Case Presentation: Undetected Hypoventilation

Hillary Loomis-King, MD
Pulmonary and Critical Care of NW MI
Munson Sleep Disorders Center
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2. I have the following relationships with entities **producing, marketing, reselling, or distributing** health care goods or services consumed by, or used on, patients.

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<th>Type of Potential Conflict</th>
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Objectives

1- Review basic pulmonary physiology.
2- Establish patient populations in which hypoventilation needs to be considered.
3- Know diagnostic criteria for sleep-related hypoventilation syndromes.
4- Understand treatment options for patients with clinical hypoventilation.
R.W.

- 66yo male visiting from Las Vegas brought to ED by family for altered mentation, somnolence and apparent dyspnea.
- Noted that he seemed to be speaking as though he was drunk when they met him at airport.
- Sought medical care when this persisted for 24 hours after his arrival with him sleeping most of the day.
- Family knows minimal history, see him a couple of times per year. Niece had noticed 7 months ago when she last saw him that his legs were quite swollen.
R.W.

ED Events

- SpO2 74% on room air
- Improved to 93% on O2 @ 3 LPM
- ABG: pH 7.01, pCO2 > 95, pO2 206
- Bi-level PAP started at 18/10 with BUR 18
- Repeat ABG 7.03, pCO2 > 95, pO2 114
- Intubated for concomitant decline in mental status
R.W.

CT Chest PE Protocol
• No acute PE
• Small right pleural effusion with adjacent atelectasis.
• Scattered areas of patchy airspace opacity
• Cardiomegaly

CT Head w/o Contrast
• No acute intracranial abnormality
R.W.

Labs

- CBC 5.8/18.1/103
- INR 1.6
- BMP 142/4.5/105/32/43/2.2
- BNP 546
- Serial troponins negative
Past Medical History

• Respiratory Failure- intubated x6 weeks in Las Vegas 7 years prior
  – Trach discussed but never done
  – Discharged on O2 but subsequently self-weaned

• Hypertension

• Chronic Lower Extremity Edema

• Polycythemia
  – Chronically anti-coagulated

• OSA, prescribed CPAP 16 months prior
  – Questionable compliance

Past Surgical History

• Roux-en-Y gastric bypass
R.W.

Social Hx
- Reformed smoker for 25 years, 30 pack-years prior
- No alcohol or drugs
- Works as a bus drive on the Las Vegas strip!

Family Hx
- Father with Hep C and heart disease

Allergies: amoxicillin

Medications:
- Warfarin
- Losartan-HCTZ
R.W.

• Admitting Diagnoses
R.W.

• Additional Studies
R.W.

Echocardiogram

• Normal LV ejection fraction at 55-60% with normal global systolic function
• Mild concentric LVH
• Normal LV filling pressures
• Severely enlarged right ventricle
• Enlarged right atrium
• Tricuspid annulus dilation
• Normal PASP, 35 mmHg
• Dilated IVD with less than 50% size variation consistent with elevated right atrial pressure
Pulmonary Function Testing: Very severe obstructive ventilatory defect with air trapping.
Was R.W.’s decompensation preventable?

Split Night PSG 12/24/16

• AHI 25.2
• Nadir O2 saturation 69%
• “For this the patient was put on nasal CPAP and titrated to a level of 16 cmH2O. At this level the respiratory events and the severe desaturation events were eliminated. The CPAP titration study was graded out as optimal.”
• Recommended follow-up nocturnal pulse oximetry study on CPAP to determine if supplemental oxygen indicated.
Was R.W.’s decompensation preventable?
Minute Ventilation: closely linked with blood CO$_2$ values

\[ V_E = V_T \times f \]
Alveolar Anatomy

- Oxygen enters red blood cells
- Diffusion of oxygen
- Carbon dioxide escapes into alveolus
- To pulmonary vein
- Film of moisture
- Epithelium of alveolus
- Capillary
- Red blood cell
- From pulmonary artery
- Ventilation
Alveolar Ventilation

\[ P_{aCO_2} = \kappa \times (V_{CO_2}/V_A) \]

where

\[ V_A = V_E - V_E \left( V_D/V_T \right) \]
Alveolar Ventilation

\[ V_D = \text{dead space} \]

**Fixed dead space**

**Alveolar dead space**
- VQ mismatch
  - Atelectasis, pulmonary embolism, pulmonary vascular disease, pneumonia
- R to L shunt
- Impaired Diffusion
  - Interstitial lung disease
Hypoxemia in Hypoventilation

$PAO_2 = (P_{atm} - P_{H_2O})FIO_2 - (P_{ACO_2}/RQ)$

*Contribution of $FIO_2$ in this equation shows why hypoxemia can be overcome by addition of supplemental oxygen.*
Renal Compensation

• Respiratory acidosis is buffered by renal compensation

\[
\text{CO}_2 + \text{HOH} \rightleftharpoons \text{H}_2\text{CO}_3 \rightleftharpoons \text{H}^+ + \text{HCO}_3^-
\]
Pulmonary Function Testing

![Graph of Flow/Volume vs Volume (Liters)]

<table>
<thead>
<tr>
<th>Oximetry at Rest: 95% at 0.21 FIO2</th>
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<tbody>
<tr>
<td>Baseline</td>
</tr>
<tr>
<td><strong>LUNG MECHANICS</strong></td>
</tr>
<tr>
<td>Actual</td>
</tr>
<tr>
<td>--------</td>
</tr>
<tr>
<td>FVC</td>
</tr>
<tr>
<td>FEV1</td>
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<p>| <strong>LUNG VOLUMES</strong>                |</p>
<table>
<thead>
<tr>
<th>Actual</th>
<th>Pred</th>
<th>%Pred</th>
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<tbody>
<tr>
<td>VC</td>
<td>3.85</td>
<td>3.64</td>
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<tr>
<td>IC</td>
<td>2.46</td>
<td>2.26</td>
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<tr>
<td>ERV</td>
<td>1.39</td>
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<tr>
<td>FRC pleth</td>
<td>3.30</td>
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<tr>
<td>RV pleth</td>
<td>1.91</td>
<td>1.46</td>
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<tr>
<td>TLC pleth</td>
<td>5.76</td>
<td>5.10</td>
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<tr>
<td>RV/TLC pleth</td>
<td>33%</td>
<td>29%</td>
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<tr>
<td>Raw</td>
<td>1.12</td>
<td>&lt;2.00</td>
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<tr>
<td>Sgaw</td>
<td>0.26</td>
<td>&gt;0.21</td>
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<p>| <strong>DIFFUSION</strong>            |</p>
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<thead>
<tr>
<th>Actual</th>
<th>Pred</th>
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<tr>
<td>DLCO</td>
<td>20.87</td>
<td>24.19</td>
</tr>
<tr>
<td>IVC</td>
<td>3.73</td>
<td>3.85*</td>
</tr>
<tr>
<td>VA</td>
<td>5.25</td>
<td>5.10</td>
</tr>
<tr>
<td>DL/VA</td>
<td>3.98</td>
<td>4.75</td>
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Mechanisms of Impairment in R.W.

- Decreased mechanical efficiency of ventilation from COPD
- Heart failure with pulmonary edema and resultant VQ mismatch
- Obesity associated atelectasis with VQ mismatch
- OSA
POPULATIONS TO CONSIDER
HYPOVENTILATION
Known Gas Abnormalities

• Sustained hypoxia on sleep study
• Supplemental oxygen requirement in wakefulness
• Prior ABG with pCO$_2$ > 45 mmHg

\[ P_{AO_2} = (P_{atm} - P_{H_2O})F_{IO_2} - (P_{ACO_2}/RQ) \]
Other Populations

• Lung disease
• Neuromuscular disease
• Chest wall disease
• Morbid obesity
• Elevated serum bicarbonate
• Polycythemia
Other Populations

• Lung disease
  – Increased dead space
• Neuromuscular disease
  – Decreased $V_T$
• Chest wall disease
  – Decreased $V_T$
• Morbid obesity
  – Decreased $V_T$, atelectasis, and VQ mismatch
• Elevated serum bicarbonate
• Polycythemia
ESTABLISHING THE DIAGNOSIS OF HYPOVENTILATION
If electing to score hypoventilation, score hypoventilation during sleep if EITHER of the below occur:

a. There is an increase in the arterial PCO$_2$ (or surrogate) to a value $>55$ mmHg for $\geq$10 minutes.

b. There is $\geq$10 mmHg increase in arterial PCO$_2$ (or surrogate) during sleep (in comparison to an awake supine value) to a value exceeding 50 mmHg for $\geq$10 minutes.
Methodologies for Measuring CO$_2$

- Arterial Blood Gas
- End Tidal CO$_2$- non-invasive measurement of partial pressure of CO2 exhaled
- TCO2- CO2 is still measured potentiometrically by determining the pH of an electrolyte layer
ETCO2

- Techniques
- Accuracy
- drawbacks
TCO2

- Technique
- Accuracy
- drawbacks
ICSD-3: Sleep Related Hypoventilation Disorders

Categories

- Obesity Hypoventilation Syndrome
- Congenital Central Alveolar Hypoventilation Syndrome
- Late-Onset Central Hypoventilation with Hypothalamic Dysfunction
- Idiopathic Central Alveolar Hypoventilation
- Sleep Related Hypoventilation Due to a Medication or Substance
- Sleep Related Hypoventilation Due to a Medical Disorder
ICSD-3: Obesity Hypoventilation Syndrome

Criteria A-C must be met

A. Presence of hypoventilation during wakefulness (PaCO$_2$ > 45 mmHg) as measured by arterial PCO$_2$, end-tidal CO$_2$, or transcutaneous CO$_2$

B. Presence of obesity (BMI > 30 kg/m$^2$)

C. Hypoventilation is not primarily due to lung parenchymal or airway disease, pulmonary vascular pathology, chest wall disorder, medication use, neurologic disorder, muscle weakness, or a known congenital or idiopathic central alveolar hypoventilation syndrome
ICSD-3: Sleep Related Hypoventilation Dues to a Medical Disorder

Criteria A-C must be met

A. Sleep related hypoventilation is present

B. A lung parenchymal or airway disease, pulmonary vascular pathology, chest wall disorder, neurologic disorder, or muscle weakness is believed to be the primary cause of hypoventilation

C. Hypoventilation is not primary due to obesity hypoventilation syndrome, medication use, or a known congenital central alveolar hypoventilation syndrome
TREATMENT OPTIONS
No back up rate, most algorithms require that a patient fail this prior to covering more advanced device.
E0471: Bi-level PAP with back-up rate

- Bi-level PAP with back up rate
  - Compared with traditional bi-level PAP, guarantees a minimal number of breaths per minute
  - Does not guarantee goal minute ventilation
E0471: AVAPS

Average Volume Assured Pressure Support

Settings

- Target $V_T$
- IPAP min & IPAP max
- EPAP (some devices now have adjusting EPAP)
- Breath rate
- Inspiratory time ($T_i$)
- Rise time
R.W. Follow-Up

• Extubated to BIPAP on hospital day #5
• Diuresed
• Discharged on his home CPAP machine at 16 cmH2O
  – Insurance constraints prevented getting more advanced therapy prior to discharge since he was from out of state.
  – Combination of diuresis and CPAP use brought pCO2 down to 64 mmHg prior to discharge
• Expedited PAP titration arranged at time of hospital discharge.
R.W. Follow-Up

- Include data from MMC PAP titration with T CO2 monitoring
References

• The AASM Manual for the Scoring of Sleep and Associated Events, Version 2.0.
• The AASM International Classification of Sleep Disorders, Third Edition.