Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

A physiological simulator for teaching complex positive airway pressure titration: development and verification against published physiological values

Samantha Faye Tina

samtinaaa@gmail.com

Independent Researcher

advancing-simulation-practice

Keywords:

Posted Date: September 2nd, 2026

DOI: https://doi.org/10.21203/rs.3.rs-10884913/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License

Additional Declarations: Competing interest reported. The author developed the simulator described in this manuscript and may seek to license or commercialize it in future. The author has no financial relationship with any device manufacturer and received no funding for this work.

Abstract

Background. Sleep technologists acquire competence in complex positive airway pressure (PAP) titration almost entirely on live patients. Simulation is established in respiratory care education, but tools available in sleep medicine teach device interfaces rather than patient responses, leaving no widely available means of rehearsing a titration in neuromuscular disease, obesity hypoventilation, chronic hypercapnic COPD, or central sleep apnea.

Objective. To develop a browser-based physiological simulator in which complex titration failures arise from the underlying model rather than from scripted events, and to assess whether its behavior reproduces published physiological relationships.

Methods. A single-compartment respiratory model was implemented with expiratory flow limitation, a Starling-resistor upper airway, sleep-stage dependent muscle tone and airway collapsibility, chemoreflex control with adjustable loop gain and circulatory delay, and separate carbon dioxide and oxygen kinetics. Seven patient phenotypes and seven PAP modes were implemented. Model output was compared against published values for six physiological relationships.

Results. The model reproduced the direction, and in most cases the approximate magnitude, of all six verified relationships, including the supine-to-lateral change in critical closing pressure, the pressure-support trade-off when expiratory pressure is raised in isolation, the emergence of treatment-emergent central apnea at sustained high pressure and its abolition by a backup rate, and the dependence of Cheyne-Stokes cycle length on circulatory delay. Two deviations were identified and are reported.

Conclusions. A model-driven simulator can reproduce the principal physiological relationships underlying complex PAP titration with sufficient fidelity for teaching. Agreement with published values establishes that the model behaves plausibly; it does not establish validity against patient recordings, and learner outcomes have not been evaluated.

1. Background

Complex PAP titration is a low-frequency, high-consequence task. A technologist may encounter advanced neuromuscular disease, treatment-emergent central sleep apnea, or Cheyne-Stokes respiration only occasionally, yet each demands a different and sometimes opposite response. Raising expiratory pressure is correct in obstruction and counterproductive in a patient whose airway does not collapse. Adding pressure support relieves hypoventilation and can lengthen a central pause. These distinctions are difficult to teach didactically because they are defined by how a patient responds, not by what a setting means.

Titration practice is codified in guidelines that specify pressure limits and adjustment intervals [1]; guidelines convey what a setting means but cannot demonstrate the consequence of choosing it wrongly. Simulation is established in respiratory care education [17], where mechanical ventilation is

routinely taught using physical test lungs that reproduce patient mechanics and respond to what the operator does. Sleep medicine training employs simulation for other purposes, and device manufacturers supply menu simulators that teach the interface of a particular machine. Neither models a patient: the settings change and nothing pushes back. PAP titration is therefore taught largely through guidelines and reference material, which convey what a setting means without demonstrating the consequence of choosing it wrongly.

This paper describes the development of a simulator built to close that gap, and reports how its behavior compares against published physiological values.

2. Methods

2.1 Standing of the formulations used

The equations below are of two kinds, and the distinction matters when judging what the model can be relied upon for. The upper airway and the chemoreflex controller use established formulations: flow limitation through a collapsible pharynx is the Starling-resistor relation [2,3,4], and ventilatory drive as a linear function of carbon dioxide sensed after a transport delay is the standard loop-gain description of periodic breathing [5]. Carbon dioxide kinetics are a conventional single-compartment mass balance against a body store.

The respiratory mechanics are phenomenological. Neither the quadratic stiffening term in the pressure-volume relation nor the volume-proportional ceiling on expiratory flow is a published form. Real pressure-volume curves are more usually represented as sigmoid or exponential, and effort-independent expiratory flow limitation is more usually derived from wave-speed or equal-pressure-point theory. The forms used here were chosen because they reproduce the behavior that matters for teaching, that compliance falls at high lung volume and that expiratory flow becomes independent of downstream pressure, at a fraction of the computational cost and with parameters that map onto quantities a technologist already reasons about. They should be read as behavioral approximations, not as physiological derivations.

2.2 Respiratory mechanics

Lung mechanics are represented as a single compartment with nonlinear compliance. Alveolar pressure is

P_alv(V) = V/C0 + k * max(0, V - V_knee)^2

so compliance falls at high lung volume, reproducing the stiffening of an overinflated lung. Inspiratory flow follows the equation of motion driven by the sum of airway pressure and inspiratory muscle pressure. Expiration is flow-limited:

Vdot_exp = -min((P_alv - P_aw)/R_exp , a * V)

The second term makes expiratory flow independent of downstream pressure during the flow-limited portion, which is why raising expiratory pressure reduces auto-PEEP relative to the set pressure without altering the trapped volume.

2.3 Upper airway

The pharynx is modeled as a Starling resistor, following the collapsible-tube description of upper airway occlusion during sleep [2,3]. During an obstructive event, inspiratory flow is capped at

Vdot_max = (P_mask - P_close) / R_us

Phenotype-specific critical closing pressures were assigned within the ranges reported from snorers through to severe obstructive disease [4]. Each event draws its own closing pressure, so a proportion occlude completely while the remainder narrow, producing a mixture of apneas and hypopneas whose composition shifts with how far mask pressure sits below the airway's opening pressure. Between events a residual resistance persists, scaled to the same deficit, reproducing continuous flow limitation and snoring.

2.4 Ventilatory control

Chemoreflex drive is a function of carbon dioxide sensed after a circulatory delay, in the tradition of loop-gain models of periodic breathing [5]:

drive = clamp(G * (PaCO2(t - tau) - P_threshold) / dP , 0, drive_max)

Loop gain G, apneic threshold, delay tau and the ceiling are phenotype-specific. Carbon dioxide is a mass balance against a body store; alveolar oxygen has its own faster balance with uptake tapering as alveolar oxygen approaches mixed venous, which bounds apneic desaturation. Central pauses terminate by arousal rather than by the return of carbon dioxide.

2.5 Sleep stage and body position

Stage modifies accessory muscle tone, airway collapsibility and chemoreflex gain. Stage-dependent chemoreflex gain was calibrated for the heart failure phenotype against reported values [9]; The opioid phenotype was constructed to reproduce the reported pattern of central apneas with ataxic breathing concentrated in non-rapid eye movement sleep [16]; the specific gain values were assigned during construction rather than fitted to published measurements. Position acts through three separate channels: airway collapsibility, functional residual capacity, which scales the gas stores [13,14], and

diaphragm loading. Each phenotype carries its own sensitivity to each channel, reflecting evidence that position acts through several distinct physiological factors rather than one [6].

2.6 Event scoring

Respiratory events are scored from the simulated signals rather than emitted by the event generator, so that a phenotype whose failure is neither airway closure nor absent effort is still detected. An obstructive event is scored when airflow is reduced against continued effort. A hypopnea is scored when tidal volume falls more than 30% below the patient's own running baseline for at least ten seconds accompanied by a desaturation of 3% or more. An apnea is scored when no breath is delivered for ten seconds while the patient continues to make inspiratory effort, whatever the reason the breath failed to open, and once per episode rather than for each ten-second interval it continues. Central apnea requires both absent effort and no delivered breath for ten seconds, so a mandatory breath falling within a pause prevents one from being scored.

Sustained hypoventilation is treated as a separate finding, flagged when carbon dioxide remains above the scoring threshold for ten minutes, since a patient may hypoventilate severely with a low event index. Hypercapnia also drives arousal in the model: above 68 mmHg the patient rouses briefly, tone returns and ventilation improves before falling away again, which reproduces the sleep fragmentation seen in untreated nocturnal hypoventilation and prevents carbon dioxide from accumulating without limit.

2.7 Phenotypes and modes

Seven phenotypes: obstructive sleep apnea at three severities, COPD-OSA overlap, obesity hypoventilation with awake hypercapnia, amyotrophic lateral sclerosis with diaphragm weakness, treatment-emergent central sleep apnea, opioid-associated ataxic breathing, and heart failure with Cheyne-Stokes respiration and adjustable ejection fraction. Seven modes: CPAP, BPAP-S, BPAP-S/T, AVAPS, AVAPS-AE, iVAPS and adaptive servo-ventilation, each exposing the controls of the corresponding device. Pressure limits follow published titration guidance [1]. Adaptive servo-ventilation is gated by ejection fraction in accordance with the contraindication established by SERVE-HF [7] and the subsequent guideline revision [8].

The consequences of these assignments are not always obvious in advance. In the neuromuscular phenotype, inspiratory muscle pressure was set so that the loss of accessory muscle support in rapid eye movement sleep leaves roughly a quarter of the waking value, following the observation that diaphragm weakness is unmasked when accessory support is removed, which is why vital capacity is measured supine as well as upright in this population [15]. At the standard bilevel starting pressure of 8 over 4, the resulting tidal volume in REM is 110 mL against an anatomical dead space of 150 mL, so no gas reaches the alveoli and carbon dioxide accumulates until arousal intervenes. Widening pressure support restores ventilation progressively:

Setting	Pressure support	Tidal volume	CO2	SpO2
8/4	4	110 mL	off scale	79%
12/4	8	244 mL	off scale	85%
16/4	12	277 mL	68	90%
20/4	16	368 mL	46	96%

Expiratory pressure contributes nothing in this phenotype, whose critical closing pressure is negative: 18 over 8 and 18 over 4 differ only in the differential, and give carbon dioxide of 73 and 54 mmHg respectively. These magnitudes were not fitted to published measurements and are reported as properties of the model rather than as verified results. Published stage-specific data in diaphragm weakness describe a different output, discrete scored events rather than sustained hypoventilation: Steier and colleagues reported a respiratory disturbance index of 8.1 +/- 10.1 in non-rapid eye movement sleep against 26.0 +/- 17.8 in REM in unilateral paralysis or severe weakness [19]. The two measures are not directly comparable, and calibration of this phenotype would require stage-specific measurements of inspiratory effort at this severity.

3. Results

3.1 Pressure support trade-off

Expiratory pressure was raised from 4 to 12 cmH2O in the COPD overlap phenotype with inspiratory pressure held at 20 and a backup rate of 14, narrowing pressure support from 16 to 8 cmH2O.

EPAP	Pressure support	Tidal volume	CO2	SpO2	Ineffective efforts
4	16	544 mL	39	95.2%	26%
8	12	331 mL	49	91.8%	33%
12	8	278 mL	62	86.7%	2%

Both sides of the trade-off appear. Narrowing pressure support halved tidal volume and raised carbon dioxide by 22 mmHg, while ineffective efforts fell from 26% to 2% as the rising expiratory pressure counterbalanced intrinsic positive end-expiratory pressure.

3.2 Obstructive severity and titration endpoint

Apnea-hypopnea index by severity and applied pressure:

Severity	CPAP 4	CPAP 7	CPAP 11	CPAP 14
Mild	24	**3**	3	3
Moderate	72	30	**6**	6
Severe	99	75	54	**6**

Each severity resolved at a different pressure, and none resolved to zero: a residual index of 3 to 6 persists once obstruction is controlled, reflecting the flow-based scoring criteria described in section 2.5, which detect minor reductions that an event-driven model would not generate. This is closer to a successful clinical titration, where a residual index below 5 is the accepted endpoint, than an index of zero would be. Event composition also shifted with severity: at CPAP 4 the mild phenotype produced predominantly hypopneas and the severe phenotype predominantly apneas, matching the clinical expectation that apnea predominance marks the severe end of the range.

3.3 Treatment-emergent central apnea

In the severe obstructive phenotype, applied pressure produced a U-shaped relationship with the apnea-hypopnea index: obstruction dominated at low pressure, events were abolished at intermediate pressure, and central events emerged once pressure was sustained above the phenotype threshold.

CPAP	AHI	Obstructive	Hypopnea	Central
4	91	27	11	0
8	58	12	12	0
12	22	1	8	0
14	**0**	0	0	0
18	98	0	0	41

Both recognised responses reproduced. From CPAP 18, adding a backup rate of 13 in bilevel spontaneous-timed mode abolished the central events, and reducing pressure to CPAP 13 returned the index to 7. Bilevel without a backup rate changed nothing, the index remaining at 98.

3.4 Cheyne-Stokes respiration and ejection fraction

Circulatory delay was scaled to ejection fraction, reaching 31 s at an ejection fraction of 20%, within the range reported for this population.

Ejection fraction	Circulatory delay	Central index
55%	12.0 s	0/h
45%	17.5 s	0/h
35%	23.0 s	10/h
30%	25.8 s	17/h
25%	28.5 s	26/h
20%	31.2 s	34/h

Cycle lengths in this range fall within the 40 to 90 seconds specified for scoring the pattern [18]. Periodic breathing appeared only below an ejection fraction of about 40% and increased monotonically as delay lengthened. Breath-by-breath tidal volumes formed a crescendo-decrescendo envelope with a twelve-breath crescendo, a rounded peak and a twelve-breath decrescendo terminating in a central pause.

3.5 Position and sleep stage in Cheyne-Stokes respiration

Sahlin and colleagues reported a mean central apnea-hypopnea index of 41 +/- 13 supine against 26 +/- 12 non-supine, a reduction of 37%, with the index highest in stages 1 and 2 and lowest in slow-wave and rapid eye movement sleep [9]. Lateral position has independently been reported to reduce the severity of central sleep apnea and Cheyne-Stokes respiration [10].

In the model at an ejection fraction of 30%, the central index fell from 17/h supine to 12/h lateral, a reduction of 29%, against the reported 37%. Head-up position produced no reduction, consistent with reports that the pattern persists upright. Stage ordering agreed with the published data, with the index highest in light non-rapid eye movement sleep and lowest in slow-wave sleep.

3.6 Position in obstructive and neuromuscular phenotypes

In the moderate obstructive phenotype at CPAP 5, the apnea-hypopnea index fell from 39/h supine to 12/h lateral and 15/h head-up, consistent with the reported fall in critical closing pressure from supine to lateral and with the observation that lower therapeutic pressure is required laterally [11]. In the neuromuscular phenotype the airway was unaffected by position while inspiratory effort rose 20% head-up, reproducing the separation between an airway mechanism and a muscle mechanism. Positional dependency differs between obstructive and central sleep-disordered breathing [12].

4. Limitations

Two limitations of the ventilatory control model are known. First, the model responds to changes in loop gain in a near-binary fashion close to its stability boundary, so a modest reduction can suppress periodic breathing entirely rather than attenuating it; the positional effect in section 3.5 was deliberately kept small for this reason and consequently understates the reported magnitude. Second, the stage ratios in section 3.5 are approximate, because the model transitions from full periodic breathing to none across a narrow range of loop gain and cannot place intermediate ratios precisely. Both reflect the same structural limitation: a single-compartment model with one chemoreflex loop produces near-binary responses close to its stability boundary. The phenomenological mechanics set out in section 2.1 are a further constraint. They reproduce the qualitative behavior of expiratory flow limitation and of a stiffening lung without being derived from it, so quantitative agreement with measured pressure-volume curves or maximal expiratory flow data should not be expected.

More broadly, this is verification against published values, not validation against patient recordings. The model was compared with figures reported in the literature; it was not compared with recordings from patients, and no expert panel formally rated its realism. Absolute magnitudes should not be relied upon. The untreated apnea-hypopnea index runs above the diagnostic figure for the severe obstructive phenotype, the positional reduction in Cheyne-Stokes falls short of the reported magnitude at 29% against 37%, and the carbon dioxide excursion during periodic breathing is wider than would be measured arterially. Carbon dioxide is bounded at 80 mmHg and displayed as off scale beyond that, since a value reached only because alveolar ventilation has fallen to zero is an artifact of leaving a scenario running that would have been interrupted in a laboratory. The synthesized waveforms are not suitable for scoring practice against AASM criteria. No learner outcomes have been evaluated.

5. Conclusion

A model-driven simulator reproduces the principal physiological relationships underlying complex PAP titration closely enough to teach the reasoning: that expiratory pressure and pressure support treat different problems, that a backup rate and pressure support are not interchangeable, that the same intervention helps one phenotype and harms another, and that a setting untested in REM has not been tested. Whether this improves technologist performance remains to be determined and is the necessary next step.

Declarations

Funding. This research received no funding.

Competing interests. The author developed the simulator described in this manuscript and may seek to license or commercialise it in future. The author has no financial relationship with any device manufacturer and received no funding for this work.

Availability of data and materials. The simulator described in this article is freely available at https://samtinapapsimulator.samtinaaa.workers.dev/. All data reported are model output and are included in this article; each figure can be reproduced by running the simulator at the settings stated in the corresponding table.

Author contributions. S.T. conceived the study, developed the simulator, designed and performed the verification analyses, and wrote the manuscript.

Ethics approval and consent to participate. Not applicable; no human subjects or human data were involved.

Consent for publication. Not applicable.

References

1. Kushida CA, Chediak A, Berry RB, Brown LK, Gozal D, Iber C, Parthasarathy S, Quan SF, Rowley JA; Positive Airway Pressure Titration Task Force, American Academy of Sleep Medicine. Clinical guidelines for the manual titration of positive airway pressure in patients with obstructive sleep apnea. *J Clin Sleep Med.* 2008;4(2):157-171. doi:10.5664/jcsm.27133
2. Remmers JE, deGroot WJ, Sauerland EK, Anch AM. Pathogenesis of upper airway occlusion during sleep. *J Appl Physiol Respir Environ Exerc Physiol.* 1978;44(6):931-938.
3. Issa FG, Sullivan CE. Upper airway closing pressures in obstructive sleep apnea. *J Appl Physiol Respir Environ Exerc Physiol.* 1984;57(2):520-527.
4. Kazemeini E, Van de Perck E, Dieltjens M, Willemen M, Verbraecken J, Op de Beeck S, Vanderveken OM. Critical to know Pcrit: a review on pharyngeal critical closing pressure in obstructive sleep apnea. *Front Neurol.* 2022;13:775709. doi:10.3389/fneur.2022.775709
5. Khoo MC, Kronauer RE, Strohl KP, Slutsky AS. Factors inducing periodic breathing in humans: a general model. *J Appl Physiol Respir Environ Exerc Physiol.* 1982;53(3):644-659.
6. Joosten SA, Edwards BA, Wellman A, Turton A, Skuza EM, Berger PJ, Hamilton GS. The effect of body position on physiological factors that contribute to obstructive sleep apnea. *Sleep.* 2015;38(9):1469-1478. doi:10.5665/sleep.4992
7. Cowie MR, Woehrle H, Wegscheider K, Angermann C, d'Ortho MP, Erdmann E, et al. Adaptive servo-ventilation for central sleep apnea in systolic heart failure. *N Engl J Med.* 2015;373(12):1095-1105. doi:10.1056/NEJMoa1506459
8. Aurora RN, Bista SR, Casey KR, Chowdhuri S, Kristo DA, Mallea JM, et al. Updated adaptive servo-ventilation recommendations for the 2012 AASM guideline: "The treatment of central sleep apnea syndromes in adults: practice parameters with an evidence-based literature review and meta-analyses". *J Clin Sleep Med.* 2016;12(5):757-761.
9. Sahlin C, Svanborg E, Stenlund H, Franklin KA. Cheyne-Stokes respiration and supine dependency. *Eur Respir J.* 2005;25(5):829-833. doi:10.1183/09031936.05.00107904

10. Szollosi I, Roebuck T, Thompson B, Naughton MT. Lateral sleeping position reduces severity of central sleep apnea/Cheyne-Stokes respiration. *Sleep.* 2006;29(8):1045-1051. doi:10.1093/sleep/29.8.1045
11. Penzel T, Moller M, Becker HF, Knaack L, Peter JH. Effect of sleep position and sleep stage on the collapsibility of the upper airways in patients with sleep apnea. *Sleep.* 2001;24(1):90-95. doi:10.1093/sleep/24.1.90
12. Yumino D, Bradley TD. Central sleep apnea and Cheyne-Stokes respiration. *Proc Am Thorac Soc.* 2008;5(2):226-236.
13. Pinna GD, Dacosto E, Maestri R, Crotti P, Montemartini S, Caporotondi A, Guazzotti G, Bruschi C. Postural changes in lung volumes in patients with heart failure and Cheyne-Stokes respiration: relationship with sleep apnea severity. *Sleep Med.* 2023;101:154-161.
14. Hurewitz AN, Susskind H, Harold WH. Obesity alters regional ventilation in lateral decubitus position. *J Appl Physiol.* 1985;59(3):774-783.
15. Lechtzin N, Wiener CM, Shade DM, Clawson L, Diette GB. Spirometry in the supine position improves the detection of diaphragmatic weakness in patients with amyotrophic lateral sclerosis. *Chest.* 2002;121(2):436-442. doi:10.1378/chest.121.2.436
16. Walker JM, Farney RJ, Rhondeau SM, Boyle KM, Valentine K, Cloward TV, Shilling KC. Chronic opioid use is a risk factor for the development of central sleep apnea and ataxic breathing. *J Clin Sleep Med.* 2007;3(5):455-461. doi:10.5664/jcsm.26908
17. Sreedharan JK, Gopalakrishnan GK, Jose AM, Albalawi IA, Alkhathami MG, Alqahtani AS. Simulation-based teaching and learning in respiratory care education: a narrative review. *Adv Med Educ Pract.* 2024;15:473-486. doi:10.2147/AMEP.S464629
18. Troester MM, Quan SF, Berry RB, et al.; for the American Academy of Sleep Medicine. *The AASM Manual for the Scoring of Sleep and Associated Events: Rules, Terminology and Technical Specifications.* Version 3. Darien, IL: American Academy of Sleep Medicine; 2023.
19. Steier J, Jolley CJ, Seymour J, Kaul S, Luo YM, Rafferty GF, Hart N, Polkey MI, Moxham J. Sleep-disordered breathing in unilateral diaphragm paralysis or severe weakness. *Eur Respir J.* 2008;32(6):1479-1487. doi:10.1183/09031936.00018808