Complex Sleep Apnea and New Positive Airway Pressure Modalities

Teofilo Lee-Chiong MD
Chief Medical Liaison
Philips Respironics

Professor of Medicine
National Jewish Health
University of Colorado

Disclosure

- Research funding: Philips Respironics
- Consulting: Elsevier, CareCore National
- Chief Medical Liaison: Philips Respironics
Learning Objectives

- Define complicated sleep disordered breathing
- Identify etiologic mechanisms
- Describe the natural history of complex sleep apnea
- Recognize the uses and limitations of ASV and AVAP

### Polysomnography

<table>
<thead>
<tr>
<th>Mode</th>
<th>Total AHI</th>
<th>OA</th>
<th>OH</th>
<th>CA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>141</td>
<td>26</td>
<td>188</td>
<td>77</td>
</tr>
<tr>
<td>CPAP 5</td>
<td>137</td>
<td>14</td>
<td>53</td>
<td>33</td>
</tr>
<tr>
<td>CPAP 7</td>
<td>124</td>
<td>10</td>
<td>25</td>
<td>11</td>
</tr>
<tr>
<td>CPAP 9</td>
<td>46</td>
<td>1</td>
<td>15</td>
<td>1</td>
</tr>
<tr>
<td>CPAP 11</td>
<td>63</td>
<td>4</td>
<td>17</td>
<td>23</td>
</tr>
<tr>
<td>CPAP 11 + 2L</td>
<td>97</td>
<td>3</td>
<td>5</td>
<td>26</td>
</tr>
<tr>
<td>BPAP 15/11</td>
<td>68</td>
<td>5</td>
<td>4</td>
<td>31</td>
</tr>
</tbody>
</table>
Management of CompSA

- Continue CPAP
  - Lower setting
  - Trial of higher setting
  - Add O2
- Stop CPAP, repeat PSG within a few days
- Switch to BPAP ± O2 ± back-up rate
- Switch to ASV

Patients

44, F, healthy, snoring, in Denver
68, M, heart failure, dyspnea
25, M, chronic pain, on narcotics
Patients

- 71, M, stroke, limited mobility
- 59, M, obese, snoring + apneas
- 69, F, snoring, insomnia, anxiety

Sleep Disordered Breathing

OSA

CSA / CSR  
Hypoventilation
Complicated Sleep Apnea

Syndrome Complexes

| OSA + CSA                  | Heart failure                                      |
|                           | Complex sleep apnea                                 |
| OSA + Hypoventilation     | COPD                                                |
| CSA + OSA + Hypoventilation | Opioids                                           |
|                           | Neuromuscular disorder                             |
|                           | Stroke                                             |
CSA: Classification

Hypercapnic

Non-hypercapnic
  • Cheyne Stokes

Hypercapnic CSA

<table>
<thead>
<tr>
<th>Clinical Features</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>High sleep PaCO2</td>
<td>Neuromuscular disorders</td>
</tr>
<tr>
<td>Often associated with high waking PaCO2</td>
<td>Chronic use of long-acting opioids</td>
</tr>
<tr>
<td>Decreased ventilatory responsiveness to hypercapnia</td>
<td></td>
</tr>
</tbody>
</table>
## Non-hypercapnic CSA

<table>
<thead>
<tr>
<th>Clinical Features</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal or low waking PaCO2</td>
<td>Idiopathic CSA</td>
</tr>
<tr>
<td>Increased ventilatory response to hypercapnia</td>
<td>Sleep-onset CSA</td>
</tr>
<tr>
<td>Brief arousals during sleep trigger a ventilatory “overshoot” that lowers PaCO2</td>
<td>CSA due to HF</td>
</tr>
<tr>
<td>below its apneic threshold</td>
<td>High altitude periodic breathing</td>
</tr>
<tr>
<td></td>
<td>Complex sleep apnea</td>
</tr>
</tbody>
</table>

## CSA vs. Cheyne Stokes

<table>
<thead>
<tr>
<th>Cycle time</th>
<th>CSA</th>
<th>CSR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Period of</td>
<td>Shorter (&lt; 45</td>
<td>Longer (&gt; 45</td>
</tr>
<tr>
<td>hyperpnea</td>
<td>seconds)</td>
<td>seconds)</td>
</tr>
<tr>
<td>Shorter</td>
<td></td>
<td>Longer</td>
</tr>
</tbody>
</table>

© 2012 American Association of Sleep Technologists
### CSA vs. Cheyne Stokes

<table>
<thead>
<tr>
<th></th>
<th>CSA</th>
<th>CSR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nadir of O2 desaturation</td>
<td>Following termination of apnea</td>
<td>More delayed</td>
</tr>
<tr>
<td>Timing of arousals</td>
<td>Termination of apnea</td>
<td>Peak of hyperpnea</td>
</tr>
</tbody>
</table>

**Cheyne-Stokes Breathing**

- **airflow**
- **SaO₂**

**Idiopathic CSA**

- **airflow**
- **SaO₂**

- A = position of arousal
- B = delay in saturation nadir

*Berry R, Sleep Medicine Pearls*
Heart Failure

OSA  CSA


- Question: Does overnight rostral fluid displacement and subsequent increase in neck circumference affect the severity of OSA and CSA in HF?
- Subjects: 57 subjects with HF (EF ≤ 45%)
- Methods: Prospective observational study
  - Subjects were divided into 2 groups:
    - Obstructive-dominant (≥ 50% of events are obstructive)
    - Central-dominant (> 50% of events are central)
  - Subjects with OSA received CPAP
Nocturnal rostral fluid shift: a unifying concept for the pathogenesis of
obstructive and central sleep apnea in men with heart failure.

• Methods:
  – Before and after PSG
    • Leg fluid volume (bio-electrical impedance)
    • Neck circumference
  – During PSG
    • TcCO2

Nocturnal rostral fluid shift: a unifying concept for the pathogenesis of
obstructive and central sleep apnea in men with heart failure.

• Outcomes:
  • Among subjects in the obstructive-dominant group,
    overnight change in leg fluid volume was inversely related to:
    – Overnight change in neck circumference
    – AHI
    – But not TcCO2

- Outcomes:
- Among subjects in the central-dominant group, overnight change in leg fluid volume was inversely related to:
  - Overnight change in neck circumference
  - AHI
  - But directly related to TcCO2

![Graph](image)

Figure 1. In both the obstructive (A) and central-dominant (B) groups, there were inverse exponential relationships between overnight changes in neck circumference and LFV.
Figure 2. In the obstructive-dominant group, there was no significant correlation between mean \( P_{\text{co2}} \), during sleep and overnight change in LFV (\( r = 0.245, P = 0.177 \)). In the central-dominant group, there was a significant correlation between mean sleep \( P_{\text{co2}} \) and the overnight change in LFV (\( r = 0.969, P = 0.009 \)).

Figure 3. Relationship between change in LFV and AHI in the obstructive- and central-dominant groups. The open circles and solid line represent the relationship between the AHI and the change in LFV in the obstructive-dominant group (\( y = 2.4 \cdot e^{0.011 \cdot x} \)). The closed circles and dashed line represent the relationship between the AHI and the change in LFV in the central-dominant group (\( y = 5.1 \cdot e^{-0.004 \cdot x} \)). The slopes of these curves differed significantly (\( P < 0.001 \)).
**Figure 3.** Relationship between change in LFV and AHI in the obstructive- and central-dominant groups. The open circles and solid line represent the relationship between the AHI and the change in LFV in the obstructive-dominant group \( (y=2.4 \cdot e^{0.511 \cdot x}) \). The closed circles and dashed line represent the relationship between the AHI and the change in LFV in the central-dominant group \( (y=5.1 \cdot e^{-0.504 \cdot x}) \). The slopes of these curves differed significantly \( (P<0.001) \).

**Figure 4.** Demonstration of a progressively greater reduction in LFV from patients with mild to no sleep apnea (M-NSA) \( (\text{AHI}<15; \; n=19) \) to OSA \( (\text{AHI} \geq 15; \; n=21) \) to CSA \( (\text{AHI} \geq 15; \; n=17) \).
Shift in sleep apnoea type in heart failure patients in the CANPAP trial.

- Question: Does improvement in heart function during CPAP therapy of CSA in persons with HF lead to conversion of respiratory events into obstructive apneas?
- Subjects: 98 subjects with HF and CSA
  - LVEF: < 40%
  - AHI: ≥ 15 (> 50% central apneas)

Shift in sleep apnoea type in heart failure patients in the CANPAP trial.

- Definitions:
  - Non-converter: > 50% of events remained central at follow-up
  - Converter: ≥ 50% of events were obstructive at follow-up
Shift in sleep apnoea type in heart failure patients in the CANPAP trial.  

- Methods: Sub-analysis of the control arm of CANPAP randomized controlled trial
- Outcomes:
  - Number of converters at follow-up: 18 subjects
    - 82% Non-converters
    - 18% Converters

**TABLE 3**  Changes in cardiovascular variables from baseline to follow-up

<table>
<thead>
<tr>
<th>Variable</th>
<th>Nonconversion group</th>
<th>Conversion group</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subjects n</td>
<td>90</td>
<td>18</td>
<td></td>
</tr>
<tr>
<td>ΔNYHA class</td>
<td>0.0 (-0.1-0.2)</td>
<td>-0.1 (-0.5-2.3)</td>
<td>0.38</td>
</tr>
<tr>
<td>ΔLVEF %</td>
<td>-0.7 (-1.9-0.6)</td>
<td>2.8 (0.4-6.0)</td>
<td>0.01</td>
</tr>
<tr>
<td>ΔCHFQ dyspnoea score</td>
<td>0.1 (0.5-1.2)</td>
<td>0.9 (0.2-1.6)</td>
<td>0.01</td>
</tr>
</tbody>
</table>

Data are presented as mean (95% CI), unless otherwise indicated. ΔNYHA: change in New York Heart Association; ΔLVEF: change in left ventricular ejection fraction; ΔCHFQ: change in Chronic Heart Failure Questionnaire score (>0.75 represents important change of moderate magnitude). p-values are for between group comparisons (ANOVA).
Complex Sleep Apnea

• Development or persistence of CSA or CSR with acute CPAP therapy in patients with predominantly OA or MA during the initial diagnostic study
• CPAP successfully eliminates OAH events but AHI remain elevated and sleep disruption persists due to CSA or CSR

Alternative Names

• CPAP-emergent CSA
• CPAP-persistent CSA
• Complicated sleep disordered breathing

• Many consider CompSA as a clinical subtype of CSA
Clinical Features

<table>
<thead>
<tr>
<th>Compared to OSA</th>
<th>Compared to CSA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Slightly lower BMI</td>
<td>Higher BMI</td>
</tr>
<tr>
<td></td>
<td>More frequent snoring</td>
</tr>
<tr>
<td></td>
<td>Less HF</td>
</tr>
<tr>
<td></td>
<td>Higher LVEF</td>
</tr>
</tbody>
</table>

Prevalence of Complex Sleep Apnea

<table>
<thead>
<tr>
<th>Author/ Site (year)</th>
<th>n</th>
<th>Complex Sleep Apnea</th>
<th>PSG</th>
<th>AHI , n/hr</th>
<th>Follow-up PSG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morgenthaler Rochester, USA (06)</td>
<td>223</td>
<td>15%</td>
<td>Split</td>
<td>32</td>
<td>-</td>
</tr>
<tr>
<td>Derniaka Oklahoma, USA (06)</td>
<td>116</td>
<td>20%</td>
<td>Split</td>
<td>51</td>
<td>2%</td>
</tr>
<tr>
<td>Lehman Adelaide, Australia (07)</td>
<td>99</td>
<td>13%</td>
<td>Mixed</td>
<td>72</td>
<td>-</td>
</tr>
<tr>
<td>Javaheri Cincinnati, USA (09)</td>
<td>1286</td>
<td>6.5%</td>
<td>Full Night</td>
<td>57</td>
<td>2%</td>
</tr>
<tr>
<td>Endo Japan (07)</td>
<td>1232</td>
<td>5.3%</td>
<td>Full Night</td>
<td>59</td>
<td>-</td>
</tr>
<tr>
<td>Yaegashi Japan (09)</td>
<td>297</td>
<td>5.7%</td>
<td>Full Night</td>
<td>56</td>
<td>-</td>
</tr>
</tbody>
</table>
The prevalence and natural history of complex sleep apnea.

• Question: What is the prevalence and natural history of CPAP-emergent CSA?
• Subjects: 1286 persons with newly diagnosed OSA
• Methods: Retrospective review
• Subjects underwent a full-night attended PSG and a full-night attended CPAP titration
• A second full-night attended CPAP titration was performed 5-6 weeks later for those who developed CPAP-emergent CSA

The prevalence and natural history of complex sleep apnea.

• Outcomes:
• Overall incidence of CPAP-emergent CSA (CAI ≥ 5) was 6.5%
• Of the 84 subjects, 42 had a second PSG, and CSA was eliminated in 33 subjects
  – Overall prevalence of CSA with long-term CPAP use was 1.5%
• Factors that were associated with persistent CPAP-emergent CSA were severe OSA, baseline CAI ≥ 5 and use of opioids
Pathophysiology

- Application of CPAP
- Reduction of PaCO2 below apneic threshold
- Development of CA

Pathophysiology

- Increase in PaCO2 above apneic threshold
- Cessation of CA / Resumption of breathing
- Hyperventilatory overshoot
Pathophysiology

Reduction of PaCO2 below apneic threshold

Development of CA

Changes in baroreflex sensitivity

Decreased work of breathing

Improved physiologic shunt
Pathophysiology

- Probability of CA developing is greater if
  - Narrow difference between baseline PaCO2 and apneic threshold (CO2 reserve)
  - Increased respiratory sensitivity to PaCO2 (controller gain)

Differential Diagnosis

- Concurrent CSA and OSA with elimination of obstructive events during CPAP titration
- Variable SRBD
  - Supine-position OA and non-supine CA
  - NREM periodic breathing and REM OA
- Acute development of anxiety with post-hyperventilatory hypocapnic CA
Management of CompSA

• Determine underlying pathophysiology
• Maximize medical therapy for comorbid disorders

Management of CompSA

• Continue CPAP
  – Lower setting
  – Trial of higher setting
  – Add O2
• Stop CPAP, repeat PSG within a few days
• Switch to BPAP ± O2 ± back-up rate
• Switch to ASV
Servo ventilation

- Assures constant ventilation based on measurements of airflow, either:
  - Peak flow
  - Minute ventilation
- Provides varying amounts of respiratory support (above expiratory pressure) during different phases of periodic breathing

Servo ventilation

- Automatically adjusts settings in response to specific respiratory events
  - Increases EPAP for obstructive events
  - Increases inspiratory PS for hypopneas
  - Decreases inspiratory PS for hyperpneas/hyperventilation
  - Back up rate for impending apneas
Servo ventilation

- 2 types of devices:
  - BiPAP AutoSV Advanced
  - VPAP Adapt ASV Enhanced
BiPAP AutoSV Advanced

- Target: 4-minute moving average of breath-by-breath peak flow
- Settings:
  - Automatic EPAP: 4-25 cmH$_2$O
  - EPAP increases by 1 cmH$_2$O q 15 seconds for obstructive apneas/hypopneas or snoring; proactive search q 5 min
  - Automatic PS: 0 to (30 minus EPAP)
  - Automatic back-up rate: 4-30

BiPAP AutoSV Advanced

- Initial settings
  - EPAPmin 4 cmH$_2$O
  - EPAPmax 15
  - Psmin 0
  - Psmax 20
  - Max pressure 25
  - Rate Auto
  - Biflex +/-
BiPAP AutoSV Advanced

• If patient is unable to fall asleep
  – Adjust Biflex
  – If UA obstruction present – increase EPAPmin by 1-2 cmH2O
  – If UA obstruction absent – increase PSmin by 1-2 cmH2O

BiPAP AutoSV Advanced

• During the night
  – If obstructive events persist – increase EPAPmin
  – If central events persist – increase PSmax or set rate to minimum of 8-10 bpm
VPAP Adapt ASV Enhanced

- Target: 90% of 3-minute moving average of MV
- Settings:
  - EPAP: 4-15 cmH₂O
  - PS[min]: 3-6 cmH₂O; PS[max]: 8-16 cmH₂O
  - By manually setting EPAP, the device automatically adjusts PS[min] (3 cmH20) first then PS[max] (8 cmH20)

VPAP Adapt ASV Enhanced

- Settings:
  - PS[max] – PS[min] ≥ 5 cmH₂O
  - EPAP + PS[max] ≤ 25 cmH₂O
  - Default back-up rate: 15/min
Servo ventilation

<table>
<thead>
<tr>
<th></th>
<th>BiPAP</th>
<th>VPAP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Target</td>
<td>Peak flow</td>
<td>Minute ventilation</td>
</tr>
<tr>
<td>EPAP</td>
<td>Automatic</td>
<td>Manual</td>
</tr>
<tr>
<td>EPAP</td>
<td>4-25 cmH20</td>
<td>4-15 cmH20</td>
</tr>
<tr>
<td>PS [min]</td>
<td>0 cmH20</td>
<td>3 cmH20</td>
</tr>
<tr>
<td>Rate [default]</td>
<td>15</td>
<td>15</td>
</tr>
</tbody>
</table>

Adaptive servoventilation versus noninvasive positive pressure ventilation for central, mixed, and complex sleep apnea syndromes.  
*Morgenthaler Ti et al. Sleep 2007*

- Comparison of efficacy of NPPV and ASV
- Prospective RCT
- Two academic sleep programs
- 21 patients
  - CSA/CSR: 6
  - Predominantly MA: 6
  - CompSAS: 9
Adaptive servoventilation versus noninvasive positive pressure ventilation for central, mixed, and complex sleep apnea syndromes.
Morgenthaler TI et al. Sleep 2007

Inclusion criteria
• Age ≥ 18 years
• Attended CPAP titration study within 12 months

Changes in AHI
• Initial diagnostic AHI: 51.9 ± 22.8/hr
• Reduction in mean AHI
  – CPAP:  34.3 ± 25.7
  – NPPV:  6.2 ± 7.6
  – ASV:    0.8 ± 2.4 (P <0.01)
Adaptive servoventilation versus noninvasive positive pressure ventilation or central, mixed, and complex sleep apnea syndromes.
Morgenthaler TI et al. Sleep 2007

<table>
<thead>
<tr>
<th></th>
<th>NPPV</th>
<th>ASV</th>
</tr>
</thead>
<tbody>
<tr>
<td>CSA/CSR</td>
<td>1.5 ± 1.5</td>
<td>0 ± 0</td>
</tr>
<tr>
<td>MA</td>
<td>10.2 ± 10.6</td>
<td>0.5 ± 0.8</td>
</tr>
<tr>
<td>CompSAS</td>
<td>6.8 ± 6.8</td>
<td>1.6 ± 3.6</td>
</tr>
</tbody>
</table>

Efficacy of adaptive servoventilation in treatment of complex and central sleep apnea syndromes.
Allam JS et al. Chest 2007

- Retrospective chart review
- First 100 patients with ASV titration
- Indications of ASV titration
  - CompSAS: 63%
  - CSA: 22%
  - CSA/CSR: 15%
- All had suboptimal response to CPAP
- Median diagnostic AHI: 48 (range 24-62)
Efficacy of adaptive servoventilation in treatment of complex and central sleep apnea syndromes.

Allam JS et al. Chest 2007

Diagnostic polysomnography

- Median AHI: 48
- Median OAI: 13
- Median CAI: 4

Efficacy of adaptive servoventilation in treatment of complex and central sleep apnea syndromes.

Allam JS et al. Chest 2007

Change in respiratory events

<table>
<thead>
<tr>
<th></th>
<th>CPAP</th>
<th>BPAP-S</th>
<th>BPAP-S/T</th>
<th>ASV</th>
</tr>
</thead>
<tbody>
<tr>
<td>AHI:</td>
<td>31</td>
<td>75</td>
<td>15</td>
<td>5</td>
</tr>
<tr>
<td>OAI:</td>
<td>1</td>
<td>5</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>CAI:</td>
<td>16</td>
<td>40</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>
If ASV Fails

- Reevaluate for underlying comorbidities
  - Maximize therapy
  - Reduce opioids
- Switch to “other” ASV device
- Increase PEEP in ASV
If ASV Fails

- Add oxygen
- Trial of hypnotic agents
- Trial of acetazolamide
- CO2 monitors
- Trial of NIPPV (BiPAP with AVAPS)

BiPAP with AVAPS

- Bi-level with Average Volume Assured Pressure Support (AVAPS)
  - Maintains a stable tidal volume when the patient is placed on either the S, ST or T mode
  - By automatically adjusting PS between IPAPmin and IPAPmax settings
  - Avoids breath by breath changes in IPAP levels (1 mbar/min)
BiPAP with AVAPS

- Indications
  - OHS
  - COPD
  - Neuromuscular weakness
  - CCHS (one case report)
  - Others requiring nocturnal ventilation?
BiPAP with AVAPS

• NOT recommended for patients with periodic breathing

BiPAP with AVAPS

• Initial settings
  – EPAP 4 cmH2O
  – IPAPmin 8
  – IPAPmax 25
  – Rate 8-10 bpm
  – I-time 1.5 sec
  – Rise time 2-3
  – Tidal volume* 8 mL/kg ideal body weight
*adjust to patient comfort to allow sleep onset
BiPAP with AVAPS

During the night
- If obstructive events persist – increase EPAP and IPAPmin (maintain PS)
- If hypoventilation persists – increase PS or rate
- If O2 desaturation is present –
  • Increase EPAP
  • Increase PS or rate
  • Add O2

3 ways to choose a starting tidal volume with AVAPS:

1. MD suggestion
2. Patient comfort
3. Ideal body weight: 8 ml/kg

AVAPS suggested tidal volume settings based on height and ideal weight:

<table>
<thead>
<tr>
<th>Height</th>
<th>59&quot;</th>
<th>61&quot;</th>
<th>63&quot;</th>
<th>65&quot;</th>
<th>67&quot;</th>
<th>69&quot;</th>
<th>71&quot;</th>
<th>73&quot;</th>
<th>75&quot;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight</td>
<td>52.0 kg</td>
<td>55.5 kg</td>
<td>59.0 kg</td>
<td>62.5 kg</td>
<td>66.5 kg</td>
<td>70.5 kg</td>
<td>74.5 kg</td>
<td>78.5 kg</td>
<td>83.0 kg</td>
</tr>
<tr>
<td>Vt (ml)</td>
<td>420 ml</td>
<td>440 ml</td>
<td>470 ml</td>
<td>500 ml</td>
<td>530 ml</td>
<td>560 ml</td>
<td>600 ml</td>
<td>630 ml</td>
<td>660 ml</td>
</tr>
</tbody>
</table>
BiPAP with AVAPS

Table 1 Clinical studies on target volume during pressure-ventilation

<table>
<thead>
<tr>
<th>Study</th>
<th>Year</th>
<th>Design</th>
<th>Cohort</th>
<th>Target volume setting</th>
<th>Main target volume outcomes*</th>
</tr>
</thead>
<tbody>
<tr>
<td>S torn et al.</td>
<td>2000</td>
<td>6-week cross-over RCT (n = 10)</td>
<td>OHS:2</td>
<td>Tidal, ETV (n = 10), T2 tidal (n = 10)</td>
<td>Increase in nocturnal PaO2, + Comparative effect on quality of life</td>
</tr>
<tr>
<td>Janas et al.</td>
<td>2000</td>
<td>1-day cross-over RCT (n = 10)</td>
<td>OHS:5</td>
<td>Tidal, ETV (n = 10), ETV (n = 2)</td>
<td>Decrease in nocturnal PaO2, + Comparative effect on polypesymmetry</td>
</tr>
<tr>
<td>Ambos et al.</td>
<td>2000</td>
<td>1-day cross-over RCT (n = 10)</td>
<td>OHS:6</td>
<td>Tidal, ETV (n = 10), ETV (n = 2)</td>
<td>Decrease in nocturnal PaO2, + Comparative effect on polyesymmetry</td>
</tr>
<tr>
<td>Osvald et al.</td>
<td>2000</td>
<td>1-day cross-over RCT (n = 10)</td>
<td>OHS:7</td>
<td>Tidal, ETV (n = 10), ETV (n = 2)</td>
<td>Decrease in nocturnal PaO2, + Comparative effect on polyesymmetry</td>
</tr>
<tr>
<td>Gisci et al.</td>
<td>2000</td>
<td>0-week cross-over RCT (n = 10)</td>
<td>OHS:8</td>
<td>Tidal, ETV (n = 10), ETV (n = 2)</td>
<td>Decrease in nocturnal PaO2, + Comparative effect on polyesymmetry</td>
</tr>
<tr>
<td>Murphy et al.</td>
<td>2000</td>
<td>1-week cross-over RCT (n = 10)</td>
<td>OHS:9</td>
<td>Tidal, ETV (n = 10), ETV (n = 2)</td>
<td>Decrease in nocturnal PaO2, + Comparative effect on polyesymmetry</td>
</tr>
</tbody>
</table>

*Comparative effects on:
- Daytime blood gases
- Longitudinal and exercise capacity
- Quality of life
- Nocturnal PaO2
- Nocturnal PaCO2
- Sleep efficiency
- Nocturnal O2 saturation
- Daytime O2 saturation
- Nocturnal PaCO2

Windisch and Storre. Thorax 2012

BiPAP with AVAPS

10 patients with OHS, failed CPAP

\[ P = 0.015 \]
\[ P = 0.004 \]
\[ P = 0.45 \]

Storre JH et al. CHEST 2006

© 2012 American Association of Sleep Technologists
Father and Son:  
The ASV Song  

It’s not time to make a change  
Just relax, take it easy  
It’s still early, it’s not your fault  
There’s so much you have to know  
Find a chair, settle down  
If you want, you can sip your coffee  
Look it’s ASV, it’s new and it’s working

I was once like you are now, and I know that  
it’s not easy  
To be calm when you’ve found apneas’  
going on  
But take you time, think a lot  
Why, think of everything the patient’s got  
For he’ll be here tomorrow, but his apneas  
might not
How can I try to relax, when I do, apneas reappear again
It’s always been the same, same old story
From the moment the study started, they were supposed to go away
Now, there’s a way, and I know that I have to let them stay
I know, I have to let apneas stay

All the times that I have tried, taking all the things I’ve learned aside
It’s hard, but it’s harder to ignore them
If ASV’s right, I’d agree, but it’s them they work, not me
Now, there’s a way and I know that I have to let them stay
I know, I have to let apneas stay.