Response of Home-Use Adaptive Pressure Modes to Simulated Transient Hypoventilation

Frédéric Lofaso, Karl Leroux, Ghilas Boussaid, Hélène Prigent, and Bruno Louis

BACKGROUND: Adaptive servoventilation (ASV) is a recently developed ventilation mode designed to stabilize ventilation in patients with central sleep apnea and Cheyne-Stokes respiration. Alternatively, modes aiming to maintain average ventilation over several breaths, such as average volume-assured pressure support (AVAPS) and intelligent volume-assured pressure support (iVAPS), could be efficient during ventilation instability by reducing central events. These modes are available on a variety of devices. This bench evaluation studied the response of these different modes and devices to simulated transient hypoventilation events. METHODS: Three home ventilation devices operating in ASV modes (AirCurve 10 CS Pacewave, ResMed; DreamStation autoSV, Philips; Prisma CR, Löwenstein) and 2 ventilators with the AVAPS mode (DreamStation BiPAP, Philips; Lumis 150 iVAPS, ResMed) were evaluated during transient central hypopnea/hypoventilation simulations characterized by a constant breathing frequency of 15 breaths/min and a progressive decrease of tidal volume (V_T) from 500 mL to 50 mL, in 18, 12, 9, and 6 breaths, respectively, followed by a progressive return to the baseline at the same rate. RESULTS: The AirCurve 10 CS Pacewave reacted to a V_T decrease between 80% and 50% of baseline V_T. DreamStation BiPAP and Prisma CR reacted when V_T decreased to between 60% and 30% of baseline V_T, whereas the AVAPS response to hypopnea occurred during the crescendo phase of hypopnea/hypoventilation V_T. The iVAPS response was between that of the AirCurve 10 CS Pacewave and the other ASV devices. Among the ASV devices, the minimum V_T was higher with AirCurve 10 CS Pacewave, followed by the Prisma CR and the DreamStation BiPAP. Minimum V_T was not influenced by AVAPS and was improved by iVAPS without outperforming the AirCurve 10 CS Pacewave. Maximum V_T was increased by iVAPS, whereas ASV devices did not induce a significant V_T overshoot. CONCLUSIONS: ASV devices improved central hypopnea/hypoventilation events without inducing hyperpnea events and therefore were better adapted than AVAPS and iVAPS devices, with notable differences in their responses to hypoventilation events. *Key words: bench study; lung model; pressure support; non-invasive ventilation; servomechanism; adaptive servo ventilation.* [Respir Care 2020;65(9):1258–1267. © 2020 Daedalus Enterprises]

Introduction

Average volume-assured pressure support (AVAPS), intelligent volume-assured pressure support (iVAPS), and adaptive servoventilation (ASV) are advanced modes of ventilation that aim to self-adjust pressure support to a patient's respiratory fluctuation in real time.[1-4] ASV is usually recommended for patients exhibiting respiratory flow instability but with a preserved ventilation (ie, normocapnic or hypocapnic), whereas AVAPS and iVAPS are recommended for patients with hypoventilation (ie, hypercapnia).[5] The widespread use of these modes is mainly limited

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by the incomplete understanding of the specific algorithms used to respond to a patient's breathing disorder. While all ASV devices are able to eliminate apneas, hypopneas may persist, mostly resulting from blunted apneas.[6] However, the objective of ASV devices is not to treat central events as soon as they occur, but rather to prevent P_{aO_2} and P_{aCO_2} oscillations and breathing instability. These devices aim to provide an adaptive support that increases inspiratory pressure support when the patient's air flow is waning, and lowers (or stops) respiratory support when the patient returns to a more stable breathing pattern or when they exhibit hyperventilation to dampen the effects of the patient's respiratory drive fluctuations. Finally, to reduce P_{aO_2} and P_{aCO_2} oscillations, which lead to recurrent central events, these devices should reduce hypoventilation as efficiently as possible to avoid the ensuing crescendo of a patient's ventilatory drive without inducing subsequent overassistance and minute ventilation overshoot. Indeed, these overcorrections may lead to an excess of the hypocapnic apnea threshold (ie, the CO_2 level below which central apneas are generated).

The objective of this bench study was to observe the dynamic response of different devices and modes to various decreasing respiratory drive speeds that could result either from central hypopneas or short hypoventilation events. Accordingly, to simulate central hypopnea and hypoventilation, we progressively decreased simulated inspiratory effort and returned to the baseline effort at the same speed without changing the breathing frequency.

QUICK LOOK

Current knowledge

Volume-assured pressure support, intelligent volume-assured pressure support, and adaptive servoventilation are advanced ventilator modes that claim to have the ability to self-adjust pressure support to a patient's respiratory fluctuation in real time.

What this paper contributes to our knowledge

Notable differences existed between devices; specifically, each device's response time to a change in breathing characteristics was markedly different. For this reason, devices, including those in the same category, differed greatly in their potential efficiency to prevent respiratory instability.

Methods

Ventilators Settings

Five ventilators were tested: AirCurve 10 CS Pacewave (ResMed, Saint Priest, France); DreamStation autoSV (Philips Healthcare, Suresnes, France); Prisma CR (Löwenstein Médical, Igny, France); DreamStation BiPAP (Philips Healthcare, Suresnes, France); and Lumis 150 iVAPS (ResMed, Saint Priest, France). The Lumis 150 iVAPS was studied at its most sensitive inspiratory trigger setting, which did not induce auto-triggering. Triggers of other devices were not adjustable. The values for ventilator settings are reported in Table 1. For all devices, PEEP was set at the lowest value (4 cm H_2O). ASV devices utilize automated algorithms to adjust expiratory pressure according to upper airway patency to control obstructive sleep apnea syndrome. Therefore, peak expiratory pressure level was set at 10 cm H_2O. Pressure support was set between the lowest possible value and 15 cm H_2O. With the iVAPS mode of the Lumis 150, the dead space was calculated for a height of 170 cm.

Experimental Bench Study

Each tested ventilator was connected via its standard circuit to the first chamber (ie, the testing chamber) of a 2-chamber test lung (MII Vent Aid TTL; Michigan Instruments, Grand Rapids, Michigan) (Fig. 1). The second chamber of the test lung (ie, the driving chamber) was connected to a flow generator that could produce various wave forms previously stored in a microcomputer. The 2 chambers were physically connected to each other by a small metal component that allowed the driving chamber to lift the testing chamber, mimicking the patient's contribution to inspiration. Because the metal component was not completely attached to the testing chamber, the latter, once pressurization completed, could rise above the driving chamber. Therefore, inspiratory flow and volume measured in the driving chamber depended only on the simulated effort, whereas flow and volume measured in the testing chamber depend on both the simulated effort and the ventilator output when the 2 chambers were connected. However, when the ventilator had to pressurize intensively, the first chamber could be disconnected from the driving chamber. At that moment, the tidal volume (V_T) results only from the ventilator output, mimicking the relaxation of inspiratory muscle before the end of insufflation and therefore inducing a delayed cycling.[7,8] To simulate the mechanical characteristics of the respiratory system, the compliance of the testing chamber was adjusted to 0.03 L/cm H_2O, and a parabolic airway resistance (Pneuflo Airway resistor Rp5, Michigan Instrument) was added between the testing chamber and the tested ventilator.

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Table 1. Ventilator Details and Settings

Mode and Device	Algorithms	PEEP, min/max cm H_2O	PS, min/max cm H_2O	Target V_T, mL/kg
ASV				
AirCurve 10 CS Pacewave	Calculation: The recent 2-min average $\dot{V}$ PS Target: 90% of the average $\dot{V}$ Backup rate: Auto – starting at recent spontaneous rate, adapting during apnea over several breaths to target rate of 15 breaths/min	4/10	0/15	
Prisma CR	Calculation: The recent 2-min average $\dot{V}$ in a moving window giving 50% weight to the previous 2 min Target for PS: Relative $\dot{V}$ of the current breath to the average* Backup rate: A mandatory breath rate applied during apneic events (defined by a drop of 80% of $\dot{V}$), adjusted automatically based on patient's breathing frequency*	4/10	0/15	
DreamStation autoSV	Calculation: The recent 4-min average peak flow PS Target: If no SDB, 95% of mean inspiratory peak flow over the last 4 min. If SDB, 60th percentile of inspiratory peak flow values over the last 4 min Backup rate: Auto – first breath if no recent SDB = inspiratory time + 8 s; first breath if recent SDB = T_{br} + 4 s	4/10	0/15	
VAPS				
DreamStation BiPAP	Calculation: Average set V_T over several breaths PS Target: If target volume differs from average recent ventilation (ie, 6 breaths), PS for next breath is changed at rate of 1 cm H_2O/min (0.5 cm H_2O/min if unstable breathing) Backup rate: Auto – 2 breaths/min below average rate of recent 6 spontaneous breaths	Fixed at 4	2/15	4–5
Lumis 150 iVAPS	Calculation: Estimates patients' VA and breathing frequency PS Target: To achieve and maintain target VA. PS adjustments are made every 0.016s throughout inspiration to achieve goal ventilation with smooth transition Backup rate: Auto – shifts between two thirds of set rate during spontaneous breathing and set rate during periods of apnea	Fixed at 4	0/15	

When settings were adjusted between minimum and maximum values, the devices started using the minimum value and the specific algorithms allowed automatic changes according to the presence of obstructive events for PEEP and hypopnea/hypoventilation for PS between the minimum and maximum setting values.

* No more details are available.

PS = pressure suppor; ASV = adaptive servoventilation; V_T = tidal volume; $\dot{V}$ = minute volume; SDB = sleep-disordered breathing; T_{br} = average time of 12 recent spontaneous breaths; VAPS = volume-assured pressure support; VA = alveolar ventilation

The flow generator, developed by our INSERM laboratory and previously described,[9,10] was built by associating pressurized air, flow measurement, and a servo valve driven by a microcomputer. This allows the continuous adjustment of the servo valve to produce the desired flows and volumes.

Accordingly, the flow generator was initially adjusted, without ventilator connection, to obtain a V_T = 500 mL and a rate of 15 cycles/min. After 10 min of a stable breathing pattern, the flow generator progressively decreased V_T to 50 mL in 18, 12, 9, and 6 cycles, and then returned V_T to 500 mL at a similar rate as the previous decrescendo. Figure 2 shows these respiratory events by presenting the inspiratory flow, which entered in the driving chamber created by the flow generator simulating the patient. To appreciate the response of the different ventilators to this breathing pattern variation, we repeated this experimental sequence with each ventilator successively connected to the first chamber of the test lung setup.

Measurements

Airway pressure was measured with a pressure differential transducer (Validyne DP45, ± 56 cm H_2O, Northridge, California) that was placed close to the testing chamber. Flow was measured using a pneumotachograph (Fleish n°2, Lausanne, Switzerland) associated with a pressure differential transducer (Validyne DP45, ± 3.5 cm H_2O). Ventilator-delivered volume was obtained from the integration of the inspiratory and expiratory flow signal.

Analysis

Hypoventilation duration (expressed in s) was the time necessary for the V_T to decrease to the baseline value, and this depended on the number of cycles dedicated to the perturbation. During each respiratory cycle perturbation, we recorded the delays at which (i) pressure started to increase in response to the V_T variation, (ii) maximum pressure support was attained, and (iii) pressure returned to baseline. These response delays were expressed as a percentage of the hypoventilation duration.

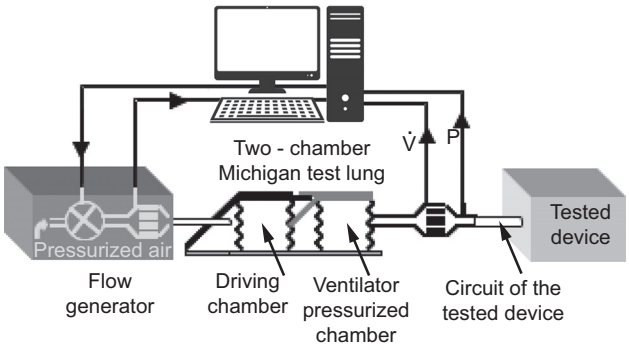

Fig. 1. Experimental setup. The flow generator simulates 10 min of normal breathing followed by hypopnea periods. These hypopneas are ideally detected by the different devices and modes; their responses are recorded via flow ($\dot{V}$) and pressure (P) measurements at the outlet of the pressurized test chamber.

During each experimental sequence, we recorded the V_T at which additional pressure support, provided by the tested device, started, attained its maximum value, and stopped; we also measured the lowest and highest V_T obtained during the breathing pattern perturbation. These volumes were expressed as percentages of the baseline V_T. Finally, we recorded the maximum pressure support (in cm H_2O) provided by the ventilator as a response to the experimental sequence.

Statistical Analysis

The devices' response delays (no., % of hypoventilation duration), V_T (no., % of baseline) at which pressure support started to increase, V_T (no., % of baseline) at which pressure support started to decrease, minimum and maximum V_T (no., % of baseline) delivered during the respiratory cycle perturbation, and maximum pressure support levels (in cm H_2O) in response to hypoventilation were compared between the different devices using a nonparametric Friedman test. Differences were considered as statistically different at an alpha level of 0.05.

Results

At baseline, pressure support remained < 2 cm H_2O for each ventilator, and the V_T was maintained at 500 mL by all the ventilators. The inspiratory trigger of the Lumis 150

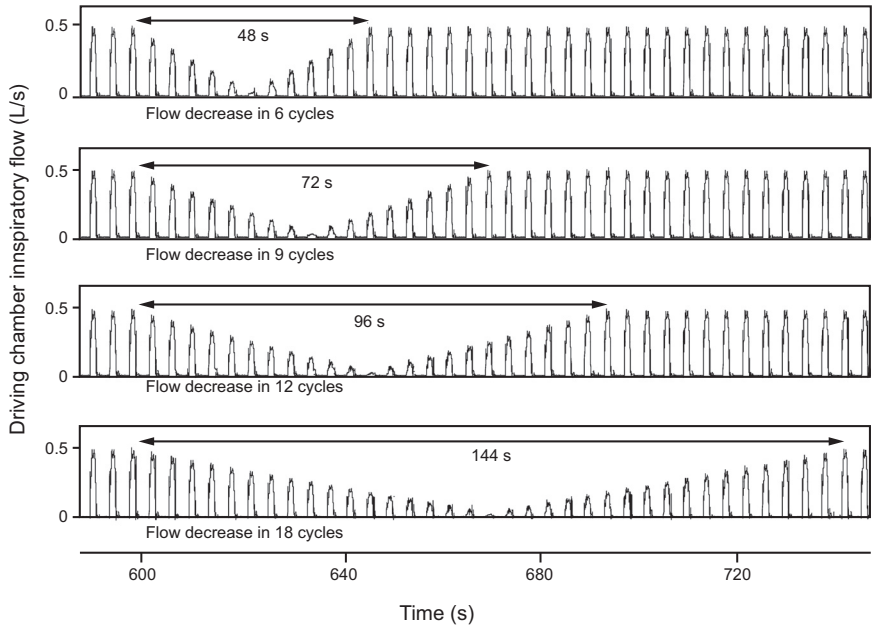

Fig. 2. Recording of inspiratory flow during the 4 central hypopnea/hypoventilation simulations. This flow is produced by the flow generator to simulate the patient and enters the driving chamber while the other chamber is connected to the tested ventilator (Lumis 150 iVAPS in this example). Note that this inspiratory flow shows what the simulated patient is able to inspire independent of ventilator assistance, considering that the ventilator's pressurized chamber (ie, testing chamber) cannot lift the driving chamber.

iVAPS was set at its most sensitive level. For each device in all conditions, end-expiratory pressure remained at its lowest level (ie, 4 cm H_2O), suggesting a lack of detection of obstructive events by the ASV devices.

Pressure support started at the lowest level (ie, zero) for all devices, except for AVAPS, which requires a minimum pressure of 2 cm H_2O. Pressure support increased only during the hypopnea periods, and only the Lumis 150 iVAPS reached the maximum level. The dynamic response of the devices to the different central hypopneas/hypoventilations is presented in Table 2. During the decreasing phase of hypopnea/hypoventilation (simulated by a progressive decrease in V_T), all ventilators except the DreamStation BiPAP responded with an increase of pressure support, although this occurred at different V_T thresholds. The AirCurve 10 CS Pacewave was the first to increase its support, followed by the Lumis 150 iVAPS and the other ASV devices.

Auto-triggering and double-triggering did not occur. Controlled breaths were observed with all devices except the Lumis 150 iVAPS. With the AirCurve algorithm, controlled breaths seemed to occur at a breathing frequency of 15 cycles/min and no unassisted breath was observed, suggesting that the ventilator took control of the respiratory frequency when minute ventilation decreased (Fig. 3).

During the hypopnea/hypoventilation event, the Prisma CR first increased its assistance and then used the oscillation technique to determine if hypopnea/hypoventilation was central or obstructive (Fig. 4). During this oscillation analysis, spontaneous breaths were not assisted, and then controlled breaths occurred once a central event was confirmed (Fig. 4). For the other devices, controlled breaths occurred after the occurrence of unassisted breaths, which were counted (Table 2). These unassisted breaths systematically happened when the inspiratory effort was at its smallest value and unable to trigger the ventilator.

During the hypopnea/hypoventilation simulation, the V_T did not reach its set nadir of 50 mL due to the intervention of the ventilation devices, with the exception of the DreamStation BiPAP device, which did not initiate an increase of pressure support before the lowest V_T was reached regardless of the simulation duration (Fig. 2). The AirCurve 10 CS Pacewave was the most efficient in maintaining V_T (Table 2). The Prisma CR was relatively constant and close to the AirCurve 10 CS Pacewave in maintaining V_T during the shortest hypopnea (48 s), whereas the Lumis 150 iVAPS immediately followed the performance of the AirCurve 10 CS Pacewave performances during the longest hypoventilation event (144 s) (Table 2).

The pressure support peak was $>$ 15 cm H_2O for the Lumis 150 iVAPS, whereas it was 12 cm H_2O for the AirCurve 10 CS Pacewave and 7–8 cm H_2O for the other devices (Table 2). During the crescendo phase of the hypopnea/hypoventilation event, each ventilator initiated a decrease of pressure support at different V_T thresholds (Table 2), except for the DreamStation BiPAP, which paradoxically started to increase pressure support once the hypopnea/hypoventilation event was over. The Prisma CR was the first device to decrease pressure support, followed by the other ASV devices (ie, AirCurve 10 CS Pacewave and DreamStation autoSV). The Lumis 150 iVAPS started decreasing pressure support while the V_T was $>$ 500 mL; this late decrease explained the important V_T overshoot of 25% above baseline, which was negligible for the other devices (Table 2).

Discussion

Noninvasive ventilation is the most appropriate approach to reverse the consequences of nocturnal hypoventilation. Although conventional pressure support ventilation efficiently corrects hypercapnia during sleep, it may produce respiratory instability by increasing the controller gain; this may facilitate sleep fragmentation and therefore sleep stage instability, which in turn enhances respiratory instability.[11-14] The devices we tested decreased pressure support when V_T or minute ventilation increased, and increased pressure support when V_T or minute ventilation decreased; in doing so, these devices are likely to reduce the controller gain and thus respiratory and sleep instability. To be effective, these devices should reduce transient hypoventilation without provoking subsequent hyperventilation when inspiratory activity returns to baseline.

AVAPS is a ventilation mode that combines aspects of pressure support ventilation with the volume-controlled mode to maintain V_T, whereas iVAPS aims to maintain stable alveolar minute ventilation. Accordingly, iVAPS defines alveolar ventilation as minute ventilation minus anatomical dead space ventilation, which was calculated with the patient's height: $120 \times (\text{height}/175)^{2,363}$.[15]

There are 2 available tools to set targets on the Lumis 150 iVAPS device. The first consists of initiating the "Learn Target" feature while the patient is awake and normocapnic under mechanical ventilation. This monitoring typically lasts 15–20 min. The alternative, which we used, is to calculate alveolar ventilation by entering the patient's height, spontaneous breathing frequency (ie, 15 cycles/min in our study) and previously measured minute ventilation or V_T (ie, 500 mL in our study). Because the metabolic rate decreases during sleep, the Lumis 150 iVAPS uses 90% of the average estimated alveolar ventilation to propose a target alveolar ventilation during sleep.[16] By simulating spontaneous breathing with a constant breathing frequency, we did not test the Lumis 150 iVAPS' ability to adjust V_T with breathing frequency changes to maintain constant alveolar ventilation. In our study, the volume-targeted mode

Table 2. Response of 5 Mechanical Ventilation Assistance Devices During 4 Different Transient Hypoventilations

		Delay, % of HD			Tidal Volume, % of baseline						
	HD, s	When Pressure Started to Increase	When P_{Imax} Occurred	When Pressure Returned to Baseline	When Pressure Started to Increase	Minimum	When Pressure Started to Decrease	Maximum	Maximum Pressure Support, cm H_2O	Unassisted Breaths, no.	Controlled Breaths, no.
AirCurve 10	144	14	33	94	75	60	78	100	12.3	0	23
CS Pacewave	96	14	33	100	70	54	78	103	12.1	0	18
	72	12	35	108	70	50	81	106	12.5	0	13
	48	19	22	114	55	37	80	110	11.6	0	8
	Mean ± SD	14.7 ± 3.0	30.7 ± 5.9	104.0 ± 8.8	67.5 ± 8.5	50.2 ± 9.7	79.2 ± 1.5	104.7 ± 4.3	12.1 ± 0.4	0 ± 0	15.5 ± 6.5
Prisma CR	144	20	47	103	50	39	65	103	8.3	4	2
	96	26	50	126	54	33	54	104	7.8	4	1
	72	30	56	130	50	36	66	104	7.3	2	1
	48	44	73	145	50	37	99	102	7.4	1	0
	Mean ± SD	30.0 ± 10.2	56.5 ± 11.6	126.0 ± 17.4	51.0 ± 2.0	36.2 ± 2.5	71.0 ± 19.4	103.2 ± 1.0	7.7 ± 0.5	2.75 ± 1.5	1.0 ± 0.8
DreamStation	144	22	53	125	40	17	70	110	9.0	3	2
autoSV	96	19	86	156	36	20	78	110	7.6	3	2
	72	25	86	154	31	20	77	110	7.4	2	1
	48	19	69	135	42	28	74	105	6.6	1	1
	Mean ± SD	21.2 ± 2.9	73.5 ± 15.8	142.5 ± 15.0	37.2 ± 4.9	21.2 ± 4.7	74.7 ± 3.6	108.7 ± 2.5	7.6 ± 1.0	2.2 ± 1.0	1.5 ± 0.6
DreamStation	144	70	111	187	NA	10	93	105	7.1	4	1
BiPAP	96	78	110	237	NA	10	97	105	7.3	4	1
	72	79	101	198	NA	8	109	110	7.0	1	0
	48	68	100	194	NA	10	105	105	4.5	1	0
	Mean ± SD	73.7 ± 5.5	105.5 ± 5.8	204 ± 22	NA	9.5 ± 1.1	101.0 ± 7.3	106.2 ± 2.5	6.5 ± 1.3	2.5 ± 1.7	0.5 ± 0.6
Lumis 150	144	14	51	147	70	46	114	124	18.0	0	0
iVAPS	96	17	53	172	60	33	118	125	17.7	0	0
	72	17	64	200	50	26	115	124	17.3	0	0
	48	19	85	244	41	22	125	125	16.8	0	0
	Mean ± SD	16.7 ± 2.1	63.2 ± 15.6	191 ± 41	55.2 ± 12.5	31.7 ± 9.1	118.0 ± 4.9	124.5 ± 5.8	17.4 ± 0.5	0 ± 0	0 ± 0
*P**		.006	.007	.006	.01	.007	.007	.02	.004	NA	NA

*Friedman test.
HD = hypoventilation duration
P_{Imax} = maximum inspiratory pressure
NA = not applicable

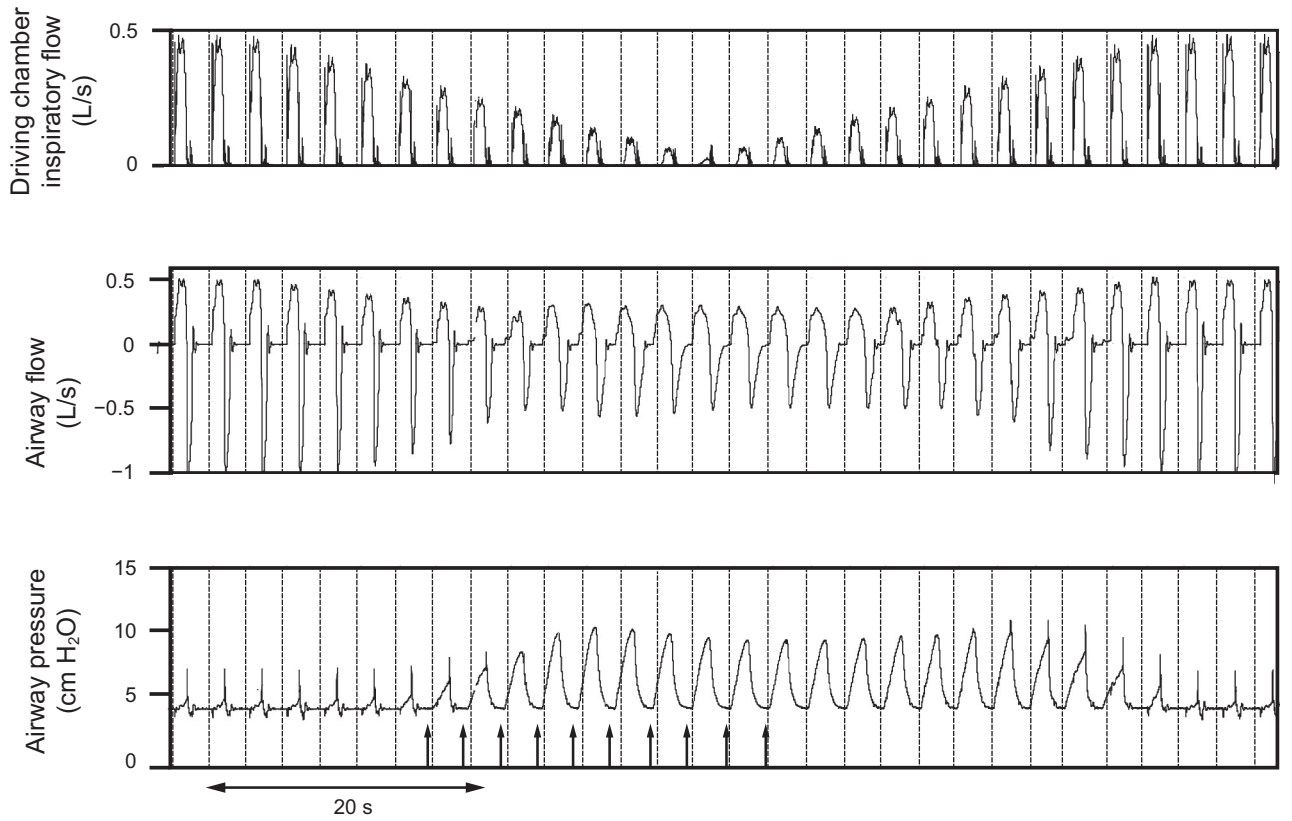

Fig. 3. Hypoventilation recording (ie, volume decreases in 12 cycles and returns to baseline in 12 cycles, 96 s) with the AirCurve 10 CS Pacewave. The dotted vertical lines represent the initiation of a patient's inspiratory effort as detected on the driving chamber inspiratory flow. Arrows pinpoint the cycles where the ventilator insufflation preceded the patient's inspiratory effort, suggesting controlled cycles initiated by the ventilator.

of the AVAPS (ie, the DreamStation BiPAP) was adjusted at 90% of baseline (ie, 450 mL for a baseline of 500 mL), which was equivalent to the Lumis 150 iVAPS targeted ventilation; because the breathing frequency did not change, one could expect the Lumis 150 iVAPS and the AVAPS to respond similarly. Therefore, we expected to observe a similar response in pressure support adjustment by the 2 devices to stabilize V_T. A direct comparison of both devices showed very different dynamic responses between the DreamStation BiPAP and the Lumis 150 iVAPS. The AVAPS response by the DreamStation BiPAP was too delayed to be adapted for the treatment of transient hypoventilation, considering that the increase of pressure support that is intended to treat hypoventilation occurred after the nadir of inspiratory activity, during the crescendo phase. To be effective, self-adjusting pressure support should increase during the decrescendo phase of V_T to dampen the fall in V_T. Conversely, during the crescendo phase of V_T, pressure assist level should decrease to dampen the rise in V_T. If pressure support increases during the crescendo, it means that this automatic increase could be counterproductive because it may accentuate V_T variation. Fortunately, because the AVAPS device's response time was very low, it did not induce a ventilation overshoot at the end of the central event. However, it should be noted that the AVAPS device used a rate of 2.5 cm H_2O/min to increase pressure support, whereas a new AVAPS model (AVAPS-AE) now allows a higher pressure change rate that can be adjusted from 1 cm H_2O/min to 5 cm H_2O/min.[16] We did not test this latest model because we limited our study to non-life-support ventilators (ie, bi-level pressure devices with a single-limb circuit configuration and an intentional leak, without battery). These devices are known to correctly estimate expiratory V_T and to successfully maintain preset minimum V_T during unintentional leaks, whereas most ventilators with a double-circuit configuration fail to evaluate the expiratory V_T and paradoxically worsen the V_T drop during unintentional leaks.[17,18] Nevertheless, AVAPS may be helpful for patients with respiratory failure due to neuromuscular and restrictive conditions in which respiratory effort varies during sleep stage changes, such as in REM sleep, and for individuals with obesity hypoventilation who may need compensation based on positional change.[19] Finally, considering that we only modified the V_T to simulate hypopnea/hypoventilation, we were surprised that the ASV devices were better at reducing

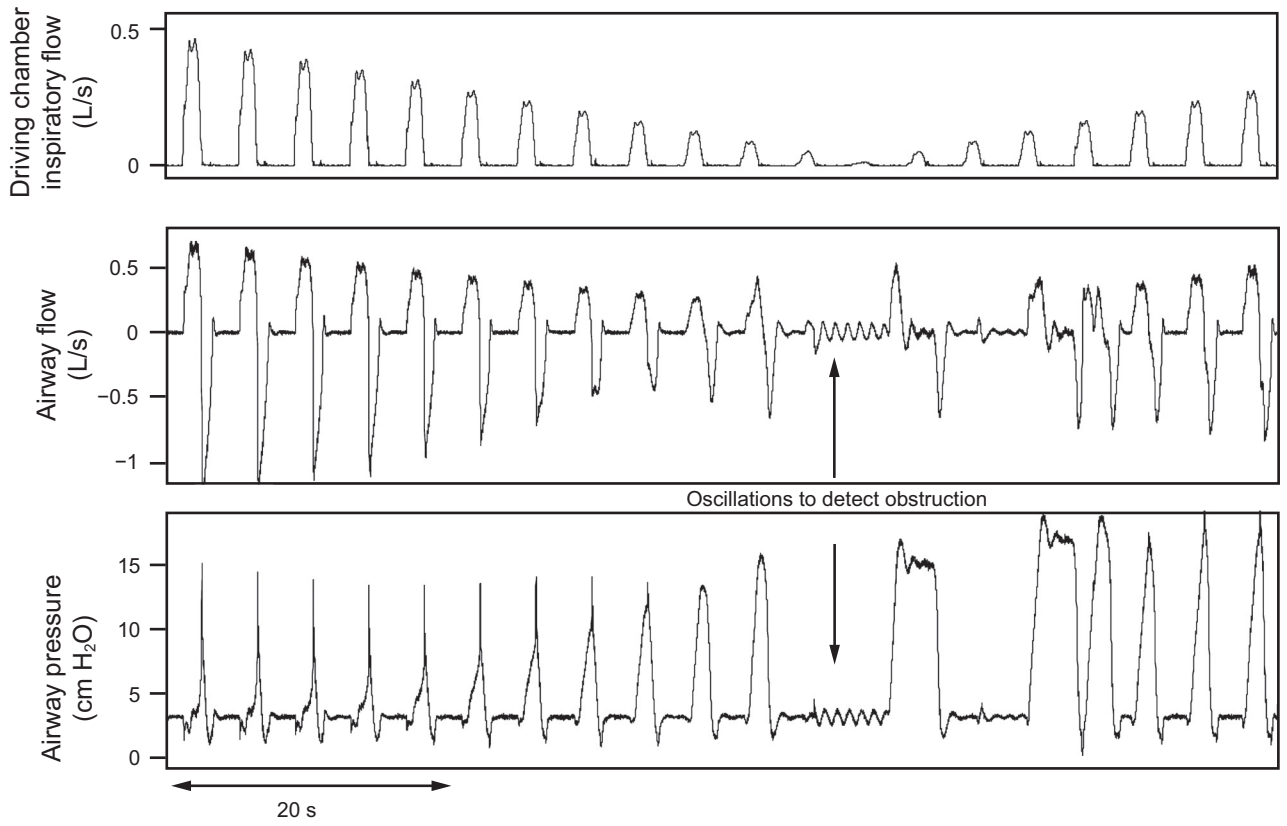

Fig. 4. Hypoventilation recording (ie, volume decreases in 12 cycles and returns to baseline in 12 cycles, 96 s) with the Prisma CR. Note that the inspiratory efforts are assisted until the device initiates oscillations to eliminate an obstructive event. Then the device maintains controlled cycles before resuming assistance to the simulated patient's efforts.

the decrease of V_T than the DreamStation BiPAP with AVAPS. Indeed, these devices have no minimum V_T objective because they only aim to provide an anticyclical ventilation to dampen a patient's drive to respiratory fluctuation; therefore, this result was unexpected.

When comparing iVAPS and ASV devices, the AirCurve S10 VAuto was the most efficient in reducing the decrease of V_T during hypopnea/hypoventilation while avoiding an overshoot when respiratory activity reached its baseline value. The Lumis 150 iVAPS outperformed the other 2 ASV devices, although, unlike the others, the Lumis 150 iVAPS induced an overshoot when respiratory activity returned to normal values. This may induce a transient reduction in arterial CO_2 pressure below the eupnea threshold and facilitate resurgence of respiratory instability by transiently increasing the controller gain.

Accordingly, the AirCurve 10 CS Pacewave had the best results in treating transient hypoventilation and central hypopnea without inducing hyperpnea during respiratory activity recovery. This device calculates target minute ventilation based on the measurement of instantaneous inspiratory flow. A low-pass filter with a time constant of 3 min provides average weighted minute ventilation. Using this continuously updated value, a target of 90–95% is calculated. When the actual ventilation decreases below the target, an integral controller increases pressure support with a rate of 0.3 cm H_2O/L/min/s. In addition, as we observed (Fig. 3), this device was able to switch to a controlled mode when the spontaneous V_T was too small, which may explain its performance.

In contrast, the ASV DreamStation autoSV monitors peak inspiratory flow in a 4-min moving window. The low ventilation limit is obtained by computing 95% of the moving window mean peak inspiratory flow value and is used in the absence of sleep-disordered breathing. Increase of pressure support is proportional to the difference between the targeted peak inspiratory flow and the current breath's peak inspiratory flow.

With the ASV Prisma CR, pressure support is regulated using an average minute ventilation calculated by a low-pass filter every 2 min in a moving window giving a 50% weight to the previous 2 min. For this device, no threshold is determined, and regulation intends to stabilize relative minute ventilation, without any predefined level. Interestingly, we observed that this device uses oscillation techniques to classify the hypopnea/hypoventilation

events as obstructive or central events; in the absence of obstructive events, the device initiates controlled cycles. This choice delayed the ventilator's response to central hypopnea; however, it could be useful for auto-piloting PEEP to maintain upper airway patency and control obstructive sleep apnea syndrome. However, our study did not evaluate this algorithm.

Finally, the results of this study were unexpected considering the algorithms provided by the device manufacturers. We observed important differences between devices: one of the ASV ventilators outperformed the other devices, including other ASV devices; iVAPS and AVAPS devices responded differently for treating nonobstructive transient hypoventilation/hypopnea. However, ASV devices are theoretically indicated for the treatment of patients without hypercapnia, whereas iVAPS and AVAPS devices are recommended for hypercapnic patients.[16] This is questionable considering our observations that the AVAPS and iVAPS devices were not the best to treat central hypopneas and transient hypoventilations. Moreover increasing the minimum value of pressure support when using the ASV mode may avoid hypercapnia and excessive pressure support, which is a cause of hypocapnia; if combined with the lack of wakefulness drive, it can lead to central apneas and sleep fragmentation.[11]

Conclusions

A growing gap exists between the development of a new generation of noninvasive ventilation modes and the knowledge needed to support their use. Our results indicate that ASV devices are liable to decrease the controller gain and thus respiratory instability by reducing hypopnea/hypoventilation severity without inducing a significant overshoot of minute ventilation. Therefore, our results confirm the appropriateness of their current indication to treat respiratory drive instability without obstruction. However, these devices are usually used only in patients with normocapnia.[5] However, because it is possible to set a minimum pressure support, if this minimum were adjusted to reduce hypercapnia, the ASV devices could be considered for patients in whom constant pressure support efficiently corrects hypercapnia but also induces respiratory instability during sleep by increasing the controller gain.[11,12]

Concerning the other so-called volume-assured pressure support devices (ie, AVAPS and iVAPS), a recent review of the literature concluded that there is no compelling data suggesting that they are superior to conventional pressure support ventilation with PEEP for chronic or acute hypercapnic respiratory failure.[5] Moreover, we were unable to confirm their usefulness for the treatment of transient hypopnea/hypoventilation resulting from respiratory drive instability or high loop gain, considering that the AVAPS response occurred after the hypoventilation nadir and the Lumis 150 iVAPS device did not outperform the AirCurve 10 CS Pacewave while facilitating overshoot of minute ventilation. Therefore, these devices cannot solve the frequent situation of respiratory instability facilitated by pressure support.[11] Clinical and bench studies are needed to better appreciate the actual benefit of these technologies in the management of complex sleep disorders, including those associated with respiratory failure.

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