Adaptive servoventilation devices are marketed to overcome sleep disordered breathing with apneas and hypopneas of both central and obstructive mechanisms often experienced by patients with chronic heart failure. The clinical efficacy of these devices is still questioned.

**Study Objectives:** This study challenged the detection and treatment capabilities of the three commercially available adaptive servoventilation devices in response to sleep disordered breathing events reproduced on an innovative bench test.

**Methods:** The bench test consisted of a computer-controlled piston and a Starling resistor. The three devices were subjected to a flow sequence composed of central and obstructive apneas and hypopneas including Cheyne-Stokes respiration derived from a patient. The responses of the devices were separately evaluated with the maximum and the clinical settings (titrated expiratory positive airway pressure), and the detected events were compared to the bench-scored values.

**Results:** The three devices responded similarly to central events, by increasing pressure support to raise airflow. All central apneas were eliminated, whereas hypopneas remained. The three devices responded differently to the obstructive events with the maximum settings. These obstructive events could be normalized with clinical settings. The residual events of all the devices were scored lower than bench test values with the maximum settings, but were in agreement with the clinical settings. However, their mechanisms were misclassified.

**Conclusion:** The tested devices reacted as expected to the disordered breathing events, but not sufficiently to normalize the breathing flow. The device-scored results should be used with caution to judge efficacy, as their validity depends upon the initial settings.

**Keywords:** obstructive apnea, central apnea, Cheyne-Stokes respiration, complex sleep apnea, adaptive servoventilation

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**BRIEF SUMMARY**

**Current Knowledge/Study Rationale:** The clinical efficacy of adaptive servoventilation devices in patients with central/complex sleep apnea is still questioned. An innovative bench test was used to challenge the detection and treatment capabilities of the three commercially available devices.

**Study Impact:** All apneas were eliminated, whereas hypopneas remained. The treatment efficacy depends partly on the device settings. The device-scored results should be used with caution to judge efficacy.

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METHODS

Materials
The principle of our bench model is illustrated in Figure 1. The system was mainly composed of a computer-controlled piston and a Starling resistor. It was able to reproduce normal and disordered breathing patterns, e.g., hypopnea and apnea, of obstructive and central mechanisms.

Flow Generator
The flow generator consisted of a 2-litre cylinder and a computer-controlled piston made from a carbon rod driven by a stepper motor (86HS35, Soprolec, Yvré-l’Évêque, France). The microcontroller received a driving flow signal in real time and transformed it into velocity and position data that directly drove the piston. In the meantime, the movement of the piston was monitored by measuring the angular position of the motor with a rotary encoder (AS5045, Austriamicrosystems AG, Premstätten, Austria).

Starling Resistor
A similar system to that described by Abdenbi et al.9 was set in the bench. In short, a cylindrical transparent chamber (200 mm long, 60 mm external diameter) containing a compliant rubber tube was connected to 2 pumps (KMP830KNDC, KNF Neuberger GmbH, Freiburg, Germany), which supplied continuous positive and negative pressures and controlled the pressure surrounding the tube inside the chamber (Pch). The opening state of the rubber tube representing the pharynx could be regulated by Pch. The Pch was monitored by a Honeywell DC001NDC4 pressure transducer (Honeywell Inc., Minneapolis, MN, USA).

Patient Circuit
The servoventilation device to be tested was connected to a conventional leak port (4 mm diameter) which was built in the patient circuit.9 A pneumotachograph (PN279331, Hamilton Medical, Bonaduz, Switzerland) was connected downstream to the mask, and the airflow was measured by a differential pressure transducer (Honeywell DC001NDC4). In addition, another 2 pressure sensors (Honeywell DC030NDC4) were placed in the circuit to record the mask pressure (Pm) and the tracheal pressure (Ptr). A balloon (Test lung 190, Maquet, Rastatt, Germany; Maximum volume 1 L, compliance = 0.03 L/cm H2O at 10 cm H2O pressure) was placed in parallel with the piston to adapt the compliance of the system to the lung of heart failure patients, which is often decreased compared to normal lungs.24 The signals measured at the mask had amplitudes and waveforms similar to patient curves (Figure 2).

All the recorded signals were sampled at 64 Hz for further analysis. A specific graphic user interface was developed.

Patient Signal
The breathing flow sequence that drove the piston was adapted from the actual flow signal recorded in a patient during a previous night. This flow was acquired by a calibrated Fleisch pneumotachograph placed in the breathing circuit, using a CID102L8 system (CIDELEC, Sainte Gemmes sur Loire, France) with a sampling frequency of 64 Hz.

Breathing Patterns
In order to mimic the respiration of a patient with coexisting central and obstructive sleep apneas and hypopneas, which is often the case in patients with heart failure,16 the reproduced flow sequence consisted of the following breathing patterns: normal breathing, and apneic/hypopneic patterns of central and obstructive mechanisms (Figure 3A).

The global breathing rate was set at 10 breaths/min (6 sec/breath), and the normal breathing amplitude with the servovoventilation device turned on was considered as the baseline. Central apneas (CA) and central hypopneas (CH) had a pattern of Cheyne-Stokes respiration (CSR) with maximum flow amplitude twice the baseline.25 Each apnea/hypopnea lasted 12 sec (equivalent to 2 breaths), and the corresponding flow amplitude decreased to 0 (i.e., the piston ceased moving completely) and 30% of the baseline, respectively. The duration of each CSR cycle was set at 1 minute (Figure 2). During either normal breathing or central events, the chamber pressure of the Starling resistor was maintained negative at -10 cm H2O and the pharyngeal tube was in an open state.

Obstructive events were induced by a total or partial collapse of the pharyngeal tube in the Starling resistor; meanwhile, the piston kept repeating normal breathing movements. A positive pressure of 9 cm H2O was imposed on the pharyngeal tube during obstructive apneas (OA). This pressure was reduced to -1 cm H2O during obstructive hypopneas (OH) in order to partially open the pharyngeal tube. Each obstructive breathing pattern lasted 24 sec (equivalent to 4 breaths) and occurred every one minute.

Reproducibility Study
The reproducibility of the bench test was evaluated using a driving sequence composed of the following breathing patterns (5 events for each): normal breathing, CA, OA, CH, and OH. The sequence was repeated 3 times on the bench without
any connection to servoventilation device. \( P_{m} \), \( P_{tr} \), and mask flow \( V' \) were measured at the peak of the inspiratory phase for each pattern if applicable. The peak-to-peak amplitude of \( V' \) (\( \Delta V' \)) was derived by calculating the upper and lower envelopes of the \( V' \) curve. One-way analysis of variance (ANOVA) was applied for comparisons. Results are shown in Table 1. There was no significant difference between the tests for \( \Delta V' \), except in \( OH \) where the coefficient of variation of measurement was 4%.

### Devices under Study and Related Software

Three commercially available ASV devices were included in the current study. Analysis reports of devices were available through the related software. Devices and corresponding software were as follows (device/software): BiPAP autoSV Advanced System One (V1.02) S/N: P03178168874C/EncorePro2 (ver. 2.8.6.4) (Philips Respironics, Murrysville, PA, USA); S9 AutoSet CS (device without auto-adjusting EPAP, which is similar to the S9 VPAP Adapt used in ASV mode with fixed EPAP, currently marketed in the United States) (SW: SX474-0903) S/N: 22112066964/ResScan (ver. 03.16.018) (Resmed, Sydney, Australia); SOMNOvent CR (V5.00) S/N: 12647/WEINMANNsupport (ver. 1.11SP1) (Weinmann, Hamburg, Germany). The operations of each device were previously described in the literature.\(^{18,26,27}\) The 3 devices are denoted as D1, D2, and D3, respectively. Two types of settings were applied: “maximum” and “clinical” for each device (Table 2). In the maximum settings, the pressure values were selected...
as the maximum values available on each device according to the manufacturer’s data. In the clinical settings, to mimic the titration method for ASV devices used in most clinical protocols, the expiratory positive airway pressure (EPAP) value was chosen as the pressure relieving the obstructive apneas, i.e., opening the pharyngeal tube at a luminal pressure.
equal to 9 cm H\textsubscript{2}O, and the maximum pressure support (PS) was chosen as 8 cm H\textsubscript{2}O.

**Protocol**

Each device was connected to the circuit with its own tubing, and was turned on after several breaths, then subjected to the sleep disordered breathing (SDB) flow sequence. The test sequence started with a 5-min normal breathing session of acclimatization, which was followed in turn by the SDB sessions composed of the previously described patterns during 1 h, including 10-min CA, 10-min OA, 5-min OH, 5-min CH, 10-min normal breathing, 10-min CA, and 9-min CH (Figure 3A). The scoring of the residual SDB events was performed over the 0- to 63.5-min period. Tests were repeated 3 times for each device setting to ensure reproducibility.

During each test, P\textsubscript{m}, V', and P\textsubscript{tr} were recorded to evaluate the efficiency of the therapy. PS values were derived from the measured expiratory and inspiratory positive airway pressures (EPAP and IPAP): PS = IPAP-EPAP. By analyzing the V’ curve, residual SDB events could be scored and compared to the original events. Moreover, the respiratory phase was indicated by the piston position and revealed the patient-device synchrony when compared to the PS curve of the device.

**Data Analysis**

To analyze the residual events with the therapy, for each test, the airflow amplitude (ΔV') was computed and normalized. Results were presented in percentage of the baseline. Residual events were scored by considering both the amplitude reduction and the corresponding duration, i.e., flow amplitude ≤ 10% of initial baseline: apnea; 10% < flow amplitude ≤ 70% of baseline: hypopneas. The duration of events is ≥ 10 sec, according to the guidelines of AASM.\textsuperscript{25} All analyses were carried out with MATLAB (MathWorks Inc., Natick, MA, USA).

**Statistical Analysis**

Data are expressed as mean ± standard deviation (SD). One-way ANOVA was applied to compare (1) the numbers of residual events between the devices; (2) the bench- and device-scored (device reports) SDB events for each device; and (3) the residual events between the devices for each SDB event. A two-way ANOVA with the factor device and the factor scor-

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**Figure 4**—Examples of the results with clinical settings: measured mask pressure and mask flow

(A) Flow of the reference test without ASV device; (B) results of BiPAP autoSV Advanced; (C) results of S9 AutoSet CS; (D) results of SOMNOvent CR. The framed parts numbered 5 and 6 are details in Figure 7E and 7F. NB, normal breathing; CA, central apnea; OA, obstructive apnea; OH, obstructive hypopnea; CH, central hypopnea. V', measured mask flow; P\textsubscript{m}, measured mask pressure.
ing method was conducted. The correspondence of breathing events between the 2 scoring methods was also investigated. A probability level < 0.05 was considered significant. Statistical analyses were performed using MedCalc statistical software (MedCalc Software, Mariakerke, Belgium).

RESULTS

Responses of Devices
Examples of the measured $V'$ and $P_m$ for each device with maximum and clinical settings are shown in Figures 3 and 4, respectively.

Pressure Responses
Figure 5 summarizes the maximum pressures reached by the devices during the tests. Maximum settings: For all the devices, PS rose with CA and then dropped when subjected to OA and to normal breathing (Figure 3). D2 reached the highest value of PS (Figure 3). D3 did not respond to the first 2 CA events (Figure 3D). On the other hand, D1 and D3 increased the EPAP during OA periods, while D2 did not. D1 reached a maximum EPAP value at the end of the obstructive disordered breathing phase and did not drop the pressure during the following central events. D3 showed the highest EPAP response and still increased the EPAP during the central events.

Clinical settings: D1 and D2 kept the EPAP at the pre-set value of 9 cm H$_2$O during the whole test (Figure 4). D3 started with a higher EPAP value than the setting, at about 12 cm H$_2$O. The highest EPAP values in D3 were observed during central SDB events. Besides, all the devices increased PS during CA and CH, where they reached their maximum values (Figure 4). These maximum PS values were slightly different between devices although the setting was 8 cm H$_2$O for all of them (Figure 5).
**Residual Flow Patterns**

At maximum settings: during CA, the flow amplitudes ($\Delta V'$) reached around 53% (D1), 52% (D2), and 43% (D3) of their baseline values, remaining below the normal baseline. Therefore, all CA were eliminated but most of them were converted to CH (Figure 6A). Original CH were partially normalized only by D2 (Figure 6A).

During the OA sequence, in D1, the EPAP did not rise high and fast enough to totally open the upper airway; therefore, OH remained. For D2, OH remained despite the rise in PS. D3 was able to suppress obstructive events at the end of the OA section with a higher EPAP level. Therefore, all OA were corrected with all the devices, but most of them were transformed to hypopneas. As a consequence, OH remained unchanged or even increased (Figure 3A). Noteworthy, the original OH sequences were fully corrected with all the devices (data not shown).

With the clinical settings, for CA, results similar to that obtained with the maximum settings were recorded: all the apneas were eliminated. All the CA were converted to CH. Original CH were not corrected with any device (Figure 6B).

D2 and D3 fully eliminated the OA and OH. Only D1 still left significant residual OH ($p < 0.01$ compared to D2 and D3). However, there was no difference in the residual OA, CA, and CH events among the devices (Figure 6B).

**Patient-Device Synchrony**

During normal breathing, the PS applied by D1 and D3 perfectly matched the rhythm of the movement of piston, whereas D2 showed double-triggering resulting in the doubling of the backup rate (Figure 7A). During hyperventilation periods, all 3 devices adequately followed the bench-simulated breathing rate. However, this auto rate differed during apneas: D1 and D3 slowed the frequency, while that of D2 was doubled (Figure 7B-7D). The maximum and clinical settings showed the same backup rate patterns.

**Residual Events on the Bench and Comparison of the Bench- and Device-Scored Residual Events**

The results of comparisons are shown in Figure 8. With the maximum device settings, all the devices reduced the number of SDB events over a 63.5-min test period, but D2 was significantly more efficient than the others ($p < 0.01$); nevertheless, none could completely suppress all the events. Two-way ANOVA revealed significant differences for the overall number of residual events between the bench test and the device reports ($p < 0.001$), and between the devices ($p < 0.001$ D2 compared to D1 and D3). For all the devices, the scoring results in the reports were lower than computed from the bench test. Average differences were, respectively, 10.7, 21.0, and 10.6 events for D1, D2, and D3 ($p < 0.01$, $p < 0.01$, $p < 0.05$; Figure 8A), approximately corresponding to the same index value per hour, as the sequence lasted 63.5 minutes.

The total number of SDB events was also reduced when the clinical settings were applied. D1 showed a higher residual event number than the others ($p < 0.05$). The scoring results in the device reports were similar to those computed from the bench test, except D1 that underestimated 3 events on average ($p < 0.05$; Figure 8B).

**Figure 6**—Residual disordered breathing events observed on the bench throughout a period of 63.5 min for the three devices with two device settings

<table>
<thead>
<tr>
<th>A. Maximum settings</th>
<th>B. Clinical settings</th>
</tr>
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<td><img src="image" alt="Graph A" /></td>
<td><img src="image" alt="Graph B" /></td>
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(A) Maximum settings, **$p < 0.01$** compared to D1 and D3. (B) Clinical settings, **$p < 0.01$** compared to D2 and D3. D1, BiPAP autoSV Advanced; D2, S9 AutoSet CS; D3, SOMNOvent CR; Ref., scoring result of the reference test.

**DISCUSSION**

This study has presented an innovative bench model that enables mimicking a patient breathing flow sequence, including all types of SDB patterns, such as apnea and hypopnea of...
Figure 7—Details of the framed parts in Figures 3 and 4

A. 1

\[
\begin{array}{c}
V' (L/s) \\
\hline
1 \\
-1 \\
\end{array}
\quad
\begin{array}{c}
P_m (cmH_2O) \\
\hline
25 \\
0 \\
\end{array}
\quad
\begin{array}{c}
0 \\
-1 \\
1 \\
\end{array}
\]

B. 2

\[
\begin{array}{c}
V' (L/s) \\
\hline
1 \\
-1 \\
\end{array}
\quad
\begin{array}{c}
P_m (cmH_2O) \\
\hline
25 \\
0 \\
\end{array}
\quad
\begin{array}{c}
0 \\
-1 \\
1 \\
\end{array}
\]

C. 3

\[
\begin{array}{c}
V' (L/s) \\
\hline
1 \\
-1 \\
\end{array}
\quad
\begin{array}{c}
P_m (cmH_2O) \\
\hline
25 \\
0 \\
\end{array}
\quad
\begin{array}{c}
0 \\
-1 \\
1 \\
\end{array}
\]

D. 4

\[
\begin{array}{c}
V' (L/s) \\
\hline
1 \\
-1 \\
\end{array}
\quad
\begin{array}{c}
P_m (cmH_2O) \\
\hline
25 \\
0 \\
\end{array}
\quad
\begin{array}{c}
0 \\
-1 \\
1 \\
\end{array}
\]

E. 5

\[
\begin{array}{c}
V' (L/s) \\
\hline
1 \\
-1 \\
\end{array}
\quad
\begin{array}{c}
P_m (cmH_2O) \\
\hline
25 \\
0 \\
\end{array}
\quad
\begin{array}{c}
0 \\
-1 \\
1 \\
\end{array}
\]

F. 6

\[
\begin{array}{c}
V' (L/s) \\
\hline
1 \\
-1 \\
\end{array}
\quad
\begin{array}{c}
P_m (cmH_2O) \\
\hline
25 \\
0 \\
\end{array}
\quad
\begin{array}{c}
0 \\
-1 \\
1 \\
\end{array}
\]

Figure 7A corresponds to frame 1 in Figure 3. Consecutively for other panels. With maximum settings: (7A) double-triggering observed during normal breathing in D2 which resulted in the doubling of the backup rate; (7B) D1 slowed down its auto rate during central apnea; (7C) double-triggering observed during central apnea in D2; (7D) D3 slowed down its auto rate during central apnea; (7E, 7F, 7D) The three devices delivered PS as expected during central apnea but the PS patterns differed between devices. With clinical settings: at the same EPAP value with D1 (7E) and D2 (7F), different airflow responses were obtained. The asynchronies were also observed with clinical settings. \( V' \), measured mask flow; \( P_m \), measured mask pressure.

Central and obstructive mechanisms, CSR (observed in numerous patients with heart failure), and CompSA. With this model, three commercially available ASV devices were evaluated and their behaviors were examined. These devices were tested with the maximum and the clinical settings separately, to investigate not only their maximum treatment capacities, but also their use in actual practice. This is important because no precise recommendations exist for the initiation of ASV treatment, and setting modes vary between clinical protocols. Most use the EPAP value previously titrated, while others used default settings or EPAP lower than efficient CPAP.

When subjected to central SDB events, the devices responded similarly: increasing PS to raise the airflow amplitude. All the devices eliminated central apneas, while hypopneas still remained, most resulting from alleviated apneas. However, they reacted differently to obstructive events with the maximum settings; whereas obstructive apneas were fully normalized by all devices using a previously titrated EPAP in the clinical settings. The number of device-scored residual events was underestimated with maximum settings for all devices, whereas this value was in agreement with the bench value with clinical settings. Moreover, the devices failed to correctly classify the mechanisms of the residual events. Therefore, the three tested devices differed from each other, and their algorithms still remained to be improved.

In most cases, ASV device algorithms that detect SDB events and determine pressure response are not explicitly described and are considered the property of the manufacturer. No published study has systematically assessed and compared such devices with specifically controlled SDB patterns. The current bench model thus allows testing device responses, as well as the residual respiratory event scoring ability of the ASV devices. This may reveal the reason for potential failures of the ASV treatment.

On the response side, from the obtained \( P_m \) results, the IPAP increased when a respiratory disturbance appeared (Figure 7B-7D) and fell during hyperventilation or normal breathing, responding to different strategies of event-detection of the manufacturers: D1 analyzed average peak flow in a 4-minute moving window and D2 concentrated on the minute-ventilation in the last 3 minutes. The strategy of D3 was not clearly described previously. Generally, all the three devices responded immediately to the respiratory events, regardless of algorithm or settings (Figure 3, 4).

When subjected to CSR and central respiratory events, the difference between IPAP and EPAP increased corresponding to increased PS. Clearly, the airflow response was largely dependent upon the PS pattern. Pressure amplitude, waveform, and duration seem to be the main factors affecting airflow and delivered volume. From Figure 7B-7D, it is apparent that each device delivered different PS patterns. First, the EPAP of D1 and D2 kept constant during the CSR cycle and only IPAP increased, while D3 also reduced its EPAP during the same cycle to enlarge the PS amplitude. Second, the pressure amplitude and its evolution also depended on the device. D2 and D3 reached their respective maximum PS value at the 9th, the 5th, and the 8th apnea event. Last, the devices applied different PS
waveforms, such as square wave for D1 and decaying exponential for D2,29 and their pressure rise time was different. These characteristics may explain why D1 and D2 resulted in different flow responses in the same conditions of EPAP and PS values (Figure 7E, 7F).

Although the manufacturers claimed that the auto backup rate of the devices during apneas applied patient’s specific rate, this point was not strictly accurate according to our observation: since the breathing rate on the bench was fixed at 10 breaths/min and the apnea period lasted 2 breaths, D1 and D3 slightly slowed their ventilation rate during the apneas (the last PS limb during apnea approached the next, as shown in Figure 7B, 7D, and 7E), whereas the breathing rate of D2 was doubled (Figure 7C, 7F).

When subjected to the obstructive events with maximum device settings, D1 and D3 adjusted their EPAP automatically to overcome the obstructions (Figure 3B, 3D). For D2, the obstruction was partially cleared with high-level PS, as its EPAP remained constant throughout the test. To eliminate the obstructive events in the clinical settings, the minimum EPAP of all the devices was set at the opening pressure of the pharyngeal tube. As a result, obstructive hypopneas remained untreated only with D1 as the EPAP of D1 did not increase (Figure 4B, 5B). It could be observed that with a doubled backup rate, D2 managed to normalize the airflow in all these OAs with a same EPAP and even an inferior PS to that of D1 (Figure 4B, 4C, and 5B). Of note, with a same titrated minimum EPAP value programmed, D3 supplied a higher EPAP pressure than the other two devices up to values which may be deleterious to some heart failure patients (13.2 cm H$_2$O for D3 vs. 9.8 and 9.3 cm H$_2$O for D1 and D2, respectively during the entire tests, Figure 5B).30,31

On the scoring side, the number of residual apneas of the three devices with the maximum settings was roughly the same as the bench value, while D1 and D3 distinguished and slightly overestimated OA (Figure 9A). However, with clinical settings, the devices overestimated apneas, except D1 (Figure 9B). All three devices failed to recognize hypopneas with maximum settings, particularly D2, which showed the largest difference between the bench and the device values and resulted in an underestimation of events (Figure 8A). Conversely, the residual hypopneas were much better scored with clinical settings although some of them were classified as apneas (Figure 9B), and total event numbers agreed with bench values (Figure 8B). This scoring difference may be due to a different definition of baseline breathing (moving window for the device vs. the first normal breathing section for the bench) and to a lower threshold than the recommendations of AASM (70% of the flow amplitude of normal breathing) apparently used by the devices, as observed with the REMstar Auto M-Series (Philips Respironics).32 For example, owing to a reduction of airflow response with clinical settings, D2 scored almost all the hypopneas as respiratory disturbances (Figure 9B D2), compared to a detection rate of 32% of bench-events with maximum settings (Figure 9A D2). The results reported here have clinical implications for the use of the number of residual events on the ASV device reports, which are often considered as efficacy criteria by practitioners.

We observed that the resulting ventilation of the devices was lower than the average normal baseline ventilation as previously described.33 Since actual airflow response was largely dependent upon PS amplitude, the reduction of flow with clinical settings was the consequence of moderate PS, of which the maximum was limited to 8 cm H$_2$O in clinical settings. Of note, flow response also depended upon lung compliance. Since the EPAP was set higher in the clinical settings, the lung faced a higher pressure and its compliance decreased according to its pressure-volume curve.34 In patients with heart failure, a reduction of lung compliance is often apparent.24 Therefore, the algorithms of the ASV devices should also take this point into consideration to allow for normalizing ventilation.

**Limitations of the Study**

ASV devices are targeted to treat patients with complicated SDB, most notably CSR. It is extremely challenging to pro-
vide a true closed-loop model because of the complex interactions associated with the delivered positive airway pressure and the breathing control of patients containing several regulation loops with different gains, e.g., plant gain for the respiratory system and controller gains for O$_2$ and CO$_2$. Therefore our bench was working on a closed-loop only in response to obstructive events. In this condition, the ASV device reacted to the obstruction of the upper airway and modified the collapsibility of the pharyngeal tube. However, the device only modified measured ventilation when subjected to central SDB events, without considering any chemoreceptor function. That is why the simulated hyperventilation during CSR could not be mitigated, but this situation may apply to patients with severe disordered breathing control. In addition, the paucity of patient-device synchrony observed here might not appear in real patients, since the breathing rate may be stabilized by ASV treatment in closed-loop conditions.

In the current test, the simulated CSR central apneas lasted 12 seconds and the cycle length of periodic breathing was 60 seconds. The apnea length may be shorter than that in average patients. However, the chemoreflex feedback loop gain of the current respiratory model could be estimated with the durations of the ventilatory phase and of the CSR cycle by using the formula proposed by Sands et al. The loop gain was thus calculated as around 1, which corresponded to a patient with moderate severity of ventilatory instability.

Further, this study only simulated common SDB events with different mechanisms independently, while the combinations of these mechanisms in the same event were not taken into account, such as central apnea with closed upper airway, which sometimes appears in the patients of heart failure. The bench may be very appropriate to test these central apneas with closed airways.

In conclusion, the tested devices reacted to disordered breathing events as expected, but not sufficiently to normalize the breathing flow. The three different devices differed in their treatment efficacy partly depending on the setting used. The device-scored results should be applied with caution to assess efficacy, as event-scoring depends upon the device settings.

Mean values are presented. For each device, the “bench bar” was considered as the reference, showing the distribution of the event count per mechanisms. The “report bar” below showed the distribution of the event count per mechanisms classified on the device reports in relationship with the mechanisms defined on the bench. Of note, the part of “report” that exceeded the range of the reference “bench” (A-D1, B-D1, D2, D3) indicated the number of false detections (normal breathing on the bench) by the device. U, event undetected by the device; A, (unclassified) apnea; CA, central apnea; OA, obstructive apnea; H, (unclassified) hypopnea; CH, central hypopnea; OH, obstructive hypopnea.

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REFERENCES