Effect of CPAP on the metabolic syndrome: a randomised sham-controlled study

A recently published editorial concluded that severity of disease, Continuous Positive Airway Pressure (CPAP) compliance and comorbidities might explain discrepancies between a randomised sham-controlled crossover study which showed that CPAP reversed metabolic syndrome (metS) and reduced weight, body mass index (BMI) and visceral abdominal fat and our findings from a randomised sham-controlled parallel-group study. Whether CPAP might be a novel method to reverse metS in those with Obstructive Sleep Apnea (OSA) is an intriguing possibility, since diagnosing and treating metS is important. We omitted to examine the effect of CPAP on metS in our

<table>
<thead>
<tr>
<th>Table 1</th>
<th>The development and regression of metS from baseline to week 12</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>CPAP</td>
</tr>
<tr>
<td></td>
<td>metS n MetS</td>
</tr>
<tr>
<td>Baseline</td>
<td>18 14</td>
</tr>
<tr>
<td>Week 12</td>
<td>12 2</td>
</tr>
<tr>
<td></td>
<td>metS n MetS</td>
</tr>
</tbody>
</table>
| Data are n metS, metabolic syndrome.
population, a typical OSA cohort with treated long-standing metabolic comorbidities and less than ideal CPAP usage. To rectify this, we retrospectively assayed stored blood for lipids and abstracted information regarding hypertension, hyperlipidaemia and its treatment to diagnosis metS.

The study design and baseline characteristics have been previously reported. MetS was defined according to international consensus guidelines, and the presence (or absence) of metS was assessed at 0 and 12 weeks. The change in the proportion of participants with or without metS from baseline were analysed by generalised linear models examining the treatment by time interaction (SAS V9.2). Analyses utilised generalised estimating equations and an exchangeable correlation structure, which were then confirmed by Bayesian methods.

Reversal of metS after 12 weeks occurred in 3 of 18 (17%) men with metS at baseline treated with CPAP compared with 1 of 14 (7%) men treated with sham; whereas metS developed in 2 of 14 (14%) men without metS at baseline compared with 3 of 17 (18%) men treated with sham (time by treatment interaction p=0.28): table 1. This indicates that 12 weeks of CPAP therapy had no effect on the development or regression of metS. Utilising Bayesian methods, restricting the analysis to the 49 men with complete data, or using the original national cholesterol education program adult treatment panel III criteria for diagnosing metS did not alter this finding.

CPAP therapy remains the standard care for OSA, however its effect on metS has only been previously examined in two contradictory randomised cross-over studies, and now by us. On the other hand, all randomised sham-controlled studies show no effect of CPAP on visceral abdominal fat, BMI and weight, except one: table 2. Our original report and these additional data support the conclusion that CPAP is unlikely to have a major effect on metabolic health in unselected individuals with OSA.

Acknowledgements We thank the men who participated in the study. We would also like to thank the research team, sleep physicians and technicians at the Woolcock Institute of Medical Research. We also thank the Sleep Disorders Unit and Biochemistry department of the Royal Prince Alfred Hospital.

REFERENCES


Table 2 Randomised sham-controlled studies examining the effect of CPAP on visceral abdominal fat (VAF)

<table>
<thead>
<tr>
<th>n</th>
<th>M</th>
<th>F</th>
<th>Design</th>
<th>AHI</th>
<th>BMI</th>
<th>Duration (weeks)</th>
<th>CPAP effect on VAF</th>
<th>CPAP effect on BMI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hoyos et al³</td>
<td>65</td>
<td>0</td>
<td>Parallel</td>
<td>40</td>
<td>31</td>
<td>12</td>
<td>−0.06 (−0.58 to 0.70) p=0.85</td>
<td>0.07 (−0.11 to 0.26) p=0.79</td>
</tr>
<tr>
<td>Sharma et al²</td>
<td>77</td>
<td>9</td>
<td>Cross-over</td>
<td>50</td>
<td>33</td>
<td>12</td>
<td>−0.20 (−0.37 to −0.06) p=0.01</td>
<td>−0.06 (−0.1 to −0.01) p=0.001</td>
</tr>
<tr>
<td>Sivam et al⁶</td>
<td>26</td>
<td>1</td>
<td>Cross-over</td>
<td>57</td>
<td>31</td>
<td>8</td>
<td>−0.03 (−0.15 to 0.08) p=0.59</td>
<td>0.07 (−0.05 to 0.05) p=0.32</td>
</tr>
<tr>
<td>Kritikou et al²</td>
<td>22</td>
<td>20</td>
<td>Cross-over</td>
<td>42</td>
<td>27</td>
<td>8</td>
<td>0.14 (−0.09 to 0.37) p=0.25</td>
<td>0.07 (−0.24 to 0.38) p=0.67</td>
</tr>
</tbody>
</table>

Data are calculated standardised effect sizes (95% CI) after treatment, unless otherwise stated.

*Values are adjusted for baseline.
M, Male; F, Female; AHI, Apnea Hypopnea Index; BMI, body mass index.
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