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Short communications

Expiratory tidal volume displayed on Bird 8400*STi* can exceed the preset tidal volume due to cardiogenic oscillation: a lung model study

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Abstract We noticed that monitored tidal volumes often exceeded preset tidal volumes in patients with large cardiogenic oscillation. To investigate whether triggering modes affect this discrepancy, we simulated cardiogenic oscillation of 90 breaths/min in a lung model, which was ventilated with a Bird 8400*STi* ventilator (Bird, Palm Springs, CA, USA). The magnitude of cardiogenic oscillation was defined as peak expiratory flow fluctuation at the lung model. Two respiratory rates (5 and 10 breaths/min) and two triggering modes (flow-triggering and pressure-triggering) were applied, while tidal volume was set at 500 ml. We recorded tidal volume on a ventilator monitor and calculated the discrepancy from the set tidal volume. We also measured fluctuation in flow and airway opening pressure created by cardiogenic oscillation. During flow-triggering, larger flow fluctuation and smaller airway pressure fluctuation were observed compared with during pressure-triggering. During flow-triggering, the discrepancy between monitored tidal volume and set value ranged from 0 to +327 ml at 5 breaths/min, and from 0 to +105 ml at 10 breaths/min. There was a linear correlation between the magnitude of cardiogenic oscillation and the overestimation of tidal volume. By contrast, during pressure-triggering, the discrepancy was small. In conclusion, tidal volume is overestimated during flow-triggering but not during pressure-triggering when cardiogenic oscillation is large.

Key Words Cardiogenic oscillation · Tidal volume · Lung model · Flow-triggering · Pressure-triggering

Cardiogenic oscillation is the fluctuation of flow or pressure created by the heartbeat at the airway opening. Cardiogenic oscillation may trigger ventilators, if its magnitude is large, even when patients do not breathe spontaneously [1,2]. Recently, in these types of patients, we noticed that the monitored tidal volume often exceeded the set tidal volume. Because the correct monitoring of tidal volume is essential in mechanically ventilated patients with respiratory failure [3], the effects of cardiogenic oscillation on tidal volume measurement must be clarified. Using a lung model, we investigated whether triggering modes affected the magnitude of cardiogenic oscillation and tidal volume overestimation.

To simulate cardiogenic oscillation, we used a two-bellows-type lung model (TTL model 1601; Michigan Instruments, Grand Rapids, MI, USA), details of which are described elsewhere (Fig. 1) [4]. We simulated cardiogenic oscillation in a driving chamber by ventilating the chamber with a V.I.P. Bird ventilator (Bird) at 90 breaths·min^{-1}, pressure-controlled ventilation, and an inspiratory-to-expiratory ratio of 1:1. A lift bar was set to transmit the oscillation generated in the driving chamber to the experimental chamber (compliance of 50 ml·cmH_2O^{-1} and resistance of 20 cmH_2O·s·l^{-1}). The magnitude of cardiogenic oscillation was defined as peak expiratory flow fluctuation (l·min^{-1}) at the airway opening of the experimental chamber when the airway was opened to the atmosphere. Then, we adjusted the level of pressure control of the V.I.P. Bird to obtain cardiogenic oscillation of 0, 2, 4, 6, and 8 l·min^{-1}, because most postcardiac surgery patients showed such a range of cardiogenic oscillation [1].

To each level of cardiogenic oscillation, we randomly applied two triggering modes and two respiratory rates: flow-triggering (FT) and pressure-triggering (PT), and 5 breaths·min^{-1} and 10 breaths·min^{-1} with a Bird 8400*STi* ventilator (Bird). The ventilator was connected to the experimental chamber via a full-length 8-mm bore endotracheal tube (Portex, Hythe, UK). We calibrated the ventilator according to the manufacturer's instructions and confirmed that there was no leakage along the ventilatory circuit (DAR Breathing System 6043; Mallinckrodt, Mirandola, Italy). Other ventilatory settings were as follows: synchronized intermittent mandatory ventilation, tidal volume of 500 ml, inspiratory time of 1.0 s, and positive end-expiratory pressure of

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4 cmH_2O. We adjusted triggering sensitivity to the level where autotriggering did not occur with the maximal cardiogenic oscillation: 10 l·min^{-1} during FT (P/N 10302R flow transducer; Bird) and −2 cmH_2O during PT (P/N 10081R flow transducer; Bird). Bias flow in the Bird 8400*STi* is fixed at 10 l·min^{-1} during FT and at zero during PT [5,6].

A pneumotachometer (model 3700A; Hans-Rudolph, Kansas City, MO, USA) was placed at the proximal end of the endotracheal tube. The pneumotachometer pressure differential was measured with a differential transducer (±5 cmH_2O), amplified, and converted to flow, using data acquisition software (Dataq Instruments, Akron, OH, USA). Airway pressure was measured by a differential transducer (±50 cmH_2O) and amplified. All signals were digitized and recorded at 100 Hz per channel. The transducer for airway pressure was calibrated at 10 cmH_2O, using a water manometer. We recorded tidal volume on a ventilator monitor and calculated the discrepancy between the monitored tidal volume and the set tidal volume. We also measured fluctuation in flow and airway opening pressure at the end of the expiratory phase during mechanical ventilation. Finally, we calculated volume displacement per heartbeat by integrating the expiratory flow fluctuation. Data values are presented as means ± SD. Using analysis of variance with repeated measures, mean values were compared across different settings. When significance was observed, the mean values were compared using paired Student's *t*-tests with Bonferroni's correction. Where applicable, we performed post-hoc analysis using a two-tailed paired *t*-test and linear regression analysis. Statistical significance was set at $P < 0.05$.

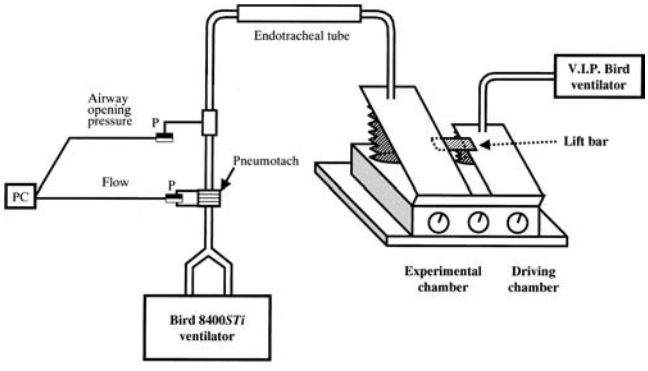

Fig. 1. Experimental setup of the lung model. Cardiogenic oscillation was created in a driving chamber at 90 breaths·min^{-1} by a V.I.P. Bird ventilator and was transmitted to an experimental chamber through a lift bar. See the text for details. *P*, pressure; *PC*, personal computer

Representative traces of flow and pressure during a breath cycle are shown in Fig. 2. While larger fluctuation in flow was observed during FT than during PT, fluctuation in the airway opening pressure was larger with PT (Table 1). During FT, the discrepancy between monitored tidal volume and set tidal volume reached 327 ml at a respiratory rate of 5 breaths·min^{-1}, and 105 ml at 10 breaths·min^{-1}. There was a linear correlation between the cardiogenic oscillation and the overestimation of tidal volume ($y = 40x - 32$; $R^2 = 0.93$ at 5 breaths·min^{-1}; $y = 14x - 12$; $R^2 = 0.95$ at 10 breaths·min^{-1}, when x = peak expiratory flow fluctuation(l·min^{-1}) caused by cardiogenic oscillation; y = overestimation of tidal volume). In contrast, during PT, the discrepancy between monitored tidal volume and set tidal volume was small (−13 to +1 ml).

The main findings of this bench study are summarized as follows: (a) during FT, exhaled tidal volume was overestimated when cardiogenic oscillation was large;

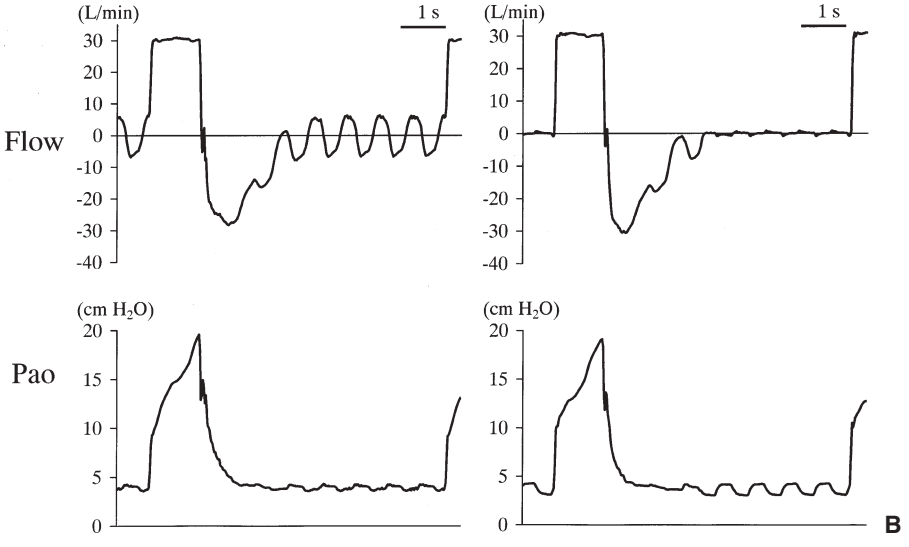

Fig. 2A,B. Representative traces of flow and pressure during a breath cycle. **A**, Flow-triggering; **B**, pressure-triggering. *Top*, flow at the airway opening; *bottom*, airway opening pressure. Ventilatory settings are: volume-controlled ventilation, 500 ml tidal volume, and respiratory rate of 10 breaths·min^{-1}

Table 1. Effects of cardiogenic oscillation on flow, pressure, and tidal volume

	Cardiogenic oscillation ($l \cdot min^{-1}$)				
	0	2	4	6	8
Fluctuation in expiratory flow ($l \cdot min^{-1}$)					
Flow-triggering	0.63	2.43	3.41	4.59	6.54
Pressure-triggering	0.00	0.28	0.49	0.63	0.90
Fluctuation in airway pressure (cmH_2O)					
Flow-triggering	0	0.38	0.47	0.59	0.71
Pressure-triggering	0	0.47	0.59	0.74	1.21
Overestimation in tidal volume at 5 breaths·min^{-1} (ml)					
Flow-triggering	0	50	104	195	327
Pressure-triggering	1	−1	−5	−3	−5
Overestimation in tidal volume at 10 breaths·min^{-1} (ml)					
Flow-triggering	0	8	43	70	105
Pressure-triggering	−7	−11	−11	−13	−12

(b) during FT, the overestimation of tidal volume increased in proportion to the magnitude of cardiogenic oscillation; (c) flow fluctuation due to cardiogenic oscillation was larger during FT than during PT. To our knowledge, this is the first description to show that triggering modes affect cardiogenic oscillation and to show the effects of cardiogenic oscillation on the monitoring function of a ventilator. Large cardiogenic oscillation caused overestimation of tidal volume maximally by 65% during FT. Overestimation of tidal volume is a serious problem, because this monitoring malfunction is not easy to notice or to correct. Patients appear to be breathing with a greater tidal volume than they actually are, leading to a mistaken decision to wean them from mechanical ventilation or to inappropriately adjust the ventilatory setting.

The overestimation in tidal volume was apparent during FT of the Bird 8400*STi*. The effect of cardiogenic oscillation on flow was smaller during PT than during FT (Table 1). This could be because of the difference in expiratory-phase continuous flow (0 $l \cdot min^{-1}$ during PT and 10 $l \cdot min^{-1}$ during FT). We supposed that, during PT, the ventilator worked like a near-closed system, and provided smaller flow fluctuation and smaller expiratory volume displacement than during FT (Table 1). We have also speculated that this ventilator sums all exhaled volumes between the mandatory breaths as a tidal volume, although we could not get detailed information from the manufacturers. Actually, when we summed exhaled volumes between two mandatory breaths, we found a close correlation ($y = 1.00x + 25$; $R^2 = 0.95$) between the monitored tidal volume and calculated tidal volume.

This study has several limitations. First, the current observation may result from the specific property of the FT function in the Bird 8400*STi* ventilator. Further study is needed to clarify whether cardiogenic oscillation affects volume monitoring in other ventilator models. Second, direct application of the data from this study to the clinical setting is limited by the fact that this was a lung model. The model did not simulate the patient situation, where many factors influence the degree of transmission of cardiogenic oscillation to the airway [1].

In conclusion, expiratory tidal volume displayed on the Bird 8400*STi* ventilator can exceed the preset tidal volume during FT when cardiogenic oscillations are large.

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