Obesity hypoventilation Syndrome

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Conflict of Interest Disclosures for Speakers

1. I do not have any relationships with any entities producing, marketing, reselling, or distributing health care goods or services consumed by, or used on, patients, OR

2. I have the following relationships with entities producing, marketing, reselling, or distributing health care goods or services consumed by, or used on, patients.

<table>
<thead>
<tr>
<th>Type of Potential Conflict</th>
<th>Details of Potential Conflict</th>
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</thead>
<tbody>
<tr>
<td>Grant/Research Support</td>
<td>NIH/NHLBI and Philips/Respironics</td>
</tr>
<tr>
<td>Consultant</td>
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<tr>
<td>Speakers’ Bureaus</td>
<td>Zephyr Medical Technologies</td>
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<tr>
<td>Financial support</td>
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<td>Other</td>
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</table>

3. The material presented in this lecture has no relationship with any of these potential conflicts, OR

4. This talk presents material that is related to one or more of these potential conflicts, and the following objective references are provided as support for this lecture:

1.
2.
3.
Objectives

- Review the definition and epidemiology of OHS
- Understand the clinical presentation and diagnosis and when to suspect OHS
- Recognize the high morbidity and mortality associated with undiagnosed and untreated OHS
  - Postoperative risk of OHS
- Discuss treatment strategies
An example of a patient with OHS
Definition and epidemiology
# Definition of OHS

## Required conditions

<table>
<thead>
<tr>
<th>Condition</th>
<th>Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Obesity</strong></td>
<td>• Body mass index ( \geq 30 \text{ kg/m}^2 )</td>
</tr>
<tr>
<td><strong>Chronic Hypoventilation</strong></td>
<td>• Awake daytime hypercapnia ( \text{PaCO}_2 \geq 45 \text{ mm Hg} )</td>
</tr>
<tr>
<td><strong>Sleep-disordered breathing</strong></td>
<td>• OSA ( \text{AHI} \geq 5 ) present in 90% of cases</td>
</tr>
<tr>
<td></td>
<td>• Sleep hypoventilation ( \text{AHI} &lt; 5 ) present in 10%</td>
</tr>
<tr>
<td><strong>Exclude other causes of hypercapnia</strong></td>
<td>• Significant obstructive airways disease</td>
</tr>
<tr>
<td></td>
<td>• Significant interstitial lung disease</td>
</tr>
<tr>
<td></td>
<td>• Severe chest wall disorders (e.g., kyphoscoliosis)</td>
</tr>
<tr>
<td></td>
<td>• Severe hypothyroidism</td>
</tr>
<tr>
<td></td>
<td>• Neuromuscular disease</td>
</tr>
</tbody>
</table>

Mokhlesi B, Tulaimat A. Chest 2007; 132:1322-1336
Obesity Hypoventilation Syndrome

Diagnosis of Exclusion!
6.4% of all adults in the US have BMI $\geq 40 \text{ kg/m}^2$ and in African Americans the prevalence of severe obesity is 12.2%.

Ogden CL. JAMA 2014
## Prevalence of OHS in obese patients being evaluated for OSA

<table>
<thead>
<tr>
<th>Author, Country</th>
<th>Year</th>
<th>Patients (No.)</th>
<th>Male (%)</th>
<th>Age (yrs)</th>
<th>BMI (kg/m²)</th>
<th>AHI</th>
<th>OHS (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leech, US&lt;sup&gt;x&lt;/sup&gt;</td>
<td>1987</td>
<td>111</td>
<td>68%</td>
<td>47</td>
<td>NR</td>
<td>58</td>
<td>37%</td>
</tr>
<tr>
<td>Resta, Italy&lt;sup&gt;x&lt;/sup&gt;</td>
<td>2000</td>
<td>219</td>
<td>64%</td>
<td>50</td>
<td>40</td>
<td>45</td>
<td>17%</td>
</tr>
<tr>
<td>Verin, France&lt;sup&gt;x&lt;/sup&gt;</td>
<td>2001</td>
<td>218</td>
<td>92%</td>
<td>55</td>
<td>34</td>
<td>51</td>
<td>10%</td>
</tr>
<tr>
<td>Akashiba, Japan</td>
<td>2002</td>
<td>143</td>
<td>100%</td>
<td>48</td>
<td>30</td>
<td>55</td>
<td>38%</td>
</tr>
<tr>
<td>Laaban, France</td>
<td>2005</td>
<td>1141</td>
<td>83%</td>
<td>56</td>
<td>34</td>
<td>55</td>
<td>11%</td>
</tr>
<tr>
<td>Mokhlesi, US&lt;sup&gt;x&lt;/sup&gt;</td>
<td>2007</td>
<td>522</td>
<td>56%</td>
<td>48</td>
<td>44</td>
<td>59</td>
<td>24%</td>
</tr>
<tr>
<td>Kawata, Japan&lt;sup&gt;x&lt;/sup&gt;</td>
<td>2007</td>
<td>1227</td>
<td>89%</td>
<td>50</td>
<td>29</td>
<td>42</td>
<td>14%</td>
</tr>
<tr>
<td>Banerjee, Australia&lt;sup&gt;t&lt;/sup&gt;</td>
<td>2007</td>
<td>74</td>
<td>54%</td>
<td>43</td>
<td>59</td>
<td>62</td>
<td>31%</td>
</tr>
<tr>
<td>Macavei, UK&lt;sup&gt;x&lt;/sup&gt;</td>
<td>2013</td>
<td>344</td>
<td>64%</td>
<td>52</td>
<td>39</td>
<td>25</td>
<td>21%</td>
</tr>
<tr>
<td>Aggregate or mean</td>
<td></td>
<td>3999</td>
<td>74%</td>
<td>50</td>
<td>38</td>
<td>50</td>
<td>17%</td>
</tr>
</tbody>
</table>

Balachandran JS, Masa JF, Mokhlesi B. Sleep Med Clin 2014
Prevalence of Obesity Hypoventilation Syndrome in patients with OSA

Balachandran JS, Masa JF, Mokhlesi B. Sleep Med Clin 2014
Estimated prevalence of OHS in the general population

General US adult population

6% with severe obesity

½ with OSA

¼ with OHS ≈

1 in 160 adults

Balachandran JS, Masa JF, Mokhlesi B. Sleep Med Clin 2014
Diagnosis and Presentation
Clinical features of OHS from 16 studies and a total of 757 patients

<table>
<thead>
<tr>
<th>Variables</th>
<th>Mean (range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, year</td>
<td>52 (42-61)</td>
</tr>
<tr>
<td>Men, %</td>
<td>60 (49-90)</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>44 (35-56)</td>
</tr>
<tr>
<td>Neck circumference, cm</td>
<td>46.5 (45-47)</td>
</tr>
<tr>
<td>pH</td>
<td>7.38 (7.34-7.40)</td>
</tr>
<tr>
<td>PaCO₂, mm Hg</td>
<td>53 (47-61)</td>
</tr>
<tr>
<td>PaO₂, mm Hg</td>
<td>56 (46-74)</td>
</tr>
<tr>
<td>Serum bicarbonate, mEq/L</td>
<td>32 (31-33)</td>
</tr>
<tr>
<td>Hemoglobin, g/dL</td>
<td>15</td>
</tr>
<tr>
<td>MRC dyspnea class 3 and 4, %</td>
<td>69</td>
</tr>
<tr>
<td>Epworth sleepiness scale</td>
<td>14 (12-16)</td>
</tr>
</tbody>
</table>

Mokhlesi B et al. Proc Amer Thorac Soc. 2008:5;221
PSG and PFT features of OHS from 16 studies and a total of 757 patients

<table>
<thead>
<tr>
<th>Variables</th>
<th>Mean (range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apnea-hypopnea index</td>
<td>66 (20-100)</td>
</tr>
<tr>
<td>SpO$_2$ nadir during sleep, %</td>
<td>65 (59-76)</td>
</tr>
<tr>
<td>Percent time SpO$_2$ &lt; 90%, %</td>
<td>50 (46-56)</td>
</tr>
<tr>
<td>FVC, % of predicted</td>
<td>68 (57-102)</td>
</tr>
<tr>
<td>FEV$_1$, % of predicted</td>
<td>64 (53-92)</td>
</tr>
<tr>
<td>FEV$_1$/FVC</td>
<td>77 (74-88)</td>
</tr>
</tbody>
</table>

Mokhlesi B et al. Proc Amer Thorac Soc. 2008:5;221
Two Patterns of Presentation

- Acute on chronic respiratory failure
  - 8% of all admissions to ICU had a diagnosis consistent with OHS
  - 75% were misdiagnosed as COPD with no evidence of obstruction on PFT

- As part of routine evaluation of OSA
- Frequently missed and diagnosed at late stage by pulmonologists or sleep specialists

Quint et al, Thorax 2007
When to suspect OHS

- Severely obese (BMI ≥ 40)
- Elevated venous bicarbonate levels from recent basic metabolic panels
- Room air hypoxemia by finger pulse oximetry
- Significant and persistent hypoxemia during PSG
- Spirometry/PFT with mild restrictive defect due to body habitus
Bicarbonate as a screening tool

OSA and BMI ≥ 30 kg/m² (n=522)

Serum HCO₃ < 27 mEq/L (n=257) 3% with OHS

Serum HCO₃ ≥ 27 mEq/L (n=265) 50% with OHS

Mokhlesi B et al. Sleep Breath 2007; 11:117
Macavei VM, et al. JCSM 2013; 9:879-84

A one unit increase in serum HCO₃ was associated with a 14% increase in the probability of having OHS

Does the current definition need revisiting?

- The current definition is based on a single one-time measurement of PaCO$_2$

- Calculated arterial or measured venous bicarbonate is a longer term guide to 24-h ventilation

- Proposed new definition:
  - Obesity
  - PaCO$_2 \geq 45$ mm Hg OR an arterial base excess $>3$ mmol/L OR a standard HCO$_3$ $>27$ mmol/L
  - absence of another cause for a metabolic alkalosis

Is a raised bicarbonate level without hypercapnia part of the spectrum of OHS?

<table>
<thead>
<tr>
<th></th>
<th>Eucapnic normal BE n=33</th>
<th>Eucapnic Elevated BE n=22</th>
<th>Hypercapnic Elevated BE n=16</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>53.6</td>
<td>48.7</td>
<td>53.7</td>
<td>0.09</td>
</tr>
<tr>
<td>BMI</td>
<td>45.2 (9.1)</td>
<td>46.5 (7.9)</td>
<td>51.6 (11.7)</td>
<td>0.056</td>
</tr>
<tr>
<td>Base Excess, mEq/L</td>
<td>0.12 (1.38)</td>
<td>3.01 (0.98)</td>
<td>4.78 (2.10)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>HCO₃, mEq/L</td>
<td>24.4 (1.18)</td>
<td>27.0 (0.87)</td>
<td>28.5 (2.11)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>pH</td>
<td>7.41</td>
<td>7.44</td>
<td>7.41</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>PaCO₂, mm Hg</td>
<td>38.6</td>
<td>40.6</td>
<td>49.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>SpO₂</td>
<td>96</td>
<td>96.3</td>
<td>92.4</td>
<td>0.007</td>
</tr>
<tr>
<td>VE, L/min</td>
<td>8.05</td>
<td>8.33</td>
<td>7.54</td>
<td>0.47</td>
</tr>
<tr>
<td>VE hypercapnic test, L/min</td>
<td>14.6</td>
<td>11.96</td>
<td>11.76</td>
<td>0.035</td>
</tr>
</tbody>
</table>

Manuel AR, Hart N, Stradling JR. Chest 2015
Pathophysiology of respiratory failure in OHS
Pathophysiology of respiratory failure

**Lung and chest wall loads**
- Decreased lung compliance
- Inspiratory threshold loading
- Supine position

**Other loads**
- CO₂ production
- Dead space

**Resistive loads**
- Upper airway obstruction
- Lower airway obstruction

**Decreased drive**
- Blunted drive in OHS
- Sedatives/Narcotics

**Decreased strength**
- Deconditioning
- Metabolic disorders
- Myopathy

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*Carr GE, Mokhlesi B, Gehlbach BK. Chest 2012 141:798*
Pathophysiology of respiratory failure

Increased Load

Decreased strength and drive
How does sleep hypoventilation lead to wake hypoventilation

- Obesity (Increased VCO₂)
  - Leptin resistance
  - Increased mechanical load/Relative weak respiratory muscles
- Obstructive sleep apnea
  - Upper airway resistance
    - Acute hypercapnia during sleep
      - Decreased CO₂ response
      - Reduced HCO₃⁻ excretion rate
- Blunted ventilatory response
- Chronic hypercapnia
- Increased serum bicarbonate
Morbidity and Mortality
Clinical Implications of OHS

- Compared to simple eucapnic OSA, patients with OHS have:
  - Lower quality of life
  - Greater healthcare expenses
  - Higher risk of pulmonary hypertension
  - Higher risk of death attributed to:
    - severe obesity
    - severe OSA
    - chronic respiratory failure

Berg G. Chest 2001; 120:377-83
Hida W. Sleep Breath. 2003; 7:1
OHS in hospitalized patients
31% of obese patients admitted to the wards were found to have undiagnosed OHS (BMI 45±9)

NO HOSPITAL DEATHS but more ICU transfers and intubations

- Mortality at 18 months was 23% vs. 9% (HR=4.0; 95% CI: 1.5 to 10.4)
- Adjusted for age, BMI, electrolytes, renal and thyroid function

Causes of acute on chronic hypercapnic respiratory failure in OHS

- Prospective study over 13 years in Spain
- 173 OHS patients with acute exacerbation
- BMI 42, age 74
- Only 9% of OHS were on home NIV but 39% on oxygen

Causes of exacerbation:
- Respiratory infection: 68%
- Cardiac: 13%
- Depressant drugs: 5%
- Trauma: 3%
- Surgery: 3%

Carrillo A, et al. AJRCCM 2012; 186:1279
Outcomes in OHS after acute hypercapnic respiratory failure treated with NIV

- OHS n=173, BMI 42, age 74
- COPD n=543, BMI 30, age 71
- Only 9% of OHS were on home NIV but 39% on oxygen
- 4% in each group required ET-T intubation
- OHS had lower ICU and hospital mortality (6% vs. 18%)
- Adjusted survival was not significantly different (p=0.11)
- At one year 45% were on CPAP and 10% on NIV

Carrillo A, et al. AJRCCM 2012; 186:1279
Long-term survival compared to OSA

◆ Retrospective study of 110 OHS vs 220 matched OSA patients
  - Similar age, sex, AHI, Epworth
  - PAP adherence ~ 6 h/night in both groups
  - Mean NIV 18/8 cm H$_2$O in OHS, mean CPAP 9 cm H$_2$O in OSA
  - Mean follow-up time of 7±4 years

◆ Five year mortality rates:
  - OHS: 15.5%
  - OSA: 4.5%
  - Risk of mortality: OR 2 (95% CI: 1.11-3.60)
  - Risk of CV event: OR 1.86 (95% CI: 1.14-3.04)
  - Strongest predictor of mortality was adherence to NIV < 4 hours
Treatment
Therapeutic options

- Positive airway pressure therapy
- Surgery
  - Tracheostomy
  - Bariatric surgery
- Pharmacological therapy
  - Medroxyprogesterone
  - Acetazolamide
  - Oxygen
PAP Therapeutic options

◆ Positive airway pressure therapy
  - CPAP
  - Bi-level PAP (spontaneous mode or S/T)
  - Volume-targeted pressure support
    ❖ AVAPS (Respironics)
    ❖ iVAPS (ResMed)
CPAP or bilevel PAP S mode in OHS

- CPAP titration failure rate can be as high as 43% in patients with OHS due to persistent hypoxemia
  

- In an RCT, 36 patients were randomized to CPAP (n=18) vs. bi-level PAP in spontaneous mode (n=18) for 3 months
  - CPAP failures were excluded
  - Change in PaCO₂ was 5.8 mm Hg with CPAP and 6.9 in bilevel PAP S mode

Volume-Targeted Pressure Support

- Automatically adjusts IPAP to guarantee a target tidal volume

Murphy PB et al. Thorax 2012;67(8):727-34
Masa JF et al. AJRCCM 2015; 192: 86
AVAPS vs. bilevel PAP/ST in OHS

RCT of 50 OHS patients to bilevel PAP/ST vs. AVAPS
- 34% enrolled during an acute-on-chronic respiratory failure

At three months there was no group differences in:
- PaCO$_2$ and PaO$_2$
- Epworth and QOL
- Decrease in BMI
- Improvement in FVC

Murphy PB et al. Thorax 2012;67(8):727-34
No differences in ventilator parameters

<table>
<thead>
<tr>
<th>Parameters</th>
<th>AVAPS (n=25)</th>
<th>Bi-level PAP/ST (n=25)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Delivered IPAP, cm H₂O</td>
<td>22±5</td>
<td>23±4</td>
</tr>
<tr>
<td>Set EPAP, cm H₂O</td>
<td>9±1</td>
<td>10±2</td>
</tr>
<tr>
<td>Leak, L/min</td>
<td>53±13</td>
<td>53±19</td>
</tr>
<tr>
<td>Patient triggered breaths, %</td>
<td>43±27</td>
<td>45±27</td>
</tr>
<tr>
<td>Mean adherence, h:min</td>
<td>4:11±2:53</td>
<td>5:08±2:22</td>
</tr>
<tr>
<td>Delta PaCO₂, mm Hg</td>
<td>- 4.5±7.5</td>
<td>- 4.5±8.2</td>
</tr>
</tbody>
</table>

AVAPS set in pressure control mode, tidal volume of 8-10 ml/kg IBW
Efficacy of Different Treatment Alternatives for Obesity Hypoventilation Syndrome: Pickwick Study

Masa JF et al. AJRCCM 2015; 192: 86
Efficacy of Different Treatment Alternatives for Obesity Hypoventilation Syndrome: Pickwick Study
NIV in patients with OHS without severe OSA

NIV in patients with OHS without severe OSA

- NIV was more effective in improving PSG parameters, ESS and QoL

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Baseline, mean (SD)/median (IQR)</th>
<th>Intra-group differences, mean (95% CI)</th>
<th>p Value of inter-group differences$</th>
</tr>
</thead>
<tbody>
<tr>
<td>NIV</td>
<td>Control</td>
<td></td>
<td>Unadjusted</td>
</tr>
<tr>
<td>PaCO$_2$, mm Hg</td>
<td>49 (4.0)</td>
<td>-6 (-7.7 to -4.2)$</td>
<td>0.006</td>
</tr>
<tr>
<td>Serum bicarbonate, mmol/L</td>
<td>30 (4.1)</td>
<td>-3.4 (-4.5 to -2.3)$</td>
<td>0.000</td>
</tr>
<tr>
<td>pH</td>
<td>7.400 (0.040)</td>
<td>0.005 (-0.005 to 0.157)</td>
<td>NS</td>
</tr>
<tr>
<td>PaO$_2$, mm Hg</td>
<td>64 (10)</td>
<td>4.6 (0.5 to 8.8)$</td>
<td>NS</td>
</tr>
<tr>
<td>FEV$_1$, %</td>
<td>72 (16)</td>
<td>1.8 (-2.7 to 6.4)</td>
<td>NS</td>
</tr>
<tr>
<td>FVC, %</td>
<td>75 (21)</td>
<td>4.7 (-4.2 to 14)</td>
<td>NS</td>
</tr>
<tr>
<td>6-MWD, m</td>
<td>309 (105)</td>
<td>29 (-16 to 74)</td>
<td>NS</td>
</tr>
<tr>
<td>Systolic BP, mm Hg</td>
<td>136 (18)</td>
<td>-4.2 (-11 to 2.5)</td>
<td>NS</td>
</tr>
<tr>
<td>Diastolic BP, mm Hg</td>
<td>80 (16)</td>
<td>0.5 (-5.3 to 6.2)</td>
<td>NS</td>
</tr>
</tbody>
</table>

## Impact of PAP Adherence on hypercapnia/hypoxemia in OHS

<table>
<thead>
<tr>
<th>Subgroup</th>
<th>N (%)</th>
<th>Change in PaCO$_2$ (Mean ± SD)</th>
<th>Change in PaO$_2$ (Mean ± SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Adherence with therapy</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average PAP use &gt; 4.5 h/day</td>
<td>34 (45%)</td>
<td>8±5</td>
<td>9±11</td>
</tr>
<tr>
<td>Average PAP use &lt; 4.5 h/day</td>
<td>41 (55%)</td>
<td>2±4</td>
<td>2±9</td>
</tr>
</tbody>
</table>

Non PAP treatment modalities

- **Oxygen**: no role as single therapy
  - At high concentrations it can increase \( \text{PaCO}_2 \) because of reduction in minute ventilation

- Recent study revealed that 20 minutes of \( \text{FiO2} \) at 50% increased PavCO2 from 53 mm Hg to 58 mm Hg with a drop in tidal volume by 89 ml.
Need for oxygen during PAP titration

- During CPAP titration 43% required supplemental oxygen (average CPAP pressures of 14 cm H₂O).
  
  Banerjee D, Chest. 2007; 131:1678
  Mokhlesi B, J Clin Sleep Med. 2006; 2:57

- Other studies of similar patients undergoing aggressive NIV titration (IPAP of ~ 13 cm H₂O above an average EPAP of 10), or volume targeted pressure support only 12%-23% required oxygen supplementation

  Murphy PB et al. Thorax 2012;67:727
  Masa JF et al. AJRCCM 2015; 192:86
The effect of supplemental oxygen in OHS in the Pickwick study

- Post-hoc analysis of a previous RCT
- 302 sequentially screened OHS patients who were randomly assigned to NIV, CPAP, or lifestyle modification.
  - 78 out of 302 (26%) were prescribed home oxygen therapy
- Oxygen therapy (1-2 L/min) was not associated with an increase in worsening ABG or hospital resource utilization in any of the groups at two months.
- Long-term studies are necessary.

Tracheostomy

- Retrospective study
- 13 patients with OSA plus OHS
- Tracheostomy improved but did not fully resolve SDB in patients with OSA plus OHS
  - NREM AHI 64 to 31
  - REM AHI 46 to 39
  - 7/13 had AHI > 20
  - Persistent SDB due to orifice obstruction b/o chin or neck adiposity or central sleep apnea
- Hypercapnia resolved in most patients

Impact of Bariatric Surgery on Respiratory Insufficiency

- 29 patients with OHS or OSA+OHS
- Mean weight loss of 50±29 kg (110±65 pounds)
- $\text{PaO}_2$ increased from 53±9 to 68±11 mm Hg
- $\text{PaCO}_2$ decreased from 51±7 to 41±4 mm Hg
- Hb decreased from 16.9 to 14.9 g/dl
- Significant improvements in ERV, FRC, FVC

Sugerman HJ. Chest 1986; 90:81
Postoperative complications in patients with unrecognized OHS: A retrospective study

Adjusted postoperative complications in patients with unrecognized OHS

<table>
<thead>
<tr>
<th>Postoperative Outcome</th>
<th>Hypercapnic OSA (n = 194)</th>
<th>OSA (n = 325)</th>
<th>OR (95% CI)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory failure</td>
<td>39 (21)</td>
<td>8 (2)</td>
<td>10.9 (3.7-32.3)</td>
<td>&lt; .0001</td>
</tr>
<tr>
<td>Heart failure</td>
<td>15 (8)</td>
<td>0</td>
<td>5.4 (1.9-15.7)</td>
<td>.002</td>
</tr>
<tr>
<td>Prolonged intubation</td>
<td>24 (13)</td>
<td>12 (4)</td>
<td>3.1 (0.6-15.3)</td>
<td>.2</td>
</tr>
<tr>
<td>Reintubation</td>
<td>12 (6)</td>
<td>5 (2)</td>
<td>1.7 (0.2-13.4)</td>
<td>.6</td>
</tr>
<tr>
<td>Tracheostomy</td>
<td>4 (2)</td>
<td>3 (1)</td>
<td>3.8 (1.7-8.6)</td>
<td>.002</td>
</tr>
<tr>
<td>ICU transfer</td>
<td>41 (21)</td>
<td>19 (6)</td>
<td>10.9 (3.7-32.3)</td>
<td>&lt; .0001</td>
</tr>
<tr>
<td>Death at 30 d</td>
<td>2 (1)</td>
<td>0</td>
<td>...^a</td>
<td>...</td>
</tr>
<tr>
<td>Death at 1 y</td>
<td>10 (5)</td>
<td>2 (0.6)</td>
<td>0.9 (0.1-7.5)</td>
<td>.9</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Variables</th>
<th>Hypercapnic OSA (n = 194)</th>
<th>OSA (n = 325)</th>
<th>β ± SE</th>
<th>P</th>
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<tbody>
<tr>
<td>ICU length of stay, d</td>
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<td></td>
</tr>
<tr>
<td>Median (IQR)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
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<td>.009</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>0.12 (0.93)</td>
<td>1.04 (3.8)</td>
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<tr>
<td>Hospital length of stay, d</td>
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<tr>
<td>Median (IQR)</td>
<td>5 (3-9)</td>
<td>0 (0-4)</td>
<td></td>
<td>.0008</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>7.3 (8.2)</td>
<td>2.8 (5.1)</td>
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</tr>
</tbody>
</table>
Postoperative complications in patients with OSA: hypercapnia may be more relevant than AHI!

Cooksey J, Mokhlesi B. Chest 2016; 149: 11
One possible preoperative approach to OHS

SUSPECTED OBESITY HYPOVENTILATION SYNDROME
(Obese patient [BMI > 30] with OSA going for elective surgery)

Prior sleep study

DONE
(AHI ≥ 5)

NOT DONE
STOP BANG score > 3

CHECK SERUM BICARBONATE

Serum bicarbonate ≤ 27

OSA PERIOPERATIVE PRECAUTIONS

Serum bicarbonate > 27, a
sPO₂ < 90% or both

Order ABG (PaCO₂ ≥ 45)

Order PSG (if not done previously) (AHI ≥ 5)

OBESITY HYPOVENTILATION SYNDROME ESTABLISHED

Research questions

- How to best screen preoperative patients for unrecognized OHS
- How to approach patients with OHS who are nonadherent to PAP therapy
- How safe is postop supplemental oxygen
- Best monitoring strategies for patients with hypercapnia
  - Oxygenation
  - Ventilation
- Avoiding management pitfalls:
  - Over diuresis
  - Excessive oxygen supplementation
Conclusions

- OHS is prevalent in patients with severe obesity and OSA
- It is frequently unrecognized and undertreated
- Untreated OHS significantly increases the risk of morbidity and mortality
- Comprehensive treatment strategies should focus on:
  - Nocturnal resolution of sleep disordered breathing
  - Weight loss
  - Increasing physical activity