

A Bench Comparison of Six Auto-Adjusting Positive Airway Pressure Devices on a Dynamic Test Bench

as Prepared by React Health 2025

ABSTRACT

Introduction: Although auto-adjusting positive airway pressure (APAP) technology has been in clinical use for several decades, recent advancements have refined its ability to deliver dynamic, real-time pressure adjustments based on detected respiratory patterns. Multiple devices utilizing different proprietary algorithms are currently available. This study aimed to evaluate the performance of six APAP devices in the presence of standardized obstructive and central respiratory events. All testing was performed at an independent testing facility.

Methods: A dynamic bench model was used to assess the latest generation of six APAP devices. Simulated obstructive and central respiratory events were applied to evaluate each device's detection capabilities. The accuracy of event detection and identification simulated obstructive and central apneic events was evaluated by comparing real bench test apnea-hypopnea index (AHI) to device-reported AHI. Efficacy was evaluated by observing the device pressure responsiveness when presented with each simulated breathing pattern. The ability to compensate for unintentional leak was also performed.

Results: All devices increased the delivered pressure in response to obstructive events. The AirSense 11 and the G3 A20 corrected the most apnea events, while the G3 A20 and PrismaSmart max demonstrated superior detection and correction of obstructive hypopneas. Snoring triggered pressure increases in three of six devices. In response to central patterns (central apneas and hypopneas and Cheyne-Stokes respiration), only three devices correctly identified the nature of the events and appropriately withheld pressure increases. Introduction of non-intentional leaks during respiratory events differently affected each device's ability to identify and correct these events.

Conclusion: This study demonstrated notable variability in the performance of APAP devices, particularly in their capacity to differentiate and respond to various respiratory event types. These findings underscore the importance of selecting the appropriate APAP device based on the specific needs and apnea characteristics of the patient to ensure optimal therapy outcomes.

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INTRODUCTION

Continuous Positive Airway Pressure (CPAP) is the first-line treatment for moderate to severe obstructive sleep apnea (OSA) syndrome (1,2). Adherence to CPAP is essential to alleviate symptoms (3) and reduce the risk cardiometabolic complications. (4) However, long-term adherence remains a significant challenge, with more than 50% of patients discontinuing therapy or demonstrating poor adherence (5).

One of the most common barriers to adherence air leak at the mask interface.(6,7) causing annoyance and sleep disruption (8). The causes of mask leak are multifactorial and include mask fit, features of facial anatomy, sleeping position and pressure settings.(9,10) In response, manufacturers have devoted substantial efforts to improving mask designs and refining device algorithms to reduce the occurrence and impact of leaks, as well as to lower the average pressure delivered.

A major technological advancement has been the development of auto adjusting positive airway pressure (APAP devices. These devices continuously monitor respiratory patterns and automatically adjust pressure in real time to treat residual obstructive events. By doing so, APAP devices help maintain airway patency regardless of sleep stage or body position (11). Initially developed as a tool for in-home pressure titration (12), APAP has since become widely adopted for long-term therapy (13). In terms of therapeutic efficacy, APAP has been shown to normalize the apnea-hypopnea index (AHI) as effectively as fixed-pressure CPAP, while delivering lower mean pressures and achieving slightly better adherence rates (14).

Previous studies (11,15–18) have demonstrated heterogeneity in the ability of different APAP devices to detect respiratory events and adjust pressure effectively. Since those evaluations, many devices have undergone algorithm updates, and new devices have entered the market. Each uses proprietary algorithms and sensor technologies to monitor airflow, detect apneas and hypopneas, and determine pressure response if needed. These differences can influence therapeutic effectiveness and patient comfort, underscoring the need for comparative evaluation. The objective of this bench study is to assess and compare the performance of six APAP devices using a mechanical lung simulator that presents each device with the same set of simulated sleep-disordered breathing patterns and leak conditions. This approach removes patient-specific variability and enables a direct, objective comparison of device behavior. The study evaluates each device's accuracy in identifying apneas and hypopneas and its ability to deliver appropriate pressure responses. A critical element of this analysis is determining whether devices can correctly distinguish between obstructive and central events, as pressure increases in response to central events are inappropriate and may worsen outcomes (1,19). Accurate detection and proper pressure modulation are essential to avoid unnecessary patient discomfort and ensure effective, individualized care.

Experimental Setup

Testing was conducted using the ASL 5000 mechanical lung simulator (IngMar Medical, Pittsburgh, PA, USA) paired with a mannequin head equipped with anatomically realistic airways and fitted with a nasal mask (Eson, Fisher & Paykel, Costa Mesa, CA, USA). Obstructive events were generated using a Starling resistor controlled by a pressure-regulation system, while central events were simulated by manipulating inspiratory effort through the mechanical lung. A non-intentional leak valve was incorporated to evaluate each device's leak compensation capability. To simulate snoring, a "snore-box" was integrated into the circuit, producing a 70 Hz sound via an embedded speaker. The complete experimental configuration is illustrated in **Figure 1**.

The lung mechanics were defined by specific parameters. Both inspiratory and expiratory resistance were set at 5 cmH₂O/L.s. System compliance was established at 60 ml/cmH₂O, with inspiratory effort amplitude ranging from -12 to -10 cmH₂O, varying uniformly to mimic natural variability in breathing amplitude. The breathing frequency was set at 15 cycles per minute (cpm), and the basal peak flow varied between 30 and 40 Liters per minute (L/min). Standardized breathing patterns and respiratory events were programmed to replicate various sleep apnea conditions.

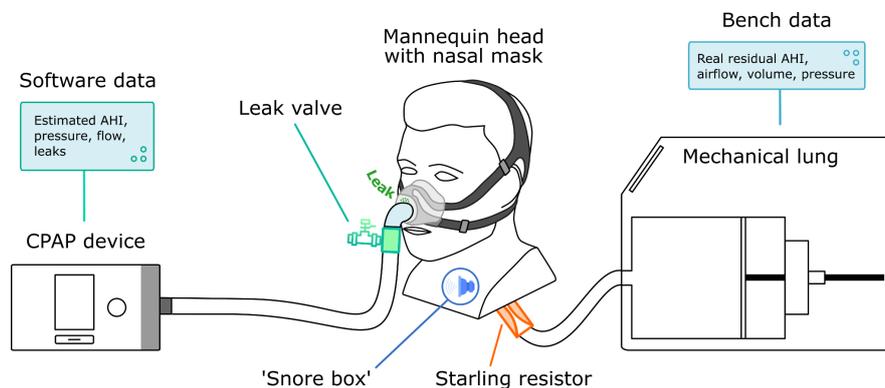


Figure 1: Experimental setup for evaluating APAP devices.

Obstructive Respiratory Events

Three obstructive patterns were simulated: obstructive apnea, obstructive hypopnea, and snoring.

- *Obstructive apnea* events lasted 20 seconds with a 1-minute interval between events; 23 events were presented over a 30-minute period. These were generated by applying 34 cmH₂O across the Starling resistor, resulting in ≥95% flow limitation.
- *Obstructive hypopnea* events lasted 50 seconds with 2-minute intervals; 11 events were delivered over 30 minutes. A resistor pressure of 28 cmH₂O created ≥60% flow limitation.
- *Snoring* was continuously simulated for 30 minutes, featuring inspiratory-phase snoring and a 20% reduction in inspiratory effort, yielding a 20% flow limitation.

Central Respiratory Events

Three central patterns were simulated: central apnea, central hypopnea, and Cheyne-Stokes respiration.

- *Central apnea* events lasted 20 seconds with a 16-second interval between events; 48 events were presented over a 30-minute period. These were generated by eliminating inspiratory effort, resulting in 100% flow limitation.
- *Central hypopnea* events lasted 50 seconds with 2-minute intervals; 11 events were delivered over 30 minutes. A 65% reduction in inspiratory effort produced $\geq 60\%$ flow limitation.
- *Cheyne-Stokes respiration* was simulated with 30-second episodes and 40-second intervals, totaling 27 events over a 30-minute period. These events featured cyclic modulation of inspiratory effort, resulting in complete (100%) flow limitation during peak hypoventilation phases.

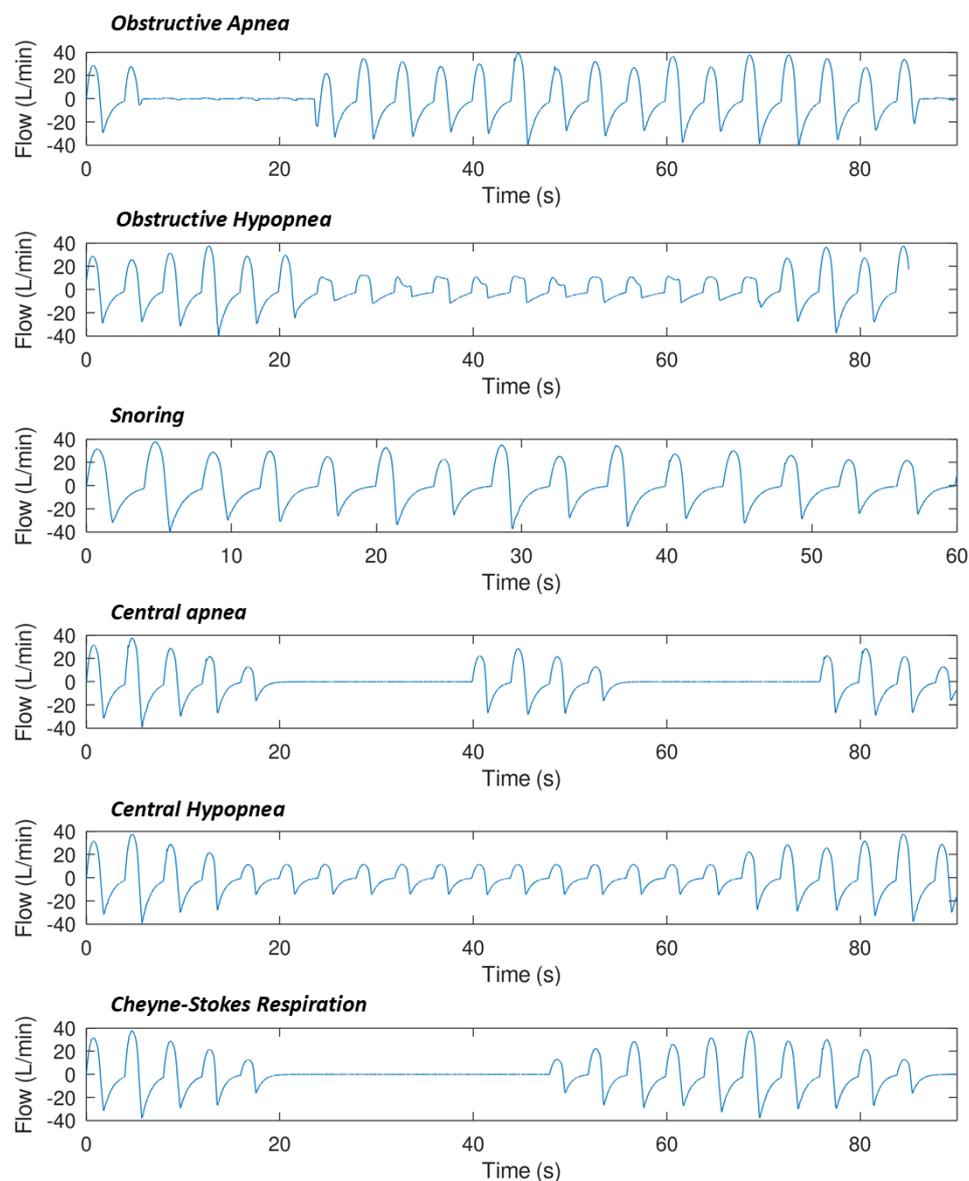


Figure 2: Respiratory flow curves for various simulated events.

Device Configuration

Six auto-adjusting CPAP devices from leading manufacturers were included in this study:

- BMC G3 A20 (Beijing, China; software version G3-2.12.04)
- ResMed AirSense 11 (San Diego, CA, USA; software version 04600.06.5.1.0)
- Philips DreamStation 2 (Amsterdam, Netherlands; software version 1.04.3830)
- Löwenstein Prisma SMART Max (Bad Ems, Germany; software version 3.13.006)
- Fisher & Paykel SleepStyle (Panmure, New Zealand; software version 1.1.1)
- Sefam Néa (Villers-lès-Nancy, France; software version A010201)

All devices were set to Auto-CPAP mode with default sensitivity settings. Pressure parameters were standardized with a minimum pressure of 4 cmH₂O and a maximum of 20 cmH₂O. Expiratory pressure relief and ramp features were disabled. Heated humidifiers were installed and filled per manufacturer instructions, but humidification was turned off. Breathing circuits were device-specific: 22 mm for the G3 A20 and SleepStyle; 15 mm for the others, with settings adjusted accordingly.

Testing Protocol

Each device was allowed to acclimate for 15 minutes under normal breathing conditions. Following this warm-up, the unit was restarted, and data acquisition commenced. Each respiratory event type was introduced after an initial 30-minute baseline period of normal breathing. The selected respiratory event (e.g., obstructive apnea, central hypopnea) was then simulated continuously for 30 minutes. Afterward, normal breathing resumed for 90 minutes to observe device recovery and stability post-event exposure.

Leak Simulation

Additional testing assessed device performance under variable leak conditions during obstructive and central apnea. Eight leak episodes were simulated each lasting either 6 or 10 minutes. The 6-minute leaks had a flow rate of 10 L/min, while the 10-minute leaks had a flow rate of 20 L/min. Two of these leaks occurred during respiratory events, while the remaining six occurred during normal breathing periods. A schematic representation of the testing procedures is shown in Figure 3.

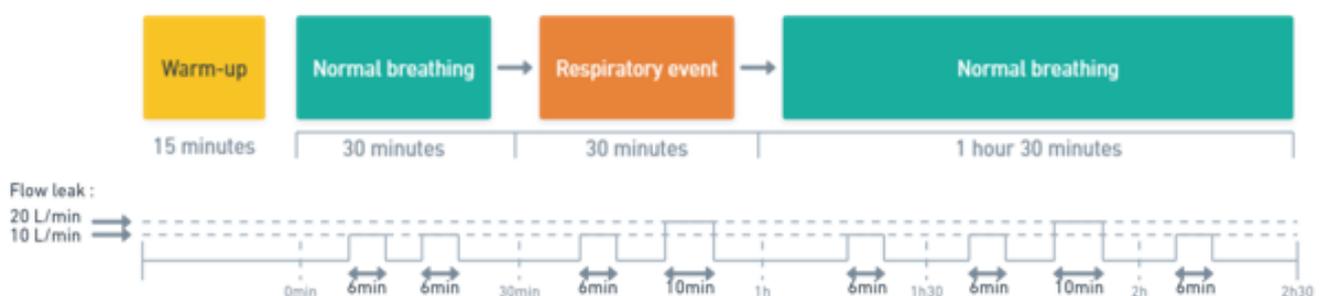


Figure 3: Above: Testing procedure. Below: Leak pattern setup for experiments involving leaks.

Outcome Measures

Continuous data collection throughout the study enabled evaluation of each device's capacity to detect and respond to simulated events. Accuracy was assessed by comparing the bench-determined residual AHI to each device's reported AHI. Because the Starling resistor allows controlled simulation of obstructive events, this setup provided a valid benchmark for detection accuracy. Pressure responsiveness was measured by observing changes in delivered pressure during each event type. Additionally, data logging and reporting features were analyzed to assess each device's clinical utility and information output.

RESULTS

The following results summarize the comparative performance of six APAP devices in response to simulated obstructive and central respiratory events, both with and without non-intentional leakage. Pressure responses and detection accuracy are presented alongside the agreement between device-reported and bench-determined data. Visual representations are provided in Figures 4 through 9, and corresponding quantitative details are outlined in Tables 1 through 5.

Obstructive Apnea

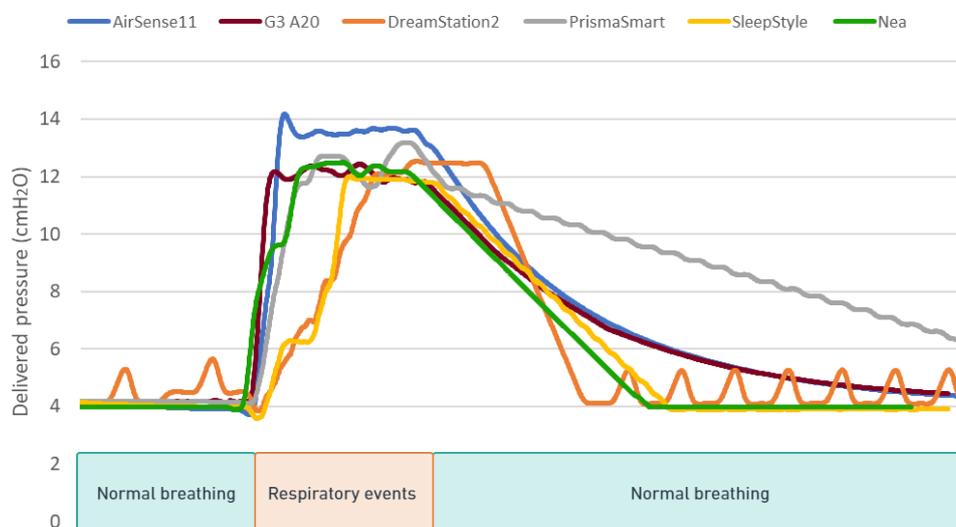


Figure 4: Pressure response of the five tested devices for obstructive apnea events.

Obstructive apnea events n=23	AirSense 11	G3 A20	Dream Station 2	PrismaSmart max	Sleep Style	Néa
Number of actual residual events (% of corrected events)	4 / 23 (83%)	4 / 23 (83%)	16 / 23 (30%)	9 / 23 (61%)	13 / 23 (43%)	11 / 23 (52%)
Estimated residual AHI / real residual AHI	4 / 4	3 / 4	15 / 16	6 / 9	12 / 13	14 / 11
Correct classification of residual events (accuracy in %)	<i>Residual apneas</i>					
	3 / 2 (150%)	2 / 2 (100%)	14 / 10 (155%)	4 / 3 (133%)	10 / 10 (100%)	4 / 4 (100%)
	<i>Residual hypopneas</i>					
	1 / 2 (50%)	1 / 2 (50%)	1 / 6 (17%)	2 / 6 (33%)	2 / 3 (67%)	10 / 7 (143%)

Table 1: Agreement between built-in software estimations and test bench objective data for residual obstructive apnea events. Actual residual events indicate the number of events that the device did not overcome. Estimated residual events represent the number of events that the device did not overcome according to the built-in software. Details of events classification are given below.

All devices responded with increased pressure following the induction of obstructive apneas (Figure 4). Maximum pressures ranged from **12 to 14.2 cmH₂O**, with the **AirSense 11** achieving the highest and the **SleepStyle** the lowest. Time to reach 10 cmH₂O varied considerably, from **3 minutes (G3 A20)** to **14 minutes (DreamStation 2)**.

- **AirSense 11** and **G3 A20** corrected 83% of events, making them the most effective.
- **DreamStation 2** showed the lowest efficacy, correcting only 30% of events.
- Residual apneas were generally overestimated, while residual hypopneas were often underestimated, except by the **Néa**, which overestimated hypopneas (Table 1).
- Most devices returned to 4 cmH₂O baseline post-event, except **PrismaSmart max**, which maintained 6 cmH₂O.

Obstructive Hypopnea

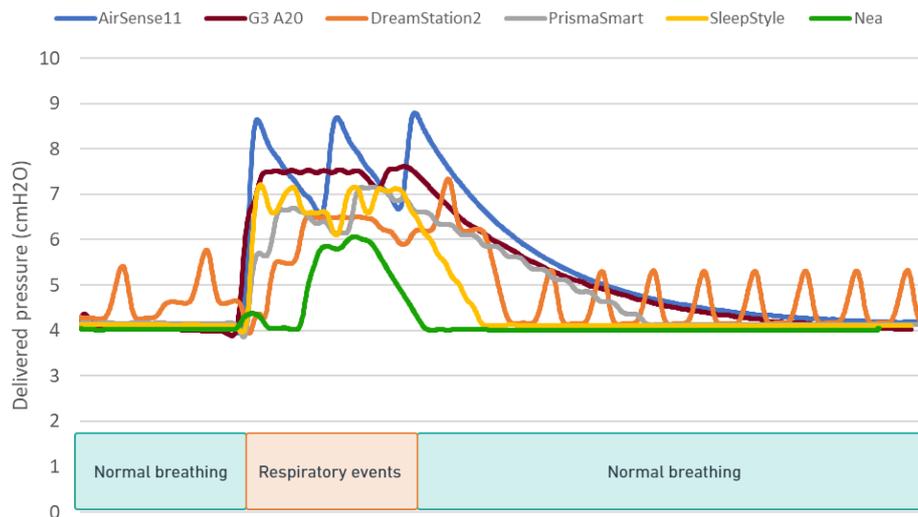


Figure 5: Pressure response of the five tested devices for obstructive hypopnea events.

Obstructive hypopnea events <i>n</i> =11	AirSense 11	G3 A20	Dream Station 2	PrismaSmart max	Sleep Style	Néa
Number of actual residual events (% of corrected events)	3 / 11 (73%)	1 / 11 (91%)	6 / 11 (45%)	1 / 11 (91%)	9 / 11 (18%)	7 / 11 (36%)
Number of estimated residual events (accuracy in %)	1 / 3 (33%)	1 / 1 (100%)	2 / 6 (33%)	1 / 1 (100%)	1 / 9 (11%)	2 / 7 (29%)

Table 2: Agreement between built-in software estimations and test bench objective data for residual obstructive hypopnea events. Actual residual events indicate the number of events that the device did not overcome. Estimated residual events represent the number of events that the device did not overcome according to the built-in software.

All devices responded with increased pressure following the induction of obstructive hypopneas, with peak pressures ranging from 6 to 8.8 cmH₂O (Figure 5).

- **G3 A20** and **PrismaSmart max** demonstrated the highest efficacy, correcting 91% of events.
- **SleepStyle** had the lowest correction rate at only 18%.
- **Airsense 11** reached the highest pressure levels followed by rapid reduction in a repetitive sawtooth pattern.
- Devices varied in accuracy when estimating residual hypopneas. **G3 A20** and **PrismaSmart max** achieved 100% match with bench data (Table 2).
- Only **G3 A20**, **PrismaSmart max**, and **Néa** provided specific classification of hypopneas.

Snoring

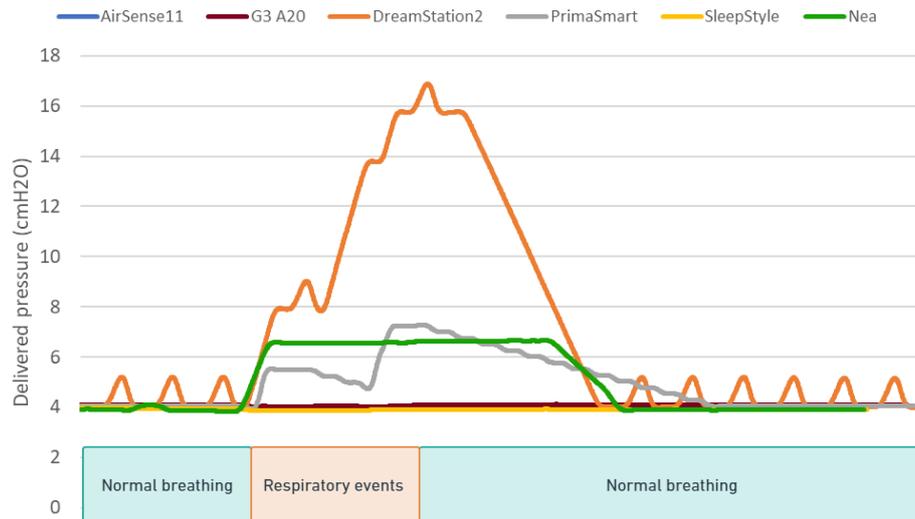


Figure 6: Pressure response of the five tested devices for snoring events.

Pressure responses to simulated snoring varied widely (Figure 6) ranging from 4 to 17 cm H₂O.

- **DreamStation 2** delivered the highest pressure (up to 17 cmH₂O), while **PrismaSmart max** and **Néa** had modest increases.
- Snoring detection methods varied:
 - **AirSense 11** provided a snoring index.
 - **DreamStation 2** and **PrismaSmart max** reported vibrational snoring events.
 - **Nea** visually tagged individual breaths.
 - **SleepStyle** and **G3 A20** did not report snoring events.

All devices returned to 4 cmH₂O baseline after the simulation.

Central Apnea

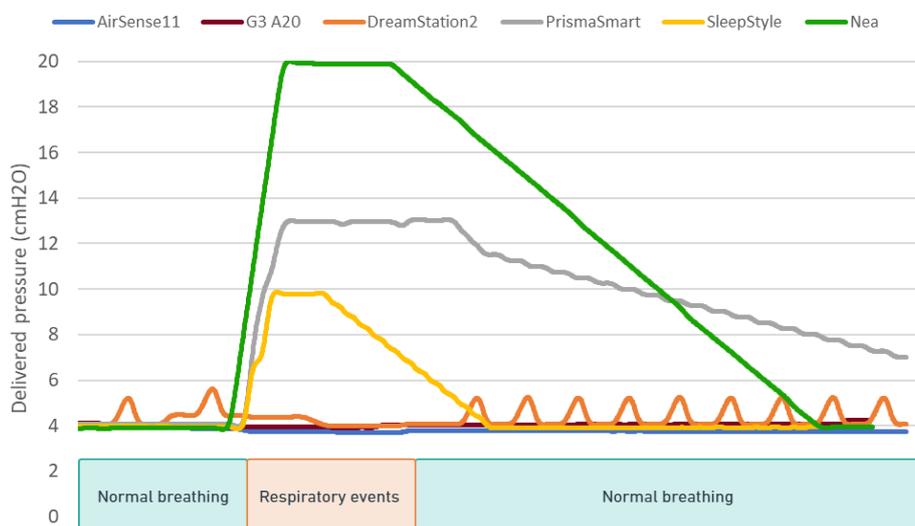


Figure 7: Pressure response of the five tested devices for central apnea events.

Central apnea events <i>n=48</i>	AirSense 11	G3 A20	Dream Station 2	PrismaSmart max	Sleep Style	Néa
Estimated number of events (accuracy in %)	48 (100%)	48 (100%)	48 (100%)	48 (100%)	48 (100%)	48 (100%)
Central origin (n, %)	48 (100%)	48 (100%)	48 (100%)	0 (0%)	45 (94%)	0 (0%)

Table 3: Built-in software reported data on the number of central apnea events and ability to categorize them as central. *The central respiratory events could not be corrected by the devices by increasing the pressure with the test bench. Consequently, the number of events processed is not shown in the table above. Instead, the nature/origin of the event as detected by the built-in software is presented.*

All devices detected 100% of respiratory events (Figure 7). However, accuracy in classification varied:

- **AirSense 11, G3 A20, and DreamStation 2** correctly identified all events as central.
- **PrismaSmart max** and **Nea** misclassified all central apneas as obstructive.
- **SleepStyle** correctly identified 94% of central events (Table 3).

Pressure increases in response to central apneas were observed in:

- **Néa** rapidly reached maximum pressure before returning to baseline.
- **PrismaSmart max** reached 13 cmH₂O and sustained elevated pressure.
- **SleepStyle** showed transient increases followed by gradual decline.

Central Hypopnea

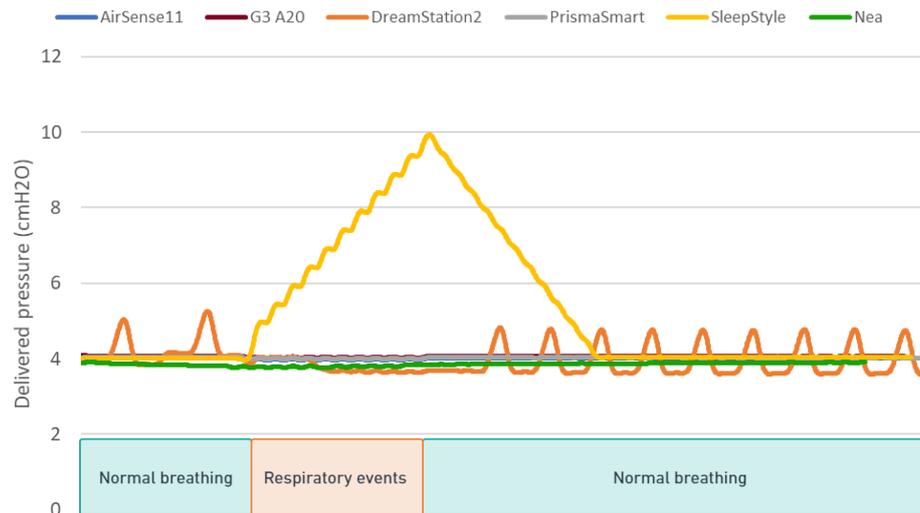


Figure 8: Pressure response of the five tested devices for central hypopnea events.

Central hypopnea events <i>n=11</i>	AirSense 11	G3 A20	Dream Station 2	PrismaSmart max	Sleep Style	Nea
Estimated number of events (accuracy in %)	0 (0%)	11 (100%)	2 (18%)	11 (100%)	11 (100%)	11 (100%)
Central origin (n, %)	NS	11 (100%)	NS	11 (100%)	NS	11 (100%)

Table 4: Built-in software reported data on the number of central hypopnea events and ability to categorize them as central (NS: not specified on the software). The central respiratory events could not be corrected by the devices by increasing the pressure with the test bench. Consequently, the number of events corrected is not shown in the table above. Instead, the nature/origin detected by the built-in software is presented.

Only **G3 A20**, **PrismaSmart max**, and **Néa** accurately detected and classified all central hypopneas (Figure 8). These devices maintained minimal pressure adjustments (Table 4).

- **SleepStyle** inappropriately increased pressure during events.
- **AirSense 11** and **DreamStation 2** did not increase pressure but left most events unspecified in the reporting.

Cheyne-Stokes Respiration (CSR)

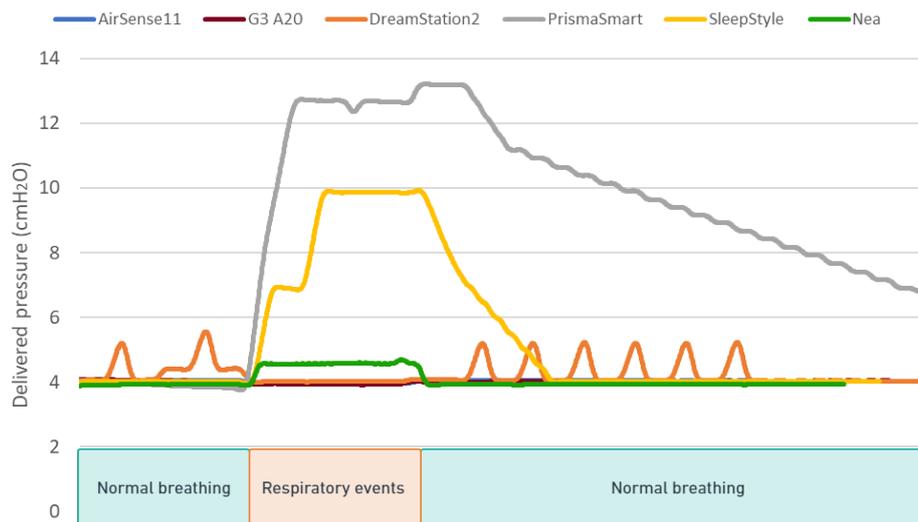


Figure 9: Pressure response of the five tested devices for Cheyne-Stokes events.

Cheyne-Stokes events n=27	AirSense 11	G3 A20	Dream Station 2	PrismaSmart max	Sleep Style	Nea
Estimated number of events (accuracy in %)	27 (100%)	27 (100%)	27 (100%)	27 (100%)	27 (100%)	27 (100%)
Central origin (n, %)	27 (100%)	27 (100%)	27 (100%)	0 (0%)	15 (56%)	27 (100%)

Table 5: Built-in software reported data on the number of Cheyne-Stokes events and ability to categorize them as central. The Cheyne-Stokes events could not be corrected by the devices by increasing the pressure with the test bench. Consequently, the number of corrected events is not shown in the table above. Instead, the nature/origin detected by the built-in software is presented.

All devices successfully detected all Cheyne Stokes Respiration (CSR) events (Figure 9), but accuracy in categorizing them as central varied:

- **AirSense 11, G3 A20, DreamStation 2, and Néa** correctly identified all events as central (Table 5).
- **SleepStyle** identified only 56% as central.
- **PrismaSmart max** did not distinguish CSR as central.

Pressure behavior reflected misclassification:

- **PrismaSmart max** reached 13 cmH₂O and maintained 7 cmH₂O at the simulation's end, similar to its response to central apneas.
- **SleepStyle** was approximately 10 cmH₂O during the event phase with the pressure decreased rapidly at the end of the events period.
- **Néa** reached only 5 cmH₂O, consistent with appropriate classification.

- **DreamStation 2** suppressed its usual pressure pulsing behavior during CSR.

CSR reporting features varied:

- **AirSense 11** indicated time percentage with CSR over 24 hours.
- **G3 A20** indicated periodic breathing with associated event times.
- **PrismaSmart max** displayed timestamped graph annotations.
- **DreamStation 2** indicated periodic breathing (though not verified in this testing).
- **SleepStyle** and **Néa** lacked CSR-specific data display.

Impact of Non-Intentional Leakage on Performance

During Obstructive Apneas

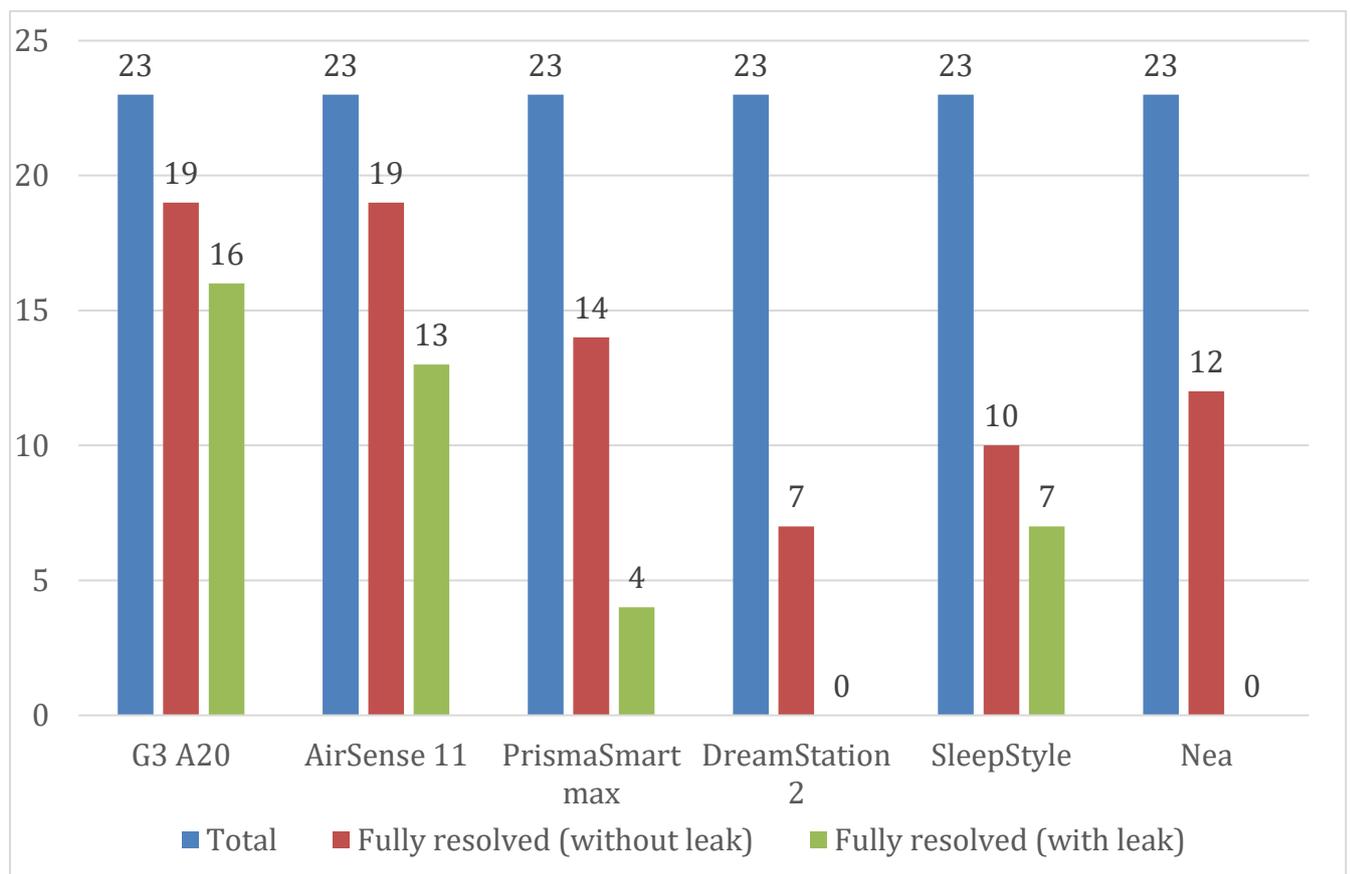


Figure 10: Impact of Non-intentional Leak on Device Performance: OSA with Leak

Leak impacted devices to varying degrees during induction of 23 obstructive apnea events:

- **G3 A20:** Minor reduction in performance with 16 out of 23 events fully resolved compared to 19 events without leak, representing a 13% reduction between the two conditions.
- **AirSense 11:** Moderate reduction with 27% fewer events corrected.
- **PrismaSmart max:** Marked reduction with 44% fewer events resolved.

- **DreamStation 2:** Unable to fully resolve any obstructive apneas with leak.
- **SleepStyle:** performance was similar to performance without leak: 10 obstructive apnea events were fully resolved without leaks versus 7 with leaks, and 13 were partially treated without leaks compared to 11 events with leak. The pressure increase was higher to compensate for leak.
- **Néa:** Performance substantially reduced with pressure delivery dropped by 4 cmH₂O.

During Central Apneas

- **AirSense 11** misclassified most central apneas as obstructive or unclassified apneas resulting in pressure increase from 4 to 10 cm H₂O.
- **G3 A20** misclassified some central apneas under leak conditions, resulting in inappropriate pressure increases (up to 10.5 cmH₂O).
- **DreamStation 2** maintained stable pressure despite leak.
- **PrismaSmart max** and **SleepStyle** continued to misclassify central events as obstructive, resulting in increased pressure. **SleepStyle** also had a delay in returning to baseline pressure after events.
- **Néa** testing under central apnea with leak was not performed due to already maximal pressure delivery under baseline conditions.

DISCUSSION

This study demonstrates the heterogeneous performance of auto-adjusting positive airway pressure (APAP) devices, even under standardized conditions involving clearly defined obstructive and central respiratory events. Despite a controlled bench setting, notable differences were observed in how each device detected and responded to obstructive apneas, hypopneas, snoring, and central events, particularly in the presence of non-intentional leak. These findings align with prior bench studies highlighting variability in algorithmic behavior and pressure response among APAP systems (11,15–18).

Devices with faster pressure response slopes, such as the AirSense 11 and G3 A20, were generally the most effective at correcting obstructive apneas. The AirSense 11 reached peak pressures exceeding 14 cmH₂O before stabilizing between 13-14 cm H₂O, raising concern for pressure-induced instability and arousals. In contrast the G3 A20 had efficacy equal to Airsense 11 in correcting obstructive apneas, but with lower delivered pressures. Devices like the G3 A20 and PrismaSmart max demonstrated high efficacy in correcting obstructive hypopneas without excessive pressurization. With its sawtooth pressure delivery pattern during obstructive hypopnea induction, the AirSense 11 appeared to inconsistently match therapeutic needs: it alternately overshot and undershot target pressures resulting in the highest overall delivered pressures among the devices tested.

Hypopnea simulation posed an additional challenge. This study applied a more stringent threshold of >60% flow reduction, more severe than the >30% criterion outlined in AASM guidelines (29) to minimize false positives. Even so, device performance in detecting and managing hypopneas varied widely, underscoring the limitations of current algorithms under non-standardized hypopnea definitions.

In response to snoring, the DreamStation 2 applied disproportionately high pressures, suggesting potential misclassification of vibratory snoring as obstructive events. In contrast, the PrismaSmart max and Néa responded more conservatively, aligning better with the known association between

snoring and flow limitation (21). While snoring itself does not always require treatment, over-response could contribute to arousals.

Several devices also exhibited inappropriate pressure increases during central apneas (Néa, PrismaSmart max, and SleepStyle) and Cheyne-Stokes respiration (PrismaSmart and SleepStyle) due to misclassification of central events as obstructive. This misidentification could potentially exacerbate central apnea or induce treatment-emergent central sleep apnea (19,22). These limitations emphasize the need for more accurate differentiation algorithms in APAP devices—particularly as they are increasingly used outside of sleep laboratories, where real-time clinical oversight is limited.

Notably, real-world device performance may differ from bench testing results due to advanced sensing modalities (e.g., forced oscillation technique, pulse wave analysis) that are not fully represented in simulated environments. Nevertheless, bench studies like this offer valuable insights into baseline algorithm behavior under controlled and reproducible conditions.

The strengths of this study include the use of a dynamic lung simulator to model real-time respiratory event transitions, the inclusion of both obstructive and central phenotypes, and the assessment of device behavior under variable leak conditions. These features offer a comprehensive, objective comparison of devices without patient-level confounders.

This work also has limitations. Bench testing does not account for individual patient variability, such as anatomical differences or unique breathing patterns, which can affect device performance in clinical settings. Also, there was use of only one representative breathing pattern per event type, and a single leak profile that may not reflect real-world leak variability and intensity. The stricter hypopnea definition may also challenge device algorithms differently than clinical scenarios.

CONCLUSION

This study confirms significant variability in the performance of commercially available APAP devices in their ability to detect and respond appropriately to different respiratory events—including obstructive apneas, hypopneas, central apneas, snoring, and Cheyne-Stokes respiration. These differences have practical implications for therapy effectiveness, patient comfort, and long-term adherence.

According to the 2019 AASM guidelines, home-based APAP therapy initiation is a viable alternative to in-lab titration for patients without major comorbidities, with comparable outcomes (23). These guidelines also emphasize the importance of early telemonitoring interventions to optimize adherence. For APAP to be effective in this paradigm, it must accurately identify respiratory event types and adapt pressure in real time, even in the presence of non-intentional leak.

Over the past two decades, APAP devices have contributed significantly to individualized care and improved compliance. However, as new devices and algorithms continue to enter the market, clinicians should remain vigilant regarding their varied capabilities and limitations. Selection of an APAP device should consider the patient's specific sleep-disordered breathing phenotype, likelihood of central events, and tolerance to pressure fluctuations.

Ongoing research, including both bench and clinical validation, is essential to guide informed device selection and to support innovation in algorithm development for improved patient outcomes.

References

1. Lévy, P., Kohler, M., McNicholas, W. T., Barbé, F., McEvoy, R. D., Somers, V. K., & Pépin, J.-L. (2015). Obstructive sleep apnoea syndrome. *Nature Reviews Disease Primers*, 1, 15015. <https://doi.org/10.1038/nrdp.2015.15>
2. Sullivan, C. E., Issa, F. G., Berthon-Jones, M., & Eves, L. (1981). Reversal of obstructive sleep apnoea by continuous positive airway pressure applied through the nares. *The Lancet*, 1(8225), 862–865. [https://doi.org/10.1016/S0140-6736\(81\)92140-1](https://doi.org/10.1016/S0140-6736(81)92140-1)
3. Weaver, T. E., Maislin, G., Dinges, D. F., Bloxham, T., George, C. F. P., Greenberg, H., & Pack, A. I. (2007). Relationship between hours of CPAP use and achieving normal levels of sleepiness and daily functioning. *Sleep*, 30(6), 711–719. <https://doi.org/10.1093/sleep/30.6.711>
4. Marin, J. M., Agustí, A., Villar, I., Forner, M., Nieto, D., Carrizo, S. J., & Barbé, F. (2012). Association between treated and untreated obstructive sleep apnea and risk of hypertension. *JAMA*, 307(20), 2169–2176. <https://doi.org/10.1001/jama.2012.3418>
5. Weaver, T. E., & Grunstein, R. R. (2008). Adherence to continuous positive airway pressure therapy: The challenge to effective treatment. *Proceedings of the American Thoracic Society*, 5(2), 173–178. <https://doi.org/10.1513/pats.200708-119MG>
6. Bachour, A., & Maasilta, P. (2004). Mouth breathing compromises adherence to nasal continuous positive airway pressure therapy. *Chest*, 126(4), 1248–1254. <https://doi.org/10.1378/chest.126.4.1248>
7. Valentin, A., Subramanian, S., Quan, S. F., Berry, R. B., & Parthasarathy, S. (2011). Air leak is associated with poor adherence to AutoPAP therapy. *Sleep*, 34(6), 801–806. <https://doi.org/10.5665/SLEEP.1054>
8. Borel, J. C., Tamisier, R., Dias-Domingos, S., Sapene, M., Martin, F., Stach, B., & Lévy, P. (2013). Type of mask may impact continuous positive airway pressure adherence in apneic patients. *PLOS ONE*, 8(5), e64382. <https://doi.org/10.1371/journal.pone.0064382>
9. Bachour, A., Hurmerinta, K., & Maasilta, P. (2004). Mouth closing device (chinstrap) reduces mouth leak during nasal CPAP. *Sleep Medicine*, 5(3), 261–267. <https://doi.org/10.1016/j.sleep.2003.11.004>
10. Lebret, M., Martinot, J. B., Arnol, N., Zerillo, D., Tamisier, R., Pépin, J. L., & Lévy, P. (2017). Factors contributing to unintentional leak during CPAP treatment: A systematic review. *Chest*, 151(3), 707–719. <https://doi.org/10.1016/j.chest.2016.09.027>
11. Farré, R., Montserrat, J. M., Rigau, J., Trepal, X., Pinto, P., & Navajas, D. (2002). Response of automatic continuous positive airway pressure devices to different sleep breathing patterns. *American Journal of Respiratory and Critical Care Medicine*, 166(4), 469–473. <https://doi.org/10.1164/rccm.2111050>
12. Gao, W., Jin, Y., Wang, Y., Sun, M., Chen, B., Zhou, N., & Shi, Y. (2011). Is automatic CPAP titration as effective as manual CPAP titration in OSAHS patients? A meta-analysis. *Sleep and Breathing*, 16(2), 329–340. <https://doi.org/10.1007/s11325-011-0514-2>
13. West, S. D., Jones, D. R., & Stradling, J. R. (2006). Comparison of three ways to determine and deliver pressure during nasal CPAP therapy for obstructive sleep apnoea. *Thorax*, 61(3), 226–231. <https://doi.org/10.1136/thx.2005.046300>
14. Ip, S., D'Ambrosio, C., Patel, K., Obadan, N., Kitsios, G. D., Chung, M., & Balk, E. M. (2012). Auto-titrating versus fixed continuous positive airway pressure for the treatment of obstructive sleep apnea: A systematic review with meta-analyses. *Systematic Reviews*, 1(1), 20. <https://doi.org/10.1186/2046-4053-1-20>
15. Isetta, V., Navajas, D., Montserrat, J. M., & Farré, R. (2015). Comparative assessment of several automatic CPAP devices' responses: A bench test study. *ERJ Open Research*, 1(1), 00031-2015. <https://doi.org/10.1183/23120541.00031-2015>

16. Hirose, M., Honda, J., Sato, E., Shinbo, T., Kokubo, K., Ichiwata, T., & Hattori, N. (2008). Bench study of auto-CPAP devices using a collapsible upper airway model with upstream resistance. *Respiratory Physiology & Neurobiology*, 162(1), 48–54. <https://doi.org/10.1016/j.resp.2008.03.014>
17. Zhu, K., Aouf, S., Roisman, G., & Escourrou, P. (2016). Pressure-relief features of fixed and autotitrating continuous positive airway pressure may impair their efficacy: Evaluation with a respiratory bench model. *Journal of Clinical Sleep Medicine*, 12(3), 385–392. <https://doi.org/10.5664/jcsm.5590>
18. Abdenbi, F., Chambille, B., & Escourrou, P. (2004). Bench testing of auto-adjusting positive airway pressure devices. *European Respiratory Journal*, 24(4), 649–658. <https://doi.org/10.1183/09031936.04.00133703>
19. Nigam, G., Pathak, C., & Riaz, M. (2016). A systematic review on prevalence and risk factors associated with treatment-emergent central sleep apnea. *Annals of Thoracic Medicine*, 11(3), 202–210. <https://doi.org/10.4103/1817-1737.185761>
20. Berry, R. B., Abreu, A. R., Krishnan, V., Quan, S. F., Strollo, P. J., & Malhotra, R. K. (2022). A transition to the American Academy of Sleep Medicine–recommended hypopnea definition in adults: Initiatives of the Hypopnea Scoring Rule Task Force. *Journal of Clinical Sleep Medicine*, 18(5), 1419–1425. <https://doi.org/10.5664/jcsm.9952>
21. Ayappa, I., Norman, R. G., Hosselet, J. J., Gruenke, R. A., Walsleben, J. A., & Rapoport, D. M. (1998). Relative occurrence of flow limitation and snoring during continuous positive airway pressure titration. *Chest*, 114(3), 685–690. <https://doi.org/10.1378/chest.114.3.685>
22. Zhang, J., Wang, L., Guo, H. J., Wang, Y., Cao, J., & Chen, B. Y. (2020). Treatment-emergent central sleep apnea: A unique sleep-disordered breathing. *Chinese Medical Journal*, 133(22), 2721–2730. <https://doi.org/10.1097/CM9.0000000000001125>
23. Patil, S. P., Ayappa, I. A., Caples, S. M., Kimoff, R. J., Patel, S. R., & Harrod, C. G. (2019). Treatment of adult obstructive sleep apnea with positive airway pressure: An American Academy of Sleep Medicine clinical practice guideline. *Journal of Clinical Sleep Medicine*, 15(2), 335–343. <https://doi.org/10.5664/jcsm.7640>