

New Ventilatory Modes for Sleep Disordered Breathing

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Sleep-related breathing disorders (SRBD)

1. Obstructive sleep apnea disorders

- (a) Obstructive sleep apnea, adult
- (b) Obstructive sleep apnea, pediatric

2. Central sleep apnea syndromes

- (a) Central sleep apnea with Cheyne–Stokes breathing
- (b) Central apnea due to a medical disorder without Cheyne–Stokes breathing
- (c) Central sleep apnea due to high-altitude periodic breathing
- (d) Central sleep apnea due to a medication or substance
- (e) Primary central sleep apnea
- (f) Primary central sleep apnea of infancy
- (g) Primary central sleep apnea of prematurity
- (h) Treatment-emergent central sleep apnea

3. Sleep-related hypoventilation disorders

- (i) Obesity hypoventilation syndrome
- (j) Congenital central alveolar hypoventilation syndrome
- (k) Late-onset central hypoventilation with hypothalamic dysfunction
- (l) Idiopathic central alveolar hypoventilation
- (m) Sleep-related hypoventilation due to a medication or substance
- (n) Sleep-related hypoventilation due to a medical disorder

4. Sleep-related hypoxemia

- (o) Disorder sleep-related hypoxemia

5. Isolated symptoms and normal variants

- (p) Snoring
- (q) Catathrenia

Hypocapnic/Hypercapnic SRBD

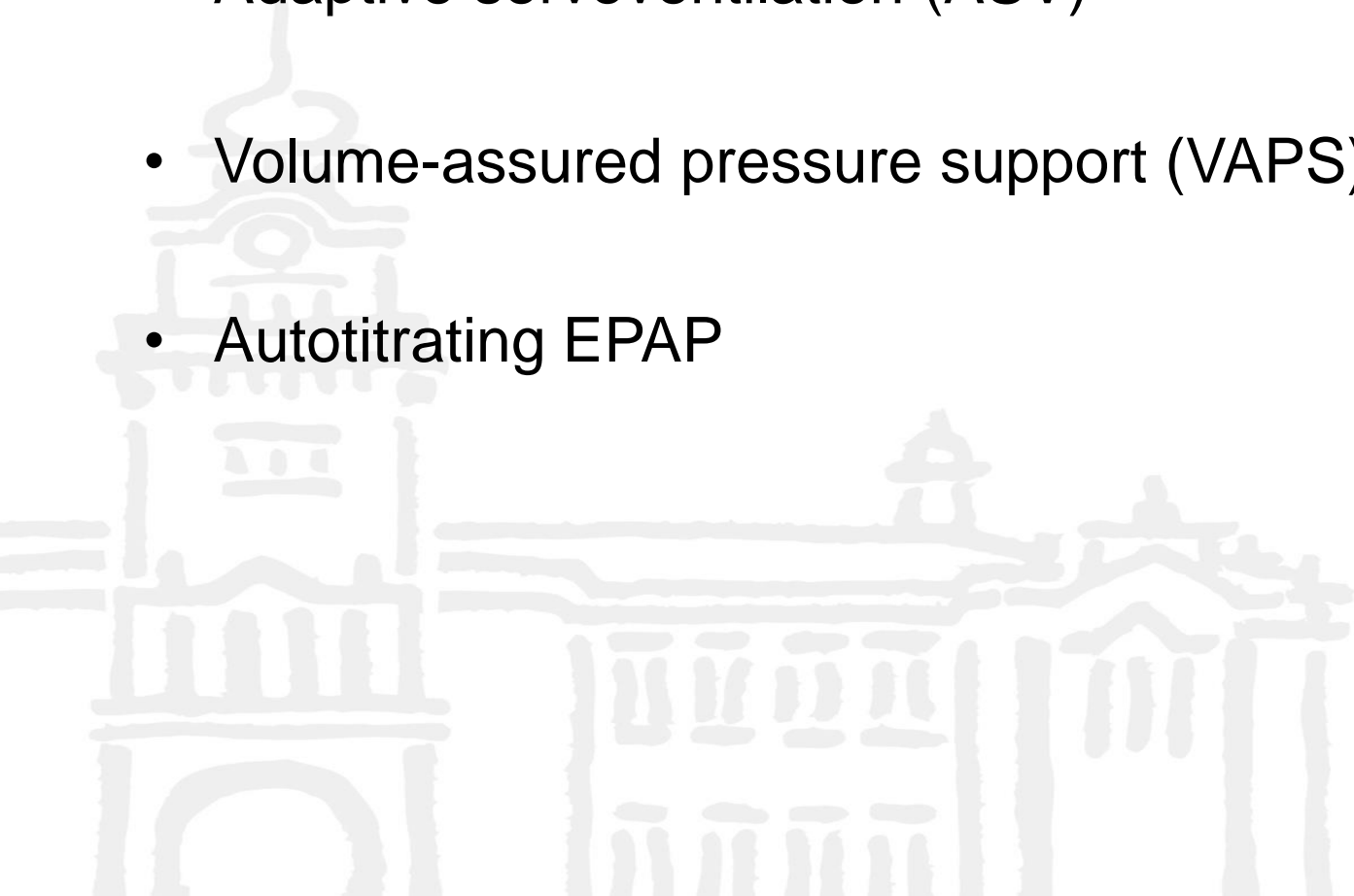
	Hypocapnic	Hypercapnic
Daytime PCO ₂	Normal or low (PCO ₂ ≤ 45mmHg)	Increased (PCO ₂ > 45mmHg)
Respiratory drive	Normal or increased	Normal or decreased
Accompanied by	Periodic breathing	Hypoventilation
Sleep stage	NREM > REM sleep	NREM < REM sleep
Disorders	<ul style="list-style-type: none"> ● Primary CSA ● Secondary <ul style="list-style-type: none"> ▪ CSA with Cheyne–Stokes breathing (CSB): associated with congestive heart failure, neurologic disease ▪ CSA due to high-altitude periodic breathing ▪ Treatment-emergent CSA 	<ul style="list-style-type: none"> ● “Won’t breathe” <ul style="list-style-type: none"> ▪ Central hypoventilation <ul style="list-style-type: none"> • Primary: primary CSA of infancy/prematurity, Congenital central hypoventilation syndrome • Secondary: brain stem tumors/infarcts ▪ CSA due to a medication or substance (narcotics/opiates) ● “Can’t Breathe” <ul style="list-style-type: none"> • Restrictive thoracic cage disorders • Neuromuscular disorders

SRBD: when CPAP is not enough

- Obesity hypoventilation syndrome: CPAP, BPAP-S, BPAP-ST, VAPS
- Sleep-related hypoventilation due to NMD: BPAP-ST, VAPS
- CSA-CSR: CPAP, BPAP-ST, ASV
- CSA due to opioids: CPAP, BPAP-ST, ASV
- Treatment-emergent CSA: CPAP, BPAP-ST, ASV

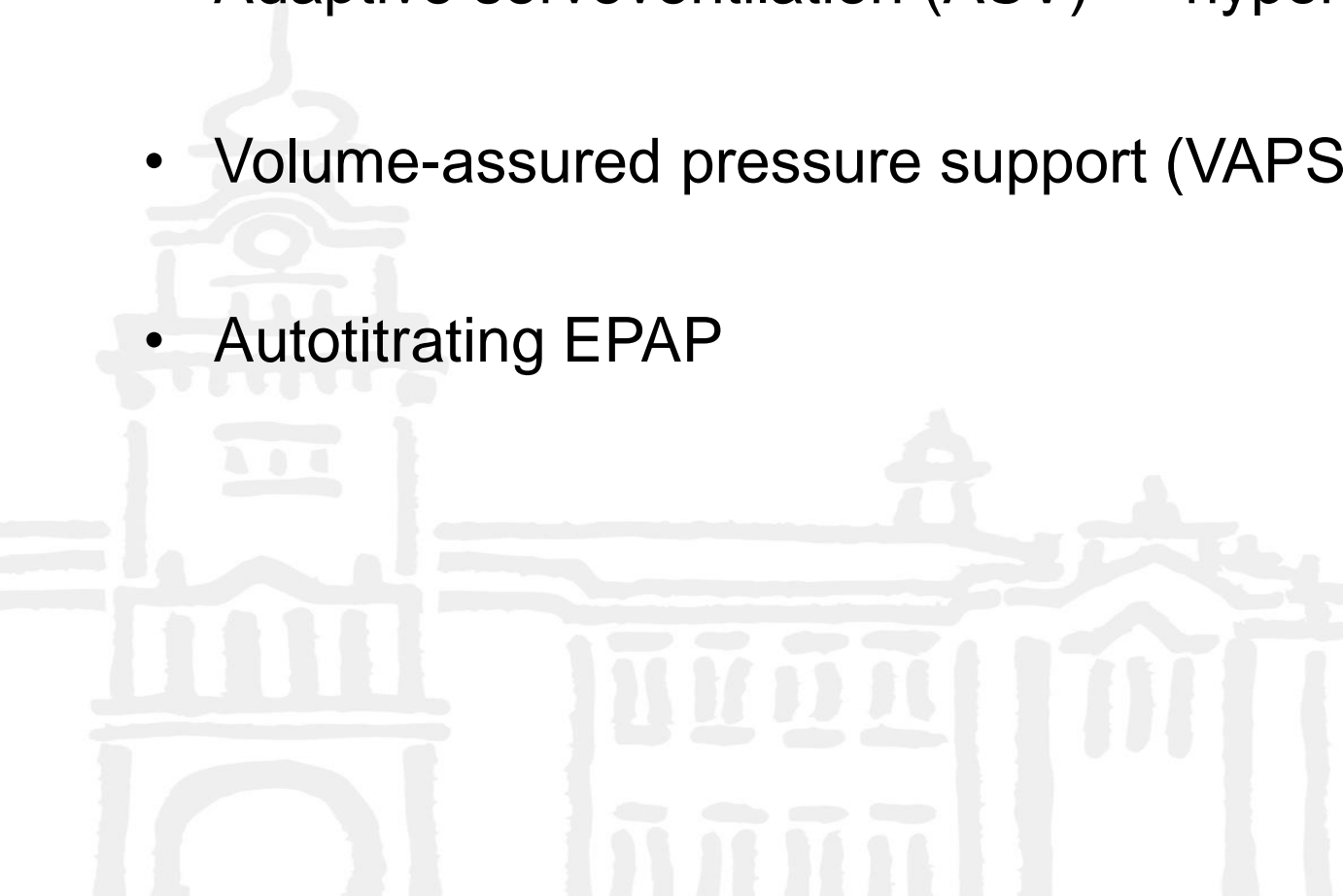
Specialized NIV for SRBD

- Adaptive servoventilation (ASV)
- Volume-assured pressure support (VAPS)
- Autotitrating EPAP



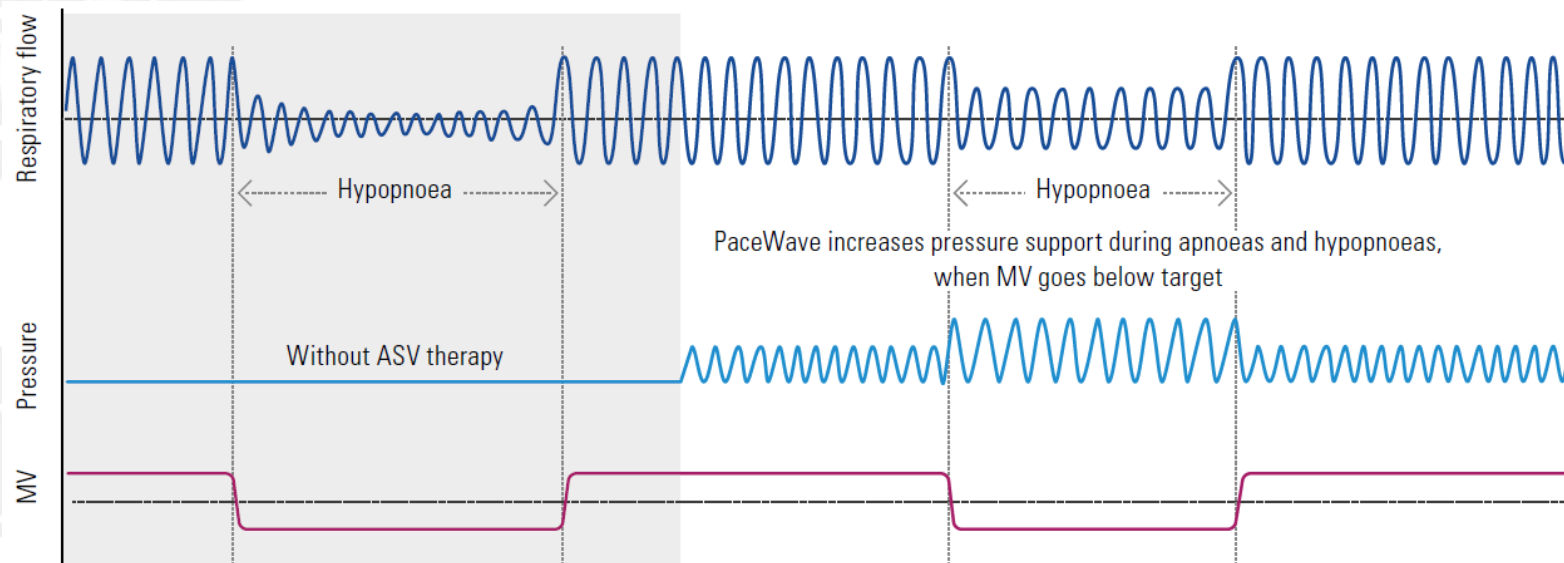
Specialized NIV for SRBD

- Adaptive servoventilation (ASV) ← hyperventilation
- Volume-assured pressure support (VAPS) ← hypoventilation
- Autotitrating EPAP



Adaptive servoventilation (ASV)

- ASV has introduced into clinical practice primarily to address CSB in the context of heart failure.
- Dynamically adjusts pressure support and respiratory rate to stabilize patient's breathing
- Controlled loop feed-back system / anticyclical ventilation



Target MV: 90% of most recent MV based on a three-minute moving average



Indication of ASV

- CSA with CSB
 - Primary CSA
 - Treatment-emergent CSA
 - Narcotic-induced CSA
 - sufficient minimum PS must be used to deal with hypoventilation if present
- } Hypocapnic CSA

TE-CSA

- Prevalence: 8% (range, 5%~20%)
- Risk factors: high baseline AHI or arousal index, hypertension, opioid use, coronary artery disease, stroke, and CHF
- Natural history: spontaneous resolution vs persistence
- Spontaneous resolution rate: 54%~86%
- Pathophysiologic mechanisms
 - Unmasking of central apnea: relief of upper airway obstruction with PAP therapy may unmask the underlying central apnea.
 - PAP-induced events: rapid changes in PAP level or mask leak may rapidly decrease arterial PCO_2 below the hypocapnic apneic threshold, leading to central apnea.
 - Effect of intermittent hypoxia: \rightarrow peripheral chemoreceptor activity \uparrow \rightarrow propensity to central apnea \uparrow

Trajectories of CSA

- U.S. telemonitoring device data, N=133,006
- Average central apnea index (CAI)

	Week 1	Week 13		
OSA	< 5/h	< 5/h		
Transient CSA	≥ 5/h	< 5/h	3.5%	55.1%
Persistent CSA	≥ 5/h	≥ 5/h		25.2%
Emergent CSA	< 5/h	≥ 5/h		19.7%

Median CPAP Usage (h/d):

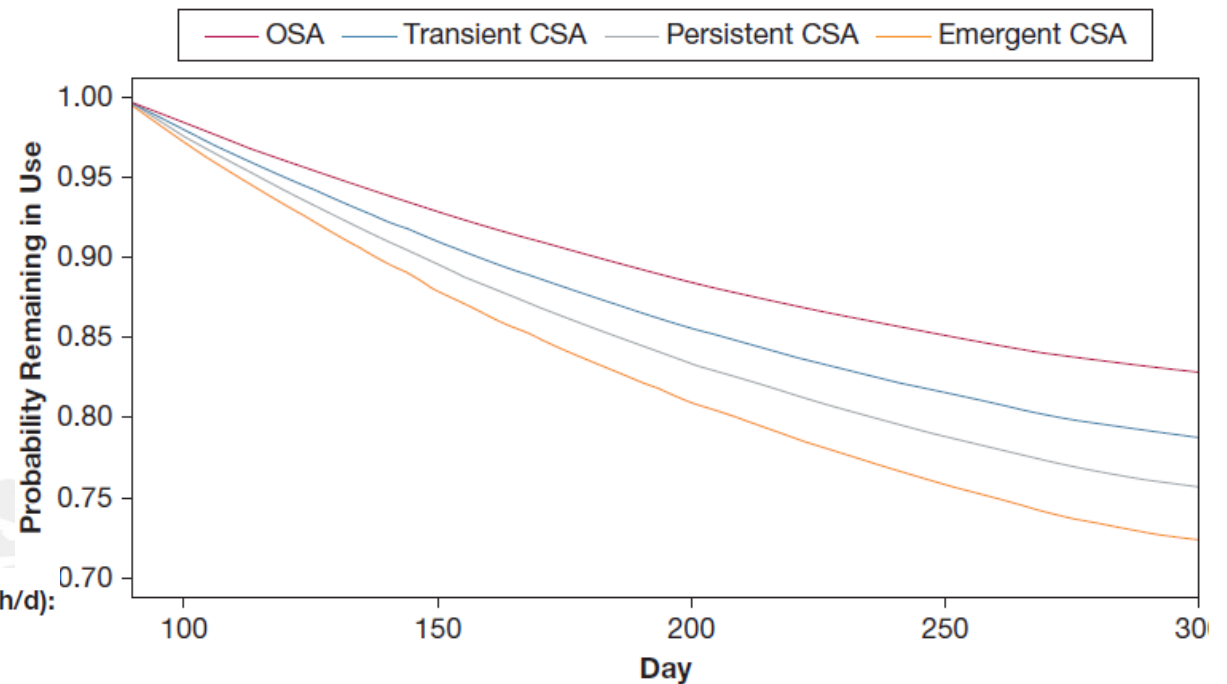
6.02 (IQR: 4.47-7.26)

5.75 (IQR: 4.27-7.03)

5.83 (IQR: 4.17-7.19)

5.68 (IQR: 3.95-7.11)

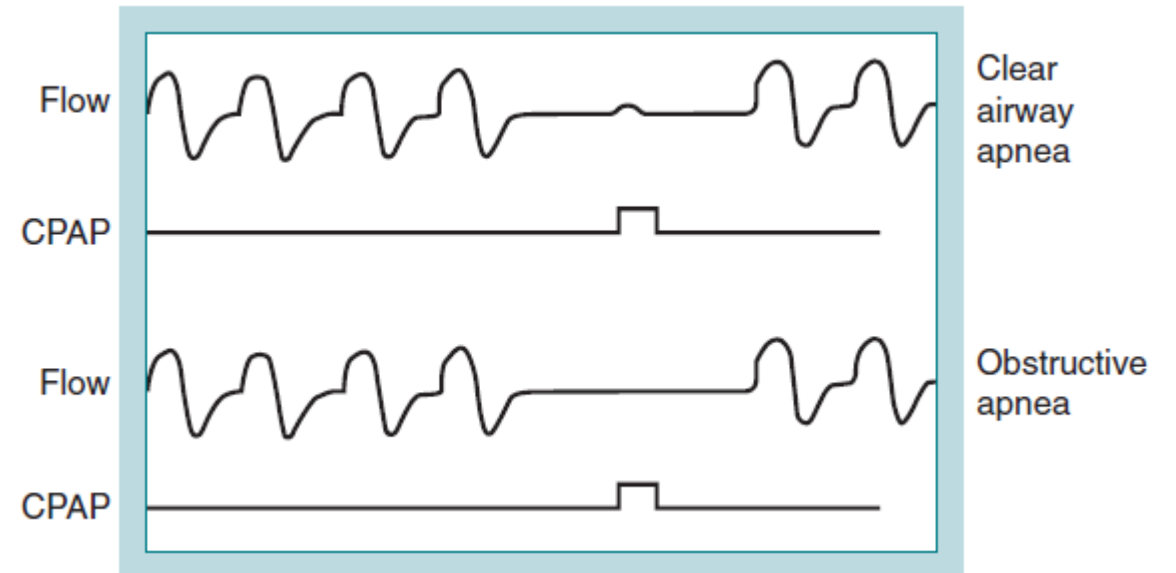
Continued use of CPAP after 90 days



How to detect central apnea?

- Differentiation of clear airway apneas versus obstructive apneas by delivering a small pressure pulse (1–2 cm H₂O pressure pulse)
- If the pressure pulse does produce an increase in flow \Rightarrow open airway (clear airway)
- If the pressure pulse does not increase flow \Rightarrow closed airway
- An APAP device using this technology does not increase pressure for “clear airway” apneas.

치료				
압력 - cmH ₂ O	중간값: 10.7	제95위 백분위수: 11.8	최대: 11.9	
누출 - L/min	중간값: 8.0	제95위 백분위수: 20.7	최대: 28.4	
시간당 이벤트	AI: 0.1	HI: 0.5	AHI: 0.6	
무호흡 지수	중양: 0.0	폐쇄성: 0.1	알 수 없음: 0.0	
체인-스토크스호흡 (야간당 평균 지속시간)				0 분 (0%)



Switching from CPAP to ASV

- U.S. telemonitoring device data, N=198,890
 - CPAP only 189,724 (95.4%)
 - ASV only 8,957 (4.5%)
 - Switch 209 (0.11%)

Table 2—Adherence rates at 90 days by patient subgroups.

	90-Day Adherence % (95% CI)
Switch CPAP to Fixed EPAP ASV (n = 127) *	
Before switch (CPAP)	67.7 (59.6, 75.8)
After switch (ASV) *	78.7 (71.6, 85.9) ^a
Switch CPAP to Variable EPAP ASV (n = 82) †	
Before switch (CPAP)	54.9 (44.1, 65.6)
After switch (ASVAuto) †	73.2 (63.6, 82.8) ^b
CPAP Only (n = 189,724)	73.8 (73.6, 74.0)
ASV Only (n = 8,957)	73.2 (72.3, 74.1)

Figure 2—Trajectories of average PAP usage before versus after the switch from CPAP to ASV.

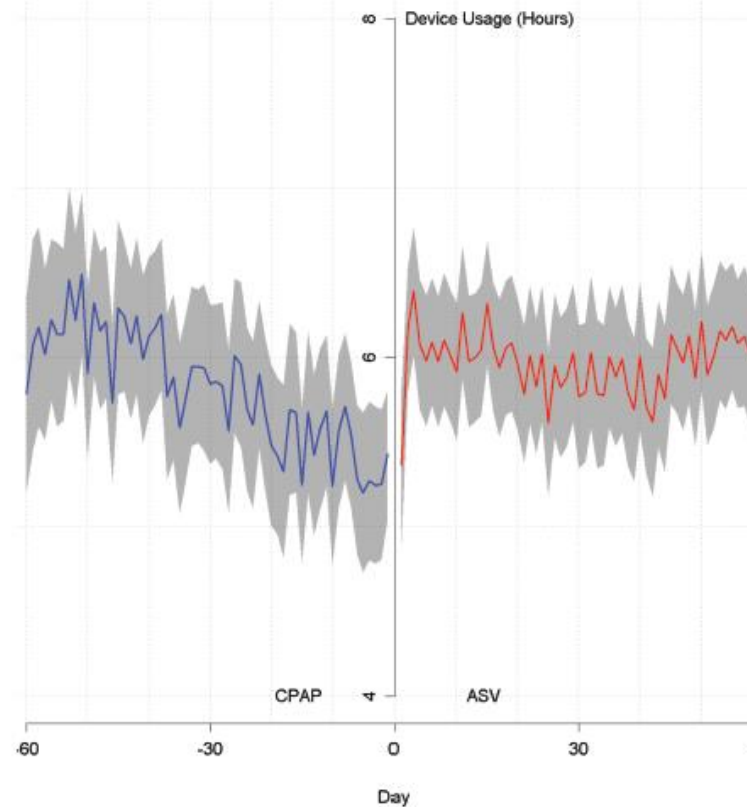
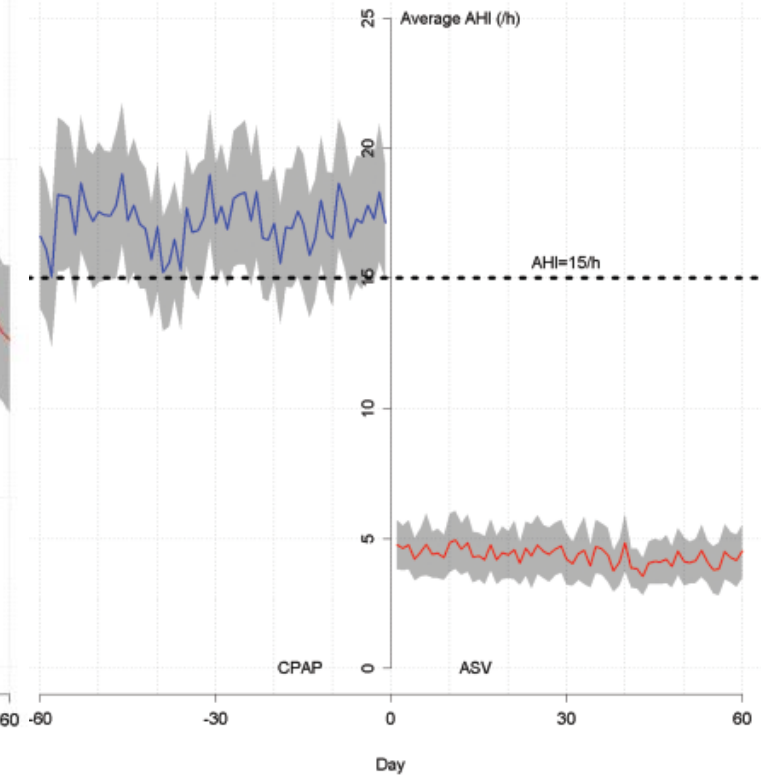
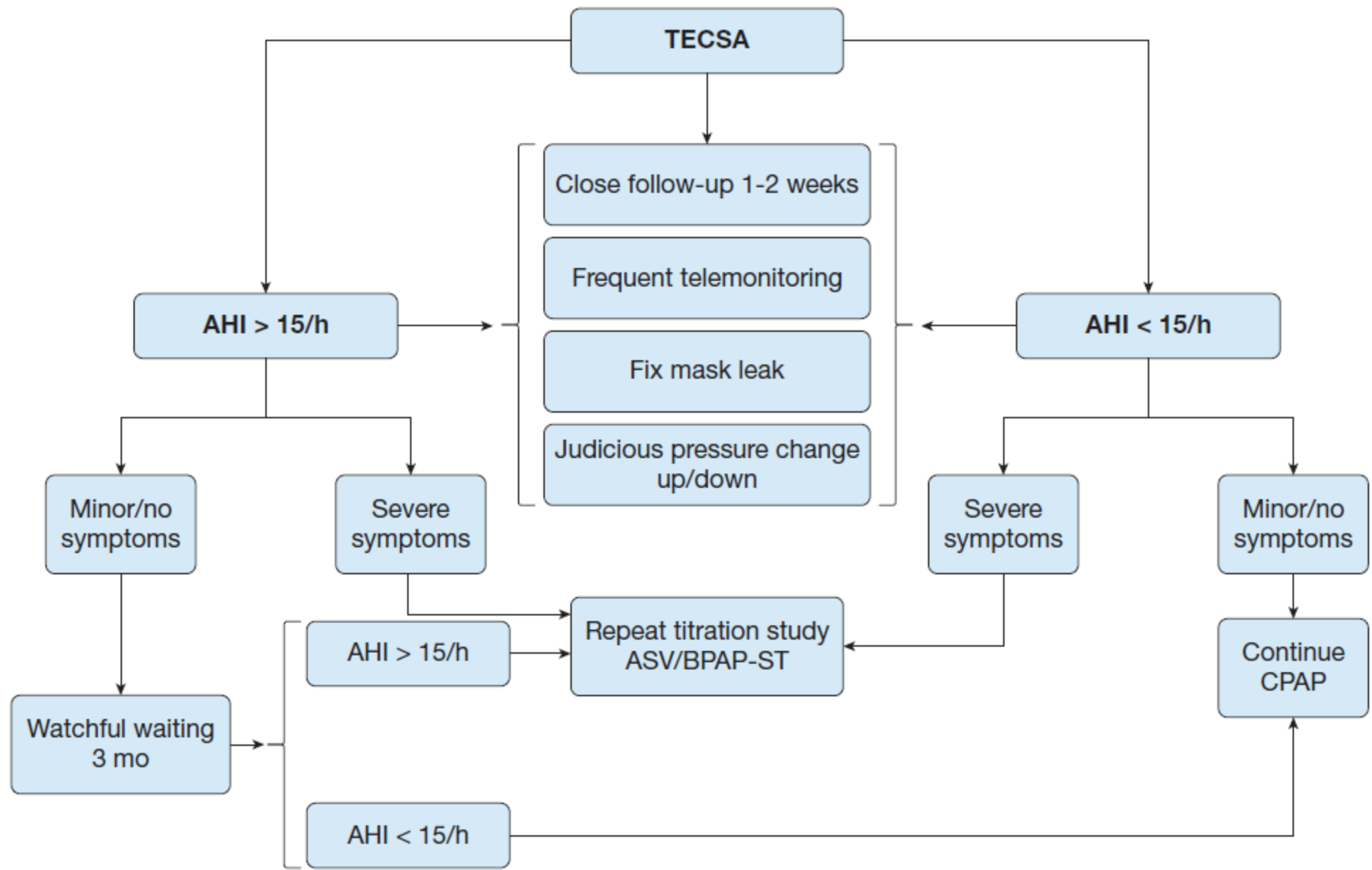


Figure 3—Average AHI before versus after the switch from PAP to ASV.

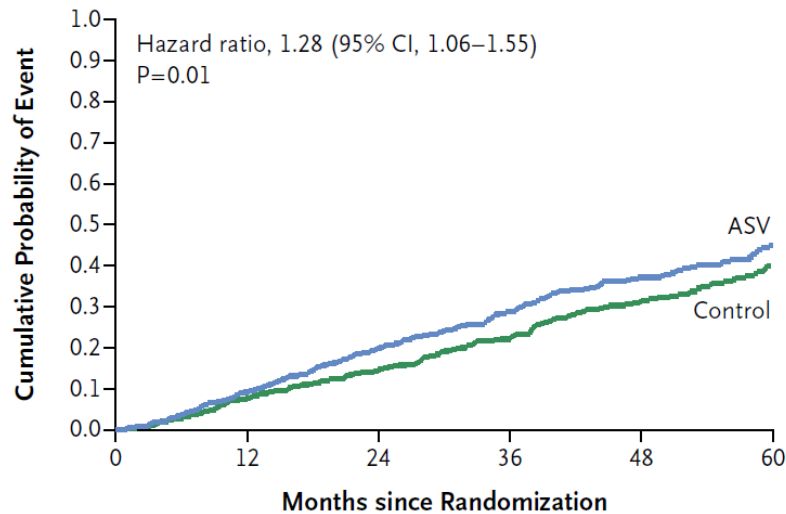




SERVE-HF

- RCT on 1,325 patients with HFrEF (LVEF $\leq 45\%$) and CSA (AHI $\geq 15/h$ with $>50\%$ central events and central AHI $\geq 10/h$)
 - ASV+ optimal medical therapy for CHF vs optimal medical therapy for CHF
 - Primary outcome: time to the first event of death from any cause, lifesaving cardiovascular intervention, unplanned hospitalization for worsening heart failure

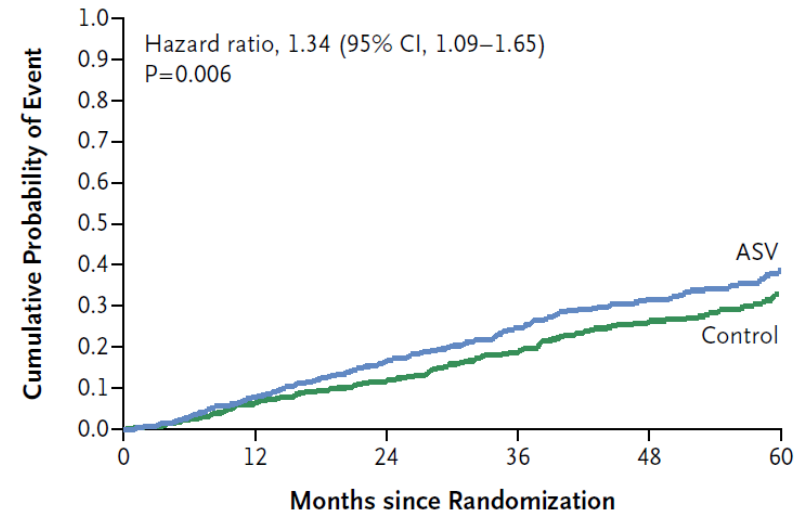
B Death from Any Cause



No. at Risk

Control	659	563	493	334	213	117
ASV	666	555	466	304	189	97

C Death from Cardiovascular Causes



No. at Risk

Control	659	563	493	334	213	117
ASV	666	555	466	304	189	97

ASV in CHF

- ASV targeted to normalize the AHI should **not** be used for the treatment of CSAS related to CHF in adults with an **ejection fraction $\leq 45\%$** and moderate or severe CSA predominant, sleep-disordered breathing.
- Ongoing RCT: **ADVENT-HF**
 - 732 participants with HFrEF (LVEF $\leq 45\%$) and sleep apnea (AHI $\geq 15/h$)
 - ASV+ optimal medical therapy for CHF vs optimal medical therapy for CHF
 - Primary outcome: the time to the composite outcome of death or first CV hospital admission or new onset atrial fibrillation/flutter requiring anti-coagulation but not hospitalization or delivery of an appropriate shock from an ICD not resulting in hospitalization

ClinicalTrials.gov Identifier: NCT01128816

Recruitment Status ⓘ : Terminated (Philips Global Field Safety Notice for PAP Devices & Ventilators)

First Posted ⓘ : May 24, 2010

Last Update Posted ⓘ : July 15, 2021

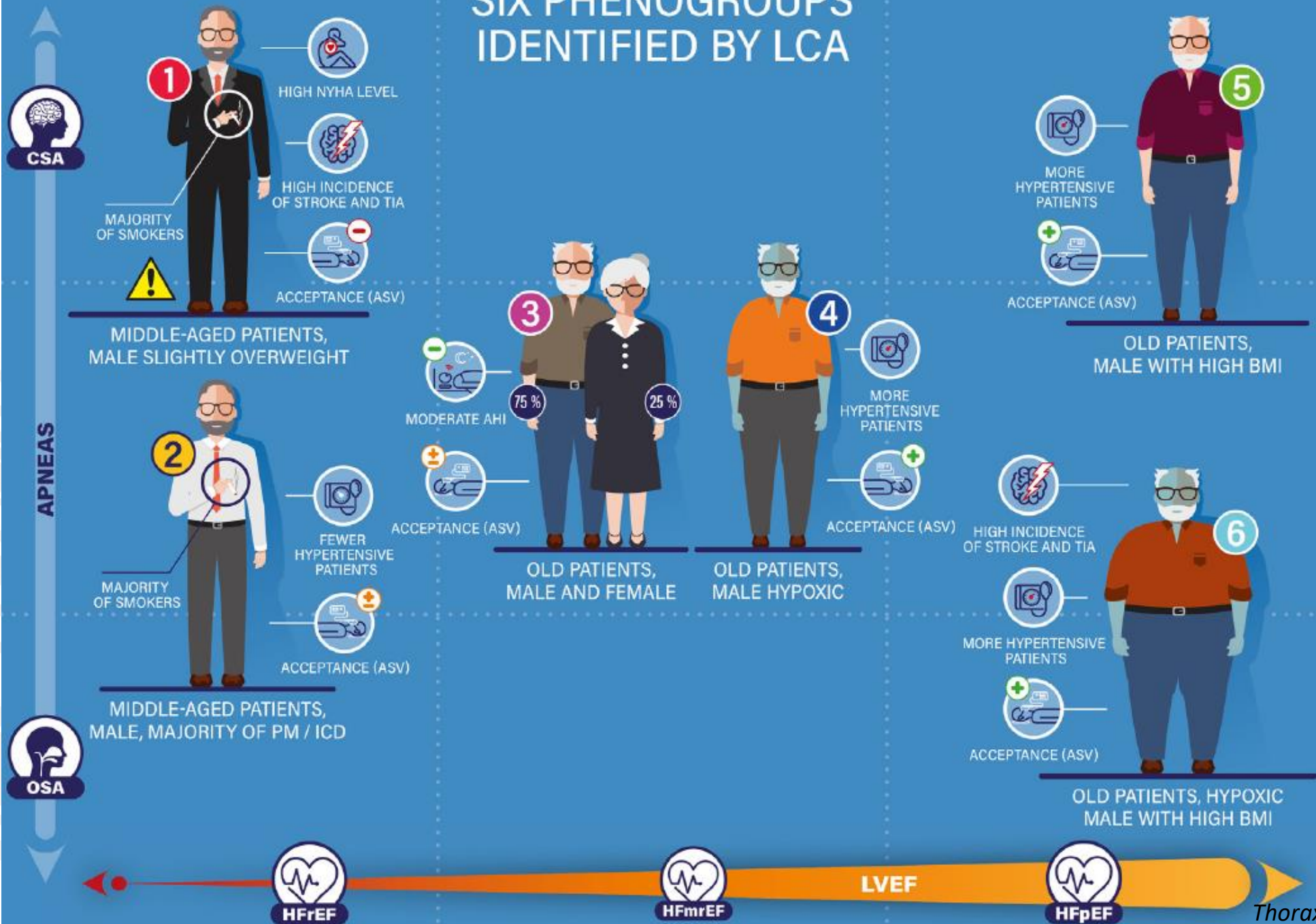
ASV in CHF: FACE study 3-mo data

- European, multicenter, prospective, observational cohort study
 - From 2009 to 2018, N=503
 - Effects of ASV therapy on clinical outcomes in patients with HF with SRBD
 - 3-month follow-up data with latent class analysis (LCA)

Table 4 Sleep-disordered breathing characteristics by cluster

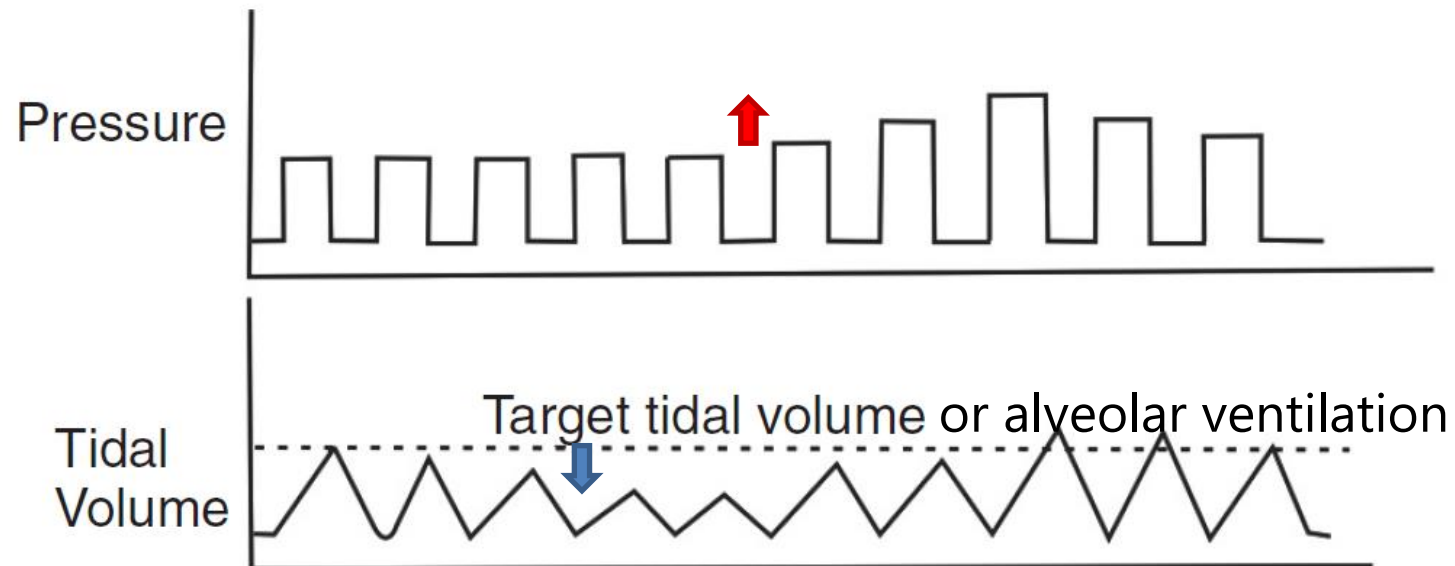
	Cluster 1 (n=79)	Cluster 2 (n=89)	Cluster 3 (n=67)	Cluster 4 (n=99)	Cluster 5 (n=119)	Cluster 6 (n=50)	P value*
Central sleep apnoea, n (%)	71 (89.9)	40 (44.9)	32 (47.8)	78 (78.8)	112 (94.1)	12 (24)	<0.01
Central AHI, /h	26 (21–32)	14 (8–19)	10 (5–16)	33 (26–41)	42 (31–55)	8.5 (3–13)	<0.01
Obstructive AHI, /h	3 (1–6)	20 (16–25)	4 (1–9)	20 (14–27)	2 (0–5)	40 (32–48)	<0.01
Time with oxygen saturation<90%, min	32 (6–87)	23 (3–62)	45 (5–103)	74 (23–141)	31 (4–83)	91 (38–113)	<0.01
CPAP usage before ASV, n (%)	8 (10.1)	8 (9.0)	20 (29.9)	14 (14.1)	37 (31.1)	18 (36.0)	<0.01
Agreed to ASV therapy, n (%)	42 (53.2)	68 (76.4)	42 (62.7)	92 (91.9)	109 (91.6)	50 (100)	<0.01

SIX PHENOGROUPS IDENTIFIED BY LCA



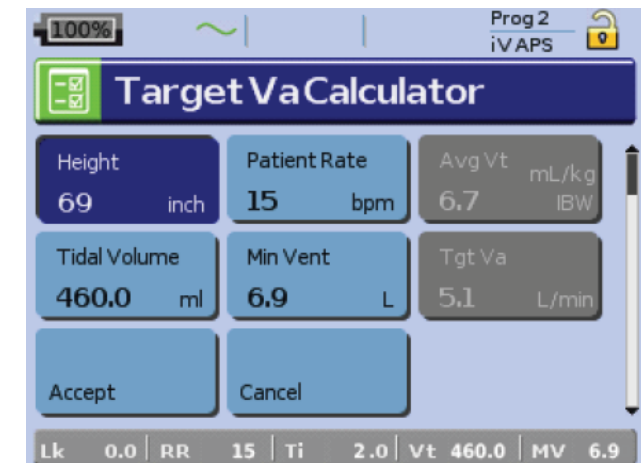
Volume-assured pressure support (VAPS)

- VAPS automatically adjusts the PS to deliver an adequate tidal volume/alveolar ventilation



VAPS

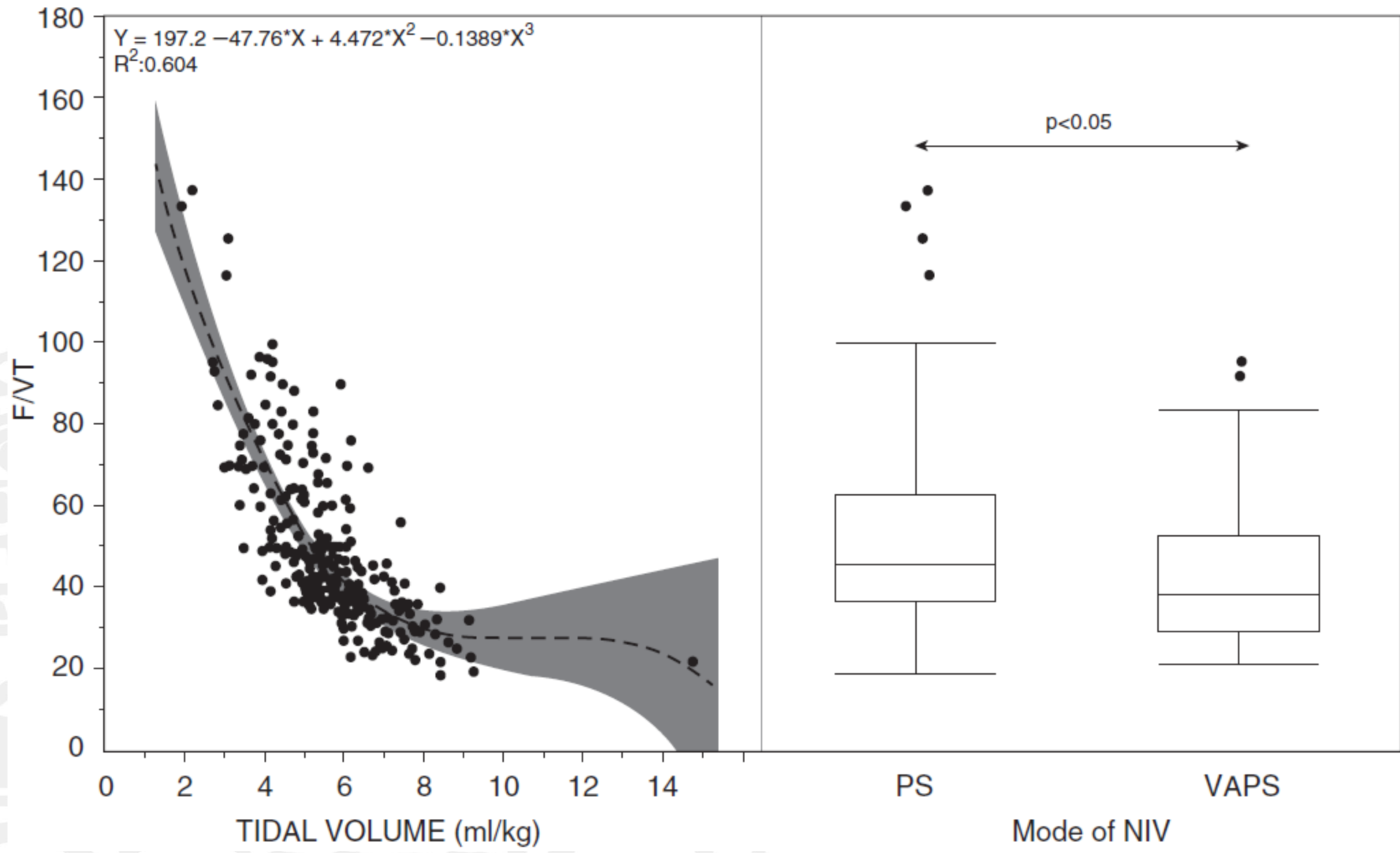
Device	Intelligent VAPS (iVAPS)	Average VAPS (AVAPS)
Target	Alveolar ventilation = minute ventilation – dead space ventilation (based on height)	Tidal volume
EPAP	Set to maintain an open airway	Same
PS	Varies	Varies
Backup rate	iBR (intelligent backup rate) varies	Fixed
Settings	Target alveolar ventilation Height	Target tidal volume
	Max PS	Max IPAP
	Min PS	min IPAP
	EPAP	EPAP
	Target backup rate	Backup rate
	Ti, Rise time, Trigger, Cycle	Ti, Rise time, Trigger, Cycle



Amyotrophic lateral sclerosis (ALS)

- Retrospective chart review of 271 patients with ALS using either PS or VAPS
 - Tidal volumes (V_T)
 - Ratio of respiratory rate to tidal volume (f/V_T): an indicator of a rapid shallow breathing pattern

	Standard PS (n = 215)	VAPS (n = 56)	P Value
Ventilator settings*			
IPAP (median)	10.2 (9.8–10.6)	12.0 (11.2–12.7)	<0.001
PSmax, cm H ₂ O	5.9 (5.5–6.2)	11.9 (11.2–12.6)	<0.0001
PSmin, cm H ₂ O	5.9 (5.6–6.2)	4.5 (4.0–5.1)	<0.0001
EPAP, cm H ₂ O	4.3 (4.2–4.5)	4.6 (4.2–4.9)	0.269
T _{imin} , s	1.07 (1.04–1.10)	1.01 (0.96–1.05)	0.009
T _{imax} , s	2.02 (1.98–2.06)	2.10 (2.03–2.16)	0.037 [†]
Back-up rate	10.8 (10.5–11.0)	12.0 (11.5–12.5)	<0.0001
Rise time, ms	746 (708–783)	660 (602–717)	0.0001 [†]
Patient-ventilator measurements			
Hours of use per day	6.6 (6.0–7.2)	6.5 (5.4–7.72)	0.703
Minute ventilation, L/min	6.03 (5.73–6.34)	6.40 (5.82–6.98)	0.26
Respiratory rate, median (range)	16 (10–29)	15 (10–31)	0.868
Tidal volume, ml	356.2 (340.2–372.2)	390.4 (359.8–420.9)	0.007 [†]
f/V_T	51.5 (45.5–54.6)	44.6 (38.8–50.5)	0.022 [†]
% triggered breaths	83.7 (80.7–86.7)	88.6 (83.6–93.7)	0.33
% cycled breaths	36.7 (31.6–41.8) [‡]	57.9 (49.4–66.4) [‡]	0.0001 [†]
V_T (ml/kg IBW), median (IQR)	5.51 (5.29–5.72)	5.90 (5.5–6.3)	0.009 [†]
V_T as % target V_T , median (IQR)	68.9 (66.2–71.5)	73.8 (68.8–78.9)	0.009 [†]



COPD

- Parallel-group RCT on 40 stable hypercapnic COPD patients
 - AVAPS vs ST for 6 months
 - Baseline FEV₁ (L) 0.60 ± 0.16 vs. 0.59 ± 0.21

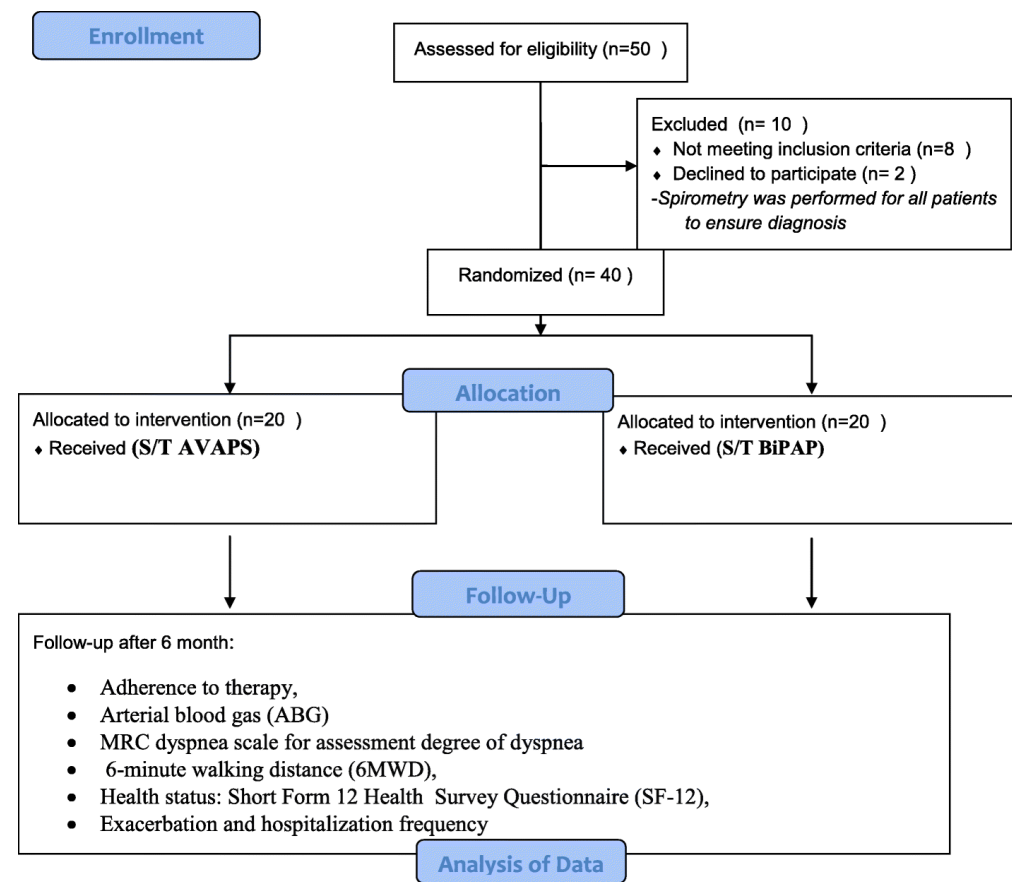


Table 2 Gase exchange

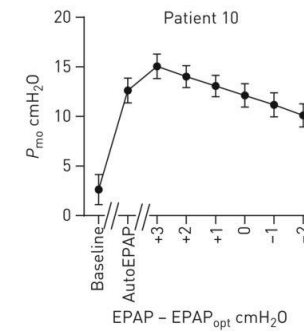
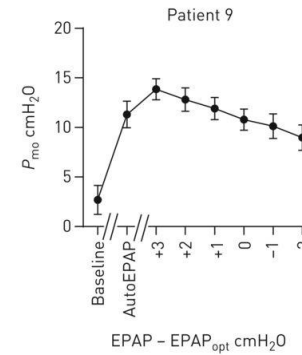
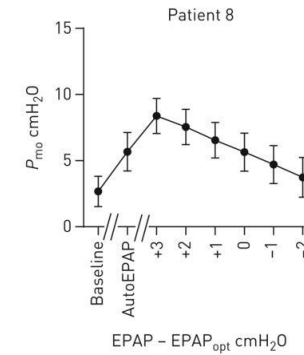
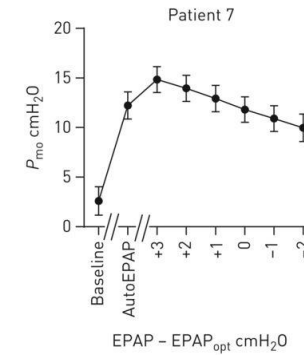
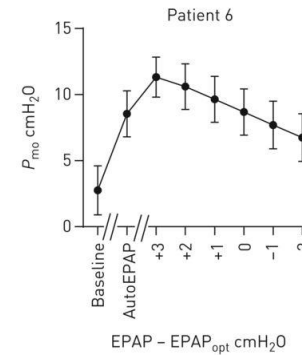
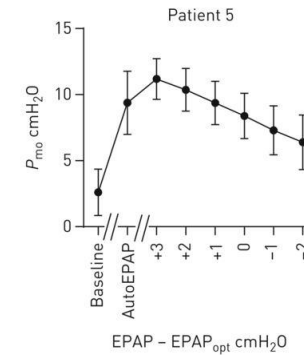
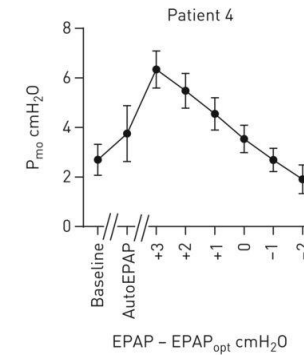
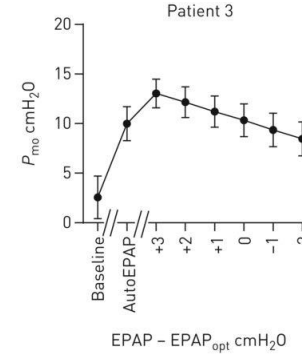
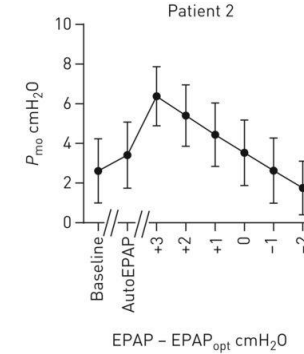
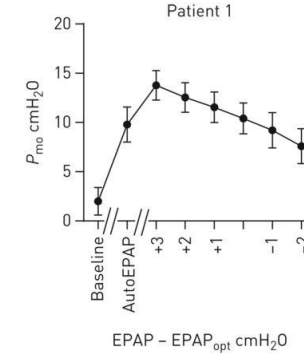
Variables	Patients treated with AVAPS		Control group		Treatment Effect (95% CI) 6 months–baseline	P-Value
	Baseline	After 6 month	Baseline	After 6 month		
Pa CO ₂ mm Hg	54.8 ± 3.5	46.6 ± 3.1	54.5 ± 3.3	48.3 ± 3.9	0.6 (–0.3 to 0.9)	0.001*
PaO ₂ mm Hg	50.7 ± 2.1	59.6 ± 2.3	50.9 ± 2.5	57.7 ± 3.2	0.6(–0.67 to 0.56)	0.001*
HCO ₃ [–] , mmol/L	34.2 ± 3.1	29.5 ± 2.1	34.9 ± 3.2	30.6 ± 2.9	0.4(–1.4 to 1.6)	0.001*

Table 3 Patient-reported outcomes (SF-12, MRC scale, and 6 MWD)

Variables	Patients treated with AVAPS		Control group		Treatment Effect (95% CI) 6 months–baseline	P-Value
	Baseline	After 6 month	Baseline	After 6 month		
SF-36 Scales #						
Vitality	55.2 ± 16.1	64.3 ± 17.5	56.1 ± 17.3	60.5 ± 16.2	5.4(1.4 to 9.3)	0.001*
General health	56.3 ± 20.5	61.3 ± 16.7	57.6 ± 19.2	58.4 ± 20.2	8.2(3.2 to 11.7)	0.001*
Physical functioning	72.4 ± 23.8	77.5 ± 21.5	69.3 ± 24.2	69.6 ± 22.1	5.5(1.1 to 9.8)	0.001*
Bodily pain	62.3 ± 23.2	65.5 ± 22.4	63.7 ± 23.6	66.7 ± 20.8	3.1 (3.4 to 8.8)	0.002*
Emotional Role Functioning	80.6 ± 22.5	83.5 ± 24.7	79.0 ± 24.8	82.3 ± 20.7	−0.5 (−6.8 to 5.3)	0.321
Physical Role Functioning	69.3 ± 23.3	73.5 ± 26.6	70.8 ± 23.7	73.2 ± 24.8	3.3(−5 to 6.6)	0.001*
Social Role Functioning	79.6 ± 23.8	83.6 ± 24.7	80.8 ± 21.9	82.9 ± 25.8	1.6 (−3.8 to 6.4)	0.143
Mental health	74.4 ± 18.6	75.8 ± 12.8	75.6 ± 15.7	75.4 ± 15.4	1.2 (−1.7 to 6.9)	0.543
PCSS	45.3 ± 9.6	51.3 ± 8.4	44.1 ± 8.4	44.7 ± 7.2	3.7 (1.2 to 5.8)	0.001*
MCSS	50.3 ± 10.8	53.5 ± 12.7	49.2 ± 11.8	52.7 ± 11.2	1.1 (−2.7 to 2.9)	0.436
MRC scale #	4.3 ± 0.5	3.1 ± 0.2	4.1 ± 0.7	4 ± 0.4	0.4(−0.2 to 3)	0.213
6MWD, m #	178.2 ± 24.3	260.5 ± 32.2	179.3 ± 32.1	255.2 ± 30.2	9.2(−1 to 15)	0.001*

Autotitrating EPAP

- Physiological study on 10 patients with COPD with chronic respiratory failure
 - Age 65 ± 6 years; male 60%; BMI $27.6 \pm 7.2 \text{ kg} \cdot \text{m}^{-2}$; FEV₁ $28.4 \pm 8.3\%$ predicted
 - Esophageal catheter for recording the diaphragm electromyogram (EMG_{di}), oximetry, capnography, parasternal EMG electrodes
 - Titration protocol: baseline EPAP level of 3 cmH₂O for 5–10 min → autotitrating EPAP up to 45 min → optimal EPAP (EPAP_{opt}) → manually, EPAP_{opt} +3 for 10 min → reduced by 1 cmH₂O every 10 min until, EPAP_{opt} –3
 - optimal EPAP = 9 (range 4–13) cmH₂O

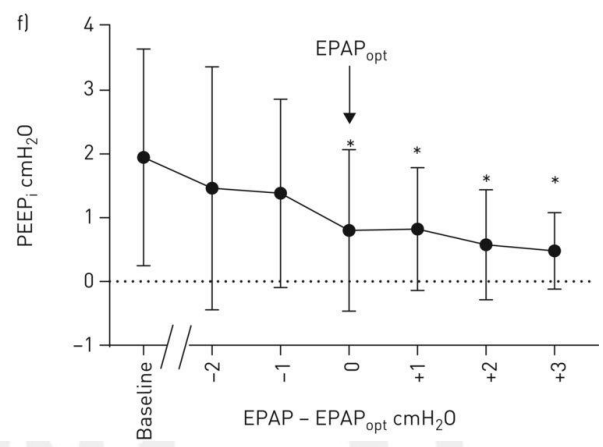
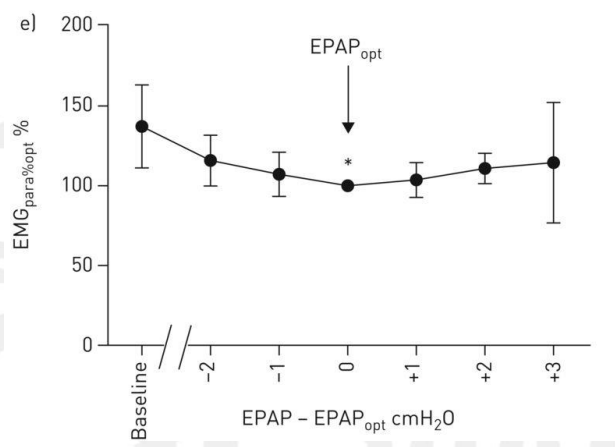
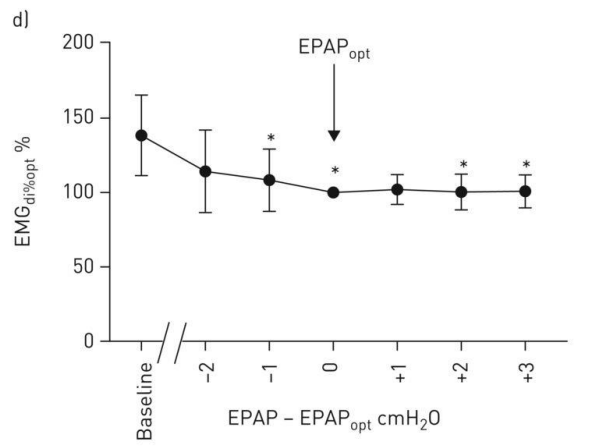
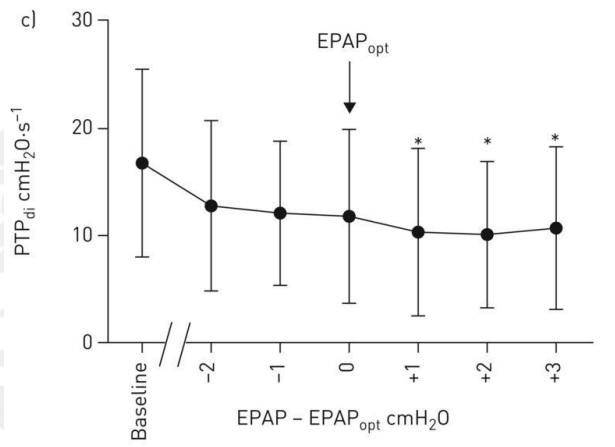
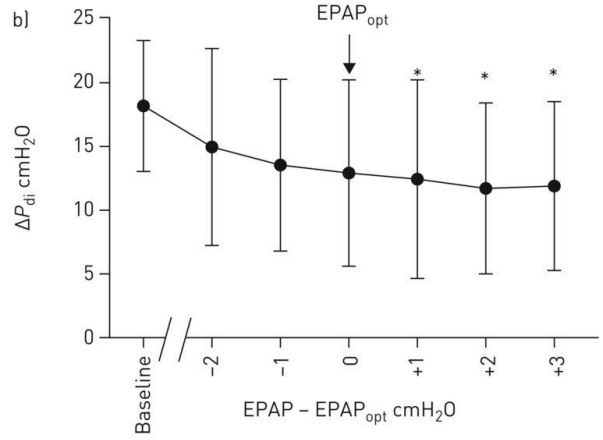
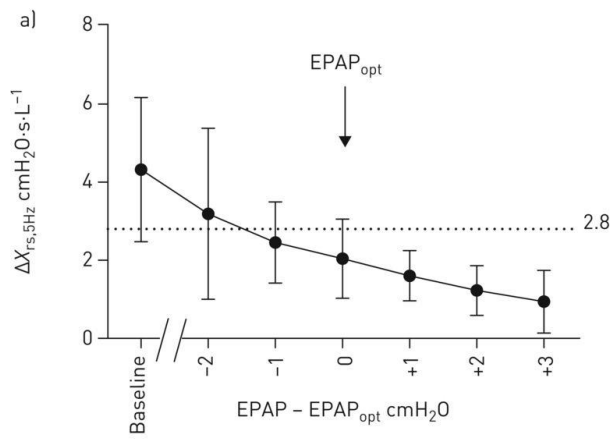


Autotitrating EPAP

TABLE 2 Physiological parameters at baseline (externally applied positive airway pressure (EPAP) 3 cmH₂O) and optimal EPAP (EPAP_{opt}) achieved at the end of the auto-titration phase

	Baseline (EPAP 3 cmH ₂ O)	EPAP _{opt}	p-value
EMG_{di%opt} %	138.5±27.1	120.9±20.7	0.049
EMG_{para%opt} %	134.3±27.0	115.6±23.8	0.008
ΔP_{di} cmH₂O	18.2±5.1	13.2±7.0	0.006
Respiratory rate bpm	19.0±3.6	20.8±3.2	0.049
P_{tccO₂} kPa	7.3±1.2	7.1±1.3	0.82
S_{pO₂} %	91.6±3.9	91.8±3.4	0.57

EMG_{di%opt}: diaphragm electromyogram as a percentage of the value at optimal EPAP; EMG_{para%opt}: parasternal electromyogram as a percentage of the value at optimal EPAP; ΔP_{di}: peak transdiaphragmatic inspiratory pressure swing



- a) within-breath change in respiratory system reactance ($\Delta X_{rs,5Hz}$),
- b) transdiaphragmatic pressure swings (ΔP_{di}),
- c) diaphragm pressure-time product (PTP_{di}),
- d) $EMG_{di\%opt}$ (diaphragm electromyogram as a percentage of the value at optimal expiratory positive airway pressure (EPAPopt)),
- e) $EMG_{para\%opt}$ (parasternal EMG as a percentage of the value at EPAPopt)
- f) intrinsic positive end-expiratory (PEEPi) during manual downtitration of EPAP from EPAPopt+3 to baseline EPAP 3 cmH₂O.

Average Volume Assured Pressure Support and auto-EPAP (AVAPS-AE): Similar efficacy than ST mode in obesity hypoventilation

RCT in patients with OHS



Evaluating sleep quality with ST or AVAPS-AE

Primary outcome

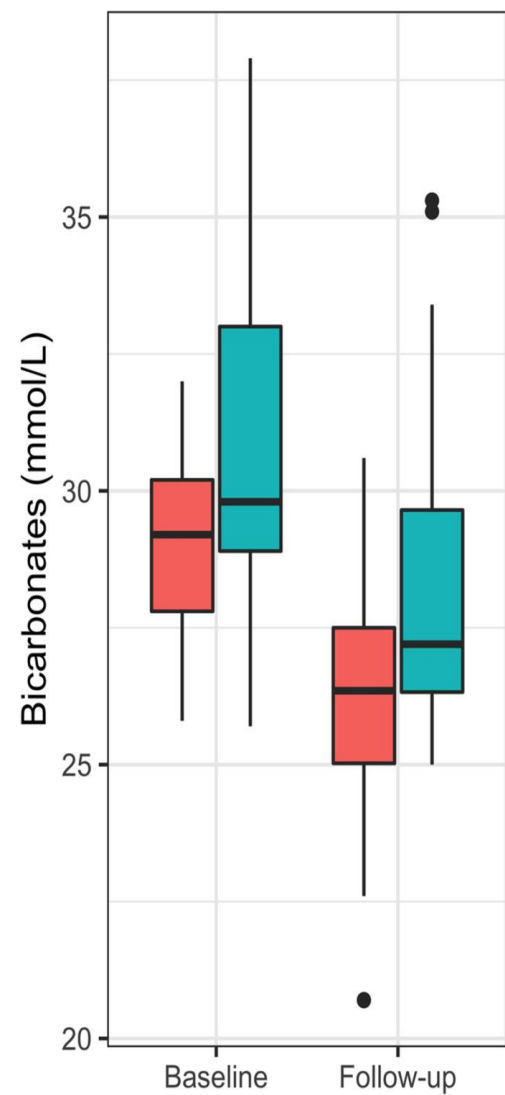
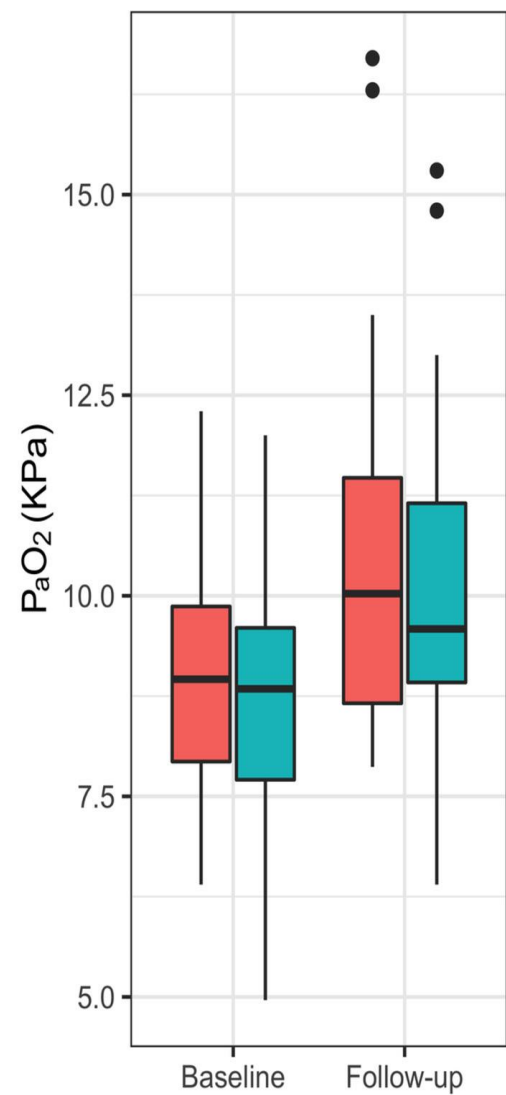
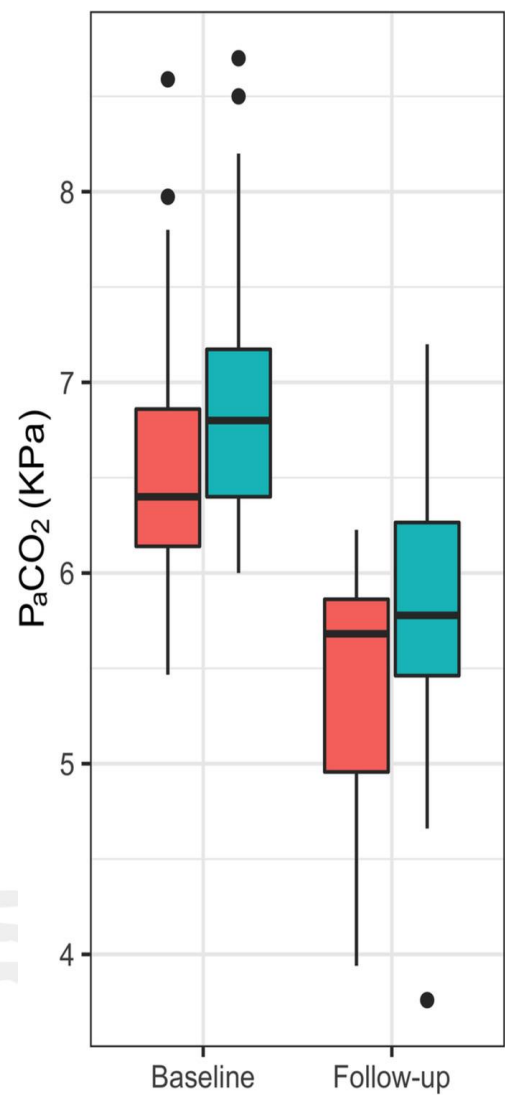
- Improvement in objective sleep quality at 2 months following NIV setup in both groups
- No difference between the two groups

Secondary outcome

- Similar improvement in gas exchange between the two groups
- Similar adherence to NIV
- Similar improvement in health-related quality of life

Table 2 Results from overnight polysomnography at baseline (room air self-venting) and at 2-months follow-up (under non-invasive ventilation) (t-test)

	ST (<i>n</i> = 26)			AVAPS-AE (<i>n</i> = 30)			Between-group comparison at follow-up
	Mean \pm SD			Mean \pm SD			
	Baseline	Follow-up	Pre-post <i>P</i> -value	Baseline	Follow-up	Pre-post <i>P</i> -value	
AHI (/h)	50 \pm 29	10 \pm 10	<0.001	56 \pm 33	12 \pm 13	<0.001	0.347
3% ODI (/h)	61 \pm 31	22 \pm 16	<0.001	72 \pm 43	22 \pm 15	<0.001	0.817
TST < 90% (%)	64 \pm 38	31 \pm 32	<0.001	62 \pm 35	33 \pm 31	<0.001	0.600
Mean saturation (%)	85 \pm 6	91 \pm 3	<0.001	86 \pm 4	91 \pm 3	<0.001	0.982
TST (min)	320 \pm 101	311 \pm 115	0.381	342 \pm 92	329 \pm 81	0.449	0.481
Sleep latency (min)	21 \pm 29	24 \pm 28	0.397	18 \pm 20	19 \pm 27	0.773	0.704
Arousal index (/h)	44 \pm 23	20 \pm 12	<0.001	48 \pm 30	19 \pm 10	<0.001	0.909
Respiratory arousal index (/h)	8 \pm 23	1 \pm 1	<0.001	7 \pm 7	1 \pm 1	<0.001	0.365
Hypoventilation (min)	63 \pm 102	5 \pm 18	0.002	35 \pm 63	7 \pm 14	0.002	0.296
WASO (min)	104 \pm 88	80 \pm 53	0.468	75 \pm 54	65 \pm 39	0.272	0.331
Awakenings (<i>n</i>)	38 \pm 24	31 \pm 23	0.085	33 \pm 19	29 \pm 24	0.070	0.634
Sleep efficacy (%)	75 \pm 17	75 \pm 22	0.897	79 \pm 13	81 \pm 10	0.365	0.534
REM (%)	11 \pm 8	16 \pm 12	0.045	11 \pm 8	19 \pm 10	0.001	0.227
N1 (%)	29 \pm 16	19 \pm 11	0.024	28 \pm 19	18 \pm 15	0.011	0.386
N2 (%)	46 \pm 11	46 \pm 11	0.752	44 \pm 12	44 \pm 9	0.996	0.408
N3 (%)	16 \pm 9	21 \pm 12	0.059	18 \pm 12	23 \pm 12	0.039	0.525



Intelligent Volume-Assured Pressure Support (iVAPS) with AutoEPAP algorithm controls upper airway obstruction in chronic respiratory failure.

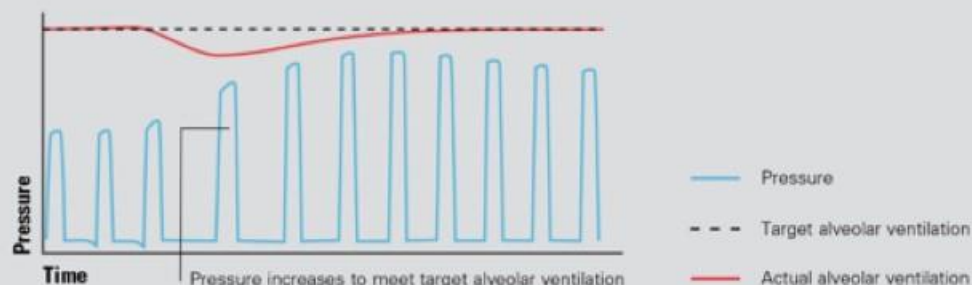
Study Population

38 patients with chronic respiratory disease on NIV therapy:

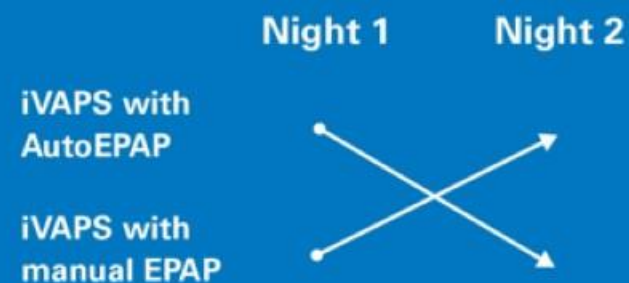
- 17 (44.7%) Neuromuscular
- 11 (28.9%) COPD
- 4 (10.5%) Obesity Hypoventilation
- 6 (15.8%) Other



All study patients were placed on NIV using iVAPS. iVAPS algorithm automatically adjusts to meet patient's target alveolar ventilation needs.



Intervention | PSG Study Design



AutoEPAP non-inferior to Manual EPAP

ODI 4% down from 7 to 2.5



AHI down from 8 to 4.X

Primary Outcome | ODI 4%

mean paired difference of ODI 4%:
iVAPS AutoEPAP – iVAPS manual EPAP is

-4.45

N=38, p value 0.0001

No difference in ODI between treatments in subgroups: Neuromuscular, COPD, Obesity Hypoventilation, Other

Secondary Outcomes

Using iVAPS AutoEPAP was just as effective as iVAPS with manually titrated EPAP for control of ventilation and sleep quality

Mean Overnight TcCO₂

45.5 iVAPS AutoEPAP
45.4 iVAPS manual EPAP

Mean Arousal Index

25.9 iVAPS AutoEPAP
31.9 iVAPS manual EPAP

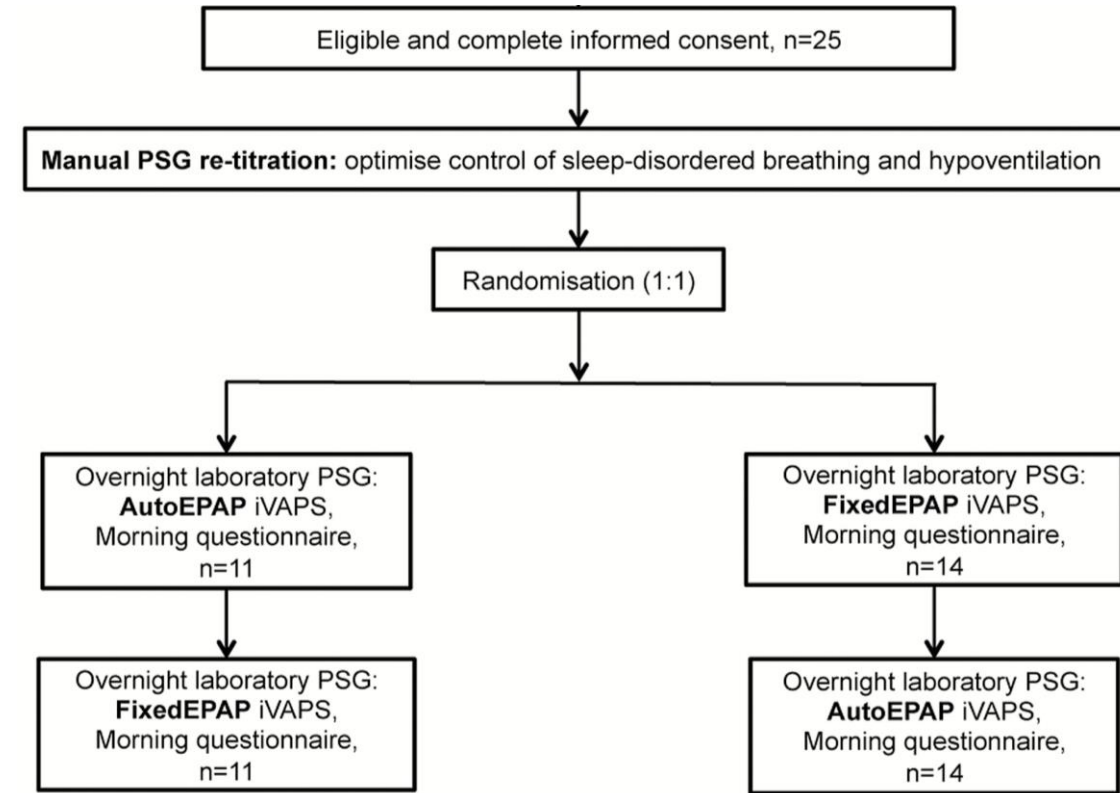
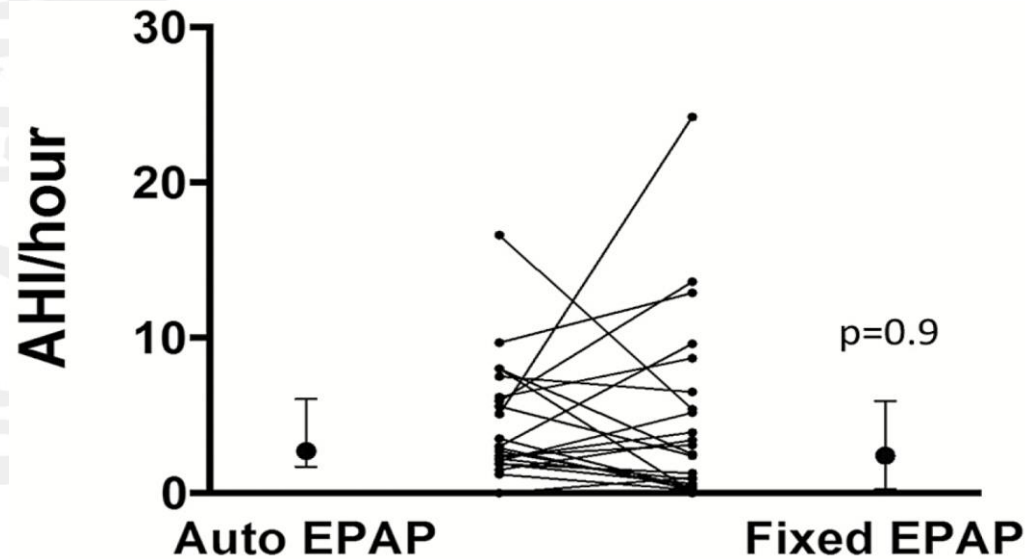
Respirology

Automatic EPAP Intelligent Volume-Assured Pressure Support is Effective in Patients with Chronic Respiratory Failure: A Randomized Trial

Orr et al. Respirology 2019; DOI: 10.1111/resp.13546

Autotitrating EPAP

- Double-blind, crossover, noninferior RCT on 25 patients with chronic hypoventilation and OSA
 - OHS (n = 11), COPD (n = 9), or NMD (n = 5)
 - autoEPAP iVAPS vs fixedEPAP iVAPS
 - Primary outcome: AHI



Clinical implications

- ASV
 - Newer generation ASV devices ? autoEPAP ?
- VAPS
 - Current evidence does not suggest any superiority of VAPS over BPAP in treating either acute or chronic hypercapnic respiratory failure
 - No safety concerns
- Autotitrating EPAP
 - May be of greatest benefit in obese patient and those with concomitant OSA
- Telemonitoring
- Outpatient set-up of NIV



Conclusions

- Specialized modes of automatically adjusting BPAP are now widely available on home devices.
 - Can aid optimizing therapy especially in complex SRBD
- However, it must also be acknowledged that robust evidence demonstrating these newer modes of ventilation provide any greater clinical benefits than fixed pressure modes is lacking.
 - They do not replace informed clinical judgment and considered manual intervention
 - Simple therapy (CPAP and fixed pressure BPAP) usually trialed first.
- Continuous long-term monitoring & advancements in technology are a very welcome, broadening our options.

Thanks for your attention

