

Determinants of Dynamic Hyperinflation in a Bench Model

Natalya Y Thorevska MD and Constantine A Manthous MD

BACKGROUND: Previous *in vivo* data suggest that high airway resistance (R_{aw}) promotes dynamic hyperinflation, especially when coupled to high minute ventilation (\dot{V}_E). However, no studies have systematically examined the relative effects of various mechanical parameters on dynamic hyperinflation. **METHODS:** Intrinsic positive end-expiratory pressure (PEEPi) was measured with a ventilator-lung model, over a range and various permutations of R_{aw} , \dot{V}_E , respiratory system compliance (C_{RS}), and duty cycles/flow regimes. **RESULTS:** Substantial dynamic hyperinflation (PEEPi > 5 cm H₂O) occurred at various \dot{V}_E , even when R_{aw} was low (4 cm H₂O/L/s) or just above normal (18 cm H₂O/L/s). A $\dot{V}_E \geq 15$ L/min was associated with increasing PEEPi in this model, across a broad range of mechanical permutations. PEEPi was significantly higher in all models during descending ramp flow than during constant flow, at equivalent peak flows (wherein duty cycle during descending ramp flow was twice that of constant flow). PEEPi was equivalent when duty cycles (and all other mechanical parameters) were equal. PEEPi was significantly greater, irrespective of duty cycle, R_{aw} , and C_{RS} , when delivered with lower tidal volume (0.6 L vs 1.0 L). The change in peak airway pressure associated with development of dynamic hyperinflation was consistently greater than the observed PEEPi. Higher \dot{V}_E , resistance, compliance, and duty cycles were all independently associated with dynamic hyperinflation. **CONCLUSIONS:** In this bench model, dynamic hyperinflation occurred with high \dot{V}_E , even at low R_{aw} . Since moderate R_{aw} and \dot{V}_E frequently occur *in vivo*, even without obstructive lung disease, occult dynamic hyperinflation is likely to occur commonly. PEEPi was greater with high frequency and small tidal volume (0.6 L) than with equal \dot{V}_E of lower frequency and larger tidal volume (1.0 L). *Key words:* positive end-expiratory pressure, barotrauma, mechanical ventilation, asthma, chronic obstructive pulmonary disease, acute respiratory distress syndrome. [Respir Care 2004;49(11):1326–1334. © 2004 Daedalus Enterprises]

Introduction

The complications of dynamic hyperinflation are well described in patients with obstructive lung diseases.^{1–3} *In vivo* data suggest that high airway resistance (R_{aw}) promotes dy-

namic hyperinflation, especially when coupled with hyperventilation.^{1,2} Previous data have suggested that dynamic hyperinflation may occur in situations other than severe airway obstruction.^{4–7} In the current study we examined systematically the relationships of R_{aw} , elastance, duty cycle, and minute ventilation (\dot{V}_E) on intrinsic positive end-expiratory pressure (PEEPi) in a ventilator-lung model.

Methods

A mechanical ventilator (7200, Nellcor-Puritan Bennett, Pleasanton, California) was connected via plastic tubing and a Y-piece to a training/test lung model (VentAid, Michigan Instruments, Grand Rapids, Michigan). This test lung model (Fig. 1) is a 2-compartment mechanical analogue of the respiratory system, consisting of 2 rubber bellows (simulating lung parenchyma and chest wall) connected to a

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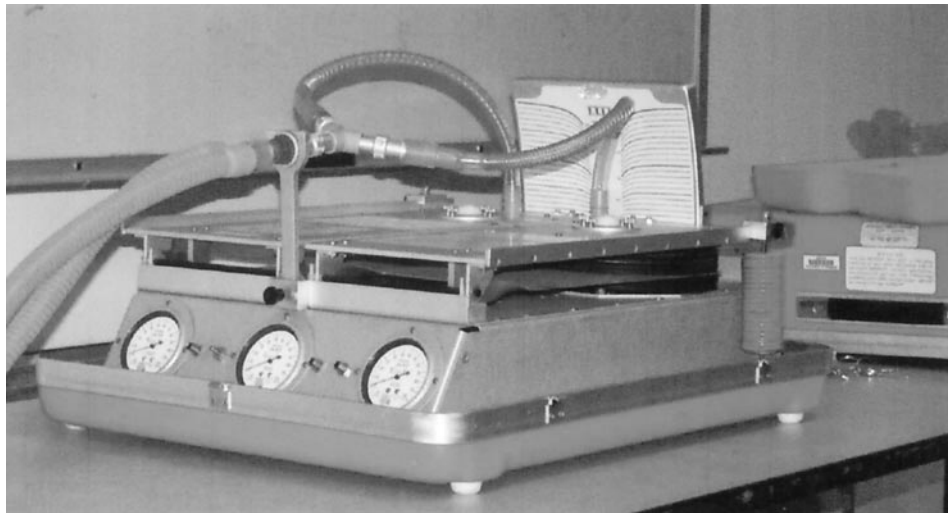


Fig. 1. The VentAid training/test lung model.

Y-shaped plastic tube (simulating the trachea and primary bronchi). Tubes of 3 different calibers, provided with the model, were used to vary R_{aw} . Total model R_{aw} , for each tube, was measured using the ventilator's resistance-measurement function. Test lung compliance (compliance of the respiratory system [C_{RS}]) was adjusted by applying springs to the model's bellows; values of compliance in various conditions were measured using the ventilator's compliance-measurement function.

A number of clinical scenarios were simulated using the following combinations of mechanical parameters delivered at 7 increments of \dot{V}_E , at constant (square) and descending ramp inspiratory flow regimes with equivalent peak inspiratory flows:

1. Normal compliance, low resistance: C_{RS} 54 mL/cm H_2O , R_{aw} 4 cm $H_2O/L/s$
2. Mild obstruction: C_{RS} 54 mL/cm H_2O , R_{aw} 18 cm $H_2O/L/s$
3. Moderate obstruction: C_{RS} 54 mL/cm H_2O , R_{aw} 28 cm $H_2O/L/s$
4. Mild restriction: C_{RS} 40 mL/cm H_2O , R_{aw} 4 cm $H_2O/L/s$
5. Moderate restriction: C_{RS} 30 mL/cm H_2O , low R_{aw} 4 cm $H_2O/L/s$
6. Severe restriction: C_{RS} 20 mL/cm H_2O , R_{aw} 4 cm $H_2O/L/s$
7. Mild restriction, mild obstruction: C_{RS} 40 mL/cm H_2O , R_{aw} 18 cm $H_2O/L/s$
8. Mild restriction, moderate obstruction: C_{RS} 40 mL/cm H_2O , R_{aw} 28 cm $H_2O/L/s$
9. Moderate restriction, mild obstruction: C_{RS} 30 mL/cm H_2O , R_{aw} 18 cm $H_2O/L/s$
10. Moderate restriction, moderate obstruction: C_{RS} 30 mL/cm H_2O , R_{aw} 28 cm $H_2O/L/s$

11. Severe restriction, mild obstruction: C_{RS} 20 mL/cm H_2O , R_{aw} 18 cm $H_2O/L/s$
12. Severe restriction, moderate obstruction: C_{RS} 20 mL/cm H_2O , R_{aw} 28 cm $H_2O/L/s$
13. Moderate inelastance, mild obstruction: C_{RS} 60 mL/cm H_2O , R_{aw} 18 cm $H_2O/L/s$
14. Moderate inelastance, moderate obstruction: C_{RS} 60 mL/cm H_2O , R_{aw} 28 cm $H_2O/L/s$

The Nellcor-Puritan Bennett 7200 ventilator delivers any given tidal volume (V_T) (and peak inspiratory flow) at twice the inspiratory time with a descending ramp, compared to the constant, inspiratory waveform. Effects of increasing \dot{V}_E (6, 9, 12, 15, 18, 21, 24 L/min) were studied for each combination of mechanical parameters. Control, volume-cycled ventilation was used with an inspiratory flow of 60 L/min. Increments of \dot{V}_E were delivered serially at V_T of both 0.6 L and 1.0 L. PEEP_i, the difference of peak airway pressure between the first breath and system equilibrium (when breath-to-breath peak pressure deviated by < 1 cm H_2O), static pressure, and mean airway pressure were measured at each \dot{V}_E . The static pressure (or plateau pressure) was measured by installing a 1.0-s inspiratory pause and recording it from the "plateau pressure" display of the ventilator. PEEP_i was measured from the ventilator's pressure gauge. A one-way valve was positioned in the inspiratory limb of the circuit; following equilibration of PEEP_i in each model, the circuit was interrupted at the one-way valve, and peak airway pressure was measured on the subsequent breath. In permutations of mechanical parameters in which PEEP_i was > 4 cm H_2O , measurements were also repeated with descending ramp flow at peak inspiratory flows adjusted to match the duty cycle measured during constant flow (60 L/min). Multiple logistic regression analyses were performed (using

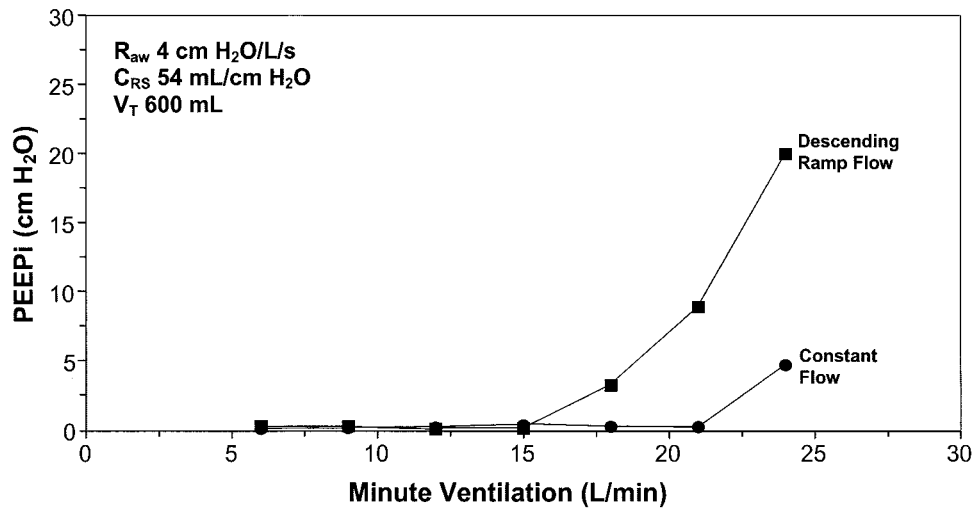


Fig. 2. Intrinsic positive end-expiratory pressure (PEEP) measured during low airway resistance (R_{aw}) and normal respiratory compliance. Even in this model of R_{aw} , which used less resistance ($R_{aw} = 4$ cm H₂O/L/s) than is observed in intubated patients, increasing intrinsic PEEP was observed at high (≥ 18 L/min) minute ventilation. C_{RS} = compliance of the respiratory system (54 mL/cm H₂O). V_T = tidal volume (600 mL).

Statistica software, StatSoft, Tulsa, Oklahoma) for mechanical variables to identify independent risk factors for dynamic hyperinflation (≥ 5 cm H₂O) and adjust for potential confounding. Variables were categorized into groups by $\dot{V}_E \leq 15$ L/min and > 15 L/min, $R_{aw} \leq 18$ cm H₂O/L/s and > 18 cm H₂O/L/s, $C_{RS} \leq 20$ mL/cm H₂O and > 20 mL/cm H₂O, and duty cycle (T_I/T_{tot}) ≤ 0.325 and > 0.325 . A p value < 0.05 signified statistical significance.

Results

Representative samples of model-derived relationships of PEEPi and \dot{V}_E are shown in Figures 2 through 9; data from all models are available on request.

Low Resistance, Normal Compliance Model

In the low resistance, normal compliance lung model ($R_{aw} = 4$ cm H₂O/L/s, $C_{RS} = 54$ mL/cm H₂O [Fig. 2]), PEEPi remained negligible until a \dot{V}_E of 24 L/min (PEEPi = 4.7 cm H₂O) during constant inspiratory flows of 60 L/min. PEEPi of nearly 9 cm H₂O developed at \dot{V}_E of 21 L/min during descending ramp inspiratory flows.

Obstructive Models

In models of mild and moderate bronchoconstriction (Figs. 3A and 3B, respectively), PEEPi developed at $\dot{V}_E \geq 15$ L/min in both models. Despite equal duty cycles (at each level of \dot{V}_E), higher PEEPi was generated during ventilation with V_T of 0.6 L, compared to 1.0 L (Fig. 4), for all $\dot{V}_E > 12$ L/min. As compliance increased (to 60 mL/cm H₂O, the maximum

compliance allowed by the model), simulating emphysema mixed with bronchoconstriction (Fig. 5), PEEPi increased, especially for the descending ramp waveform for which it was immeasurably high at $\dot{V}_E = 24$ L/min.

Restrictive Models

Much lower PEEPi were generated across various \dot{V}_E and ranges of resistance in restrictive models (≤ 30 mL/cm H₂O). In general, PEEPi remained negligible until $\dot{V}_E > 15$ L/min.

Mixed Models

PEEPi developed at $\dot{V}_E \geq 15$ L/min for combinations of elevated R_{aw} (18 cm H₂O/L/s) and decreased C_{RS} (40 mL/cm H₂O, Fig. 6) for V_T of 0.6 L, and 18 L/min for V_T of 1.0 L. Again, all PEEPi were greater for descending ramp than for constant flow regimes and higher for low V_T (0.6 L) as compared to high V_T (1.0 L) (Table 1).

When the peak inspiratory rate of the descending ramp flow regime was increased to yield equivalent duty cycles to that of the constant flow regime, PEEPi was similar for the 2 flow regimes until high \dot{V}_E , > 21 L/min, after which PEEPi was greater in the descending ramp flow model (Fig. 7).

Increments of peak airway pressure during dynamic hyperinflation, from the first delivered breath to that of system equilibrium, exceeded measured PEEPi for all models (Fig. 8). Increments of peak airway pressure were highly dependent on resistance and not affected by compliance, even at high \dot{V}_E (Fig. 9).

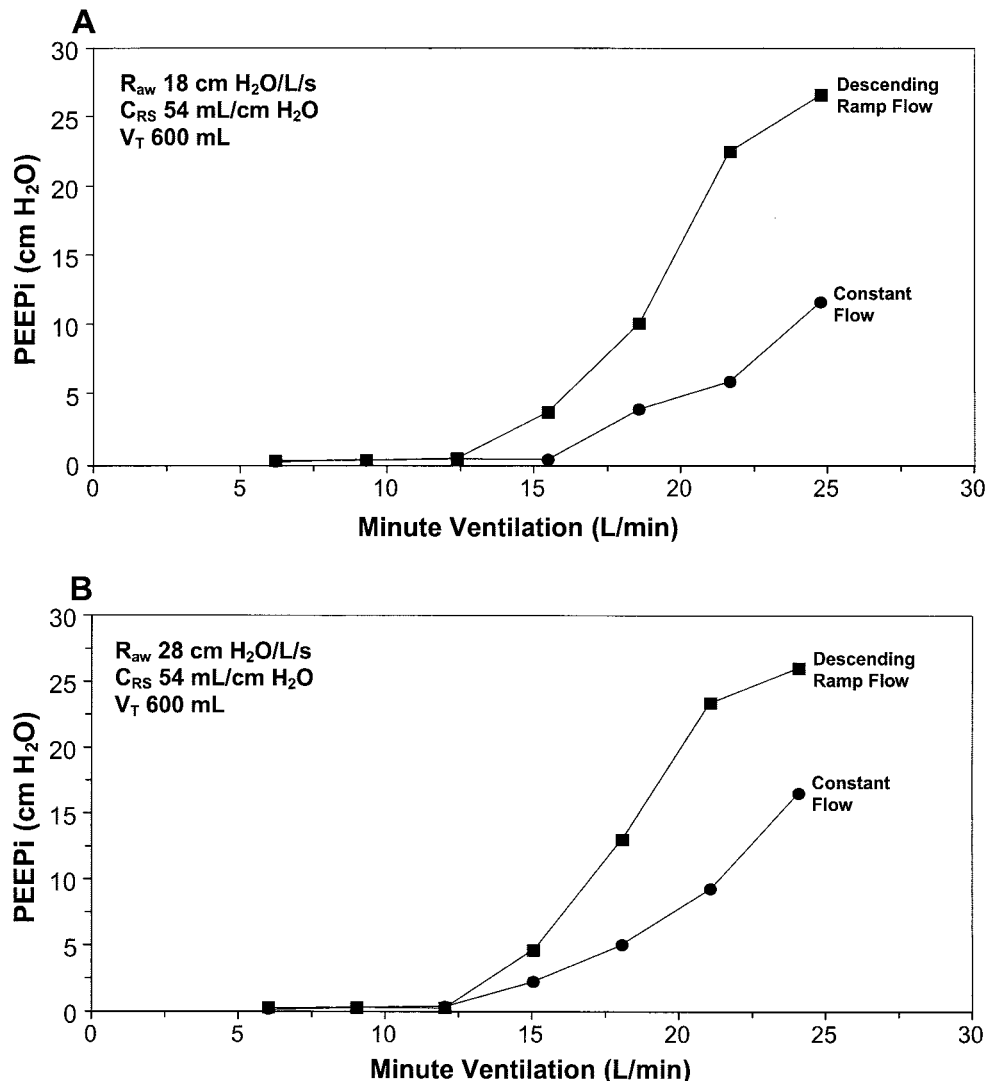


Fig. 3. Intrinsic positive end-expiratory pressure (PEEPi) during normal compliance (C_{RS} = compliance of the respiratory system [54 mL/cm H₂O]) and (A) mild (airway resistance [R_{aw}] = 18 cm H₂O/L/s) and (B) moderate obstruction (R_{aw} = 28 cm H₂O/L/s). C_{RS} = compliance of the respiratory system (54 mL/cm H₂O). V_T = tidal volume (600 mL).

In multivariable logistic regression models, \dot{V}_E ($p = 0.0003$), C_{RS} ($p = 0.002$), T_I/T_{tot} ($p = 0.002$), and R_{aw} ($p = 0.04$) were independently associated with PEEPi. The model could not be described by a single mathematical equation (ie, to predict PEEPi given \dot{V}_E , C_{RS} , T_I/T_{tot} , and R_{aw}).

Discussion

Our study demonstrates that PEEPi is positively associated with \dot{V}_E , R_{aw} , C_{RS} , and duty cycle. Although these principles could be derived from simple physical principles, these findings highlight several concepts that are useful at the bedside in routine practice. First, PEEPi occurred in many simulated mechanical conditions, even without airflow obstruction. This has been demonstrated before;⁴⁻⁷

however, our data examine this phenomenon more extensively and systematically. Across various combinations of R_{aw} and C_{RS} (and fixed peak inspiratory flow), PEEPi was always higher and occurred at lower \dot{V}_E with descending ramp than with constant flow. However, this could be attributed entirely to the duty cycle during descending ramp flow (on this ventilator), being twice that as during constant flow at a given peak flow. When peak inspiratory flows were adjusted to generate equivalent duty cycles, PEEPi was roughly the same with the descending ramp and constant waveforms. Dynamic hyperinflation was greater in all mechanical combinations when delivered with 0.6 L than with 1.0 L, despite equivalent duty cycles. Finally, increments in peak airway pressure associated with dynamic hyperinflation were not entirely related to PEEPi.

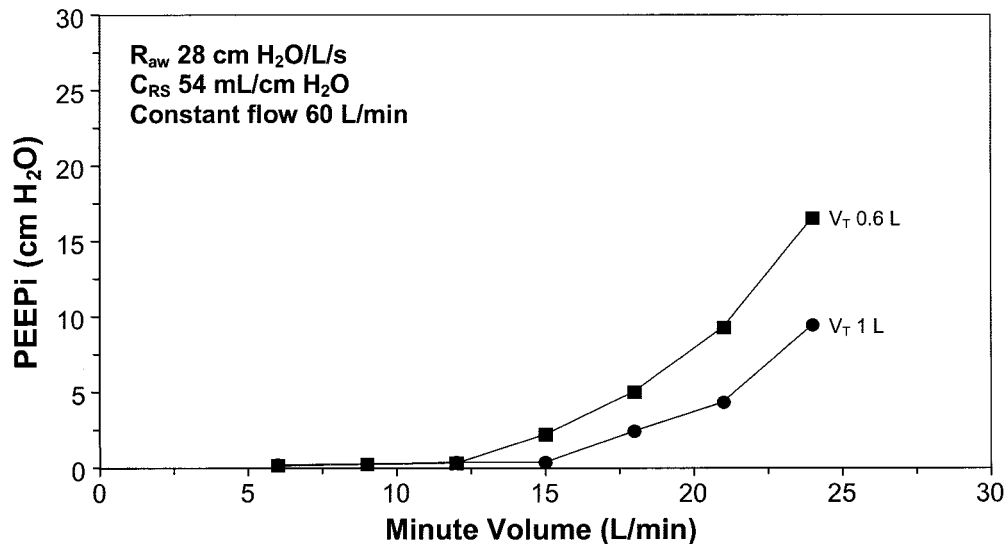


Fig. 4. Intrinsic positive end-expiratory pressure (PEEP) at tidal volume (V_T) of 0.6 versus 1.0 L, given equivalent mechanics, flows, and minute ventilation. All models had significantly higher intrinsic PEEP at 0.6 L than at 1.0 L. R_{aw} = airway resistance (28 cm H₂O/L/s). C_{RS} = compliance of the respiratory system (54 mL/cm H₂O).

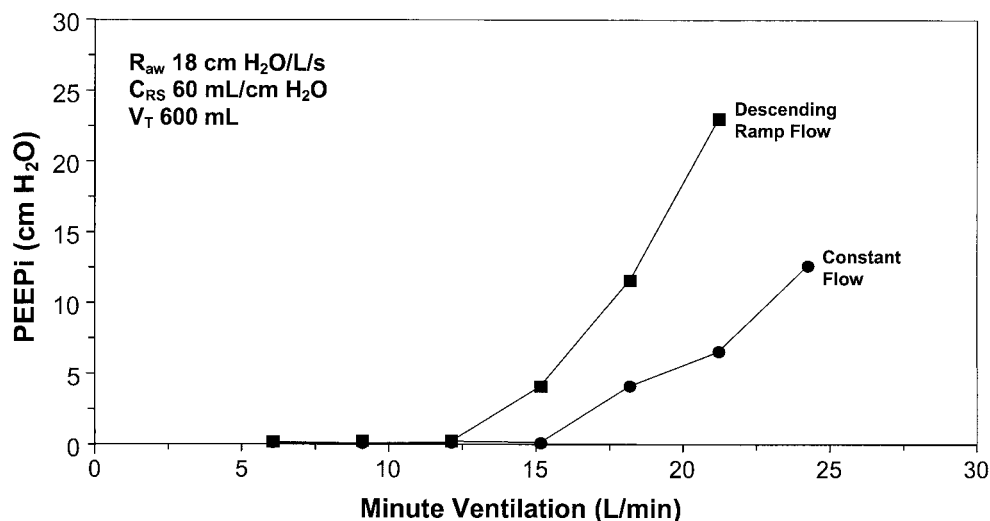


Fig. 5. Intrinsic positive end-expiratory pressure (PEEP) measured at the model's maximum compliance (C_{RS} [60 mL/cm H₂O]) and mildly elevated airway resistance (R_{aw} [18 cm H₂O/L/s]). V_T = tidal volume (600 mL).

These data are generally consistent with *in vivo* data from Tuxen and Lane¹ and Williams et al² describing dynamic hyperinflation associated with high airway resistance. In those now-classic studies, dynamic hyperinflation was measured in a cohort of patients with "severe airway obstruction" (R_{aw} was not reported but appears to be roughly 27 cm H₂O/L/s, from figures). Total exhaled volume was used to measure dynamic hyperinflation and, as in our bench study, increased dramatically at $\dot{V}_E \geq 16$ L/min. However, they also found that dynamic hyperinflation could be reduced by reducing V_T (at any given \dot{V}_E)—the opposite of our results. Indeed, they also com-

pared dynamic hyperinflation during ventilation with 0.6 L to both 1.0 L and 1.6 L (keeping \dot{V}_E fixed) in patients with asthma.¹ Accordingly, they examined similar \dot{V}_E and the same V_T combinations *in vivo* and demonstrated opposite results (ie, less dynamic hyperinflation with lower V_T /higher rates). There may have been subtle differences in duty cycle that could have contributed to their observations. Our findings are consistent with those of de Durante et al,⁸ who demonstrated 4 times greater PEEPi (5.8 vs 1.4 cm H₂O) among patients with acute respiratory distress syndrome ventilated with 6–8 mL/kg versus 10–12 mL/kg (again, keeping \dot{V}_E and T_I/T_{tot} fixed). One possible expla-

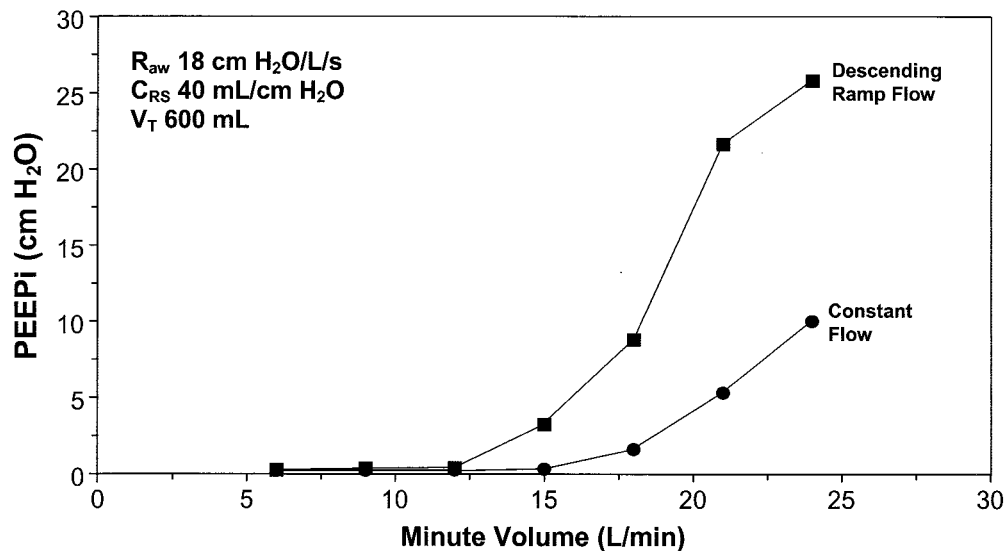


Fig. 6. Intrinsic positive end-expiratory pressure (PEEP) with mild obstruction and mild restriction. R_{aw} = airway resistance (18 cm H₂O/L/s). C_{RS} = compliance of the respiratory system (40 mL/cm H₂O). V_T = tidal volume (600 mL).

Table 1. Samples of Intrinsic Positive End-Expiratory Pressure*

\dot{V}_E (L/min)	R_{aw} (cm H ₂ O/L/s)	T_I/T_{tot}	PEEPi (cm H ₂ O)	
			V_T 600 mL	V_T 1000 mL
6	18	0.1	0.1	0.1
9	18	0.15	0.2	0.2
12	18	0.2	0.2	0.1
15	18	0.25	0.2	0.2
18	18	0.3	3.9	0.3
21	18	0.35	5.9	2.7
24	18	0.4	11.6	6.6
6	28	0.1	0.2	0.2
9	28	0.15	0.3	0.3
12	28	0.2	0.3	0.4
15	28	0.25	2.2	0.4
18	28	0.3	5	2.4
21	28	0.35	9.3	4.3
24	28	0.4	16.5	9.4

*Values were generated at constant duty cycles (T_I/T_{tot}), with tidal volumes of 600 mL or 1000 mL, at increments of minute volume (\dot{V}_E) and respiratory-system compliance (C_{RS}) of 54 mL/cm H₂O. Note that PEEPi is systematically higher for tidal volume (V_T) of 600 mL than 1000 mL. This was the pattern across various mechanical permutations.

nation to explain lower dynamic hyperinflation at higher V_T is that expiratory flow is not linear with respect to time. The higher V_T could yield a higher elastic recoil pressure and greater flows in early (eg, during exhalation of the first 400 mL of a 1,000 mL V_T) than in late (ie, during exhalation of the last 600 mL) exhalation.

We also observed that peak airway pressure increased *more* than the observed PEEPi from the first delivered

breath to system equilibrium. Since inspiratory flow and V_T were not changed in the 5–10 breaths it took to reach equilibrium, it is possible that dynamic resistance or compliance changed in the system, yielding a larger increment of total (peak) pressure than of PEEPi. This could also represent some measurement artifact (vide infra). Irrespective of the mechanism, increments of peak airway pressure during hyperinflation appear to overestimate PEEPi.

This study has a number of notable limitations. First, the lung model had set increments of resistance, one of which was subphysiologic (yielding a total system resistance of 4 cm H₂O/L/s, which is well below the values noted in intubated adults). Accordingly, all in vitro experiments were limited by the increments of resistance provided by the model manufacturer.⁹ Second, calculations of system compliance and resistance depended on the accuracy of sensor measurements and ventilator software. Idiosyncrasies of the ventilator (eg, exhalatory valve resistance) may also affect dynamic hyperinflation,¹⁰ especially at very high respiratory frequencies. Expiratory resistance, which is more likely to affect dynamic hyperinflation, was not measured. Indeed, the specific relationships of dynamic hyperinflation and respiratory mechanics noted in this study may only apply to the single ventilator we tested. Nonetheless, the exponential increase in PEEPi (with increasing \dot{V}_E), in all models, is unlikely to be ventilator-specific. Although the inflection point may vary with ventilator characteristics, the general relationships are a function of mechanics and duty cycle. Obviously, these data do not reflect in vivo conditions. First, we could study only model-determined increments of compliance and resistance (eg, the highest compliance of the model was 60 mL/cm H₂O). Also, the degree to which this single-compartment model mimics

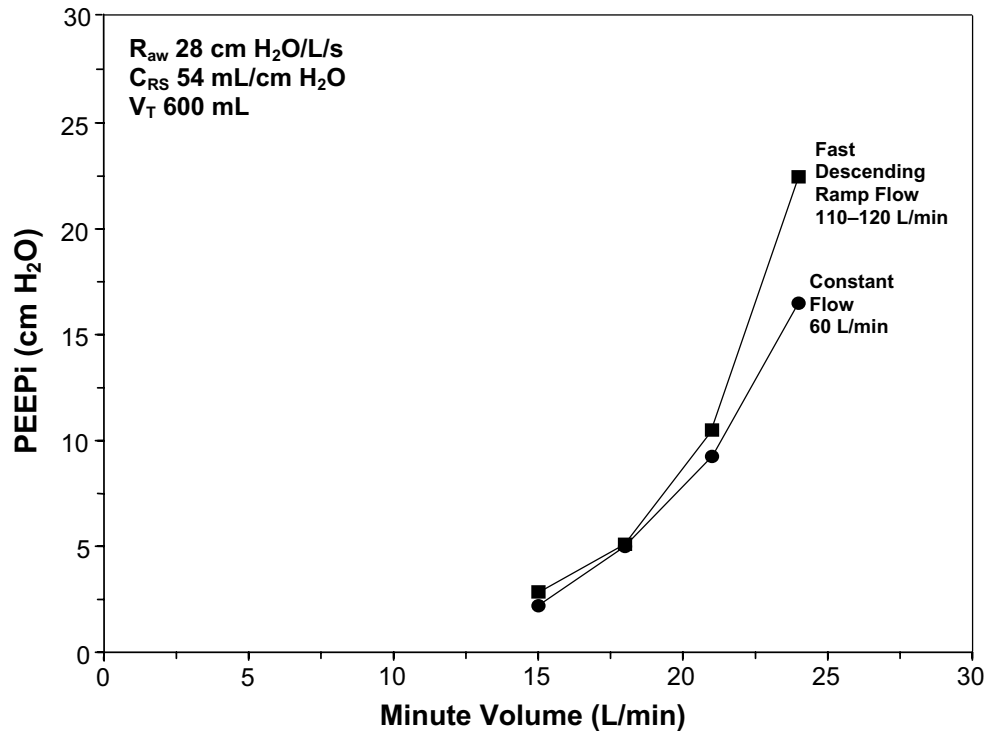


Fig. 7. Intrinsic positive end-expiratory pressure (PEEPi) with constant and descending ramp inspiratory flow and equal duty cycles. R_{aw} = airway resistance (28 cm H₂O/L/s). C_{RS} = compliance of the respiratory system (54 mL/cm H₂O). V_T = tidal volume (600 mL).

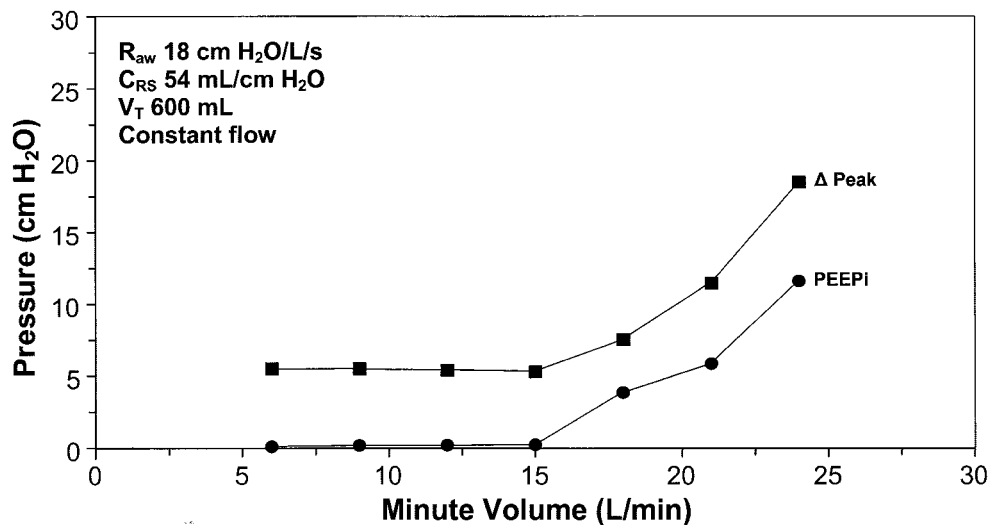


Fig. 8. Intrinsic positive end-expiratory pressure (PEEPi) and increments of peak inspiratory pressure (Δ peak) associated with dynamic hyperinflation. Note that the increment of peak airway pressure consistently exceeded that of PEEPi, irrespective of minute volume. Similar results were reproduced across various models of resistance and compliance. R_{aw} = airway resistance (18 cm H₂O/L/s). C_{RS} = compliance of the respiratory system (54 mL/cm H₂O). V_T = tidal volume (600 mL).

complex lung physiology, with millions of respiratory units, each with discrete time constants, is uncertain. Previous data have demonstrated that local heterogeneity of time constants is likely to promote underestimation of PEEPi.¹¹ To determine whether the model accurately predicts PEEPi in a similar variety of clinical scenarios would require measurements

in hundreds of mechanically ventilated (and muscle relaxed) patients, which was not possible for this study. Nonetheless, the model demonstrates principles that may be of clinical utility in routine practice.

First, clinicians should be aware that dynamic hyperinflation, and its associated pulmonary and hemodynamic

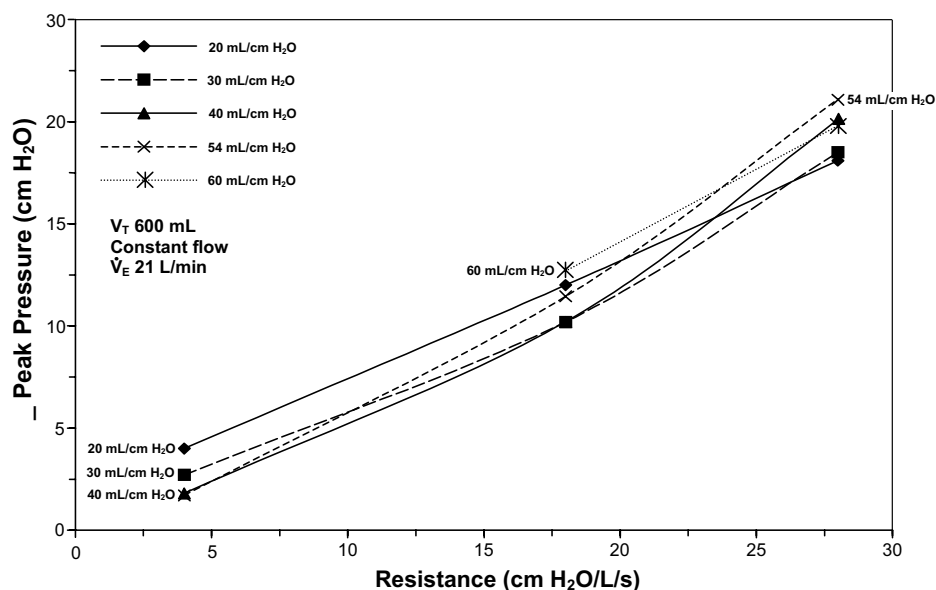


Fig. 9. Increments of peak airway pressure during dynamic hyperinflation, across various models of compliance, plotted as a function of airway resistance. Note that increments in peak airway pressure during dynamic hyperinflation were independent of respiratory compliance. V_T = tidal volume (600 mL). \dot{V}_E = minute ventilation.

risks,^{1,2,7} may contribute to a number of diverse clinical scenarios. For example, patients who are being resuscitated during shock or who have substantial acidosis usually hyperventilate to compensate. Since dynamic hyperinflation increases exponentially at high \dot{V}_E , its effect would be to decrease the gradient for venous return, thereby exacerbating the hypoperfused state (ie, worsening the acidosis unto a “vicious cycle”).⁷ Dynamic hyperinflation could likewise contribute to hypotension and/or risk of barotrauma of patients who become transiently tachypneic due to pain, anxiety, or other causes of patient-ventilator asynchrony. Our findings also serve a cautionary function, since a not-uncommon approach to high peak airway pressure is to reduce inspiratory flow or switch to descending ramp waveforms, both of which increase duty cycle, thereby promoting dynamic hyperinflation. A high peak airway pressure is not necessarily dangerous unless reflective of dangerous transalveolar pressures, which, with obvious limitations, may be reflected best by the plateau pressure.^{12,13} Efforts to reduce peak pressure, due to increased airway resistance—indicated by a high peak-plateau gradient—at the expense of increased duty cycle can be harmful or even life-threatening.¹⁴ A previous study suggested that 18 L/min is the threshold at which PEEP_i is produced.⁶ Our data suggest that physicians and respiratory therapists should consider the possibility that dynamic hyperinflation is present when \dot{V}_E much exceeds 15 L/min, especially when coupled to long duty cycles, elevated resistance, and/or elevated compliance. When substantial levels of PEEP_i are detected, caregivers can examine the degree to which it is deleterious to any given patient’s cardiopulmonary profile

and respond (or not) accordingly.⁷ However, since muscle relaxants are infrequently used because of associated neuromuscular complications, detection of PEEP_i is difficult if not impossible. Accordingly, clinicians must suspect (and combat) PEEP_i in those clinical circumstances (see above) in which it is expected.

Conclusions

This study demonstrates that PEEP_i is generated by complex relationships of \dot{V}_E , duty cycle, resistance, and elastance. In the permutations we examined, dynamic hyperinflation was greater during ventilation with 0.6 L than with 1.0 L at each combination of \dot{V}_E , duty cycle, resistance, and compliance. Although the general relationships demonstrated in this model can be predicted by elementary physiologic principles, the data illustrate that PEEP_i is likely to be generated under a variety of conditions in which it is not ordinarily suspected or detected.

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