Update on Adaptive Servo-Ventilation

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September 23, 2016

Focus on Advanced Sleep Medicine
WSS Annual Meeting 2016

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About one o'clock

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Disclosures

- No financial conflicts of interest
- Associate Professor of Medicine
  University of Wisconsin School of Medicine and Public Health
- Chair, CHEST ACCP) Guidelines Oversight Committee
- Co-author of AASM Guidelines related to treatment of CSA

- **Opinions expressed do not necessarily reflect positions or policies of the Department of Veterans Affairs or the US Government.**

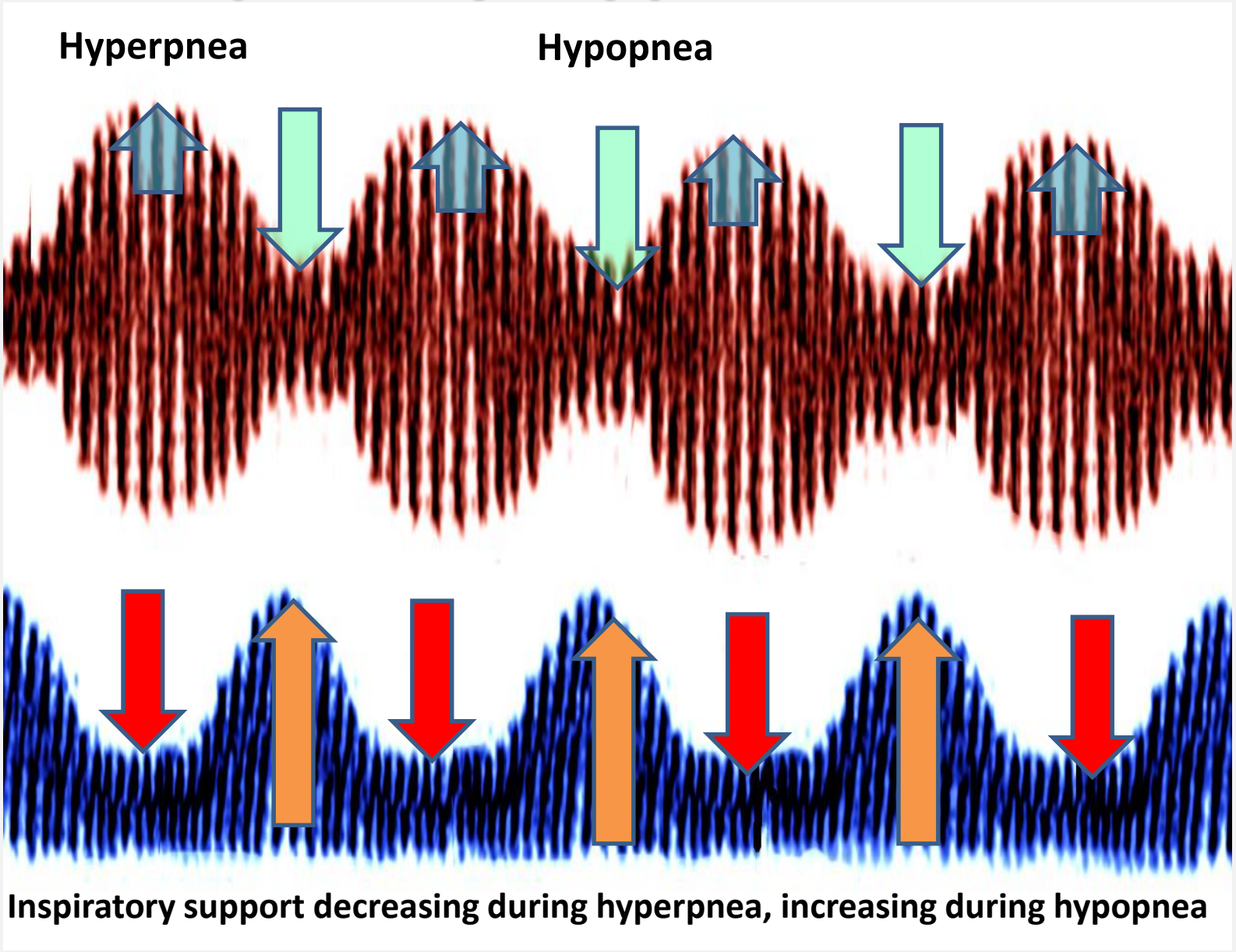
Definition of Adaptive Servo-Ventilation

- ASV is an advanced form of pressure supported non-invasive ventilation, which
- Monitors the patient's respiration and "**adapts**" the degree of pressure support,
- Assessing by means of a "**servo**" feedback loop, it
- Increases or decreases pressure support to achieve a target "**ventilation**"

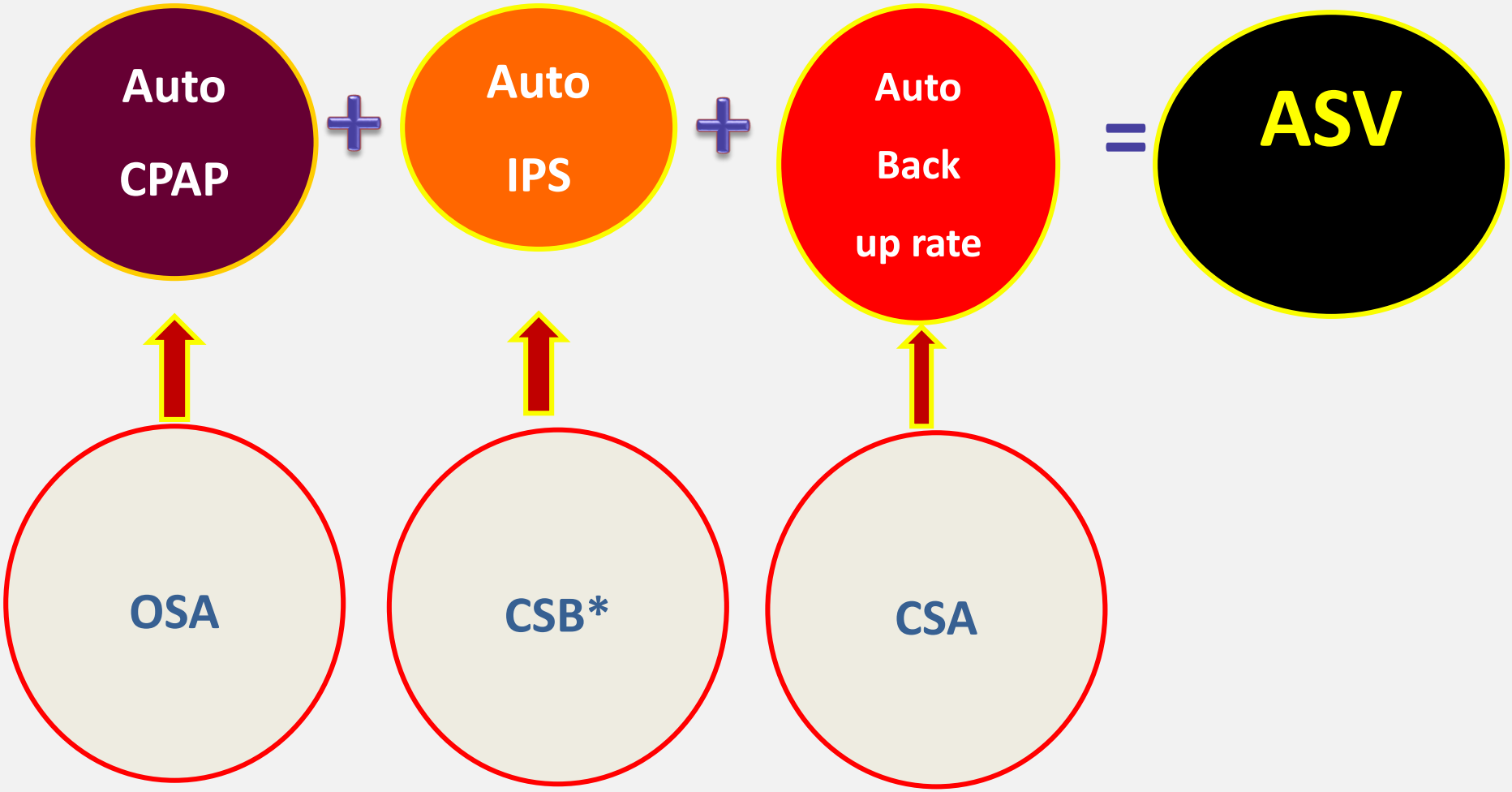
Components of ASV Devices

- Auto variable inspiratory support
  - ↗ Increases with hypopnea
  - ↘ Decreases with hyperpnea
- Auto back up rate to abort any impending apnea
- Auto CPAP (EPAP) to eliminate obstructive events

Variable inspiratory support



After Harris and Javaheri . Advanced PAP therapies , Fundamentals of Sleep Technology, 2012



* Periodic breathing.

Slide courtesy of S Javaheri .

2 ASV devices in US

Philips Respironics	ResMed
4.25 lbs	3.35 lbs
10.7 in	11.2 in
6.5 in	6.2 in
4.0 in	3.4 in

Adaptive Servo Ventilation

Respironics - Auto SV		ResMed – VPAP Adapt
EPAP	⟷	EPAP (*aka* EEP)
$IPAP_{MIN}$	⟷	EPAP + PS_{MIN}
$IPAP_{MAX}$	⟷	EPAP + PS_{MAX}

Adaptive Servo-ventilation (ASV) How does it Work?

Both compute patient airflow and minute ventilation from machine flow and mask pressure
Both are bilevel positive airway pressure devices

ResMed VPAP adapt SV

- Calculates recent average minute ventilation
- Targets a percent of recent average minute ventilation (90-95%)
- For each delivered breath, adjusts inspiratory positive airway pressure (IPAP) and thus pressure support (PS) accordingly
- Employs back-up rate

VPAP adapt SV

BiPAP Auto SV

Respironics BiPAP Auto SV (± "Advanced")

- Calculates recent average peak inspiratory flow
- Targets a percent of recent peak inspiratory flow using a combination of parameters based on index of Cheyne-Stokes severity, pattern of breathing compared to stored severe pattern and recently required amount of PS
- For each delivered breath, adjusts inspiratory positive airway pressure (IPAP) and thus pressure support (PS) accordingly
- Employs back-up rate
- "Advanced" model adds independent auto-titration of expiratory positive airway pressure (EPAP)

Slide courtesy of Lee Brown ,MD

VPAP adapt SV Technology: Estimating Instantaneous Respiratory Airflow

Compute leakage conductance (inverse of resistance) from instantaneous mask airflow divided by the square root of instantaneous mask pressure (measured from mask sampling tube)

Compute mask leak from leakage conductance multiplied by the square root of instantaneous mask pressure

Subtract mask leak from total airflow delivered to mask to determine instantaneous respiratory airflow

Slide courtesy of Lee Brown ,MD

VPAP adapt SV Technology: Determining Value of Pressure Support

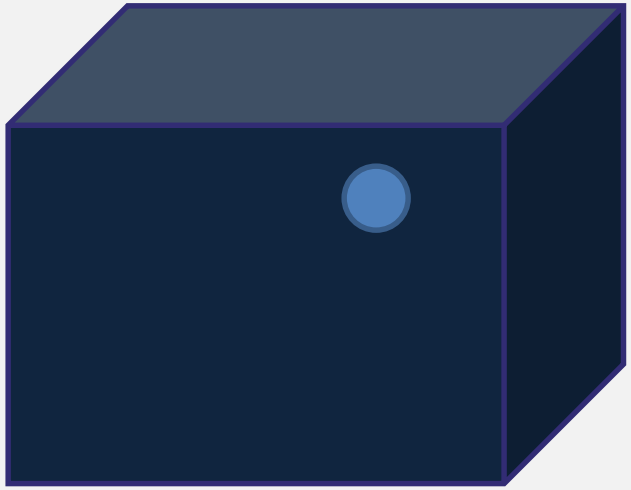
Integrate instantaneous airflow signal using low pass filter with TC ~100 seconds to determine recent average ventilation. TC is chosen to exceed the typical lung-chemoreceptor delay and CSR cycle times

Use percentage of recent average ventilation (90-95%) as the target value in the regulatory control loop (clipped integral controller)

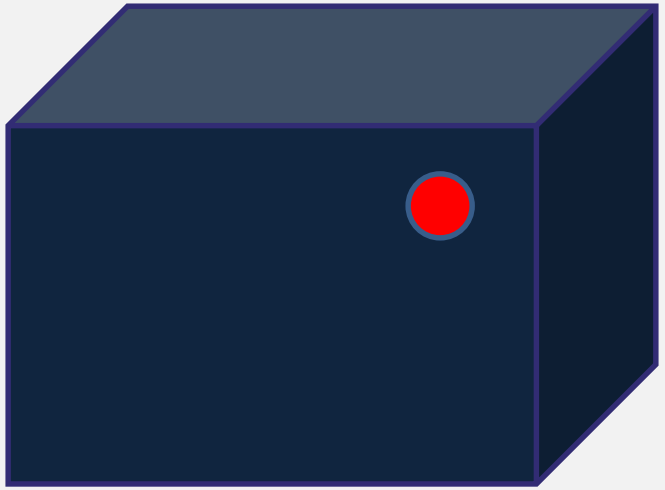
Controller subtracts absolute value of the instantaneous respiratory airflow from the target ventilation, multiplies it by a constant (typically, -0.3) and integrates the result over a few breaths. This error term is used to determine the value of pressure support

Slide courtesy of Lee Brown ,MD

2 ASV devices in US



Black box 1
(with a blue light)



Black box 2
(with a red light)

Indications for ASV

- Complex pattern of obstructive, central, and mixed apnea and hypopnea. (aka “Complex Sleep-disordered Breathing”)
- Idiopathic primary central sleep apnea
- Treatment-induced central sleep apnea (aka “Complex Sleep Apnea”)
- Opiate related central sleep apnea (aka Biot’s breathing, ataxic breathing)
- CSA in patients with **HFrEF**
- CSA in patients with **HFpEF**

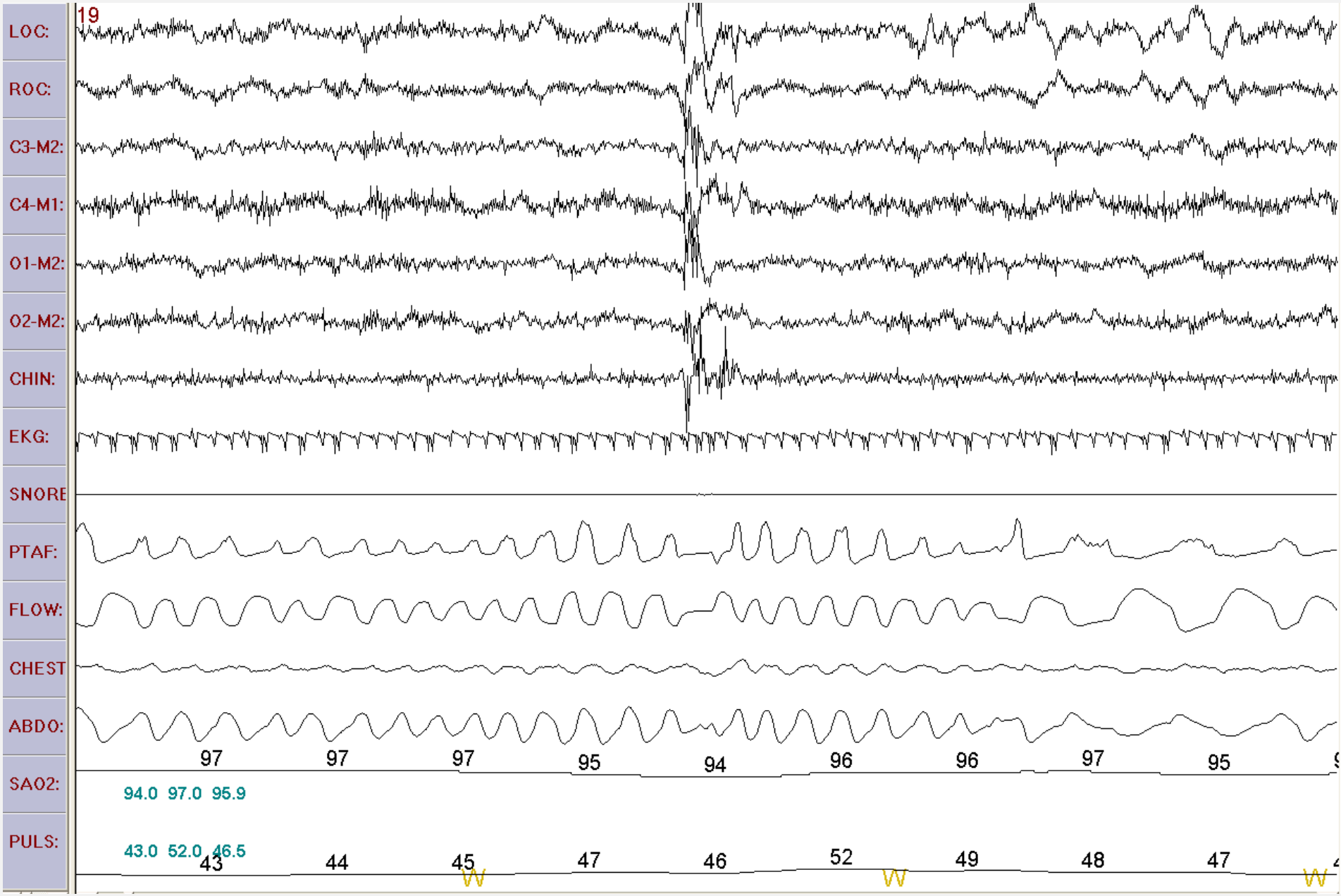
Complex Sleep-Disordered Breathing

Case Report

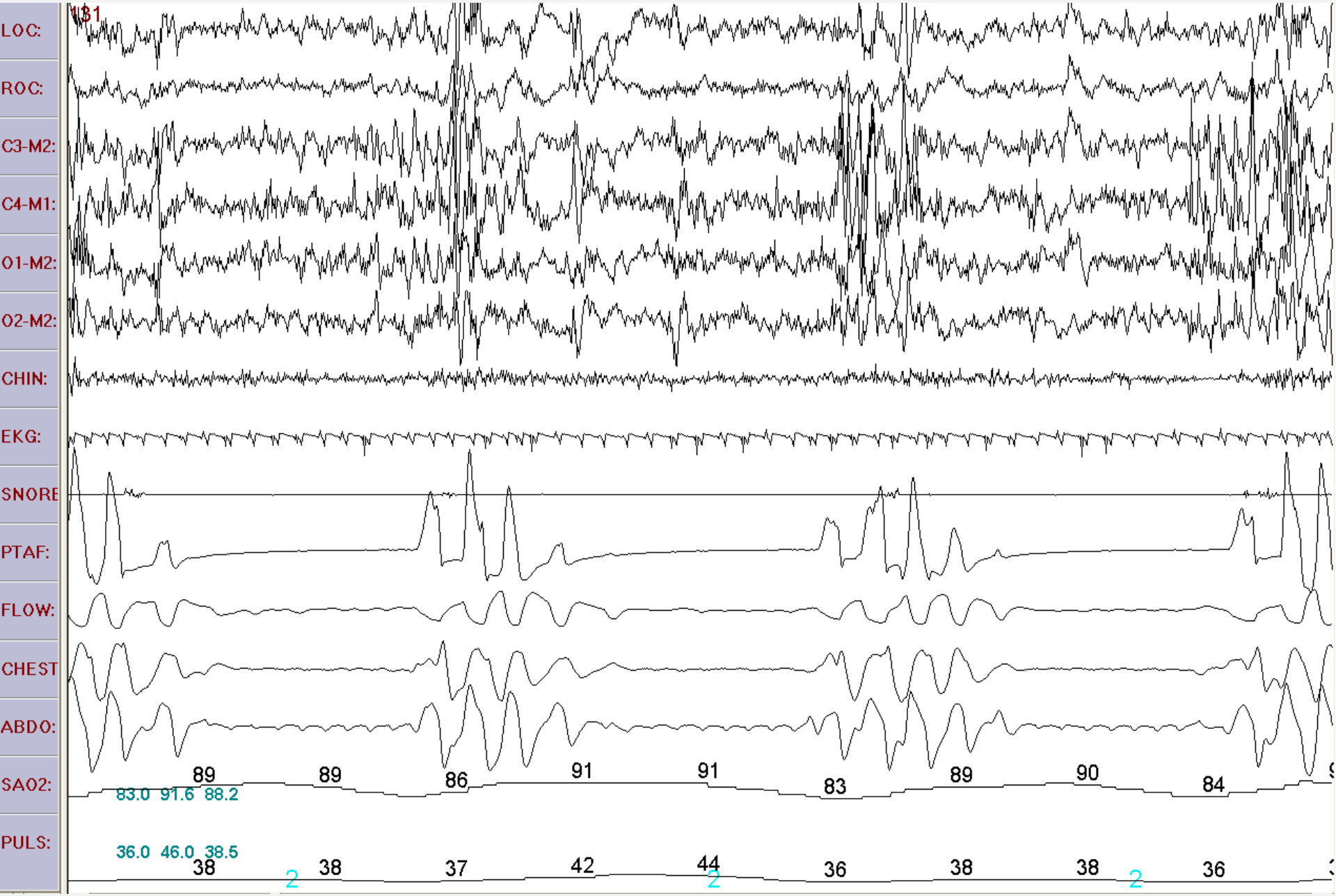
- 26 year old man, Ht. 76 in. Wt. 235 lbs.
- Generally good health, active military
- Complains of snoring, witnessed apnea, daytime sleepiness
- Previously diagnosed with “obstructive sleep apnea” but he was intolerant of CPAP. Underwent an unsuccessful surgical treatment of the upper airway

Brown LK, Casey KR. Curr Opin Pulm Med 2007; 13:473.

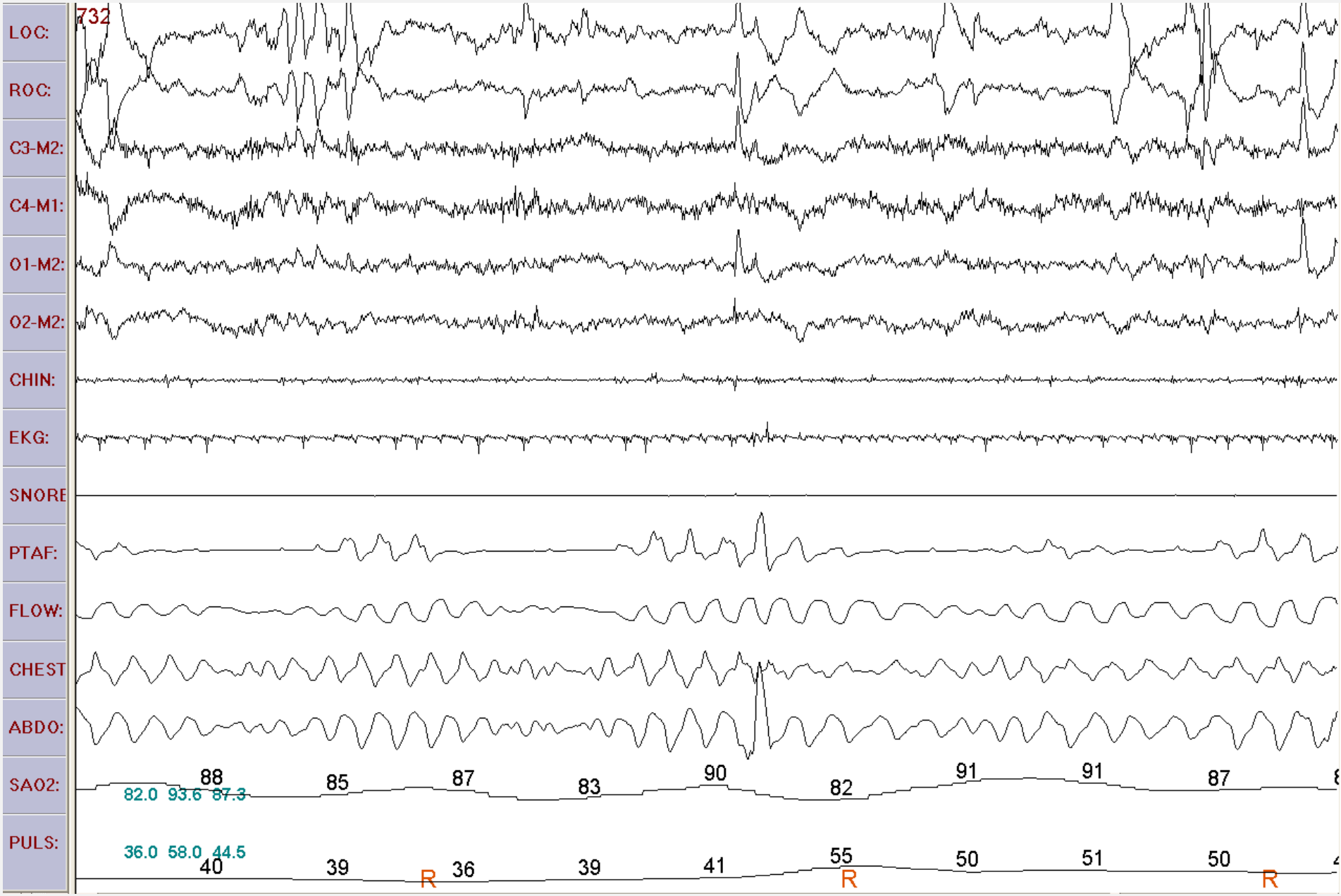
Baseline, awake, drowsy



Stage 2 NREM sleep



REM sleep


LOC:
ROC:
C3-M2:
C4-M1:
O1-M2:
O2-M2:
CHIN:
EKG:
SNORE
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CHEST
ABDO:
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732
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82.0 93.6 87.3
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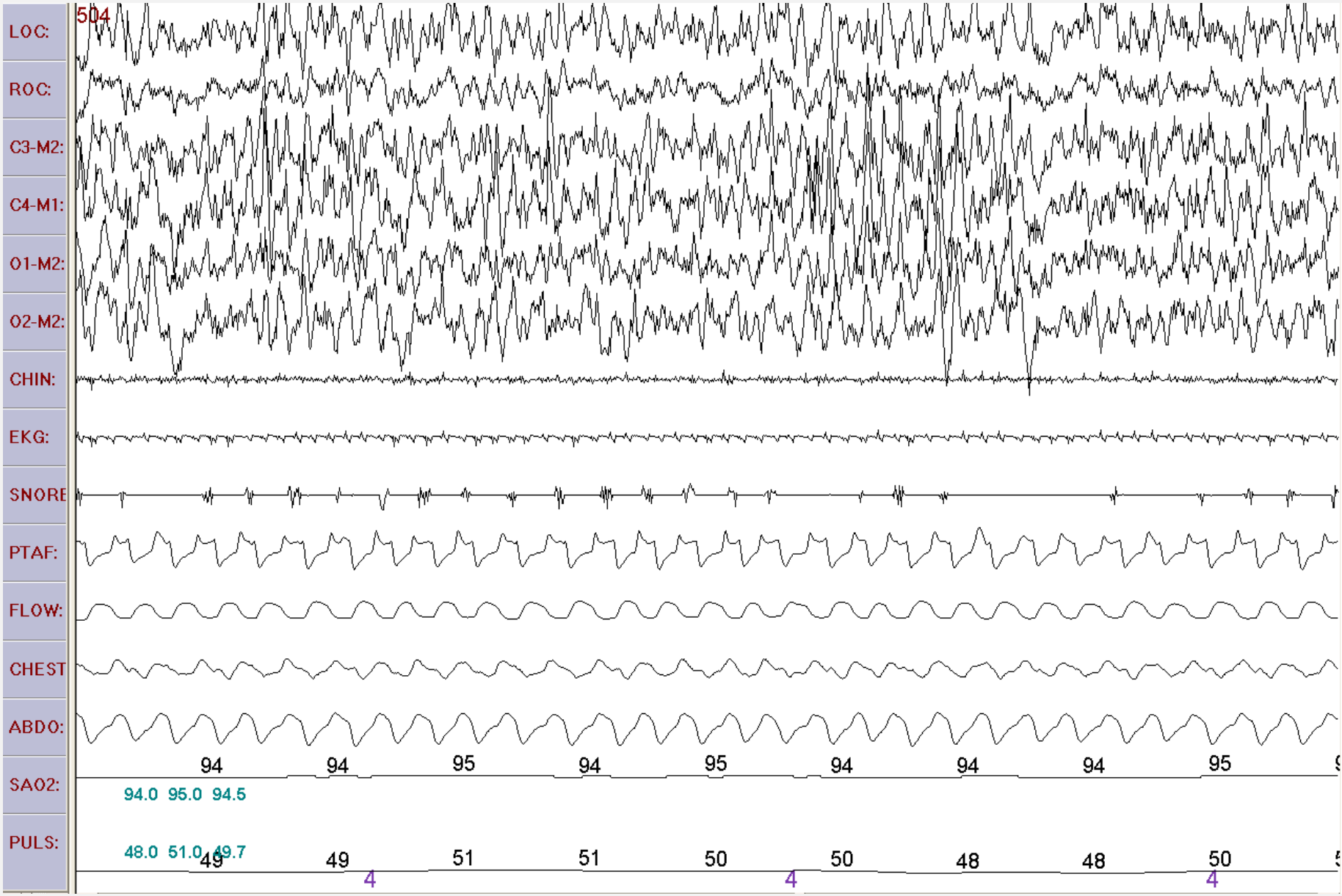
Stage 3/4 NREM Sleep



Brown LK, Casey KR. Curr Opin Pulm Med 2007; 13:473.

Patterns of Complex Sleep-disordered Breathing

1. Mixed apneas
2. Periodic breathing intermixed with obstructive events
3. Position dependence of mechanism (obstructive supine, central non-supine)
4. Sleep stage dependence
5. Time of night dependence

Gilmartin GS, Daley RW, Thomas RJ. Curr Opin Pulm Med 2005; 11:485.

Complex Sleep-disordered Breathing

1. Presumably related to a combination of upper airway obstruction and ventilatory control abnormalities
2. Quite uncommon
3. Represent a challenge to treatment
   - Supplemental oxygen
   - Sedative-hypnotic medication
   - Exogenous/endogenous CO_2
   - ASV

Treatment-induced Central Sleep Apnea

Case Report

- 65 year old, obese male smoker with coronary artery disease (s/p PCI to RCA in 2007). No active complaints. BMI 36.2, BP 142/75. Recent stress test was negative.
- Medications:
  - AMLODIPINE 10 MG ONCE DAILY
  - HYDRALAZINE 25 MG THREE TIMES DAILY
  - HCTZ 12.5 MG ONCE DAILY
  - ISOSORBIDE 60 MG SA ONCE DAILY
  - METOPROLOL 50MG TWICE DAILY
  - PRAVASTATIN 40 MG ONCE DAILY

Case Report

- Echocardiogram:
  - LV size normal. Normal systolic function. Ejection fraction 55%-60%. Abnormal LV relaxation (grade 1 diasolic dysfunction.
- No symptoms of sleep apnea except snoring. Difficult to control hypertension.
- Recommendations:
  - Increase metoprolol, continue other medications.
  - Advised to quit smoking
  - Weight loss program, exercise
  - Refer for sleep study

Polysomnography findings (2 min window)

PSG Findings

SLEEP ARCHITECTURE AND STAGING DATA:

- The time in bed (TIB) was 387.5 minutes. Total sleep time (TST) was 254.0 minutes. Thus, sleep efficiency was 65.5% related to an initial sleep latency of 7.0 minutes and wakefulness after sleep onset (WASO) of 126.5 minutes. Latency to REM from sleep onset was 70.5 minutes.
- Stage N1 sleep accounted for 29.3%, stage N2 60.6%, stage N3 0.0%, and REM 10.0% of TST. There were 47.5 stage changes per hour of sleep.

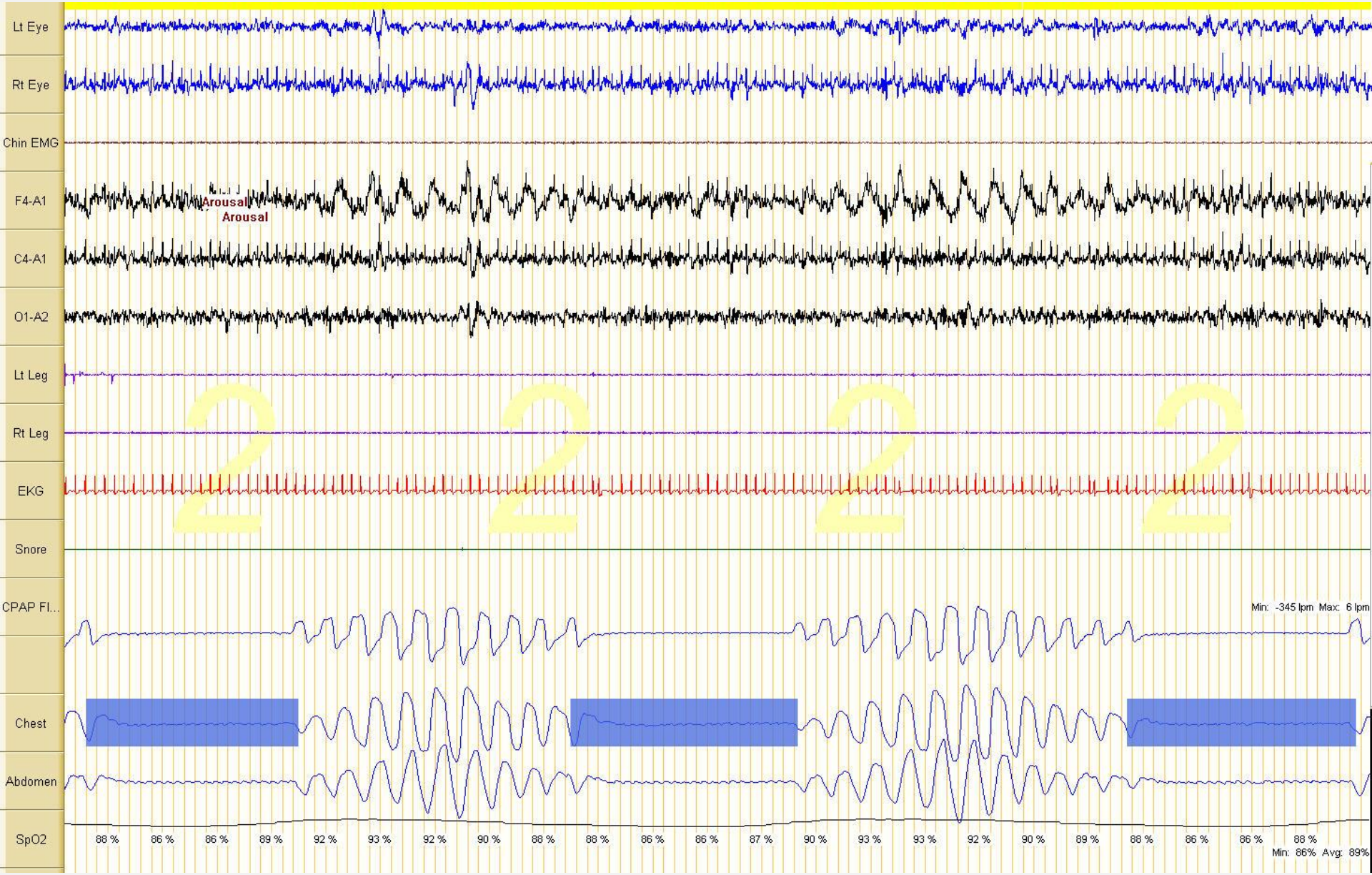
RESPIRATORY EVENTS:

- There were a total of 209 apneas recorded: 4 obstructive, 186 central, and 19 mixed in character. There were also 40 hypopneas noted.
- The overall AHI was 58.8 events/hr of sleep. The Central Apnea Index was 43.9/hr. The AHI was 64.9 /hr in NREM sleep versus 4.7 /hr during REM sleep. During 102.5 minutes of sleeping supine the AHI was 69.6 /hr compared to 53.6 /hr non-supine.
  - AHI: 58.8 /hr
  - Supine: 69.6 /hr
  - Non Supine: 53.6 /hr
  - REM: 4.7 /hr
  - NREM: 64.9 /hr
  - CAI: 43.9 /hr

What should be done next?

1. Supplemental oxygen at 2 L/min
2. Titration of CPAP/BPAP in the sleep laboratory
3. Home auto-titration of CPAP for 2 weeks
4. Titration of Adaptive Servo Ventilation

CPAP Titration Polysomnography findings (2 min window)



Complex Sleep Apnea Syndrome: Is It a Unique Clinical Syndrome?

Timothy I. Morgenthaler, MD[1,2]; Vadim Kagramanov, MD[3]; Viktor Hanak, MD[2]; Paul A. Decker, MS[4]

[1]Mayo Clinic Sleep Disorders Center, Rochester, MN; [2]Division of Pulmonary and Critical Care Medicine, Mayo Clinic, Rochester, MN; [3]Michigan Medical PC, Grand Rapids, MI; [4]Division of Biostatistics, Mayo Clinic, Rochester, MN

Study Objectives: Some patients with apparent obstructive sleep apnea hypopnea syndrome (OSAHS) have elimination of obstructive events but emergence of problematic central apneas or Cheyne-Stokes breathing pattern. Patients with this sleep-disordered breathing problem, which for the sake of study we call the "complex sleep apnea syndrome," are not well characterized. We sought to determine the prevalence of complex sleep apnea syndrome and hypothesized that the clinical characteristics of patients with complex sleep apnea syndrome would more nearly resemble those of patients with central sleep apnea syndrome (CSA) than with those of patients with OSAHS.

Design: Retrospective review

Setting: Sleep disorders center.

Patients or Participants: Two hundred twenty-three adults consecutively referred over 1 month plus 20 consecutive patients diagnosed with CSA.

Interventions: NA.

Measurements and Results: Prevalence of complex sleep apnea syndrome, OSAHS, and CSA in the 1-month sample was 15%, 84%, and 0.4%, respectively. Patients with complex sleep apnea syndrome differed in gender from patients with OSAHS (81% vs 60% men, $p < .05$) but were otherwise similar in sleep and cardiovascular history. Patients with complex sleep apnea syndrome had fewer maintenance-insomnia complaints (32% vs 79%; $p < .05$) than patients with CSA but were otherwise not significantly different clinically. Diagnostic apnea-hypopnea index for patients with complex sleep apnea syndrome, OSAHS, and CSA was 32.3 ± 26.8, 20.6 ± 23.7, and 38.3 ± 36.2, respectively ($p = .005$). Continuous positive airway pressure suppressed obstructive breathing, but residual apnea-hypopnea index, mostly from central apneas, remained high in patients with complex sleep apnea syndrome and CSA (21.7 ± 18.6 in complex sleep apnea syndrome, 32.9 ± 30.8 in CSA vs 2.14 ± 3.14 in OSAHS; $p < .001$).

Conclusions: Patients with complex sleep apnea syndrome are mostly similar to those with OSAHS until one applies continuous positive airway pressure. They are left with very disrupted breathing and sleep on continuous positive airway pressure. Clinical risk factors don't predict the emergence of complex sleep apnea syndrome, and best treatment is not known.

Keywords: Sleep apnea, mixed central and obstructive; sleep-disordered breathing; sleep hypopnea

Citation: Morgenthaler TI; Kagramanov V; Hanak V et al. Complex sleep apnea syndrome: is it a unique clinical syndrome? *SLEEP* 2006;29(9):1203-1209.

Complex sleep apnea: unique clinical syndrome?

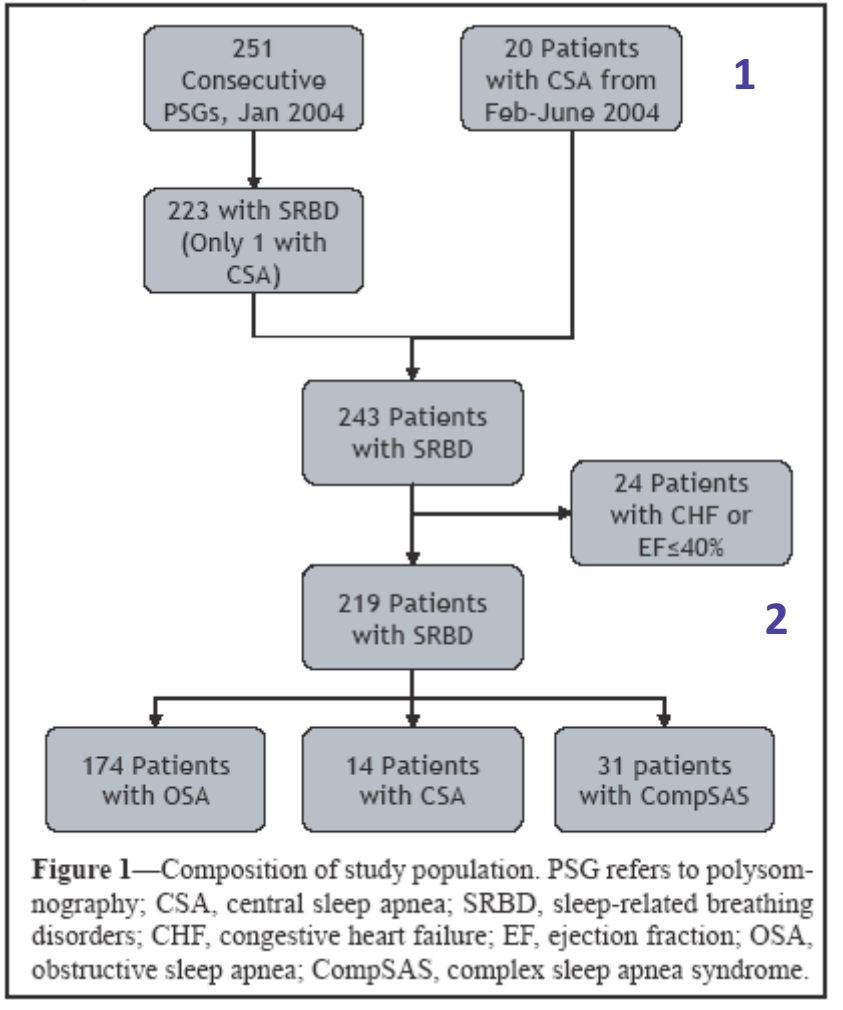


Figure 1—Composition of study population. PSG refers to polysomnography; CSA, central sleep apnea; SRBD, sleep-related breathing disorders; CHF, congestive heart failure; EF, ejection fraction; OSA, obstructive sleep apnea; CompSAS, complex sleep apnea syndrome.

- "CompSAS" defined by emergence of CSR or CSA during CPAP titration using a "split-night" protocol.
- Prevalence of Complex Sleep Apnea in one month sample was 15.2%.
- Prevalence of Primary CSA was 0.1%.

1 - Study population was "enriched" with patients diagnosed with CSA

2 – Patients with EF≤40% were excluded

Morgenthaler et al. Sleep 2006; 29:1203.

CPAP-induced CSA

Table 1—Demographics and Physical Findings of Patients with Sleep-Related Breathing Disorders

Characteristic	OSAHS (n=174)	CSA (n=14)	CompSAS (n=31)	p Value[a]
Age, y	56.7±13.1	56.1±15.4	52.3±15.2	.247
Men	105 (60)	6 (43)	25 (81)	.027[bc]
Body mass index, kg/m^2	34.7±9.8	29.3±6.0	33.0±6.0	.083[d]
ESS score	11.0±5.3	12.1±7.9	11.4±5.5	.728
Hypertension	96 (55)	8 (57)	16 (52)	.935
Habitual snoring	159 (91)	13 (93)	30 (97)	.709
Witnessed apneas	87 (50)	7 (50)	20 (65)	.340
Initial insomnia complaints	45 (26)	5 (36)	8 (26)	.739
Sleep maintenance insomnia	79 (45)	11 (79)	10 (32)	.016[cd]
Nocturnal dyspnea	34 (20)	5 (36)	7 (23)	.323
Peripheral edema	34 (20)	1 (7)	9 (29)	.236
Echo EF	0.60±0.06	0.62±0.04	0.60±0.06	.686
Echo RVSP	36.2±11.0	39.0±14.5	30.3±5.0	.247
Atrial fibrillation, past or present	15 (9)	1 (7)	2 (6)	1.000

- Few differences between groups
- CompSAS more likely to be male
- CompSAS less likely to complain of sleep-maintenance insomnia

Morgenthaler et al. Sleep 2006; 29:1203.

CPAP-induced CSA

Lehman S, et al. Central sleep apnea on commencement of CPAP in patients with a primary diagnosis of OSA/Hypopnea. J Clin Sleep Med 2007; 3:462-6.

- 13.1% (13/99)of patients demonstrated emergence or persistence of CSA at or near prescribed CPAP.
- Separate baseline and CPAP titration studies as well as split-night studies were included.
- Retrospective study.

CPAP-induced CSA

Javaheri S, Smith J, Chung E. The prevalence and natural history of complex sleep apnea. J Clin Sleep Med 2009; 205-11.

- Monthly incidence ranged from 2% to 10%. Overall, 6.5% (84/1286) in a one year period.
- Studies were all full night baseline and full night CPAP titration studies – no split-night studies were included.
- Retrospective study.

CPAP-induced CSA

- Endo Y, et al. Prevalence of complex sleep apnea among Japanese patients with SAS. Tohoku J Exp Med 2008; 215:349-54.

  Prevalence: 5.0% (males 5.3%, females 1.1%)

- Yaegashi H, et al. Characteristics of Japanese patients with complex sleep apnea syndrome: a retrospective comparison with OSAS. Intern Med 2009; 48:427-32.

  Prevalence: 5.7%

Author Site (year)	n	CompSA	PSG	AHI (/hr)	Follow-up PSG
Morgenthaler USA (2006)	223	15%	Split	32	-
Derniaka USA (2006)	116	20%	Split	51	2%
Lehman Australia (2007)	99	13%	Mixed	72	-
Javaheri USA (2009)	1286	6.5%	Full Night	57	2%
Endo Japan (2007)	1232	5.3%	Full Night	59	-
Yaegashi Japan (2009)	297	5.7%	Full Night	56	-
Cassel Germany (2011)	675	12.2%	Full Night	36	3%

Cassel W, et al. A prospective polysomnographic study on the evolution of complex sleep apnea. Eur Resp J 2011; 38:329-337.

Complex sleep apnea occurs in more severe OSA

AHI with and without CPAP-induced CSA

Author	No Complex SA	Complex SA
Morgenthaler	21	32
Derniaka	47	51
Lehman	53	72
Javaheri	39	79
Endo	49	59
Yaegashi	49	56
Cassel	26	36

CPAP-induced CSA: Prevalence

- Overall prevalence (all published series) is ***9.8%***
  - Split-night and mixed ***16.0%***
  - Full night titration ***7.1%***
- More likely to be ***male***
- More likely with ***severe*** obstructive sleep apnea
- More common in patients with co-morbid ***cardiac disease***
- May have ***fewer*** complaints of sleep-maintenance insomnia that other patients with OSAS
- Clinically similar in all other respects including age, BMI, snoring, witnessed apnea, daytime sleepiness

CPAP-induced CSA: Treatment

- Several studies have reported that Adaptive Servo Ventilation (ASV) is effective for treatment of Complex Sleep Apnea:

1. Morgenthaler TI, Gay PC, Gordon N, Brown LK. ASV versus noninvasive positive pressure ventilation for central, mixed, and complex sleep apnea syndromes. Sleep 2007; 30:468
2. Allam JS, Olson EJ, Gay PC, Morgenthaler, TI. Efficacy of ASV in treatment of complex and central sleep apnea syndromes. Chest 2007; 132:1839-46.

CPAP-induced CSA: Natural History

- Javaheri S, et al. 2009: Polysomnography repeated in 5 to 6 weeks
  - **CSA resolved in 79% (33/42)**
- Dernaika T, et al. Chest 2007; 132:81-7: PSG repeated in 2-3 months
  - **Complete or near complete resolution in 92% (12/14)**
- Kuzniar TJ, et al: 13 patients who had a 2nd PSG were identified. PSGs were often performed because of an abnormal oximetry study.
  - **Mean AHI decreased from 26 to 7 but 6 had AHI≥10**

Eur Respir J 2011; 38: 329–337
DOI: 10.1183/09031936.00162009

A prospective polysomnographic study on the evolution of complex sleep apnoea

W. Cassel*, S. Canisius*, H.F. Becker#, S. Leistner*, T. Ploch*, A. Jerrentrup*, C. Vogelmeier*, U. Koehler* and J. Heitmann*

- Prospective study of 675 OSA patients (mean age 55.9 yr, 13.9% female)
- Full night PSG at diagnosis, 1st night with stable CPAP, and after 3 months CPAP treatment
- 12.2% (82/675) had initial CompSA
- 6.9% (30/436) had CompSA at follow up

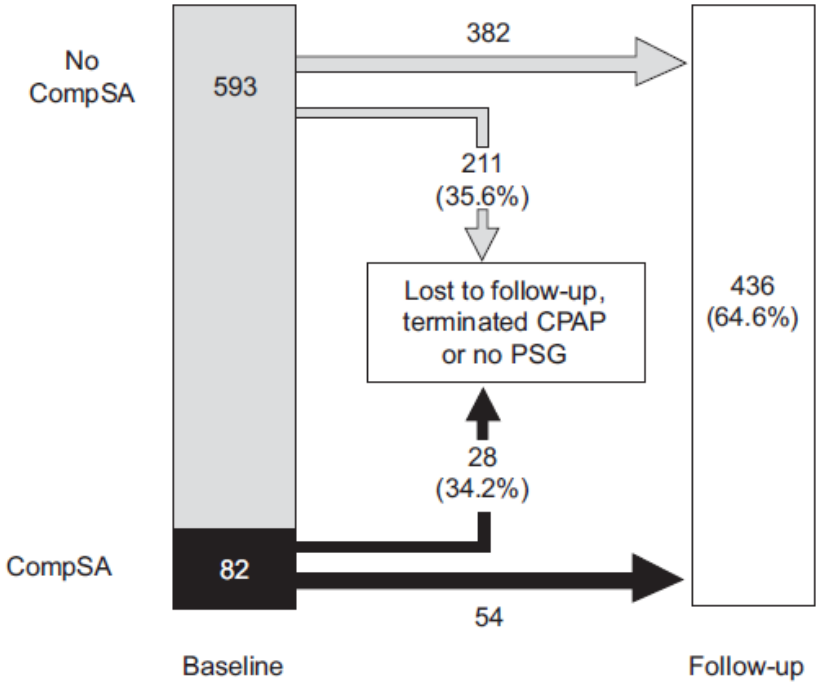


FIGURE 1. Number of patients available for and lost to the 3-month follow-up. CompSA: complex sleep apnoea; CPAP: continuous positive airway pressure; PSG: polysomnography.

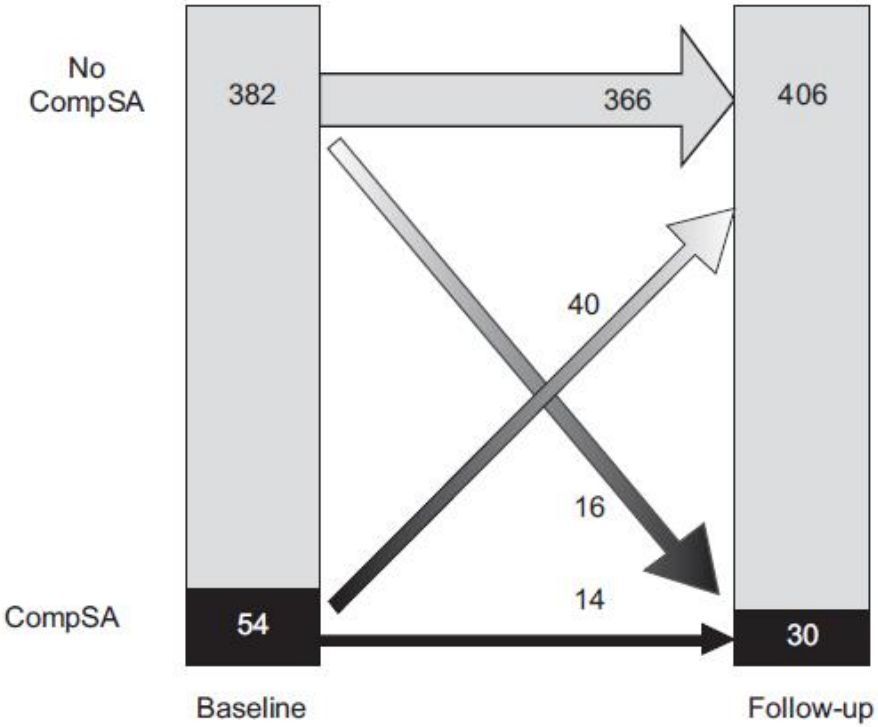


FIGURE 2. Number of patients with complex sleep apnoea (CompSA) and no CompSA over the 3-month study period.

- Of the 82 patients initially diagnosed, 28 lost to follow up, of the remaining 54 patients 14 met criteria for CompSA
- **16 of the 382 patients <u>not initially diagnosed</u> with CompSA exibited CompSA at follow up**

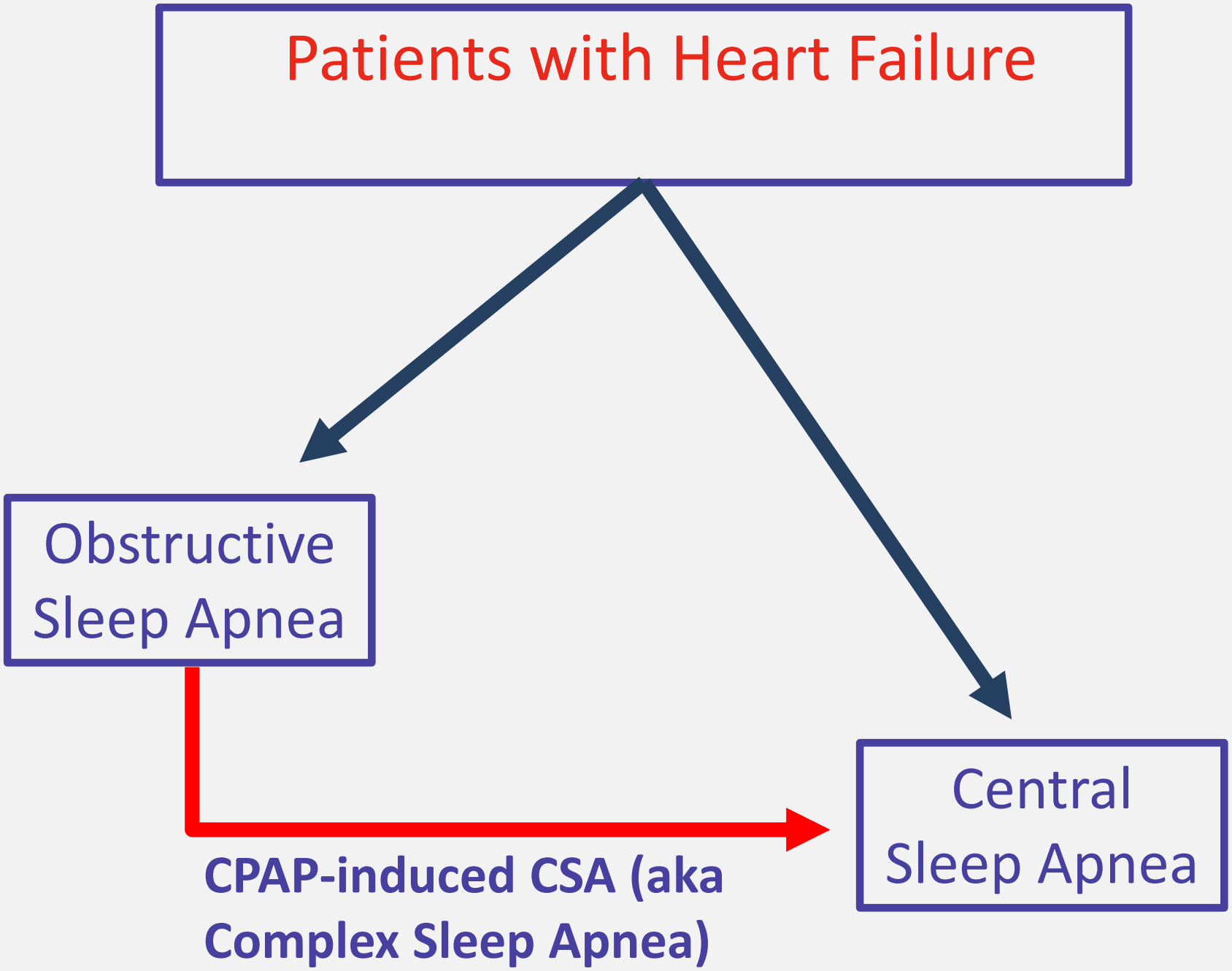
Cassel W, et al. A prospective polysomnographic study on the evolution of complex sleep apnea. Eur Resp J 2011; 38:329-337.

CPAP-induced CSA: Pathogenesis

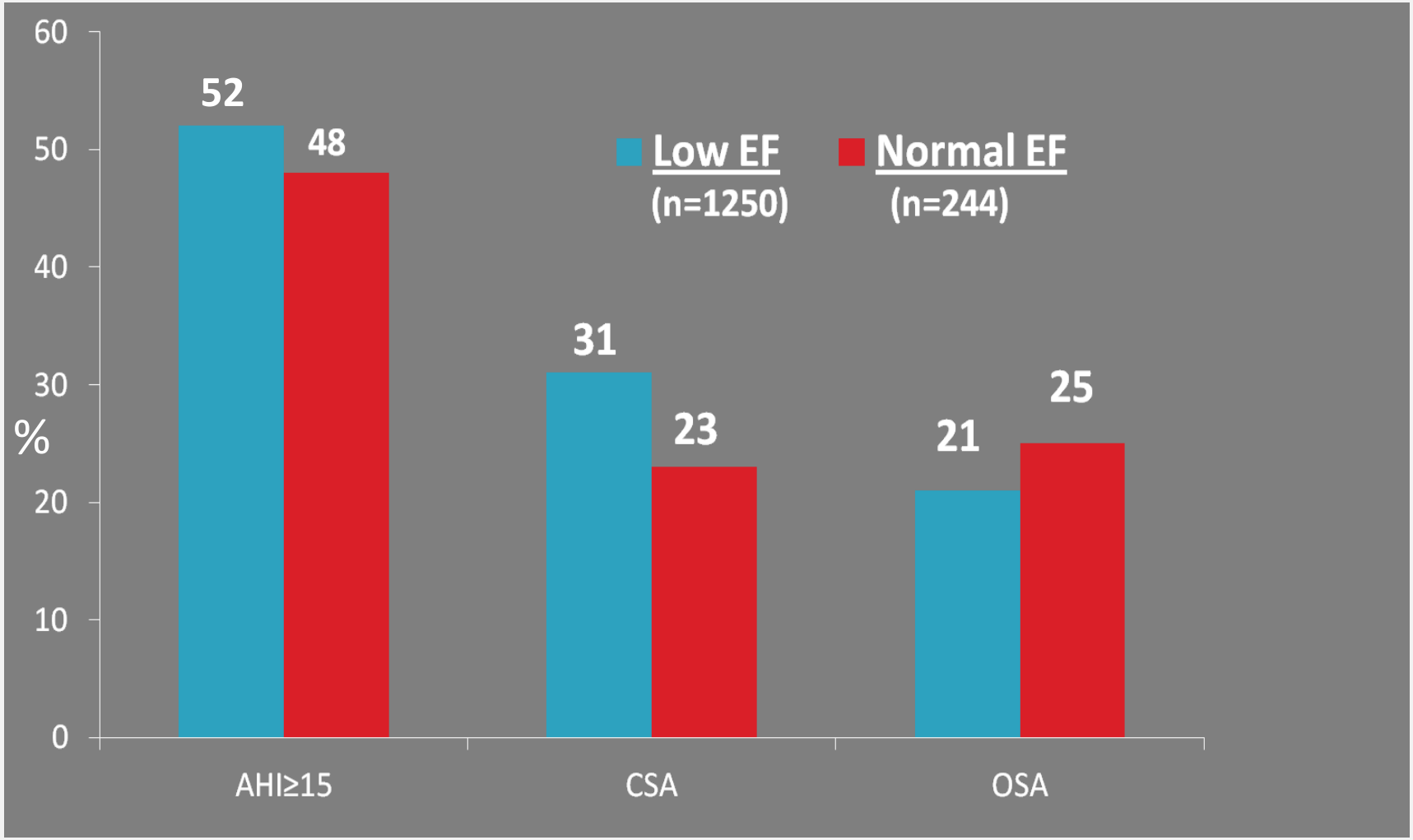
- Early studies of OSA patients treated with tracheostomy occasionally demonstrated transient development of CSA which subsequently resolved(Guilleminault et al. *Arch Intern Med* 1981; Coccagna et al. *Physiopath Resp* 1972)
- Emergence of CSA after surgical relief of nasal obstruction in OSA. Goldstein C; Kuzniar TJ. *J Clin Sleep Med* 2012;8:321-322.
- Complex sleep apnea unmasked by the use of a mandibular advancement device. Kuzniar et al. *Sleep Breath* 2011; 15:249-52.

Sleep Apnea in CHF

- **Approximately 40% of patients with CHF have CSA or CSR.**
- Among patients referred to a sleep clinic, using an AHI cutoff of 10,15, and 20 per hour of sleep:
  - Overall prevalences of SDB in CHF patients were 72%, 61%, and 53%.
  - CSA prevalences were 33%, 29%, and 25% respectively;
  - OSA were 38%, 32%, and 27%.
- Using an AHI cutoff of 10/hour of sleep
  - 148 patients with CSA
  - 168 patients with OSA
  - 134 patients with no SDB
- Risk factors for central and obstructive sleep apnea in 450 men and women with congestive heart failure. Sin DD, Fitzgerald F, Parker JD, Newton G, Floras JS, Bradley TD. AJRCCM 1999;160:1101–1106.


Patients with Heart Failure
Obstructive Sleep Apnea
Central Sleep Apnea
CPAP-induced CSA (aka Complex Sleep Apnea)

Worldwide prevalence of sleep apnea in HF consecutive patients



What do we know (or think we know) about sleep disordered breathing in HF

- Obstructive sleep apnea is common in patients with heart failure.
- Central sleep apnea in patients with HF is associated with a poor prognosis.
- Less severe HF tends to demonstrate more OSA versus more severe demonstrates more CSA.

What do we know (or think we know) about sleep disordered breathing in HF

- Obstructive sleep apnea is common in patients with heart failure.
  - Oxidative stress related to intermittent hypoxemia
  - Systemic inflammation
  - Metabolic dysregulation
  - Endothelial dysfunction
  - Sympathetic excitation
  - Increased risk of hypertension (potentially treatable)

Effects of Continuous Positive Airway Pressure on Cardiovascular Outcomes in Heart Failure Patients With and Without Cheyne-Stokes Respiration

Don D. Sin, MD, MPH; Alexander G. Logan, MD; Fabia S. Fitzgerald, RN;
Peter P. Liu, MD; T. Douglas Bradley, MD

Background—Continuous positive airway pressure (CPAP) improves cardiac function in patients with congestive heart failure (CHF) who also have Cheyne-Stokes respiration and central sleep apnea (CSR-CSA). However, the effects of CPAP in CHF patients without CSR-CSA have not been tested, and the long-term effects of this treatment on clinical cardiovascular outcomes are unknown.

Methods and Results—We conducted a randomized, controlled trial in which 66 patients with CHF (29 with and 37 without CSR-CSA) were randomized to either a group that received CPAP nightly or to a control group. Change in left ventricular ejection fraction (LVEF) from baseline to 3 months and the combined mortality-cardiac transplantation rate over the median 2.2-year follow-up period were compared between the CPAP-treated and control groups. For the entire group of patients, CPAP had no significant effect on LVEF, but it was associated with a 60% relative risk reduction (95% confidence interval, 2% to 64%) in mortality–cardiac transplantation rate in patients who complied with CPAP therapy. Stratified analysis of patients with and without CSR-CSA revealed that those with CSR-CSA experienced both a significant improvement in LVEF at 3 months and a relative risk reduction of 81% (95% confidence interval, 26% to 95%) in the mortality–cardiac transplantation rate of those who used CPAP. CPAP had no significant effect on either of these outcomes in patients without CSR-CSA.

Conclusions—CPAP improves cardiac function in CHF patients with CSR-CSA but not in those without it. Although not definitive, our findings also suggest that CPAP can reduce the combined mortality–cardiac transplantation rate in those CHF patients with CSR-CSA who comply with therapy. (***Circulation***. **2000;102:61-66.**)

Sin DD, et al., *Circulation*. 2000;102:61-66.

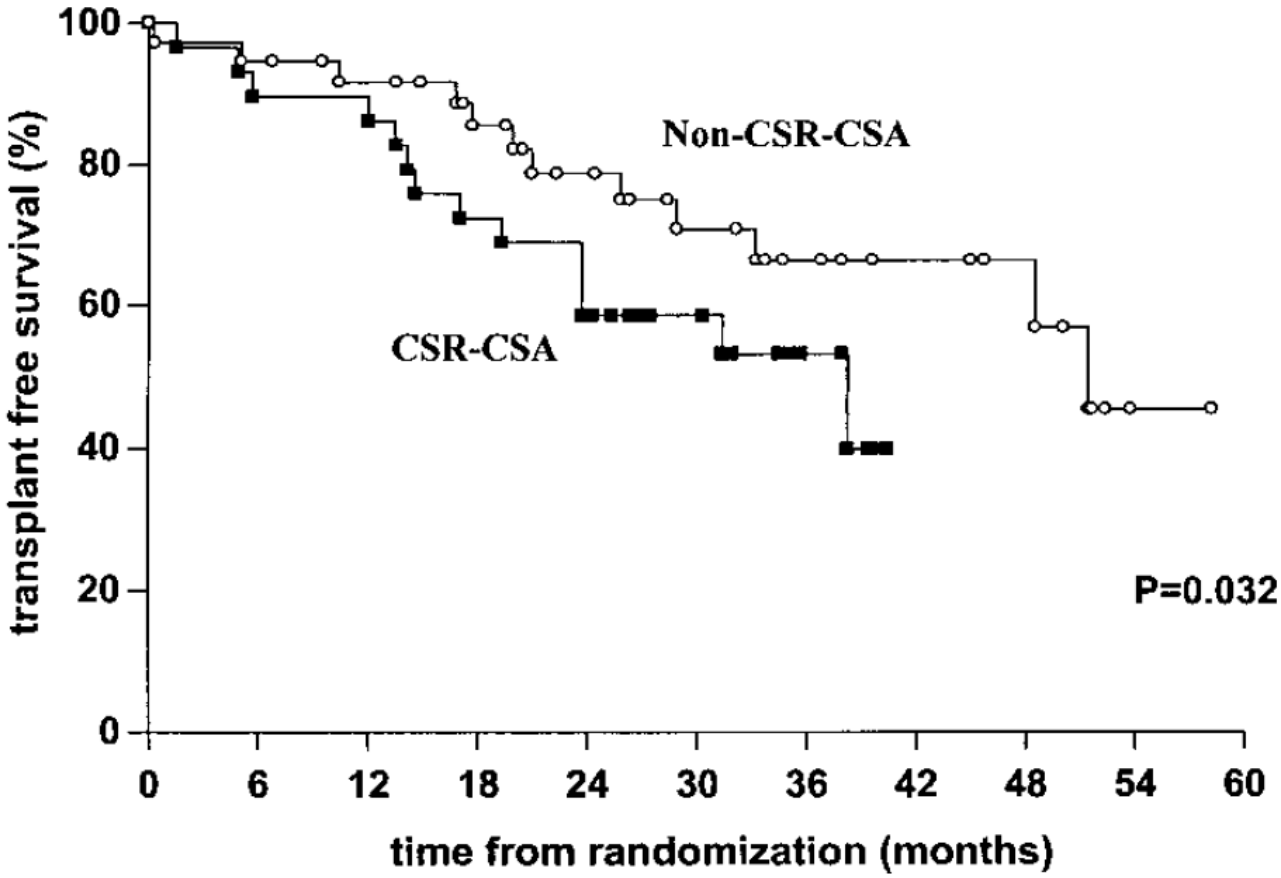


Figure 2. Transplant-free survival in CHF patients with CSR-CSA was significantly worse than in those without CSR-CSA, independent of the use of CPAP.

Sin DD, et al., *Circulation*. 2000;102:61-66.

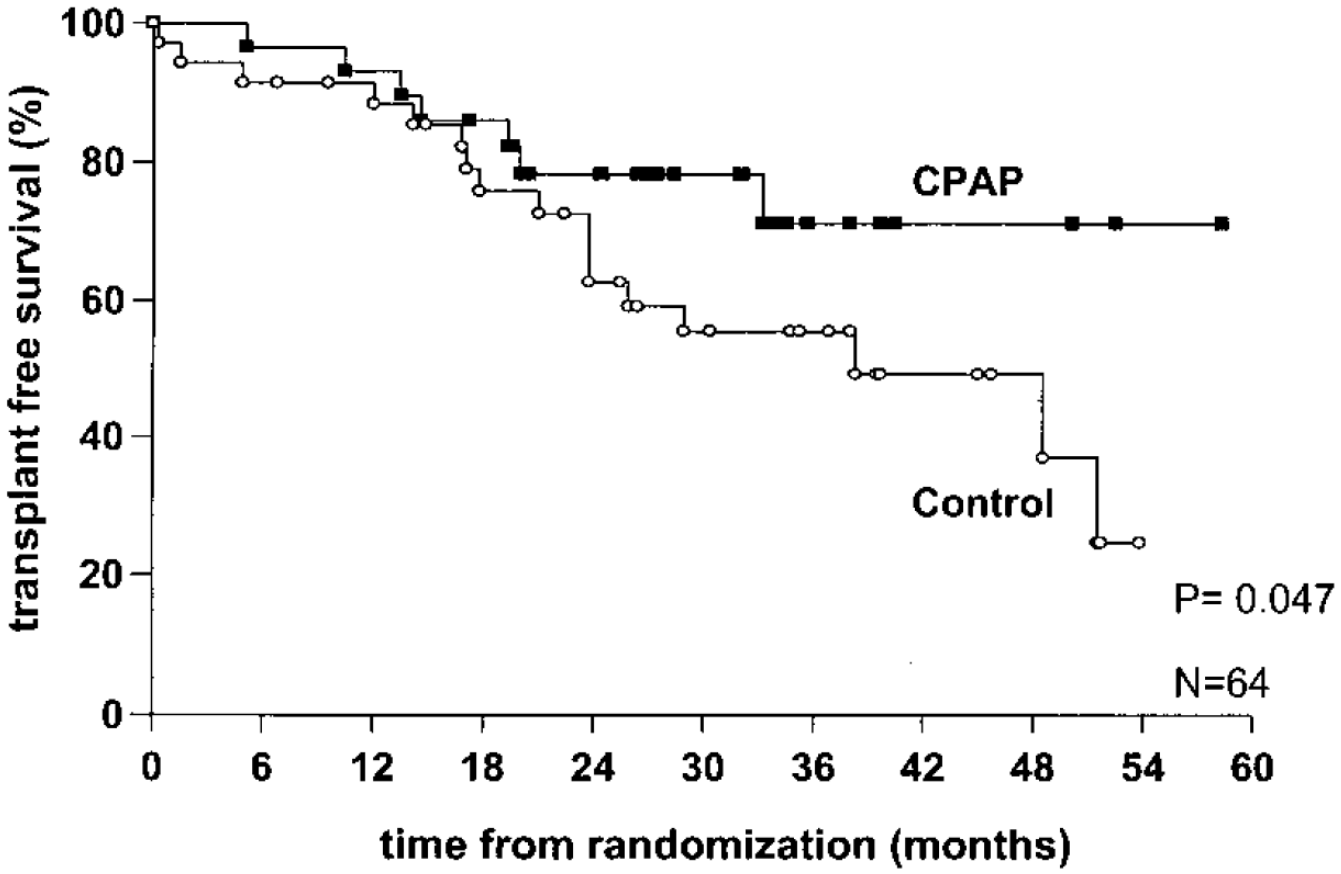


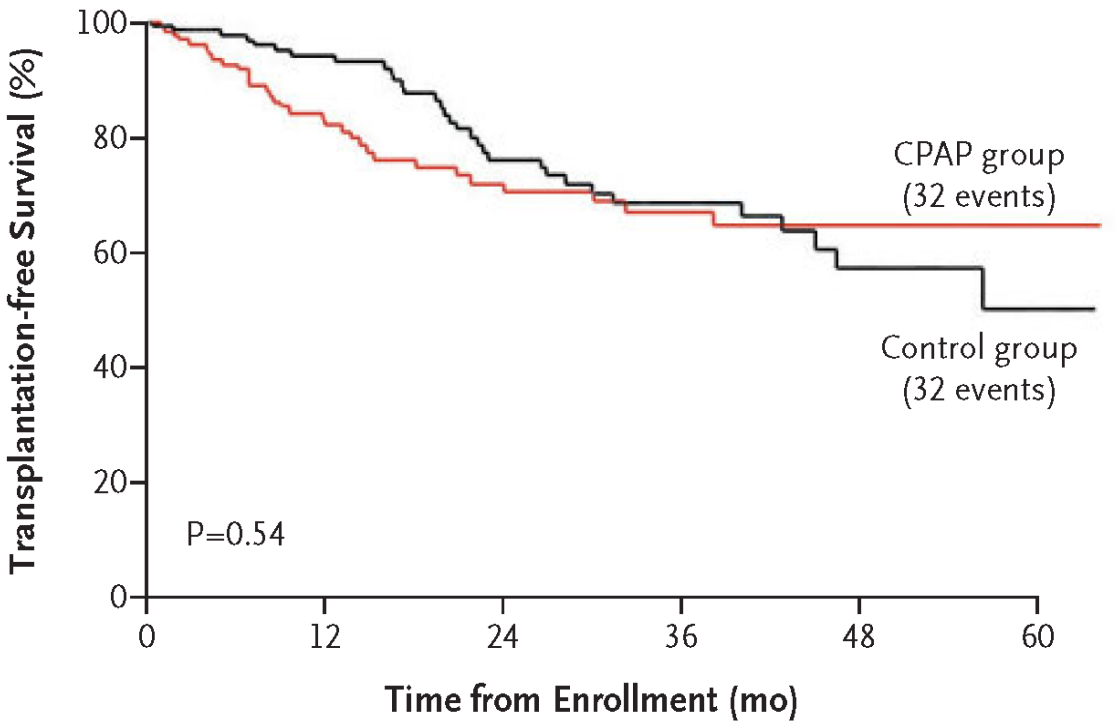
Figure 3. Treatment analysis revealed that overall transplant-free survival was significantly greater in patients randomized to CPAP who complied with therapy than in control subjects.

CPAP for Central Sleep Apnea and Heart Failure

NEJM 2005; 353:2025-33.

Bradley TD, Logan AG, Kimoff RJ, Sériès F, Morrison D, Ferguson K, Belenkie I, Pfeifer M, Fleetham J, Hanly P, Smilovitch M, Tomlinson G, Floras JS, for the CANPAP Investigators

Canadian Continuous Positive Airway Pressure for Patients with Central Sleep Apnea and Heart Failure (CANPAP) trial



No. at Risk

CPAP group	128	104	79	59	49	42	33	24	20	12	6
Control group	130	117	96	79	59	46	37	27	19	12	4

Figure 3. Heart-Transplantation–free Survival.

There was no difference in transplantation-free survival rates between the control group and the CPAP group (hazard ratio for transplantation-free survival, 1.16; P=0.54). However, there was an early divergence in the event rates that favored the control group (hazard ratio for transplantation-free survival, 1.5; P=0.02) that altered after 18 months to favor the CPAP group (hazard ratio for transplantation-free survival, 0.66; P=0.06).

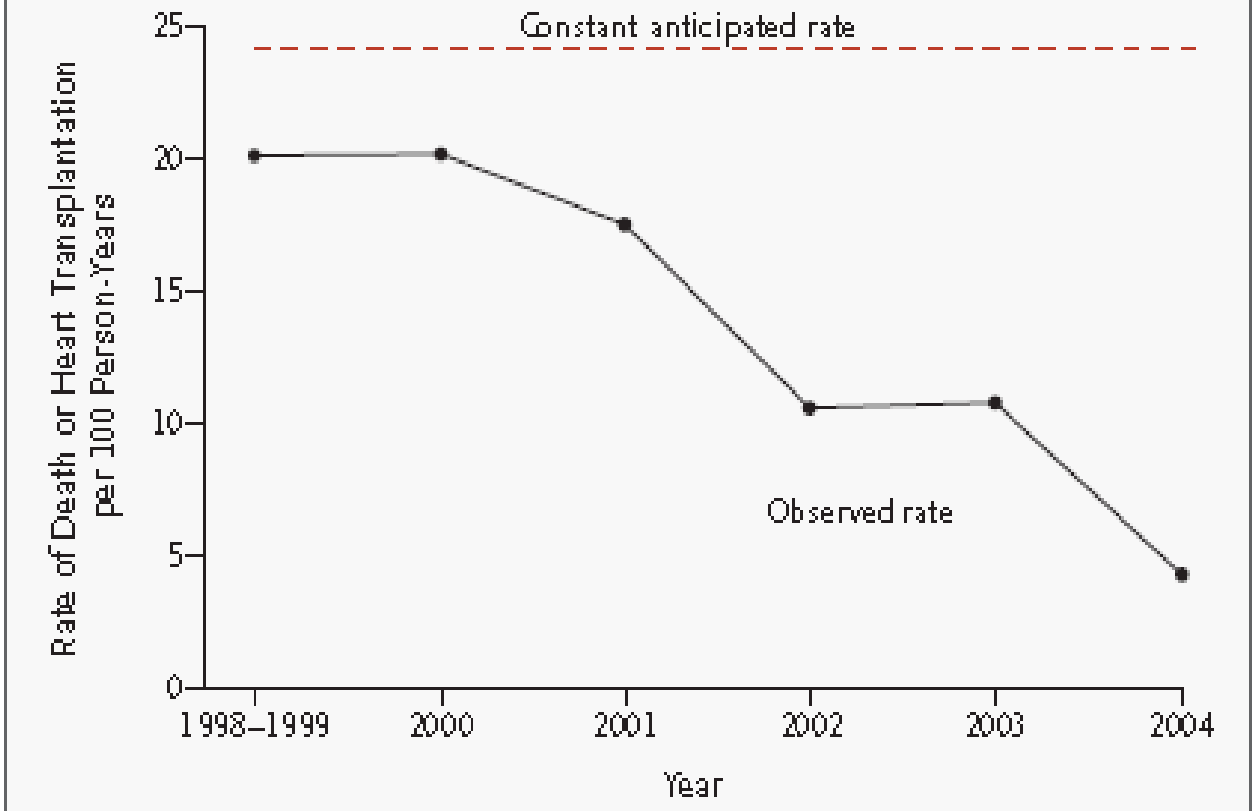


Figure 2. The Primary Event Rate (Death or Heart Transplantation per 100 Person-Years) for the Two Groups Combined.

The primary event rate fell from 83 percent to 17 percent of the predicted rate over the course of the trial.

Adaptive Pressure Support Servo-Ventilation

A Novel Treatment for Cheyne-Stokes Respiration in Heart Failure

HELMUT TESCHLER, JENS DÖHRING, YOU-MING WANG, and MICHAEL BERTHON-JONES

- Am J Respir Crit Care Med 2001; 164:614–619.
- N=14 subjects, stable cardiac failure receiving optimal medical treatment
- tested untreated and on four treatment nights in random order
  - nasal oxygen (2 L/min),
  - CPAP) (mean 9.25 cmH2O)
  - BilevelPAP (mean 13.5/5.2 cmH2O
  - ASV largely at default settings (mean pressure 7 to 9 cmH2O)

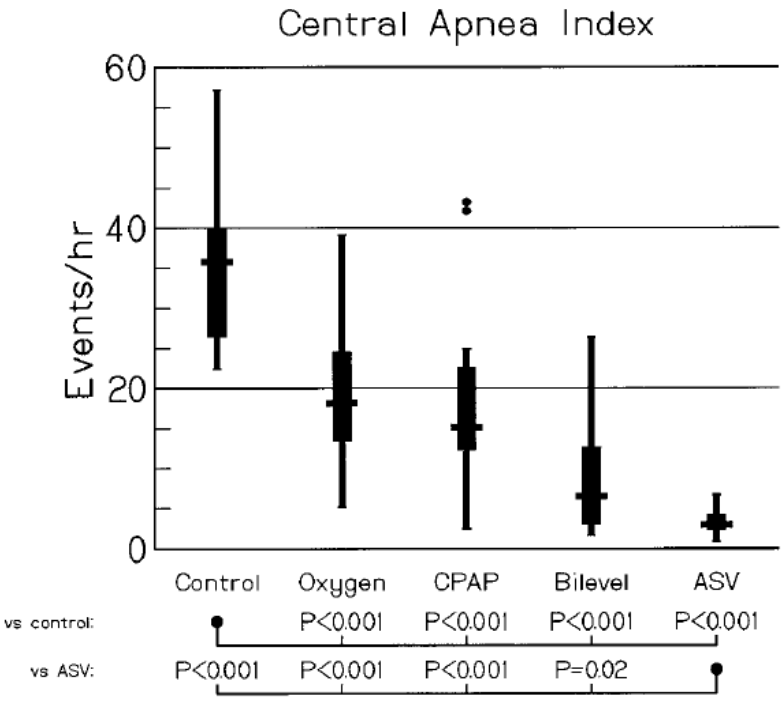
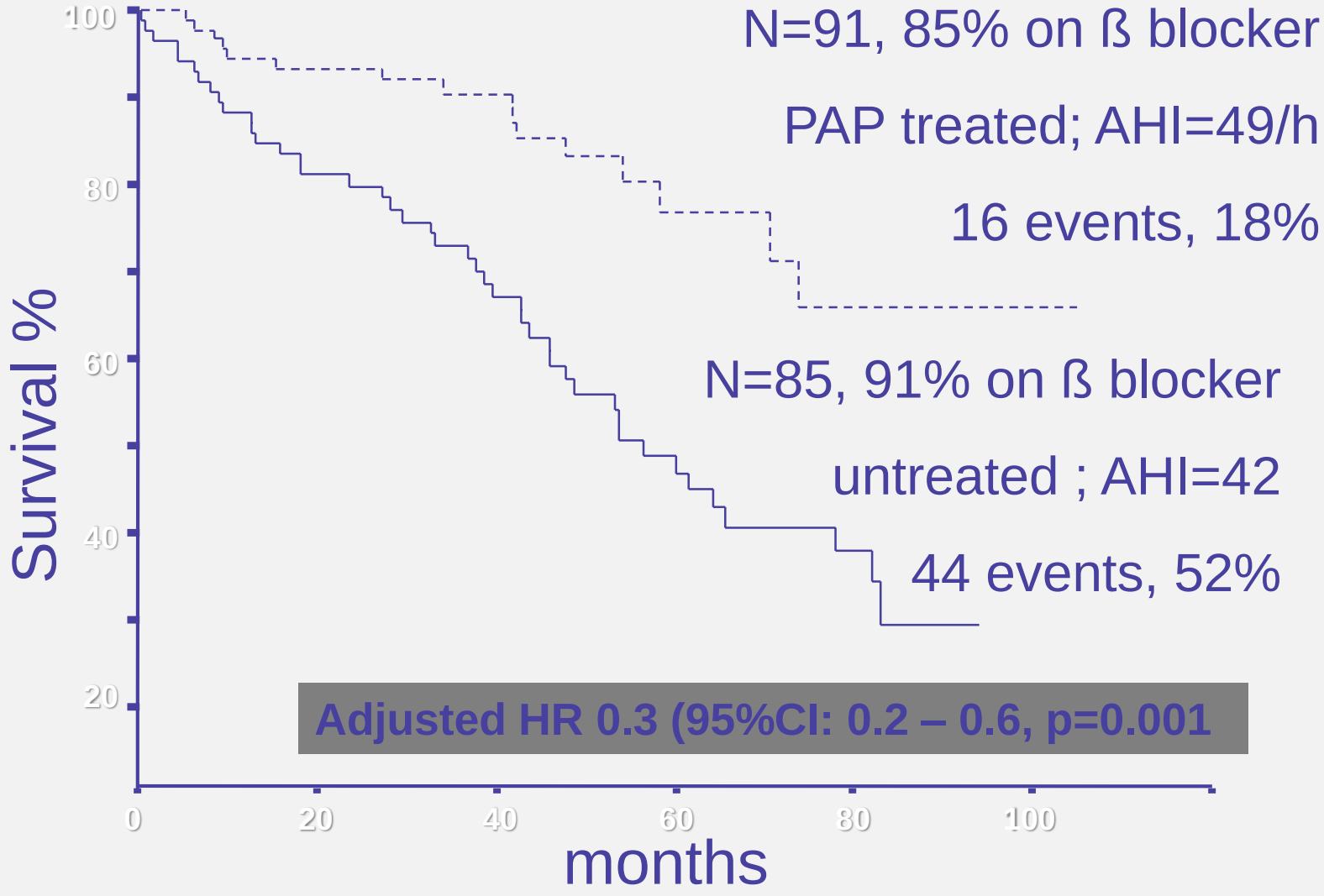


Figure 2. *Box plots* of effect of treatment on central apnea index. *Horizontal bar:* median; *thick vertical line:* interquartile range; *circles:* outliers; *thin bar:* range excluding outliers. Also shown are statistical significance of comparisons between control and each of the four treatments, and between ASV and the other four conditions.

Effects of PAP treatment* on survival in SHF and severe sleep apnea (*Jilek et al. EJHF, 2011)*

**CPAP. BPAP, and ASV*



Adaptive Servoventilation for Treatment of Sleep-Disordered Breathing in Heart Failure

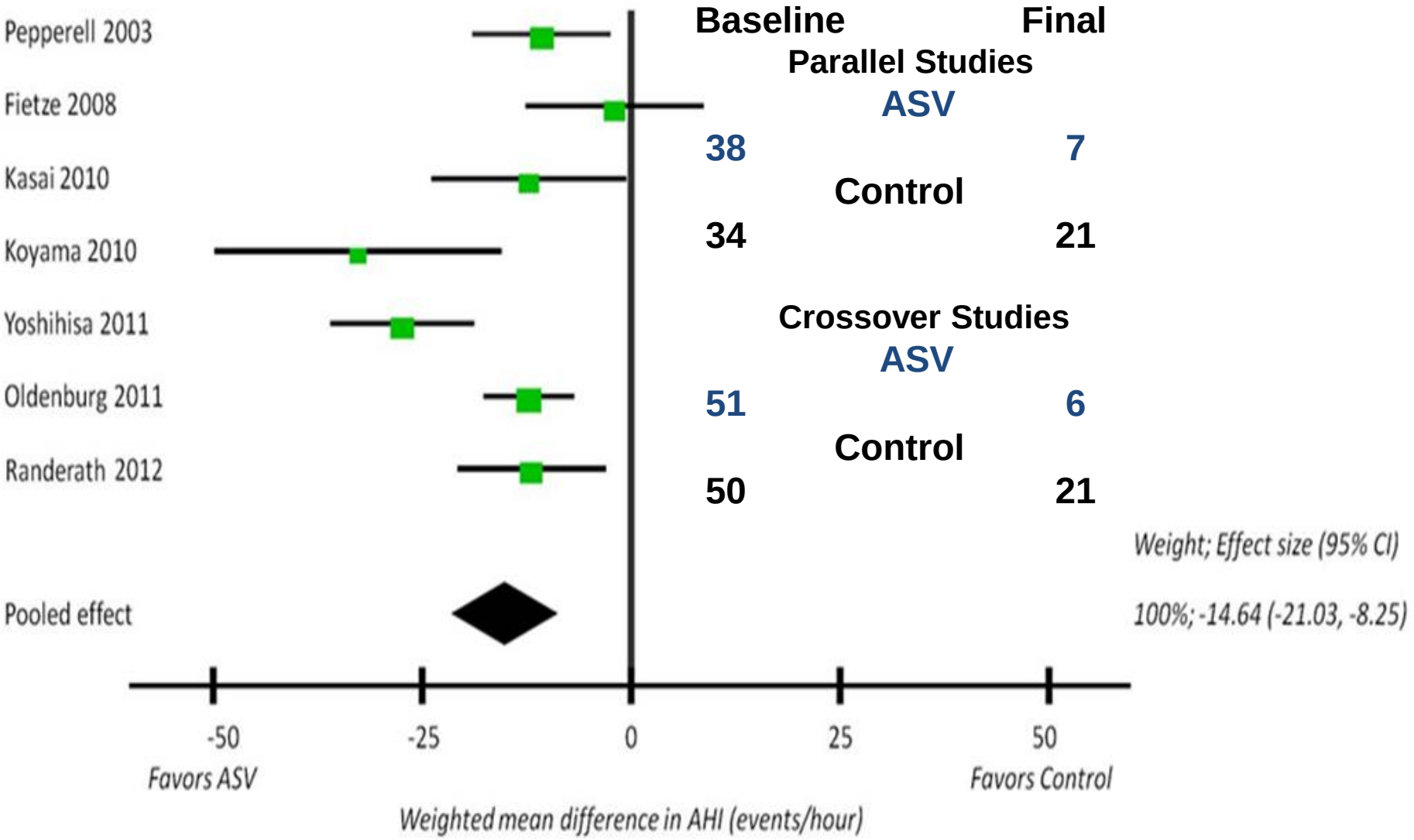
A Systematic Review and Meta-analysis

Bhavneesh K. Sharma, MD; Jessie P. Bakker, PhD; David G. McSharry, MB; Akshay S. Desai, MD, MPH; Shahrokh Javaheri, MD, FCCP; Atul Malhotra, MD, FCCP

Background: **Adaptive servoventilation (ASV) has demonstrated efficacy in treating sleep-disordered breathing (SDB) in patients with heart failure (HF), but large randomized trials are lacking. We, therefore, sought to perform a systematic review and meta-analysis of existing data.**
Methods: **A systematic search of the PubMed database was undertaken in March 2012. Publications were independently assessed by two investigators to identify studies of ≥ 1-week duration that compared ASV to a control condition (ie, subtherapeutic ASV, continuous or bilevel pressure ventilation, oxygen therapy, or no treatment) in adult patients with SDB and HF. Mean, variability, and sample size data were extracted independently for the following outcomes: apnea-hypopnea index (AHI), left ventricular ejection fraction (LVEF), quality of life (SF-36 Health Survey; Medical Outcomes Trust), 6-min walk distance, peak oxygen consumption ($\dot{V}O_2$) % predicted, and ventilatory equivalent ratio for CO_2 ($\dot{V}E/\dot{V}CO_2$) slope measured during exercise. Random effects meta-analysis models were applied.**
Results: **Fourteen studies were identified (N = 538). Comparing ASV to control conditions, the weighted mean difference in AHI (−14.64 events/h; 95% CI, −21.03 to −8.25) and LVEF (0.40; 95% CI, 0.08-0.71) both significantly favored ASV. ASV also improved the 6-min walk distance, but not peak $\dot{V}O_2$ % predicted, $\dot{V}E/\dot{V}CO_2$ slope, or quality of life, compared with control conditions.**
Conclusions: **In patients with HF and SDB, ASV was more effective than control conditions in reducing the AHI and improving cardiac function and exercise capacity. These data provide a compelling rationale for large-scale randomized controlled trials to assess the clinical impact of ASV on hard outcomes in these patients.**
CHEST 2012; 142(5):1211–1221

Abbreviations: AHI = apnea-hypopnea index; BPV = bilevel pressure ventilation; CAI = central apnea index; CSA = central sleep apnea; CSB = Cheyne-Stokes breathing; HF = heart failure; LVEF = left ventricular ejection fraction; OAI = obstructive apnea index; OSA = obstructive sleep apnea; SDB = sleep-disordered breathing; $\dot{V}E/\dot{V}CO_2$ = ventilatory equivalent ratio for CO_2; $\dot{V}O_2$ = oxygen consumption

Effect of ASV on AHI compared to the control in heart failure patients
Modified from Sharma et al, Chest, 2012



TREATMENT OF CENTRAL SLEEP APNEA SYNDROME IN ADULTS

Sleep 2012; 35:17-40

The Treatment of Central Sleep Apnea Syndromes in Adults: Practice Parameters with an Evidence-Based Literature Review and Meta-Analyses

R. Nisha Aurora, MD[1]; Susmita Chowdhuri, MD[2]; Kannan Ramar, MD[3]; Sabin R. Bista, MD[4]; Kenneth R. Casey, MD, MPH[5]; Carin I. Lamm, MD[6]; David A. Kristo, MD[7]; Jorge M. Mallea, MD[8]; James A. Rowley, MD[9]; Rochelle S. Zak, MD[10]; Sharon L. Tracy, PhD[11]

- CPAP therapy targeted to normalize the apnea-hypopnea index (AHI) is indicated for the initial treatment of CSAS related to CHF. (STANDARD)
- Nocturnal oxygen therapy is indicated for the treatment of CSAS related to CHF. (STANDARD)
- **Adaptive Servo-Ventilation (ASV) targeted to normalize the apnea-hypopnea index (AHI) is indicated for the treatment of CSAS related to CHF. (STANDARD)**
- BPAP therapy *in a spontaneous timed (ST)* mode targeted to normalize the apnea-hypopnea index (AHI) may be considered for the treatment of CSAS related to CHF only if there is no response to adequate trials of CPAP, ASV, and oxygen therapies. (OPTION)
- The following therapies have limited supporting evidence but may be considered for the treatment of CSAS related to CHF after optimization of standard medical therapy, if PAP therapy is not tolerated, and if accompanied by close clinical follow-up: acetazolamide and theophylline. (OPTION)

Study ID	Year	Exposed n[e]/M[e]/SD[e]	Control n[c]/M[c]/SD[c]	Weight (%)	Association measure with 95% CI
Kasai	2010	15/1.9/2.1	15/37.4/19.5	11.00%	-35.5 (-45.43 to -25.57)
Arzt	2008	14/4/3.7	14/46.4/15	12.00%	-42.4 (-50.49 to -34.31)
Fietze	2008	15/11.1/9.9	15/31/10	13.00%	-19.9 (-27.02 to -12.78)
Oldenburg	2008	29/3.8/4.1	29/37.4/9.4	15.00%	-33.6 (-37.33 to -29.87)
Morgenthaler	2007	6/0/0	6/46/22.7	6.00%	-46 (-64.16 to -27.84)
Philippe	2006	9/3/4	9/47/18	9.00%	-44 (-56.05 to -31.95)
Szollosi	2006	10/14/12	10/30/20.9	7.00%	-16 (-30.94 to -1.06)
Zhang	2006	14/6.5/0.8	14/34.5/6.1	15.00%	-28 (-31.22 to -24.78)
Pepperell	2003	15/5.4/7.4	15/24.7/11.3	13.00%	-19.3 (-26.14 to -12.46)
				100%	-30.82 (-36.36 to -25.28)

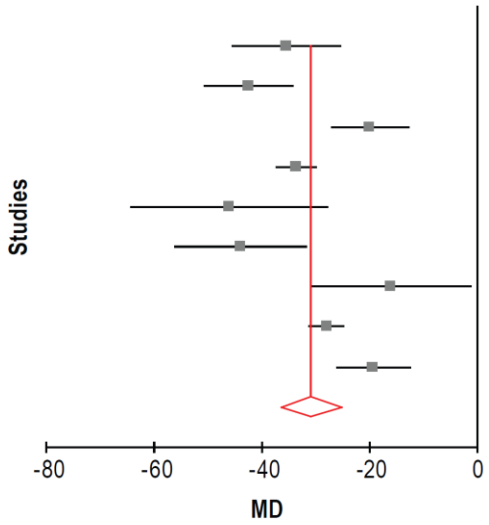


Figure 6—Meta-analysis of AHI from before-after ASV treatment trials

Table 7—Summary of quality and findings for ASV

Quality assessment							Summary of findings			
No of studies	Design	Limitations	Inconsistency	Indirectness	Imprecision	Other considerations	No of patients	Effect Absolute	Quality	Importance
LVEF (follow-up 0.5-6 months; measured with: %; Better indicated by higher values)										
6	4 randomized[51,54,60,61] and 2 non-randomized[52,57] trials	2 RCTs-no limitations; 2 RCTs – limitations; 2 NRTs – no other limitations	no serious inconsistency	no serious indirectness	no serious imprecision	Generally funded by manufacturers	95[1]	MD 6.1 higher (3.9 to 8.4 higher)	⊕⊕⊕○ MODERATE	IMPORTANT
AHI (follow-up 0.005 - 6 months; measured with: No./hr sleep; Better indicated by lower values)										
9	6 randomized[51,53-55,60,61] and 3 non-randomized[52,56,57] trials	2 RCTs – no limitations; 4 RCTs – limitations / 1 night of study / small n; 2 NRTs no other limitations; 1 NRT – only 1 night	no serious inconsistency	no serious indirectness	no serious imprecision	Generally funded by manufacturers	127[1]	MD 30.8 lower (36.4 to 25.3 lower)	⊕⊕⊕○ MODERATE	IMPORTANT

[1]Results vs. baseline, patients served as their own controls.

Values and Tradeoff for ASV:

- The overall quality of evidence for ASV is moderate. While there is no survival or long-term data available for ASV at this time, there is a sufficient amount of data consistently demonstrating improvement in both the AHI and LVEF.
- Additionally, there was a study suggesting overall better compliance with ASV compared with CPAP.
- It is worth noting that most of the available studies are industry sponsored, and different manufacturers
- utilize different algorithms to detect respiratory events and determine characteristics of pressure delivery.
- Therefore, generalizability is not possible or appropriate. There is also some uncertainty as to what are the optimum settings, reflecting an overall lack of experience with using these devices.
- It should be mentioned that the cost of these devices is several-fold greater than the cost of CPAP, and availability is not universal.
- Nonetheless, the data for ASV is consistent and is at least comparable if not better than the data supporting CPAP use.

Sleep 2012; 35:17-40.

Summary (as of 2012)

- Uncertainties related to identification of obstructive apnea versus central apnea versus hypopnea are important.
- Sleep apnea adversely effects heart disease *and* heart disease adversely effects sleep apnea.
- Patients with heart failure share many etiologic factors with sleep apnea patients. As a result, obstructive sleep apnea is common.
- Central sleep apnea is common, particularly in severe patients and is a marker for poor prognosis.
- Underlying cardiac disease is one of the risk factors for CPAP-induced central sleep apnea.
- While simple CPAP and even oxygen alone may be beneficial in some patients, there is a definite trend in the landscape of treatment toward more frequent use of ASV (adaptive servo ventilation).

SERVE-HF Trial – NEJM 2015; 373:1095-105.

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Adaptive Servo-Ventilation for Central Sleep Apnea in Systolic Heart Failure

Martin R. Cowie, M.D., Holger Woehrle, M.D., Karl Wegscheider, Ph.D., Christiane Angermann, M.D., Marie-Pia d'Ortho, M.D., Ph.D., Erland Erdmann, M.D., Patrick Levy, M.D., Ph.D., Anita K. Simonds, M.D., Virend K. Somers, M.D., Ph.D., Faiez Zannad, M.D., Ph.D., and Helmut Teschler, M.D.

SERVE-HF Trial

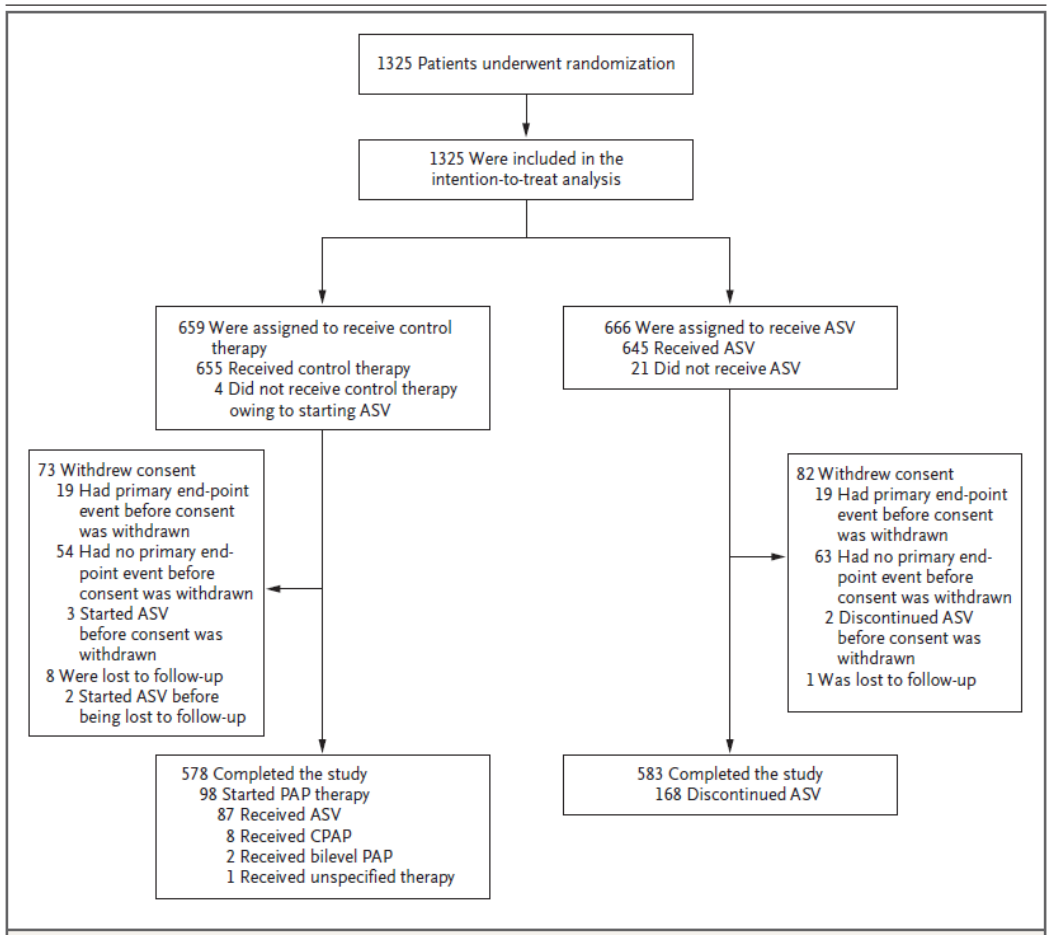


Figure 1. Randomization, Treatment, and Follow-up of the Patients.

Patients who withdrew consent did so for both study participation and follow-up (see the Supplementary Appendix). Of the 73 patients who withdrew consent in the control group, 3 had started adaptive servo-ventilation (ASV), and of the 82 who withdrew consent in the ASV group, 2 had discontinued ASV. CPAP denotes continuous positive airway pressure, and PAP positive airway pressure.

SERVE-HF Trial

Table 1. Demographic and Clinical Characteristics of the Patients at Baseline.*

Characteristic	Control (N=659)	Adaptive Servo-Ventilation (N=666)
Age — yr	69.3±10.4	69.6±9.5
Male sex — no. (%)	599 (90.9)	599 (89.9)
Body weight — kg	86.1±17.5	85.6±15.8
Body-mass index†	28.6±5.1	28.4±4.7
NYHA class — no./total no. (%)		
II	194/654 (29.7)	195/662 (29.5)
III	454/654 (69.4)	456/662 (68.9)
IV	6/654 (0.9)	11/662 (1.7)
Left ventricular ejection fraction — %‡		
Mean	32.5±8.0	32.2±7.9
Range	9.0–71.0	10.0–54.0
Diabetes mellitus — no./total no. (%)	252/653 (38.6)	254/660 (38.5)
Cause of heart failure — no./total no. (%)		
Ischemic	366/642 (57.0)	390/653 (59.7)
Nonischemic	276/642 (43.0)	263/653 (40.3)
Blood pressure — mm Hg		
Systolic	122.1±19.6	122.3±19.0
Diastolic	73.3±11.5	73.7±11.3

SERVE-HF Trial

Table 1. Demographic and Clinical Characteristics of the Patients at Baseline.*

Characteristic	Control (N=659)	Adaptive Servo-Ventilation (N=666)
Electrocardiographic finding — no./total no. (%)		
Left bundle-branch block§	65/295 (22.0)	79/304 (26.0)
Sinus rhythm	395/646 (61.1)	372/650 (57.2)
Atrial fibrillation	147/646 (22.8)	178/650 (27.4)
Other	104/646 (16.1)	100/650 (15.4)
Implanted device — no. (%)	364 (55.2)	362 (54.4)
No device	295 (44.8)	304 (45.6)
Non-CRT pacemaker	29 (4.4)	32 (4.8)
ICD	161 (24.4)	163 (24.5)
CRT-P	21 (3.2)	14 (2.1)
CRT-D	153 (23.2)	153 (23.0)
Hemoglobin — g/dl	13.9±1.5	13.8±1.6
Creatinine — mg/dl¶	1.4±0.6	1.4±0.6
Estimated GFR — ml/min/1.73 m^2	59.3±20.8	57.8±21.1
6-Min walk distance — m	337.9±127.5	334.0±126.4

SERVE-HF Trial

Table 1. (Continued.)

Characteristic	Control (N=659)	Adaptive Servo-Ventilation (N=666)
Concomitant cardiac medication — no./total no. (%)		
ACE inhibitor or ARB	603/659 (91.5)	613/666 (92.0)
Beta-blocker	611/659 (92.7)	612/666 (91.9)
Aldosterone antagonist	325/659 (49.3)	316/666 (47.4)
Diuretic	561/659 (85.1)	561/666 (84.2)
Cardiac glycoside	124/657 (18.9)	149/666 (22.4)
Antiarrhythmic drug	89/659 (13.5)	128/666 (19.2)

SERVE-HF Trial

Table 2. Respiratory Characteristics at Baseline.*

Characteristic	Control (N=659)	Adaptive Servo-Ventilation (N=666)
Epworth Sleepiness Scale score†	7.1±4.6	7.0±4.3
AHI — no. of events/hr	31.7±13.2	31.2±12.7
Central apnea index/total AHI — %	46.5±30.0	44.6±28.9
Central AHI/total AHI — %	81.8±15.7	80.8±15.5
Oxygen desaturation index — no. of events/hr‡	32.8±19.0	32.1±17.7
Oxygen saturation — %		
Mean	92.8±2.5	92.8±2.3
Minimum	80.3±7.5	80.7±7.0
Time with oxygen saturation <90% — min	55.7±73.9	50.5±68.2

SERVE-HF Trial

Table 1. (Continued.)

Characteristic	Control (N=659)	Adaptive Servo-Ventilation (N=666)
Concomitant cardiac medication — no./total no. (%)		
ACE inhibitor or ARB	603/659 (91.5)	613/666 (92.0)
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Cardiac glycoside	124/657 (18.9)	149/666 (22.4)
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SERVE-HF Trial

Table 1. (Continued.)

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Antiarrhythmic drug	89/659 (13.5)	128/666 (19.2)

SERVE-HF Trial

Table 3. Incidence of End-Point Events.*

Event	Control (N=659)		Adaptive Servo-Ventilation (N=666)		Hazard Ratio (95% CI)	P Value
	No. of Patients (%)	No. of Events/Yr (95% CI)	No. of Patients (%)	No. of Events/Yr (95% CI)		
Primary end point†	335 (50.8)	0.212 (0.190–0.236)	360 (54.1)	0.245 (0.220–0.272)	1.13 (0.97–1.31)	0.10
First secondary end point†	317 (48.1)	0.200 (0.179–0.224)	345 (51.8)	0.235 (0.211–0.261)	1.15 (0.98–1.34)	0.08
Second secondary end point†	465 (70.6)	0.405 (0.369–0.444)	482 (72.4)	0.441 (0.403–0.483)	1.07 (0.94–1.22)	0.28
Death from any cause	193 (29.3)	0.093 (0.081–0.107)	232 (34.8)	0.119 (0.104–0.135)	1.28 (1.06–1.55)	0.01
Cardiovascular death	158 (24.0)	0.076 (0.065–0.089)	199 (29.9)	0.102 (0.088–0.117)	1.34 (1.09–1.65)	0.006
Hospitalization for any cause	448 (68.0)	0.384 (0.349–0.421)	452 (67.9)	0.411 (0.374–0.451)	1.05 (0.92–1.20)	0.47
Unplanned hospitalization for worsening heart failure	272 (41.3)	0.164 (0.145–0.185)	287 (43.1)	0.190 (0.169–0.214)	1.13 (0.95–1.33)	0.16
Heart transplantation	12 (1.8)	0.006 (0.003–0.010)	8 (1.2)	0.004 (0.002–0.008)	0.70 (0.28–1.70)	0.43
Implantation of long-term VAD	10 (1.5)	0.005 (0.002–0.009)	16 (2.4)	0.008 (0.005–0.013)	1.67 (0.76–3.68)	0.20
Resuscitation	19 (2.9)	0.009 (0.006–0.014)	25 (3.8)	0.013 (0.008–0.019)	1.40 (0.77–2.54)	0.27
Resuscitation for cardiac arrest	16 (2.4)	0.008 (0.004–0.013)	18 (2.7)	0.009 (0.005–0.015)	1.19 (0.61–2.34)	0.61
Appropriate shock	65 (9.9)	0.033 (0.026–0.043)	45 (6.8)	0.024 (0.017–0.032)	0.71 (0.48–1.04)	0.08
Noncardiovascular death	35 (5.3)	0.017 (0.012–0.024)	33 (5.0)	0.017 (0.012–0.024)	1.00 (0.62–1.62)	0.99

SERVE-HF Trial

Table 3. Incidence of End-Point Events.*

Event	Control (N=659)		Adaptive Servo-Ventilation (N=666)		Hazard Ratio (95% CI)	P Value
	No. of Patients (%)	No. of Events/Yr (95% CI)	No. of Patients (%)	No. of Events/Yr (95% CI)		
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Second secondary end point†	465 (70.6)	0.405 (0.369–0.444)	482 (72.4)	0.441 (0.403–0.483)	1.07 (0.94–1.22)	0.28
Death from any cause	193 (29.3)	0.093 (0.081–0.107)	232 (34.8)	0.119 (0.104–0.135)	1.28 (1.06–1.55)	0.01
Cardiovascular death	158 (24.0)	0.076 (0.065–0.089)	199 (29.9)	0.102 (0.088–0.117)	1.34 (1.09–1.65)	0.006
Hospitalization for any cause	448 (68.0)	0.384 (0.349–0.421)	452 (67.9)	0.411 (0.374–0.451)	1.05 (0.92–1.20)	0.47
Unplanned hospitalization for worsening heart failure	272 (41.3)	0.164 (0.145–0.185)	287 (43.1)	0.190 (0.169–0.214)	1.13 (0.95–1.33)	0.16
Heart transplantation	12 (1.8)	0.006 (0.003–0.010)	8 (1.2)	0.004 (0.002–0.008)	0.70 (0.28–1.70)	0.43
Implantation of long-term VAD	10 (1.5)	0.005 (0.002–0.009)	16 (2.4)	0.008 (0.005–0.013)	1.67 (0.76–3.68)	0.20
Resuscitation	19 (2.9)	0.009 (0.006–0.014)	25 (3.8)	0.013 (0.008–0.019)	1.40 (0.77–2.54)	0.27
Resuscitation for cardiac arrest	16 (2.4)	0.008 (0.004–0.013)	18 (2.7)	0.009 (0.005–0.015)	1.19 (0.61–2.34)	0.61
Appropriate shock	65 (9.9)	0.033 (0.026–0.043)	45 (6.8)	0.024 (0.017–0.032)	0.71 (0.48–1.04)	0.08
Noncardiovascular death	35 (5.3)	0.017 (0.012–0.024)	33 (5.0)	0.017 (0.012–0.024)	1.00 (0.62–1.62)	0.99

SERVE-HF Trial

Death from any cause	193 (29.3)	0.093 (0.081–0.107)	232 (34.8)	0.119 (0.104–0.135)	1.28 (1.06–1.55)	0.01
Cardiovascular death	158 (24.0)	0.076 (0.065–0.089)	199 (29.9)	0.102 (0.088–0.117)	1.34 (1.09–1.65)	0.006

Updated Adaptive Servo-Ventilation Recommendations for the 2012 AASM Guideline: "The Treatment of Central Sleep Apnea Syndromes in Adults: Practice Parameters with an Evidence-Based Literature Review and Meta-Analyses"

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An update of the 2012 systematic review and meta-analyses were performed and a modified-GRADE approach was used to update the recommendation for the use of adaptive servo-ventilation (ASV) for the treatment of central sleep apnea syndrome (CSAS) related to congestive heart failure (CHF). Meta-analyses demonstrated an improvement in LVEF and a normalization of AHI in all patients. Analyses also demonstrated an increased risk of cardiac mortality in patients with an LVEF of ≤ 45% and moderate or severe CSA predominant sleep-disordered breathing. These data support a *Standard level recommendation against* the use of ASV to treat CHF-associated CSAS in patients with an LVEF of ≤ 45% and moderate or severe CSAS, and an *Option level recommendation* for the use of ASV in the treatment CHF-associated CSAS in patients with an LVEF > 45% or mild CHF-related CSAS. The application of these recommendations is limited to the target patient populations; the ultimate judgment regarding propriety of any specific care must be made by the clinician.

Keywords: central sleep apnea, adaptive servo-ventilation, clinical practice guideline

Citation: Aurora RN, Bista SR, Casey KR, Chowdhuri S, Kristo DA, Mallea JM, Ramar K, Rowley JA, Zak RS, Heald JL. Updated adaptive servo-ventilation recommendations for the 2012 AASM guideline: "The Treatment of Central Sleep Apnea Syndromes in Adults: Practice Parameters with an Evidence-Based Literature Review and Meta-Analyses". *J Clin Sleep Med* 2016;12(5):757–761.

Recommendation 1: Adaptive servo-ventilation (ASV) targeted to normalize the apnea-hypopnea index (AHI) should not be used for the treatment of CSAS related to CHF in adults with an ejection fraction ≤ 45% and moderate or severe CSA predominant, sleep-disordered breathing. (STANDARD AGAINST)

Recommendation 2: Adaptive servo-ventilation (ASV) targeted to normalize the apnea-hypopnea index (AHI) can be used for the treatment of CSAS related to CHF in adults with an ejection fraction > 45% or mild CHF-related CSAS. (OPTION)

Treatment of OSA in HF

- Optimisation of CV function
- Promotion of sleep hygiene
- Avoid ETOH, benzodiazepines, opioids
- Cessation of smoking
- Weight loss
- PAP devices: CPAP, bilevel
- Negative intraoral pressure device
- Positional therapy
- Mandibular advancement devices
- Modification of upper airway (in highly selected patients)
- Hypoglossal nerve stimulation
- Nocturnal use of supplemental oxygen

Treatment of CSA in HF

- Optimisation of CV function
- Promotion of sleep hygiene
- Avoid ETOH, benzodiazepines, opioids
- Cessation of smoking
- PAP devices: CPAP, bilevel, ASV
- Positional therapy
- Phrenic nerve stimulation
- Nocturnal use of supplemental oxygen
- Acetazolamide
- Theophylline

Thank you for your attention!

Questions,
Comments, Complaints?

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