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Research Article

Development and internal verification of an open-source mechanical ventilation simulator for teaching ventilatory trade-offs in pediatric critical asthma: A respiratory-care education tool

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Abstract

Background

In mechanically ventilated children with critical asthma, increasing minute ventilation can improve carbon dioxide clearance but may shorten expiratory time and worsen gas trapping, intrinsic positive end-expiratory pressure (auto-PEEP), airway-pressure exposure, and hemodynamic burden. Because invasive ventilation is uncommon, respiratory therapists and other critical-care learners have limited opportunities to observe these trade-offs at the bedside.

Methods

We developed an open-source, computer-based mechanical ventilation simulator for teaching the physiological consequences of ventilator adjustments in pediatric critical asthma. Internal verification included automated physiological-direction checks, a same-patient respiratory-rate sweep, predefined teaching scenarios, one-at-a-time sensitivity analyses, and reproducibility testing. No patient data were used for clinical validation.

Results

In a fixed virtual patient with severe airflow obstruction, the simulated PaCO2 response improved as respiratory rate increased to approximately 22 breaths/min and then worsened at higher rates as expiratory time shortened. Auto-PEEP and plateau pressure increased progressively, while the mean arterial pressure surrogate declined at higher rates. Teaching scenarios and sensitivity analyses showed the expected directional behaviour.

Discussion

The simulator makes clinically relevant ventilatory trade-offs visible while keeping its assumptions open to inspection. The direction of the principal responses is consistent with established obstructive physiology, but absolute values and numerical thresholds remain educational surrogates rather than patient predictions.

Conclusion

This internally verified simulator provides a faculty-guided environment for teaching ventilatory trade-offs in pediatric critical asthma. It is an educational tool and has not been clinically validated against patient data.

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INTRODUCTION

Pediatric critical asthma is an important cause of admission to the pediatric intensive care unit (PICU), but progression to invasive mechanical ventilation is uncommon.[1-4] In a large multicenter database of children admitted to North American PICUs for asthma between 2009 and 2019, intubation occurred in 4.6% of admissions and decreased from 6.9% to 3.4% over the study period.[4] Most children can therefore be managed without invasive ventilation, but those who require intubation present a particularly challenging combination of severe airflow obstruction, dynamic hyperinflation, and potential cardiovascular compromise. For respiratory therapists and other members of the critical-care team, the relative rarity of these cases also limits opportunities to gain repeated bedside experience with their ventilatory management.

Mechanical ventilation in severe asthma requires balancing carbon dioxide clearance against adequate expiratory time and limitation of dynamic hyperinflation.[5-14] When airflow obstruction is severe, expiration may remain incomplete before the next breath begins. Increasing respiratory rate in an attempt to increase minute ventilation and lower PaCO2 can further shorten expiratory time, promoting gas trapping, intrinsic positive end-expiratory pressure (auto-PEEP), higher airway pressures, and impaired venous return.[5-9] Consequently, permissive hypercapnia may be preferable to pursuing rapid normalization of PaCO2 when the latter requires ventilator settings that worsen hyperinflation or pressure exposure.[5,6] These competing effects are central to the clinical management of mechanically ventilated severe asthma but can be difficult for learners to visualize simultaneously.

This educational challenge extends beyond respiratory therapists in clinical practice. Respiratory therapy students and trainees, medical residents and fellows, and other clinicians learning pediatric critical care, emergency medicine, or anesthesiology may encounter invasively ventilated pediatric asthma only infrequently. Some clinically important signals can also be counterintuitive. For example, a higher respiratory rate may initially improve carbon dioxide clearance but subsequently worsen it as expiratory time becomes inadequate, while a low end-tidal CO2 value may not reliably indicate adequate ventilation in the presence of marked ventilation heterogeneity or increased dead space. A teaching environment in which these relationships can be repeatedly explored without patient risk may therefore complement bedside and case-based education.

Simulation-based education provides an opportunity to examine rare, high-risk clinical situations through repeated practice, structured discussion, and feedback.[15-21] Computer-based simulation has the additional advantage of allowing learners to change individual ventilator settings and immediately examine their expected physiological consequences. An open-source simulator can also make the assumptions underlying those responses available for review and modification, rather than presenting them as unexplained outputs.

We therefore developed an open-source, computer-based mechanical ventilation simulator for teaching ventilatory trade-offs in pediatric critical asthma. The simulator was designed to demonstrate how changes in respiratory rate, expiratory time, tidal volume, airway obstruction, and positive end-expiratory pressure can influence gas trapping, auto-PEEP, airway pressures, carbon dioxide clearance, and hemodynamic consequences. The aim of this report is to describe the simulator, its physiological and educational design, and the internal verification used to determine whether its responses follow the expected direction of established obstructive respiratory physiology.

MATERIALS AND METHODS

Ethics approval was not required because this study did not involve human participants, patient data, biological material, or animal research.

SIMULATOR OVERVIEW AND INTENDED EDUCATIONAL USE

We developed an open-source, computer-based mechanical ventilation simulator designed to support faculty-guided teaching of ventilatory management in pediatric critical asthma. The simulator allows users to modify patient characteristics and ventilator settings and to observe the resulting changes in respiratory mechanics, gas trapping, intrinsic positive end-expiratory pressure (auto-PEEP), airway pressures, carbon dioxide clearance, and a simplified hemodynamic response.

The simulator was designed around the clinical problem of severe airflow obstruction, in which changes intended to improve ventilation may produce competing physiological effects. For example, increasing respiratory rate may initially improve carbon dioxide clearance but may also shorten expiratory time and promote dynamic hyperinflation when lung emptying is incomplete. The software therefore displays several physiological outputs together so that learners can explore these trade-offs rather than focus on a single ventilatory target.

The current scenario library and educational framework were developed for pediatric critical asthma. The Streamlit interface restricts age inputs to 1–18 years and weight inputs to 8–80 kg, whereas the underlying scenario schema permits ages 0–25 years and weights greater than 0 up to 150 kg. These technical limits do not establish clinical validity across those age or weight ranges, nor do they imply that wheeze in infancy represents diagnosed asthma. The scenarios evaluated in this report are pediatric, and the primary reference patient used for internal verification is a child. The same general software architecture could potentially be adapted to other age groups or obstructive respiratory conditions, but such use would require population-specific scenarios, assumptions, and separate evaluation.

The simulator is intended for education rather than selection of ventilator settings for individual patients. No patient data were used to calibrate or clinically validate its numerical outputs. These scope limitations are stated here

Table 1. Terminology used in this report

Term	General meaning	Meaning in this study
Simulator	A system that allows users to explore the behavior of a represented clinical or physiological situation	The complete computer-based educational application through which users modify patient or ventilator settings and observe simulated responses
Physiological model	A mathematical or conceptual representation of physiological relationships	The equations and assumptions linking expiratory time, airway obstruction, gas trapping, auto-PEEP, airway pressures, ventilation, and hemodynamic effects
Software	The computer code that implements the simulator and its functions	The open-source code package containing the physiology engine, scenario files, interface, analysis functions, and automated tests
Verification	Assessment of whether a system behaves as specified by its design and implementation	Internal checks that the software calculates the programmed relationships consistently and changes in the expected physiological direction when inputs are modified
Calibration	Selection or adjustment of model parameters to produce a defined or plausible response	Selection of fixed educational coefficients used to produce plausible teaching behavior. These coefficients were not fitted to patient data and are not instructor-selected ventilator settings unless explicitly exposed as simulator inputs
Clinical validation	Assessment of how closely model outputs agree with observations from real patients	Comparison of simulated outputs with measured patient data. This was not performed in the present study

to define the intended use of the software and are considered further in the Discussion.

TERMINOLOGY AND SCOPE

To maintain consistent terminology throughout the manuscript, we use simulator to refer to the complete educational application, physiological model to refer to the mathematical representation of respiratory physiology within the simulator, and software to refer to the underlying code and computational implementation. We also distinguish internal verification from calibration and clinical validation, because these processes answer different questions (Table 1).

DESIGN AND REPORTING PRINCIPLES

The simulator was developed as an educational tool in which interpretability and reproducibility were prioritized over physiological complexity. We followed established principles for reproducible computational research and software citation.[22,23] Patient characteristics and ventilator settings are stored in human-readable scenario files, and the software includes an interactive user interface, predefined teaching scenarios, automated verification tests, and functions for comparing simulated responses across different settings.

The software version evaluated in this study was version 1.8.1. The archived maintenance snapshot, v1.8.1-post1, included updates to scenario metadata, documentation, release testing, and reproducibility files but did not alter the physiological calculations. Simulation outputs, figures, and the reported numerical results can be regenerated from the archived software release. Detailed equations, fixed educational coefficients, display limits, scenario parameters, and software-verification procedures are provided in the Supplementary Material.

REFERENCE VIRTUAL PATIENT AND VENTILATOR SETTINGS

Internal verification of the main ventilatory trade-off was performed using the same virtual patient while changing respiratory rate (respiratory-rate sweep) and holding the other patient and ventilator settings constant. The reference scenario represented a passive 8-year-old child weighing 25 kg with severe airflow obstruction. Volume-controlled ventilation was used with a tidal volume of 6.5 mL/kg, external PEEP of 3 cmH2O, and an inspiratory-to-expiratory ratio of 1:3. Respiratory rate was progressively varied from 6 to 40 breaths/min. The 1:3 I:E ratio was retained as a fixed experimental input rather than a recommended clinical setting; its modelled inspiratory time decreases from 2.5 s at 6 breaths/min to 0.375 s at 40 breaths/min.

Respiratory-system compliance was set at 1.0 mL/cmH2O/kg, corresponding to a total baseline compliance of 25 mL/cmH2O in the reference patient. Inspiratory airway resistance was 48 cmH2O·s/L, the patient was passive, and no bronchodilator or adjunct modifiers were active. The complete reference-patient parameter set is provided in **Supplementary Table S1.**

The respiratory-rate sweep was chosen to test a predefined physiological expectation. At lower rates, increasing respiratory rate was expected to increase alveolar ventilation and improve carbon dioxide clearance. As respiratory rate increased further, the resulting reduction in expiratory time was expected to promote incomplete lung emptying, gas trapping, increasing auto-PEEP, and eventually worsening carbon dioxide clearance despite the higher set rate. All patient and ventilator inputs other than respiratory rate were kept unchanged during this analysis.

REPRESENTATION OF OBSTRUCTIVE RESPIRATORY PHYSIOLOGY

The simulator represents severe airflow obstruction by linking expiratory time with airway resistance and respiratory-system compliance. Together, resistance and compliance determine how rapidly the respiratory system can empty during expiration. When expiratory time is sufficiently long, most of the delivered tidal volume can leave the lungs before the next breath. When expiratory time becomes too short, a progressively larger volume remains trapped at end expiration.

This retained volume is used to estimate dynamic hyperinflation and intrinsic positive end-expiratory pressure (auto-PEEP). As hyperinflation increases, the model also applies a progressive reduction in effective respiratory-system compliance to represent the increasing mechanical cost of lung overdistension. Plateau pressure therefore reflects external PEEP, auto-PEEP, and the elastic pressure required to deliver the tidal volume. It is calculated as external PEEP plus modelled auto-PEEP plus delivered tidal volume divided by effective compliance, not measured through a simulated end-inspiratory occlusion. The stored inspiratory-pause field is 0 s and is not incorporated into inspiratory/expiratory timing in version 1.8.1. During volume-controlled ventilation, peak airway pressure additionally reflects the pressure required to overcome inspiratory airway resistance. This distinction allows the simulator to demonstrate the clinically important situation in which peak pressure may be high because of severe airway resistance while plateau pressure remains considerably lower.

GAS EXCHANGE AND HEMODYNAMIC RESPONSE

Carbon dioxide clearance is based on effective alveolar ventilation rather than set minute ventilation alone. The simulator accounts for anatomical dead space and applies additional reductions in effective ventilation when obstruction, gas trapping, ventilation heterogeneity, or inadequate expiratory time become more severe. Consequently, increasing respiratory rate can initially increase effective ventilation but may eventually become counterproductive if the accompanying reduction in expiratory time causes substantial gas trapping.

The simulated PaCO2 response is calculated from carbon dioxide production and effective alveolar ventilation using the standard alveolar-ventilation relationship. The corresponding pH is estimated using an acute respiratory acid-base relationship. End-tidal CO2 is estimated from PaCO2 and the modelled alveolar dead-space fraction. In mechanically ventilated pediatric patients, the PaCO2-to-end-tidal-CO2 gradient increases as physiologic dead space increases[24]; the simulator uses this relationship to create teaching scenarios in which end-tidal CO2 may underestimate the severity of hypercapnia when modelled dead space is high.

The simulator also provides a simplified mean arterial pressure (MAP) surrogate to make the cardiovascular consequences of dynamic hyperinflation visible during teaching. This response incorporates the virtual patient's baseline pressure and preload together with the effects of increasing auto-PEEP, high plateau pressure, acidosis, and excessive applied PEEP. It is intended to represent the expected direction of hemodynamic change rather than to predict an individual patient's arterial pressure.

APPLIED PEEP AND INSPIRATORY THRESHOLD LOAD

Applied PEEP is represented using a simplified counterbalancing-PEEP teaching approximation. When applied PEEP remains at or below the simulated intrinsic PEEP, the model treats it as counterbalancing the pressure that must be overcome at the beginning of inspiration rather than as an additional hemodynamic burden. In spontaneously breathing scenarios, this is represented by a progressive reduction in the inspiratory threshold-work surrogate as applied PEEP approaches intrinsic PEEP.

When applied PEEP exceeds intrinsic PEEP, the excess is treated as additional pressure exposure and progressively contributes to the simulated hemodynamic penalty. This approximation was included to demonstrate the clinical concept of counterbalancing PEEP in airflow obstruction; it does not reproduce the full interaction among airway closure, flow limitation, lung volume, and applied PEEP in an individual patient. Detailed mathematical relationships, fixed educational coefficients, parameter limits, and the complete computational implementation of these pathways are provided in the Supplementary Material.

INTERNAL VERIFICATION PROCEDURES

Internal verification was performed to determine whether the simulator implemented the programmed relationships consistently and responded in the expected physiological direction when selected inputs were changed. Verification was not intended to determine whether the absolute numerical outputs accurately predict measurements in individual patients.

The verification process included four complementary components. First, automated software checks assessed input validity, calculation consistency, interface operation, and release integrity. Second, predefined physiological direction checks examined whether changing a single relevant input produced the expected response. These checks included confirming that shorter expiratory time or higher respiratory rate increased gas trapping and auto-PEEP under severe obstruction, that larger tidal volumes increased pressure exposure, that lower respiratory-system compliance increased plateau pressure, and that higher airway resistance increased peak airway pressure.

Third, the principal physiological verification experiment used the fixed reference patient described above. Respiratory rate was increased from 6 to 40 breaths/min while all other patient and ventilator settings were held constant. We predefined the expected response as an initial improvement in carbon dioxide clearance followed, at sufficiently high respiratory rates, by worsening gas trapping, increasing auto-PEEP, and loss of the initial carbon dioxide benefit as expiratory time became progressively shorter.

Fourth, local one-at-a-time sensitivity analyses were performed by varying selected patient and ventilator characteristics around the reference scenario while leaving the remaining inputs unchanged. Variables examined included airway resistance, respiratory-system compliance, ventilation heterogeneity, tidal volume, respiratory rate, and inspiratory-to-expiratory ratio. These analyses were used to determine whether the direction of change remained consistent with the physiological relationships represented in the simulator.

Reproducibility was additionally assessed by repeating calculations with different simulation time steps and by comparing generated outputs with a stored reference snapshot. In this deterministic model, the reference scenario and three teaching scenarios were each assessed at time steps of 0.5, 1, 2, and 5 s (16 scenario–time-step configurations). For maximum auto-PEEP, plateau pressure, and PaCO2, the prespecified acceptance criterion was a relative spread below 5% across time steps. Separately, five archived scenario snapshots checked four summary outputs each against tolerances of the greater of 0.5 units or 3% of the stored reference value. These automated tests assess numerical stability and implementation consistency, not repeated patient measurements or clinical validation. The detailed automated test architecture, acceptance criteria, and numerical tolerances are provided in the Supplementary Material.

TEACHING SCENARIOS

In addition to the controlled verification procedures, the simulator includes predefined teaching scenarios designed to illustrate recognizable clinical problems in obstructive mechanical ventilation. Unlike the respiratory-rate sweep, these scenarios are not controlled single-variable experiments; several patient or ventilator settings may differ simultaneously to create the intended teaching situation. They are therefore used to demonstrate clinical concepts rather than to establish quantitative relationships between individual variables.

Controlled hypoventilation. This scenario represents a strategy using a relatively low respiratory rate and adequate expiratory time in a severely obstructed patient. It is intended to demonstrate the acceptance of hypercapnia in exchange for reduced gas trapping and lower plateau-pressure exposure.

High respiratory rate. This scenario represents an attempt to increase minute ventilation by raising respiratory rate. The shorter expiratory time promotes incomplete lung emptying and increasing auto-PEEP, illustrating why a higher set minute ventilation does not necessarily produce better effective carbon dioxide clearance in severe airflow obstruction.

Excessive tidal volume. This scenario uses a larger tidal volume while preserving sufficient expiratory time. It is intended to separate the consequences of excessive volume and pressure exposure from those caused primarily by dynamic hyperinflation. In this scenario, carbon dioxide clearance may improve while plateau pressure rises into an undesirable range.

Excessive applied PEEP. This passive-patient scenario illustrates the simplified representation of excessive external PEEP. Applied PEEP above the simulated intrinsic PEEP increases pressure exposure and contributes to the hemodynamic penalty. The scenario is intended for discussion of the potential cost of excessive PEEP rather than for determining an optimal bedside PEEP value.

Spontaneous breathing and counterbalancing PEEP. This scenario represents a spontaneously breathing patient with intrinsic PEEP. Applied PEEP is progressively increased to illustrate the modeled reduction in inspiratory threshold work as external PEEP approaches intrinsic PEEP and the appearance of an additional hemodynamic penalty when applied PEEP exceeds intrinsic PEEP.

Silent-chest and low end-tidal CO2 pitfall. This scenario represents very severe airflow obstruction with marked impairment of effective ventilation and increased modeled alveolar dead space. It was designed to demonstrate that a low end-tidal CO2 value can appear falsely reassuring despite substantial simulated hypercapnia when communication between ventilated lung units and the airway is severely impaired.

Post-intubation collapse demonstration. A separate deliberately unsafe configuration represents the combination of severe obstruction and ventilator settings producing extreme gas trapping, pressure exposure, hypercapnia, and hemodynamic compromise. When predefined safety limits are exceeded, the simulator displays capped values and an unsafe-physiology warning. This scenario is therefore an unsafe-configuration demonstration, not a quantitative prediction of post-intubation physiology.

Optional pediatric pharmacotherapy modifiers are available in the software as separate demonstration features but were not part of the primary physiological verification reported here; their implementation is described in the Supplementary Material.

SOFTWARE AVAILABILITY AND REPRODUCIBILITY

The simulator is open-source and distributed under the Apache License 2.0. The software version evaluated in this study was version 1.8.1, with the archived maintenance snapshot identified as v1.8.1-post1. This maintenance release updated scenario metadata, documentation, release testing, and reproducibility files without changing the physiological calculations used for the analyses reported here.[25]

The software package includes the physiological model, predefined scenario files, an interactive user interface, comparison and sensitivity functions, and automated verification tests. Scenario inputs are stored in human-readable files, and the numerical outputs and figures reported in this manuscript can be regenerated from the archived software release. The GitHub repository and Zenodo archived release are identified in the Code availability section. Detailed equations, fixed educational coefficients, scenario parameters, verification criteria, numerical tolerances, and additional reproducibility information are provided in the Supplementary Material.

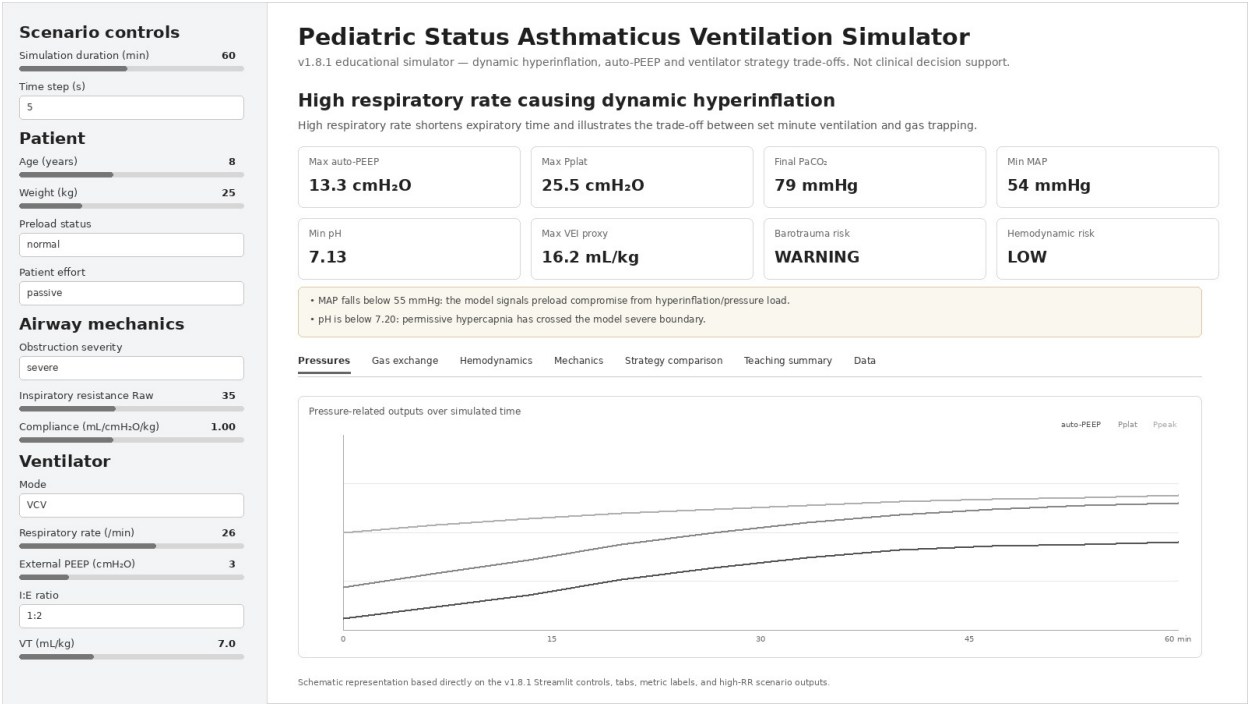

Figure 1. Schematic representation of the simulator user interface

The figure reproduces the principal v1.8.1 Streamlit interface elements—patient and ventilator controls, displayed metrics, teaching alerts, and output tabs—using values generated by the high-respiratory-rate teaching scenario. It is provided to illustrate how the simulator presents competing respiratory-mechanics, gas-exchange, and hemodynamic outputs during a faculty-guided session.

RESULTS

SIMULATOR INTERFACE

The simulator provides an interactive interface through which users can select patient characteristics and ventilator settings and observe the resulting changes in respiratory mechanics, gas exchange, and hemodynamic outputs. The principal displayed variables include auto-PEEP, plateau and peak airway pressures, PaCO2, end-tidal CO2, pH, and the MAP surrogate. **Figure 1** provides a schematic view of the v1.8.1 interface based directly on its implemented controls, tabs, metric labels, and scenario outputs.

SAME-PATIENT RESPIRATORY-RATE SWEEP

In the fixed 8-year-old, 25-kg reference patient with severe airflow obstruction, increasing respiratory rate produced the predefined non-linear carbon dioxide response. PaCO2 decreased as respiratory rate increased from 6 to 22 breaths/min, reaching its lowest simulated value at approximately 22 breaths/min. With further increases in respiratory rate, PaCO2 rose again as progressively shorter expiratory time was accompanied by greater gas trapping.

Over the same sweep, auto-PEEP and plateau pressure increased progressively with respiratory rate. Auto-PEEP increased from 0.7 cmH2O at 6 breaths/min to 29.3 cmH2O at 40 breaths/min, while plateau pressure increased from 10.2 to 43.3 cmH2O. The MAP surrogate showed a small initial improvement at low respiratory rates followed by a progressive decline at higher rates. At 40 breaths/min, PaCO2 reached the simulator's upper display limit of 180 mmHg; this capped value indicates that the simulated configuration exceeded the model's predefined display range rather than representing a quantitative clinical prediction. The complete respiratory-rate sweep is shown in **Figure 2**, with selected values summarized in **Table 2**.

TEACHING SCENARIOS

The teaching scenarios differed in several inputs and illustrate physiological trade-offs rather than controlled single-variable comparisons. Their settings and representative outputs are summarized in **Supplementary Tables S2** and **S3**, respectively.

Controlled hypoventilation maintained lower auto-PEEP and plateau pressure while accepting hypercapnia; the high-rate scenario increased auto-PEEP without improving carbon dioxide clearance.

The excessive-tidal-volume scenario increased plateau pressure despite a comparatively low PaCO2, whereas excessive applied PEEP increased the simulated hemodynamic burden.

The silent-chest scenario illustrated a potentially misleading end-tidal CO2 value of approximately 14 mmHg despite a simulated PaCO2 of approximately 105 mmHg (**Figure 3**).

Complete representative scenario outputs are provided in **Supplementary Table S3.**

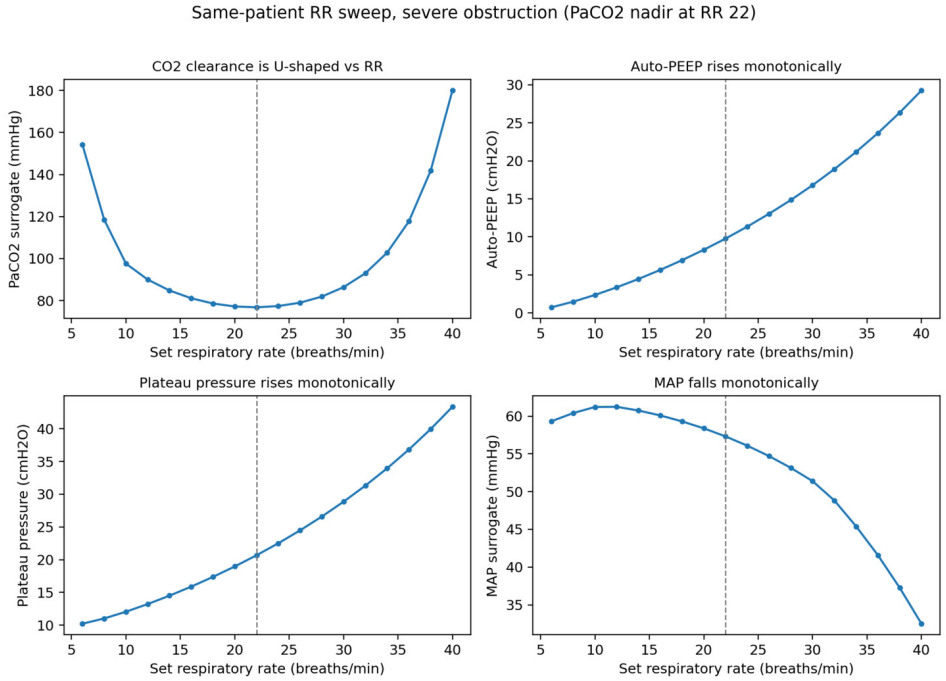

Figure 2. Effect of respiratory rate in the same severely obstructed virtual patient

Respiratory rate was increased from 6 to 40 breaths/min while all other patient and ventilator settings were held constant. Increasing respiratory rate initially improved the PaCO2 response, with the lowest value occurring near 22 breaths/min. At higher rates, shorter expiratory time was associated with increasing auto-PEEP and plateau pressure and a subsequent worsening of the PaCO2 response. The MAP surrogate declined progressively at higher respiratory rates. Values are simulated educational outputs and should be interpreted according to their direction of change rather than as predicted patient measurements.

Abbreviations: auto-PEEP, intrinsic positive end-expiratory pressure; MAP, mean arterial pressure; PaCO2, arterial partial pressure of carbon dioxide; RR, respiratory rate.

Table 2. Same-patient respiratory-rate sweep. All numerical values are simulated educational outputs and are not predicted patient measurements

Respiratory rate (breaths/min)	Auto-PEEP (cmH2O)	Plateau pressure (cmH2O)	PaCO2 (mmHg)	MAP surrogate (mmHg)
6	0.7	10.2	154.2	59.3
12	3.4	13.2	90.0	61.2
18	6.9	17.4	78.6	59.3
22	9.8	20.7	76.8	57.3
28	14.9	26.6	81.9	53.1
34	21.2	34.0	102.8	45.4
40	29.3	43.3	180.0*	32.5

*Upper display limit of the simulator.

Abbreviations: auto-PEEP, intrinsic positive end-expiratory pressure; MAP, mean arterial pressure; PaCO2, arterial partial pressure of carbon dioxide.

APPLIED PEEP AND INSPIRATORY THRESHOLD LOAD

In the spontaneous PEEP-titration scenario, applied PEEP reduced the inspiratory threshold-work surrogate as it approached intrinsic PEEP; above intrinsic PEEP, an additional MAP-surrogate penalty appeared. This is an educational approximation, not a patient-specific PEEP prescription.

SENSITIVITY ANALYSES

Local one-at-a-time sensitivity analyses produced the expected directional responses: auto-PEEP varied most with respiratory rate, airway resistance, and I:E ratio, whereas PaCO2 varied particularly with tidal volume and respiratory rate. These findings reflect local directional sensitivity, not population-level uncertainty.

UNSAFE-CONFIGURATION DEMONSTRATION

The post-intubation collapse scenario reached display caps and generated unsafe-physiology warnings for auto-PEEP, airway pressures, PaCO2, and the MAP surrogate. Its capped outputs were excluded from quantitative scenario comparisons because they indicate an unsafe configuration rather than predict further physiological deterioration.

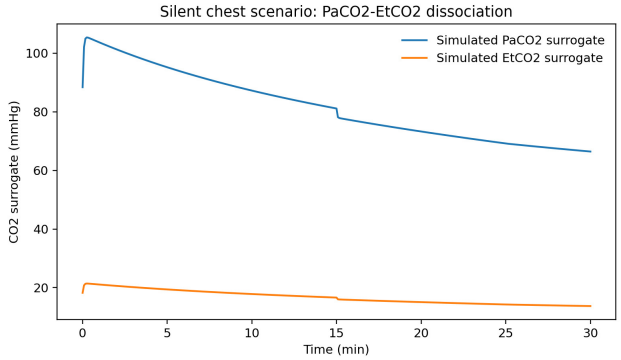

Figure 3. Low end-tidal CO2 despite severe simulated hypercapnia

In the silent-chest teaching scenario, severe airflow obstruction and increased modelled alveolar dead space produce an end-tidal CO2 value of approximately 14 mmHg despite a PaCO2 of approximately 105 mmHg. The scenario illustrates why a low end-tidal CO2 signal may be misleading when effective communication between ventilated lung units and the airway is markedly impaired.
Abbreviations: EtCO2, end-tidal carbon dioxide; PaCO2, arterial partial pressure of carbon dioxide.

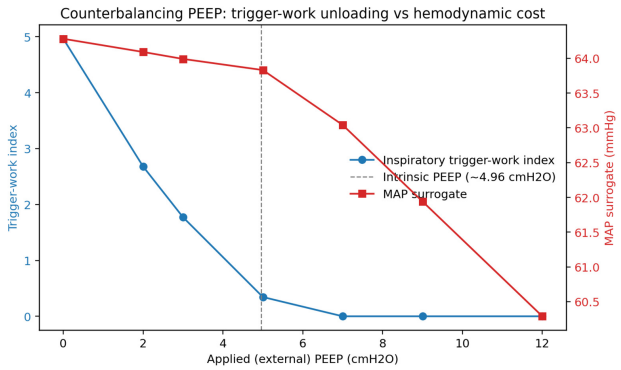

Figure 4. Counterbalancing PEEP in a spontaneously breathing virtual patient

Applied PEEP was progressively increased in a patient with fixed intrinsic PEEP. The simulated inspiratory threshold-work burden decreased as applied PEEP approached intrinsic PEEP. When applied PEEP exceeded intrinsic PEEP, an additional hemodynamic penalty appeared. The figure illustrates the simulator's simplified teaching representation of counterbalancing PEEP rather than a bedside PEEP-titration rule.
Abbreviations: MAP, mean arterial pressure; PEEP, positive end-expiratory pressure.

DISCUSSION

PRINCIPAL FINDINGS

We present an open-source simulator for teaching pediatric critical asthma with predefined scenarios and automated internal-verification checks. In the fixed severely obstructed virtual patient, increasing respiratory rate initially improved simulated carbon dioxide clearance but subsequently shortened expiratory time sufficiently to increase gas trapping, auto-PEEP, and plateau pressure and worsen PaCO2. The accompanying decline in the hemodynamic surrogate illustrates the competing mechanical and cardiovascular costs of ventilation in severe airway obstruction.

The same general physiological direction was observed across the automated checks, the respiratory-rate sweep, and the predefined teaching scenarios. These findings should be interpreted as evidence of internal consistency and physiological directionality rather than as evidence that the simulator predicts the magnitude of physiological responses in individual patients.

PHYSIOLOGICAL PLAUSIBILITY OF THE SIMULATED RESPONSES

The central pathway represented by the simulator has direct support from clinical physiology literature. Stather and Stewart describe gas trapping in severe asthma as a consequence of low expiratory flow requiring long expiratory times; if the next breath interrupts exhalation, intrinsic PEEP and dynamic hyperinflation result.[5] In Tuxen and Lane's mechanically ventilated patients with severe airflow obstruction, reducing expiratory time by increasing respiratory rate increased measured hyperinflation; pressures rose in parallel with lung volume, and hypotension occurred at the highest lung volumes.[7] In a separate study of 12 mechanically ventilated patients with status asthmaticus, lowering respiratory rate from 18 to 12 and 6 breaths/min prolonged expiratory time and reduced plateau pressure, used as an indicator of dynamic hyperinflation.[8] These observations support the direction of the simulator's respiratory-rate/expiratory-time to hyperinflation and pressure/hemodynamic pathway, but not its absolute values or the location of any numerical threshold.

Other programmed relationships are also qualitatively consistent with published physiology. Tuxen and Lane observed greater hyperinflation with larger tidal volumes and found peak airway pressure to be predominantly related to inspiratory flow rather than changes in lung volume.[7] Stather and Stewart explain that during an inspiratory pause peak airway pressure falls to plateau pressure as the resistive component dissipates, supporting the distinction between resistance-dominated peak pressure and static plateau pressure.[5] The exact U-shaped PaCO2 curve generated by the present simulator—including the nadir near 22 breaths/min—is not a clinically validated curve. It emerges from the programmed interaction between increasing set ventilation and worsening effective ventilation as gas trapping becomes severe and should therefore be interpreted as an educational demonstration of a trade-off rather than as a patient-derived response curve.

The plateau pressure of approximately 10 cmH2O at the lowest respiratory rate in the reference sweep further illustrates the distinction between numerical output and clinical prediction. The reference patient was assigned a respiratory-system compliance of 1.0 mL/cmH2O/kg, a tidal volume of 6.5 mL/kg, external PEEP of 3 cmH2O, and a long expiratory time at 6 breaths/min. Under those programmed conditions, little dynamic hyperinflation develops, and the resulting plateau pressure is low within the model. This value should not be interpreted as representative of bedside plateau pressures in children with critical asthma; it reflects the specific educational reference settings used to isolate the effect of progressively increasing respiratory rate.

Available pediatric evidence supports the clinical relevance of ventilator rate and inspiratory/expiratory timing but does not validate the numerical relationships used in this simulator. In a retrospective pediatric series of mechanically ventilated status asthmaticus, pressure-control settings, respiratory rate, and inspiratory and expiratory time were titrated using blood gases, flow waveforms, and exhaled tidal volume.[10] Formal clinical validation of the present simulator would require comparison of simulated outputs with prospectively or retrospectively measured patient data across relevant ventilator settings. Such validation was outside the scope of this development study and remains necessary before any quantitative clinical interpretation of the simulator can be considered.

EDUCATIONAL RELEVANCE

For respiratory therapists and other critical-care learners, the main educational value of the simulator is the ability to display competing physiological consequences at the same time. A learner can increase respiratory rate and observe that an initially favorable change in carbon dioxide clearance may later be accompanied by increasing auto-PEEP, pressure exposure, and hemodynamic burden. Similarly, increasing tidal volume may improve the carbon dioxide response while increasing plateau pressure. The silent-chest scenario provides a separate monitoring example: as physiologic dead space increases, the PaCO2-to-end-tidal-CO2 gradient can widen in mechanically ventilated children,[24] so a low end-tidal CO2 value should not be interpreted in isolation.

These relationships are difficult to teach using isolated numerical examples because the relevant variables are interdependent. The simulator allows learners to change ventilator settings, observe the resulting pattern, and then discuss why improving one physiological target may worsen another. In this context, the intended educational question is not which simulated ventilator setting should be applied to a patient, but which physiological trade-off a particular strategy creates and which bedside measurements would be required to judge that trade-off clinically.

The open-source design also allows educators and learners to inspect the assumptions underlying the displayed responses. This provides an opportunity for faculty-guided discussion not only of ventilator strategy but also of the limitations of simplified physiological representations. Such transparency may be particularly useful when teaching rare, high-risk situations in which repeated clinical exposure is limited.

CURRENT SCOPE AND POTENTIAL EXTENSION

The present simulator was developed around invasive mechanical ventilation in pediatric critical asthma. It does not currently model non-invasive ventilation as a distinct clinical system. Extending the simulator to non-invasive support would require additional assumptions related to spontaneous breathing, patient–ventilator interaction, interface leak, pressure delivery, and the effects of continuous aerosol therapy. For this reason, the present results should not be extrapolated directly to non-invasively ventilated patients.

The underlying educational approach, however, is not necessarily limited to pediatric asthma. The same framework of modifying ventilator settings and observing competing physiological consequences could potentially be adapted to other forms of severe airflow obstruction or to other age groups. Such extensions would require condition- and population-specific parameterization, teaching scenarios, and separate internal and clinical evaluation rather than simply applying the current pediatric asthma scenarios unchanged.

FUTURE EDUCATIONAL EVALUATION

The next stage of evaluation should focus on how the simulator performs as an educational intervention. A pilot study could examine whether a faculty-guided session improves learners' understanding of expiratory time, dynamic hyperinflation, auto-PEEP, permissive hypercapnia, and ventilatory trade-offs. Potential outcomes include knowledge acquisition, ventilator-strategy selection in standardized cases, ability to explain physiological consequences, usability, and perceived educational value.

A stronger subsequent design could compare simulator-assisted teaching with conventional case-based instruction and assess retention of knowledge or transfer to standardized clinical scenarios. Expert review by respiratory therapists, pediatric intensivists, anesthesiologists, and simulation educators would also help evaluate scenario realism, interface usability, debriefing prompts, and risk messaging before broader curricular implementation.

LIMITATIONS

The simulator has several important limitations. First, it uses a simplified, lumped representation of respiratory physiology and does not reproduce regional airway closure, multiple interacting time constants, secretion plugging, detailed endotracheal-tube effects, patient–ventilator asynchrony, or full cardiopulmonary interaction. It should therefore be understood as an educational representation of selected physiological relationships rather than as a digital replica of an individual patient. The fixed I:E input is not independently reconciled with the selected inspiratory flow, and plateau pressure is an algebraic surrogate rather than an occlusion-derived measurement.

Second, several internal coefficients controlling ventilation loss, dead-space expansion, pressure amplification, compliance change, occult auto-PEEP, and the hemodynamic response were selected as fixed educational calibration values rather than derived from patient-level data. These coefficients help produce coherent teaching behaviour but do not establish quantitative clinical accuracy.

Third, the treatment of applied PEEP is deliberately simplified. In the passive-patient pathway, external PEEP influences pressure and the hemodynamic surrogate but does not dynamically increase modelled end-expiratory lung volume or intrinsic PEEP. In real patients with expiratory flow limitation, the response to external PEEP depends on

airway closure, critical closing pressure, lung heterogeneity, spontaneous effort, and other factors not represented fully here.

Fourth, gas exchange is represented as an instantaneous steady-state response. The PaCO2 output does not include the time required for equilibration among alveolar gas, blood, and tissue carbon dioxide stores, and the acid-base response does not incorporate renal compensation. The simulator therefore represents the expected direction of change rather than the time course of arterial blood gases after a ventilator adjustment.

Finally, the simulator has not been calibrated or validated against patient data, and its educational effectiveness has not yet been tested in learners. These limitations restrict interpretation of absolute numerical outputs but do not prevent the current use of the simulator as a faculty-guided tool for exploring predefined physiological relationships and discussing their clinical implications.

CONCLUSIONS

We developed an open-source, computer-based mechanical ventilation simulator to support faculty-guided teaching of ventilatory trade-offs in pediatric critical asthma. In a fixed virtual patient with severe airflow obstruction, the simulator reproduced the expected qualitative relationship between increasing respiratory rate, shortening expiratory time, worsening gas trapping, rising auto-PEEP and airway pressures, and eventual loss of the initial benefit in carbon dioxide clearance. The tool is intended to help learners visualize how attempts to improve one ventilatory target may create competing mechanical and hemodynamic consequences.

The simulator has undergone internal verification of its programmed physiological relationships but has not been validated against patient data. Its current role is therefore educational rather than clinical decision support. Future work should evaluate usability and educational effectiveness and determine whether the same approach can be adapted to other obstructive conditions or patient populations.

FUNDING

No funding was received for conducting this study.

COMPETING INTERESTS

PT is the developer of the open-source simulator described in this manuscript. The authors declare no financial competing interests and no other competing interests relevant to the content of this article.

ETHICS APPROVAL

This study did not involve human participants, their data, or biological material, and no patient data were used; ethics approval was therefore not required.

DATA AVAILABILITY

All data generated or analyzed during this study are included in this article and in the project's public repository and archived release. The scenario library, generated output tables, and figures are openly available, and the reported numerical results and figures can be regenerated from the provided code. Repository and archive links are provided in the Code availability section.

CODE AVAILABILITY

The source code is open source and released under the Apache License 2.0. The software package version analyzed in this manuscript was 1.8.1, and the archived maintenance snapshot was the release tag v1.8.1-post1. Pediatric status asthmaticus ventilation simulator, version 1.8.1 / release tag v1.8.1-post1. Open-source software (Apache-2.0).
GitHub repository: https://github.com/taffache-hash/pediatric-asthma-ventilation-simulator
Zenodo archived release: https://doi.org/10.5281/zenodo.20848420

AUTHOR CONTRIBUTIONS

All authors contributed to model design, scenario selection, clinical interpretation, and critical revision of the manuscript for important intellectual content; all authors read and approved the final manuscript and agree to be accountable for all aspects of the work.

USE OF AI-ASSISTED TOOLS

AI-assisted tools were used during software drafting, code review, documentation refinement, and manuscript language editing. All equations, scenarios, outputs, references, and claims were reviewed by the authors, who remain responsible for the accuracy, integrity, reproducibility, and interpretation of the work. AI tools were not used as authors and were not treated as primary sources.

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SUPPLEMENTARY MATERIALS

Supplementary materials

Download: https://cjrt.ca/article/174248-development-and-internal-verification-of-an-open-source-mechanical-ventilation-simulator-for-teaching-ventilatory-trade-offs-in-pediatric-critical-ast/attachment/376866.docx