Short Term Effect of Adaptive Servo-Ventilation Compared with Continuous Positive Airway Pressure on Muscle Sympathetic Nerve Activity in Patients with Heart Failure

This study was supported in part by a research grant from the Fukuda Foundation for Medical Technology in Tokyo.
Short Term Effect of Adaptive Servo-Ventilation Compared with Continuous Positive Airway Pressure on Muscle Sympathetic Nerve Activity in Patients with Heart Failure

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ESC CONGRESS 2012   29/Aug./2012
Mechanisms of adverse effects of sleep apnoea

1) repetitive episodes of hypoxia
2) arousal from sleep
3) lack of lung extension

- activating sympathetic nervous system
- predisposition to arrhythmias and poor outcome
Central sleep apnoea is a predictor of poor outcome in patients with heart failure.


Effect of CPAP is inconsistent in patients with central sleep apnoea

CPAP non-responder: AHI ≥ 15/h during CPAP

We need other type of positive airway pressure with greater suppression of central sleep apnoea.

Adaptive servo-ventilator (ASV) (2007- in Japan)

End expiratory pressure (EEP)
Minimal pressure support (Min PS)
Maximal pressure support support (Max PS)

Pressure of ASV (cmH₂O)
Respiration

airway pressure

Max PS
Min PS
EEP

Max PS
Min PS

set pressure

0
0
ASV can abolish central sleep apnoea

Is this beneficial effect of ASV related to decrease in sympathetic nerve activity?

It remains unknown whether the sympatho-inhibitory effect of ASV is via positive end-expiratory pressure or servo-ventilation.

Aim

To compare the short term effect between ASV and CPAP on respiratory pattern and MSNA in patients with heart failure.
Methods

Subject: patients with chronic heart failure (n=38)
(NYHA I-III, LVEF <45%, OAI <5/h)

- MSNA
- heart rate, blood pressure (Jentow), \( \text{SpO}_2 \) (Sentec Digital Monitor System)
- respiratory pattern (bioimpedance method)
- cardiorespiratory polygraphy (Somté®, Compumedics)
- echocardiography
- specific activity scale, neurohumoral factors, medications etc.
Protocol

Data sampling

Before

-10

Start recording

Start device

During

ASV (n=20)

6.6cmH₂O

Stop device

Stop recording

CPAP (n=18)

6.3cmH₂O

actual mean pressure

Time (minutes)
Measurement of MSNA

Amplifier

Band-pass filter (500-5000 Hz)

AD converter (PowerLab)

Computer

MSNA
burst rate (bursts/min)
burst incidence (bursts/100 beats)

Tungsten microelectrode
Peroneal nerve
Assessment of respiratory instability

CV-RR: coefficient of variation of respiratory rate
CV-TV: coefficient of variation of tidal volume

Coefficient of variation (CV) = standard deviation / mean

NYHA I
CV-RR 8.0%, CV-TV 7.4%

NYHA III
CV-RR 26.4%, CV-TV 35.3%

10min
Patients’ characteristics

<table>
<thead>
<tr>
<th></th>
<th>ASV</th>
<th>CPAP</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>63 ± 14</td>
<td>60 ± 15</td>
<td>n.s.</td>
</tr>
<tr>
<td>Men/women</td>
<td>17/3</td>
<td>11/7</td>
<td>n.s.</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>22.0 ± 3.5</td>
<td>21.9 ± 3.0</td>
<td>n.s.</td>
</tr>
<tr>
<td>Specific activity scale (Mets)</td>
<td>5.0 ± 1.2</td>
<td>4.8 ± 1.3</td>
<td>n.s.</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>31 ± 9</td>
<td>31 ± 10</td>
<td>n.s.</td>
</tr>
<tr>
<td>BNP (pg/ml)</td>
<td>240 ± 219</td>
<td>223 ± 179</td>
<td>n.s.</td>
</tr>
<tr>
<td>Plasma norepinephrine (pg/ml)</td>
<td>227 ± 91</td>
<td>257 ± 75</td>
<td>n.s.</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>5</td>
<td>3</td>
<td>n.s.</td>
</tr>
</tbody>
</table>

Mean ± SD or number of patients
## Patients’ characteristics

<table>
<thead>
<tr>
<th>Medication</th>
<th>ASV</th>
<th>CPAP</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>RAS-inhibitors (%)</td>
<td>90</td>
<td>94</td>
<td>n.s.</td>
</tr>
<tr>
<td>β-blockers (%)</td>
<td>70</td>
<td>78</td>
<td>n.s.</td>
</tr>
<tr>
<td>Digitalis (%)</td>
<td>25</td>
<td>11</td>
<td>n.s.</td>
</tr>
<tr>
<td>Loop-diuretics (%)</td>
<td>70</td>
<td>94</td>
<td>n.s.</td>
</tr>
<tr>
<td>Aldosterone antagonists (%)</td>
<td>55</td>
<td>44</td>
<td>n.s.</td>
</tr>
<tr>
<td>Amiodarone (%)</td>
<td>30</td>
<td>28</td>
<td>n.s.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Cardiorespiratory polygraphy</th>
<th>ASV</th>
<th>CPAP</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apnoea hypopnoea index (/h)</td>
<td>15  ± 13</td>
<td>13  ± 14</td>
<td>n.s.</td>
</tr>
<tr>
<td>Central apnoea index (/h)</td>
<td>7   ± 9</td>
<td>7   ± 11</td>
<td>n.s.</td>
</tr>
<tr>
<td>Obstructive apnoea index (/h)</td>
<td>2   ± 2</td>
<td>1   ± 1</td>
<td>n.s.</td>
</tr>
<tr>
<td>Hypopnoea index (/h)</td>
<td>6   ± 7</td>
<td>4   ± 5</td>
<td>n.s.</td>
</tr>
</tbody>
</table>
45y.o. man, DCM, ASV

Before

ECG
MSNA raw
MSNA integ
SpO2
Resp

During

1min
1min
52y.o. man, DCM, CPAP

Before vs During CPAP:

ECG

MSNA raw

MSNA integ

SpO2

Resp

1min
Respiratory instability

CV-TV

Before During
ASV

Before During
CPAP

CV-RR

Before During
ASV

Before During
CPAP

p < 0.01
p < 0.01

p < 0.01
p < 0.05
MSNA

**Burst rate**

- Before During ASV
- Before During CPAP

**Burst incidence**

- Before During ASV
- Before During CPAP

$p < 0.001$
Relationship between change in respiratory instability and change in burst incidence

ΔBurst incidence
(bursts/100beats)

ΔBurst incidence
(bursts/100beats)

Improvement of respiratory instability by ASV might relate to sympathoinhibition in patients with heart failure.
Summary

Short term effect of ASV compared with CPAP on respiratory pattern and MSNA was examined in patients with heart failure.

1) ASV improved respiratory instability, but CPAP did not.
2) ASV decreased MSNA, but CPAP did not.
3) Improvement of respiratory instability correlated with decrease in MSNA.
Conclusion

• ASV, but not CPAP, could reduce MSNA in patients with heart failure.

• These different responses of respiratory pattern might influence the difference in clinical effectiveness between ASV and CPAP.