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EDITORIAL

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Why does airway pressure rise sometimes near the end of inflation during pressure support?

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Sir: Patient-ventilator non-synchrony is common [1–6] and may increase morbidity [4]. Some possible reasons for its adverse effects include increased need for sedation, sleep disruption, respiratory muscle injury and errors in assessing weanability (see [7] for review).

Extension of the ventilator's inflation phase much beyond the end of inspiratory effort (delayed cycling-off) is a very common form of non-synchrony, particularly with pressure support (PSV) [2, 3, 5, 6; see 8–10 for mechanisms]. With delayed cycling-off, the lungs are being forcibly inflated when the patient wants them to deflate. When this occurs in alert individuals, it results in considerable discomfort. Often, expiratory muscles are activated in an effort to force cycling-off [11–13].

Airway pressure (P_{AW}) frequently overshoots the set PSV level before the ventilator cycles off [13]. Given the reported expiratory muscle activation in association with delayed cycling-off [11, 12], some researchers interpret the P_{AW} overshoot as indicative of expiratory recruitment in response to delayed cycling-off (i.e. patient is fighting the ventilator). Whereas expiratory muscle activation may result in pressure overshoot, an overshoot need not reflect expiratory muscle activation or even excessive delayed cycling-off. This is important to recognize, since concluding that the patient is fighting the ventilator, or that cycling-off delay is excessive when it is not, may lead to unnecessary sedation or to increasing the cycling-off flow threshold when this is not indicated. The latter situation may result in the equally undesirable premature termination of the inflation cycle.

To understand the mechanism of P_{AW} overshoot it is necessary to review how the ventilator controls P_{AW}. When gas is flowing into the patient, P_{AW} (i.e. upstream pressure) is higher than downstream pressure (alveolar pressure; P_{ALV}) by an amount corresponding to flow rate and respiratory resistance (R_{RS}; Fig. 1):

$$P_{AW} = P_{ALV} + \text{Flow} \times R_{RS} \,. \qquad \text{Eq. (1)}$$

Thus, if P_{ALV} increases, P_{AW} must increase unless the ventilator decreases flow appropriately. Flow output of ventilators changes in response to an error signal, which

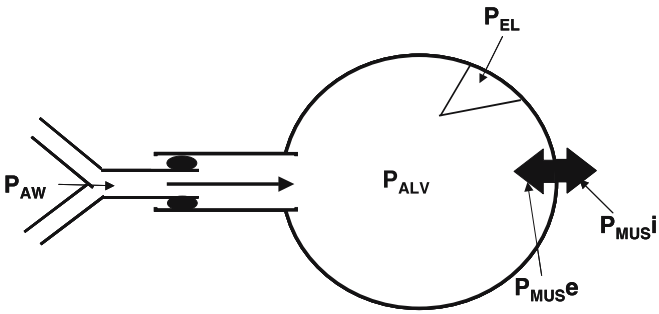

Fig. 1 Dynamic relation between airway pressure (P_{AW}), alveolar pressure (P_{ALV}) and flow, and determinants of P_{ALV}. P_{EL} elastic recoil pressure of the respiratory system, P_{MUS}i and P_{MUS}e are pressures generated by inspiratory and expiratory muscles, respectively

in PSV is a function of the difference between current pressure and target pressure. A gain factor (error gain) determines how much flow changes per unit change in error signal. A high error gain achieves the desired control with less deviation from target, but it also promotes oscillation in flow and P_{AW}. Complex handling of the error signal has evolved to optimize performance. The error signal to which the valves respond is made up of different components, one related to current error magnitude (proportional component), one to the integral of error over a specified previous interval (integral component) and one to the rate of change in error (derivative component), each with its own gain (the PID system). Such systems can maintain pressure close to target if current error changes slowly. But for rapid reduction in flow, as would be needed if P_{ALV} were rising rapidly, deviation from target pressure (current error) must be large. Increasing the gain of the proportional component would attenuate the pressure overshoot, but this promotes oscillatory behaviour. The error gains in effect in a given ventilator represent the manufacturer's compromise between fidelity of response and stability.

What determines P_{ALV} during the ventilator's inspiratory phase? In the passive state, and ignoring the usually small chest-wall resistance [14], P_{ALV} is essentially the passive recoil pressure of the lung/chest-wall complex (P_{EL}; Fig. 1), which is a function of lung volume and respiratory elastance. When spontaneous efforts are present (e.g. during PSV), P_{ALV} is additionally determined by inspiratory muscle pressure ($P_{MUS}i$), which reduces P_{ALV} below P_{EL}, and by expiratory muscle pressure ($P_{MUS}e$), which increases P_{ALV} above P_{EL} (Fig. 1):

$$P_{ALV} = P_{EL} - P_{MUS}i + P_{MUS}e \; . \qquad \text{Eq. (2)}$$

Neural inspiration continues for a while following active triggering. During this time $P_{MUS}i$ continues to rise, tending to reduce P_{ALV}, but lung volume, and hence P_{EL}, also rises tending to increase P_{ALV}. Since active expiration, if it happens at all, begins during neural expiration, the time course of P_{ALV} during this phase is determined by the balance between rate of increase in $P_{MUS}i$ and rate of increase in P_{EL}. With low respiratory drive and high PSV level, the increase in P_{EL} dominates quickly, and P_{ALV} begins rising soon after triggering, whereas with high drive and relatively low assist P_{ALV} may continue to fall for a while after triggering. In either case, because rates of increase in $P_{MUS}i$ and in P_{EL} are relatively slow, and the two forces tend to cancel each other, the net rate of change in P_{ALV} during the phase of neural inspiration is relatively slow and can be easily handled by the ventilator's slow control system; thus, flow increases or decreases appropriately with P_{ALV}, and P_{AW} is maintained close to target.

At the end of neural inspiration $P_{MUS}i$ falls. During the phase of declining $P_{MUS}i$ the earlier antagonistic relation between $P_{MUS}i$ and P_{EL} becomes complimentary in that both are now operating to increase P_{ALV}. P_{ALV} will increase at a faster rate, determined primarily by the rate of decline in $P_{MUS}i$. Control of this rate of decline is quite complex [15, 16]. In general, however, the rate of decline in $P_{MUS}i$ is greater when inspiratory muscle activation is higher [17–19].

For a given rate of increase in P_{ALV} at end-inspiration the rate at which the ventilator must reduce flow to keep P_{AW} constant is inversely related to resistance (Eq. (1)). Accordingly, all else being the same, less error (i.e. less overshoot) will develop if resistance is high.

Activation of expiratory muscles at any point in the respiratory cycle will also result in an increase in P_{ALV} (Eq. (2)), necessitating a reduction in flow to maintain P_{AW} close to target. As with loss of $P_{MUS}i$ at end-inspiration, whether or not an error (i.e. overshoot) develops, its magnitude will depend on how fast expiratory muscle pressure ($P_{MUS}e$) is increasing, and on R_{RS}.

It is clear from the above that a pressure overshoot will develop when there is a fast decline in $P_{MUS}i$ or a fast increase in $P_{MUS}e$, particularly when resistance is low. Until recently, it was not known whether the occurrence of an overshoot during PSV is due to normal relaxation of inspiratory muscles or to expiratory muscle activation. In this issue, Prinianakis et al. [20] report on the mechanism of overshoot in 15 patients who displayed an overshoot. In all patients onset of the overshoot coincided with onset of decline in $P_{MUS}i$. Expiratory activation was observed in only half the patients and, when it occurred, began well beyond the onset of the overshoot. This does not mean that expiratory activity is never responsible for the overshoot; instead, their data indicate that, in practice, when an overshoot is present during PSV it is much more likely the result of the normal relaxation of inspiratory muscles than activation of expiratory muscles. As expected, the rate of increase in P_{AW} during the overshoot correlated with the rate of decline in $P_{MUS}i$. An even better correlation was observed between the rate of increase in P_{AW} and the rate of decline in flow, likely because the required rate of decrease in flow incorporates both the rate of increase in P_{ALV} and R_{RS}, and not the change in P_{ALV} alone (Eq. (1)).

What to do if there is an overshoot? The data of Prinianakis et al. [20] clearly teach that it is not prudent to presume that expiratory muscles are active simply because an overshoot is present. Furthermore, even if expiratory muscles are contributing to the overshoot, this need not reflect fighting or even a reflex response to continued lung inflation; expiratory muscles are routinely activated at increased levels of respiratory drive (e.g. during exercise [21] and with CO_2 and hypoxic stimulation [22]), even in the absence of any feedback from the lungs [22]. To conclude that there is fighting, there must be other indications of fighting, for example, agitation or if the overshoot is inconsistent and erratic-looking, as may be expected of behavioural responses; however, can one at least conclude that there is excessive delayed

cycling-off and raise the cycling-off flow threshold? Here, again, the answer is no, unless there is other evidence of excessive cycling-off delay. During normal breathing without a ventilator expiratory flow does not begin until sometime after P_{MUS}i starts declining [15, 18, 23; see 5 for recent discussion]. Accordingly, a reasonable cycling-off delay is neither abnormal nor undesirable and does not warrant intervention. As indicated above, an excessive overshoot will tend to occur primarily when respiratory drive is high and resistance is low. Under these conditions, excessive cycling-off delay is extremely unlikely [3, 8, 10]. In this respect it is relevant to note that the patients of Prinianakis et al. [20] were receiving a relatively low level of PSV ($\approx 8\,cmH_2O$) and had a relatively high inspiratory drive ($\Delta P_{DI}/\Delta t = 19.2\,cmH_2O/s$). As may be expected under these conditions, the overshoot phase was brief (estimated to be 0.18 s, on average, based on an overshoot amplitude of $2.6\,cmH_2O$ and a rate of increase in P_{AW} of $14.7\,cmH_2O/s$). This is well within the normal physiological delay between end of neural inspiration and onset of expiratory flow; thus, advancing cycling-off is indicated only if the delay (i.e. duration of the overshoot) is excessive (e.g. $> 10\%$ of respiratory cycle duration, or as indicated indirectly by the presence of ineffective efforts) and, if this is done, care must be taken not to eliminate the overshoot completely as this may result in premature termination of the ventilator cycle. Since the overshoot occurs primarily when respiratory drive is high, the presence of an important overshoot should invite an assessment of whether the current level of support is adequate.

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