering phase of the ventilator, is a reliable surrogate of the decay in esophageal pressure during the first 100 ms of the effort. The difference between the two measurements is small and clinically acceptable (− 0.3 ± 0.5 cmH_2O).

Specific aspects are important when interpreting $P_{0.1}$ displayed by ventilators: decompression of air in the circuit can result in underestimation of $P_{0.1}$, and ventilators use different methods to measure $P_{0.1}$. Some ventilators perform a short end-expiratory occlusion when a manoeuver is activated, but others display a breath-to-breath estimation based on the trigger phase. The trigger phase in modern ventilators is often less than 50 ms, which could result in underestimation of $P_{0.1}$ especially if the inspiratory effort is high [11].

$P_{0.1}$ under mechanical ventilation

$P_{0.1}$ can be useful to adjust the level of ventilatory support due to its close correlation with inspiratory effort. Higher values of $P_{0.1}$ indicate insufficient levels of support while lower values correspond to excessive assistance [5], both during assist-controlled and spontaneous modes of ventilation. We recently showed that $P_{0.1}$ can detect excessive levels of inspiratory effort in patients under pressure control, intermittent mandatory and synchronized intermittent mandatory ventilation [12]. The optimal threshold of $P_{0.1}$ was 3.5 cmH_2O with a sensitivity of 92% and a specificity of 89%. Pletsch-Assuncao et al. [13] recently reported an optimal threshold of $P_{0.1} \leq 1.6$ cmH_2O to diagnose overassistance defined by WOB < 0.3 J/L or > 10% ineffective efforts (with a sensitivity of 62% and a specificity of 87%). This had a lower performance than the presence of a respiratory rate ≤ 17 bpm, possibly related to the technique to measure $P_{0.1}$ (manual occlusion) and the definition of overassistance. Interestingly, a closed-loop algorithm to automatically adjust the level of support based on a target $P_{0.1}$ has proved feasible [14].

$P_{0.1}$ can be used to adjust external positive end-expiratory pressure (PEEP) in patients with hyperinflation. Mancebo et al. [6] proved that a decrease in estimated $P_{0.1}$ with the addition of external PEEP indicates a drop in PEEPi and in WOB with reasonable sensitivity and specificity.

More recently, Mauri et al. [15] found that $P_{0.1}$ is a sensitive indicator of respiratory drive in patients with severe ARDS undergoing venous–venous extracorporeal membrane oxygenation. They showed that a change in sweep gas flow resulting in a change in $PaCO_2$ was well reflected by $P_{0.1}$, varying on average between 0.9 and 3.0 cmH_2O.

$P_{0.1}$ has extensively been studied as a predictor of weaning success or failure. Originally, a high $P_{0.1}$ during a spontaneous breathing trial was associated with failure, suggesting that a high respiratory drive could predict weaning failure. As elegantly proved by Bellani et al. [16], patients failing a trial of decrease in support during weaning are unable to increase oxygen consumption in response to an increased drive (i.e. higher $P_{0.1}$).

However, overlap in $P_{0.1}$ values between success and failure groups was evidenced as more data were published, and no threshold accurately predicts weaning outcome using $P_{0.1}$ alone or combined with other parameters [17]. This is explained by the complex pathophysiology of weaning failure and the design of experiments. $P_{0.1}$ was often measured during pressure support, known to underestimate inspiratory effort after extubation [18]. Despite having no magic value to predict weaning outcome, clinicians can still get information concerning the respiratory drive (high or low). In this context, very high values (for example, higher than 6 cmH_2O) are associated with failure.

Conclusions and future directions

$P_{0.1}$ is a useful and valid measure of respiratory drive in mechanically ventilated patients. Work is needed to build a bridge between research and clinical practice since this parameter is now easily available. The accuracy of $P_{0.1}$ displayed by modern ventilators (using flow or pressure trigger), and in different clinical conditions (e.g., presence of PEEPi) needs to be studied.

Additionally, a practical approach to the use of $P_{0.1}$ displayed by ventilators needs to be evaluated. $P_{0.1}$ can be used to detect excessive or insufficient levels of inspiratory effort and better guide muscle- and lung-protective ventilation strategies. The latter should include adjusting ventilator settings, CO_2 removal and titration of sedative drugs to achieve an acceptable range of inspiratory effort for a given clinical condition. In particular, $P_{0.1}$ could be of great value during a sensitive period: transition from fully controlled to assisted modes of ventilation.

$P_{0.1}$ can already provide clinicians with information regarding the drive of their patients, it is sensitive to ventilator settings, and may be useful during weaning. Considering the importance of patients' respiratory drive under mechanical ventilation, it seems that it is time to start using it in the clinical setting.

Author details
[1] Interdepartmental Division of Critical Care Medicine, University of Toronto, Toronto, Canada. [2] Keenan Research Centre, Li Ka Shing Knowledge Institute, St. Michael's Hospital, 209 Victoria Street (Room 408), Toronto, ON M5B 1T8, Canada. [3] Division of Respirology, Department of Medicine, University Health Network and Sinai Health System, Toronto, Canada. [4] Sanatorio Mater Dei, Buenos Aires, Argentina. [5] Departamento de Medicina Intensiva, Pontificia Universidad Católica de Chile, Santiago, Chile.

Acknowledgements
Funding was provided by Keenan Chair in Critical Care and Acute Respiratory Failure.

Compliance with ethical standards

Conflicts of interest
IT received consulting fees from MBMed SA. LB's research laboratory received research grants and/or equipment from Covidien, General Electric, Fisher Paykel, Maquet, Air Liquide, and Philips.

Received: 29 November 2017 Accepted: 4 January 2018
Published online: 19 January 2018

References

1. Brochard L, Slutsky A, Pesenti A (2017) Mechanical ventilation to minimize progression of lung injury in acute respiratory failure. Am J Respir Crit Care Med 195:438–442. https://doi.org/10.1164/rccm.201605-1081CP
2. Goligher EC, Dres M, Fan E et al (2017) Mechanical ventilation-induced diaphragm atrophy strongly impacts clinical outcomes. Am J Respir Crit Care Med. https://doi.org/10.1164/rccm.201703-0536OC
3. Tobin MJ, Gardner W (1998) Monitoring the control of breathing. In: Tobin M (ed) Principles and practice of intensive care monitoring. McGraw-Hill, New York, pp 415–464
4. Whitelaw WA, Derenne JP, Milic-Emili J (1975) Occlusion pressure as a measure of respiratory center output in conscious man. Respir Physiol 23:181–199. https://doi.org/10.1016/0034-5687(75)90059-6
5. Alberti A, Gallo F, Fongaro A et al (1995) $P_{0.1}$ is a useful parameter in setting the level of pressure support ventilation. Intensive Care Med 21:547–553. https://doi.org/10.1007/BF01700158
6. Mancebo J, Albaladejo P, Touchard D et al (2000) Airway occlusion pressure to titrate positive end-expiratory pressure in patients with dynamic hyperinflation. Anesthesiology 93:81–90. https://doi.org/10.1097/00000542-200007000-00016
7. Holle RH, Schoene RB, Pavlin EJ (1984) Effect of respiratory muscle weakness on $P_{0.1}$ induced by partial curarization. J Appl Physiol 57:1150–1157. https://doi.org/10.1152/jappl.1984.57.4.1150
8. Kera T, Aihara A, Inomata T (2013) Reliability of airway occlusion pressure as an index of respiratory motor output. Respir Care 58:845–849. https://doi.org/10.4187/respcare.01717
9. Murciano D, Aubier M, Bussi S et al (1982) Comparison of esophageal, tracheal, and mouth occlusion pressure in patients with chronic obstructive pulmonary disease during acute respiratory failure. Am Rev Respir Dis 126:837–841. https://doi.org/10.1164/arrd.1982.126.5.837
10. Conti G, Cinnella G, Barboni E et al (1996) Estimation of occlusion pressure during assisted ventilation in patients with intrinsic PEEP.

Am J Respir Crit Care Med 154:907–912. https://doi.org/10.1164/ajrccm.154.4.8887584
11. Telias IG, Junhasavasdikul D, Rittayamai N et al (2017) Accuracy of $P_{0.1}$ displayed by modern ventilators—a bench study. Am J Respir Crit Care Med 195:A1881
12. Rittayamai N, Beloncle F, Goligher EC et al (2017) Effect of inspiratory synchronization during pressure-controlled ventilation on lung distension and inspiratory effort. Ann Intensive Care 7:100. https://doi.org/10.1186/s13613-017-0324-z
13. Pletsch-Assuncao R, Caleffi Pereira M, Ferreira JG et al (2017) Accuracy of invasive and noninvasive parameters for diagnosing ventilatory overassistance during pressure support ventilation. Crit Care Med. https://doi.org/10.1097/CCM.0000000000002871
14. Iotti GA, Brunner JX, Braschi A et al (1996) Closed-loop control of airway occlusion pressure at 0.1 second ($P_{0.1}$) applied to pressure-support ventilation: algorithm and application in intubated patients. Crit Care Med 24:771–779
15. Mauri T, Grasselli G, Suriano G et al (2016) Control of respiratory drive and effort in extracorporeal membrane oxygenation patients recovering from severe acute respiratory distress syndrome. Anesthesiology 125:159–167. https://doi.org/10.1097/ALN.0000000000001103
16. Bellani G, Foti G, Spagnolli E et al (2010) Increase of oxygen consumption during a progressive decrease of ventilatory support is lower in patients failing the trial in comparison with those who succeed. Anesthesiology 113:378–385. https://doi.org/10.1097/ALN.0b013e3181e81050
17. Fernandez R, Raurich JM, Mut T et al (2004) Extubation failure: diagnostic value of occlusion pressure ($P_{0.1}$) and $P_{0.1}$-derived parameters. Intensive Care Med 30:234–240. https://doi.org/10.1007/s00134-003-2070-y
18. Sklar MC, Burns K, Rittayamai N et al (2017) Effort to breathe with various spontaneous breathing trial techniques. a physiologic meta-analysis. Am J Respir Crit Care Med 195:1477–1485. https://doi.org/10.1164/rccm.201607-1338OC