ACOI
Clinical Challenges in Inpatient Care
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Chicago Marriott Downtown
Magnificent Mile, Chicago, IL
Registration now open

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Disclaimers: Remember when evaluating this lectures quality

• Dr. Barreiro has no financial or industry disclosures.

FULL DISCLOSURES:
• I have no relationship with Russia.

• Dr. Barreiro is responsible for the contents of this lecture, although he will adamantly blame Dr. Greco for any failures.
Objectives

• Review the pulmonary pathophysiology associated with obesity.

• Discuss what is the diagnostic criteria for obesity hypoventilation (OHS) and how to identify patients with OHS better.

• Discuss the acute care treatment errors in patients with OHS.

• Overview the treatments and modalities for obesity hypoventilation syndrome.
Fig 1. Joe the “Fat Boy” illustration by S. Etyinge, Jr.
(from Dickens C. The posthumous papers of the Pickwick Club. Boston: Ticknor and Fields: Boston; 1867.)

Prevalence of Obesity Among Adults: 1989

- < 10%
- 10-15%
- > 15%

Prevalence of Obesity Among Adults: 1998

< 10%  10-15%  > 15%

Prevalence of Obesity Among Adults

Weight Of The Nation: A Look At The Most (And Least) Obese States In America. Posted by publichealthwatch - September 23, 2015
Obesity

↓ Lung Volumes  ▲ Work of Breathing  ▲ CO₂ production  Leptin Resistance

Impaired respiratory muscle performance

↓ tidal volume

Airway Obstruction  ▲ PaCO₂  Hypoxia  Altered Neurotransmitters Chemoreceptors

Sleep Disordered Breathing  ▲ Bicarb

Sleep hypoventilation

Hypoventilation

Reduced ventilator arousals

Source: Sleep-Related Hypoventilation Syndromes, *Fishman’s Pulmonary Diseases and Disorders, 5e*

Citation: Grippi MA, Elias JA, Fishman JA, Kotloff RM, Pack AI, Senior RM, Siegel MD. *Fishman’s Pulmonary Diseases and Disorders, 5e; 2015*
Body Mass Index
Prevalence of OHS

- BMI 25.0-29.9 kg/m² Overweight
- BMI 30.0-34.9 kg/m² Grade I
- BMI 35.0-39.9 kg/m² Grade II (morbid)
- BMI >40 kg/m² Grade III (extreme)

## Respiratory System Mechanics in Health Subjects, Obese and OHS

<table>
<thead>
<tr>
<th>Variable</th>
<th>Units</th>
<th>Healthy</th>
<th>Obese</th>
<th>OHS</th>
<th>% Change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crs (Compliance)</td>
<td>L/cmH₂O</td>
<td>0.11</td>
<td>0.05</td>
<td>0.06</td>
<td>37% Δ</td>
</tr>
<tr>
<td>Rrs (Resistance)</td>
<td>cmH₂O/L/sec</td>
<td>1.2</td>
<td>4.0</td>
<td>7.8</td>
<td>44% Δ</td>
</tr>
<tr>
<td>P&lt;sub&gt;i&lt;/sub&gt;&lt;sub&gt;max&lt;/sub&gt;</td>
<td>cmH₂O</td>
<td>100</td>
<td>95</td>
<td>60</td>
<td>30% Δ</td>
</tr>
<tr>
<td>P&lt;sub&gt;e&lt;/sub&gt;&lt;sub&gt;max&lt;/sub&gt;</td>
<td>cmH₂O</td>
<td>150</td>
<td>125</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>MVV</td>
<td>L/min</td>
<td>159</td>
<td>129</td>
<td>89</td>
<td>55% Δ</td>
</tr>
</tbody>
</table>

Crs, respiratory system compliance; Res, respiratory system resistance.

P<sub>i</sub> max, maximal inspiratory pressure; P<sub>e</sub> max, maximal expiratory pressure; MVV, maximal voluntary ventilation.

Morbidity of OHS

- Groups with OHS carry combined diagnoses including:
  - congestive heart failure (odds ratio 9; 95% confidence interval 2.3 - 35),
  - angina (odds ratio 9; 95% confidence interval 1.4 - 57),
  - cor pulmonale (odds ratio 9; 95% confidence interval 1.4 - 57)

- No difference was found in the likelihood of having osteoarthritis, diabetes, hypertension, hypothyroidism.

- Same group found OHS more likely to be:
  - Hospitalized,
  - Greater admitted to the intensive care unit (40% versus 6%),

Schema depicting a proposed inadequate compensatory ventilatory response to CO₂ loading during obstructive respiratory events in the obesity hypoventilation syndrome. The dark-shaded areas depict CO₂ loading due to reduced CO₂ excretion during respiratory events while the light-shaded areas depict CO₂ unloading due to compensatory hyperventilation between respiratory events. (Reproduced with permission from Berger KI, Goldring RM, Rapoport DM. Obesity hypoventilation syndrome. Semin Respir Crit Care Med. 2009;30(3):253–261.)
A. Nocturnal oximetry in a patient with severe obstructive sleep apnea demonstrating recurrent desaturations in nonrapid eye movement (NREM) and rapid eye movement (REM) sleep. Each apneic event results in an oxyhemoglobin desaturation that improves as the apnea is terminated. The degree of desaturation is greater in REM sleep and resolves when continuous positive airway pressure (CPAP) of 5 cm H₂O is applied. B. In contrast to A, this nocturnal oximetry demonstrates prolonged desaturation in a patient with obesity hypoventilation syndrome (OHS). The desaturation corresponds to periods of prolonged hypoventilation. Although not diagnostic, this pattern is highly suggestive of OHS.
# The Role of Leptin in OHS

![Diagram of Adipose Tissue](image)

<table>
<thead>
<tr>
<th>Reference (year)</th>
<th>Main message</th>
<th>Main limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ip et al (2000)</td>
<td>Leptin significantly correlated with AHI</td>
<td>Only males/Limited number of patients/Potential influence by comorbidities/No adjustment for FM</td>
</tr>
<tr>
<td>Campo et al (2007)</td>
<td>Higher leptin is associated with reduced respiratory drive and reduced hypercapnic response</td>
<td>Conditions of blood sampling unknown/Potential influence by comorbidities</td>
</tr>
<tr>
<td>Philips et al (2000)</td>
<td>Increased leptin in OSAHS</td>
<td>Only males/Limited number of patients/Low statistical power</td>
</tr>
<tr>
<td>Barcelo et al (2005)</td>
<td>Decrease in leptin after nCPAP treatment in non-obese OSAHS</td>
<td>Only males/Limited number of patients/No adjustment for FM</td>
</tr>
<tr>
<td>Shimizu et al (2002)</td>
<td>Significant decrease in leptin after 1 day of nCPAP The decrease of leptin correlated with cardiac sympathetic function</td>
<td>Only males/Limited number of patients/Potential influence by comorbidities/No adjustment for FM</td>
</tr>
<tr>
<td>Phipps et al (2002)</td>
<td>Leptin is a predictor for the presence of hypercapnia</td>
<td>Limited number of patients/Sex unknown</td>
</tr>
</tbody>
</table>

**Abbreviations:** FM: Fat Mass

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Diagnosis of Obesity Hypoventilation

• Body mass index (BMI) > 30  kg/m$^2$
• Sleep disorder breathing
• Chronic daytime alveolar hypoventilation (PaCO$_2$ > 45 mmHg)
• Serum HCO$_3^-$ >27 mEq/l
• Rule out other causes of hypoventilation
  • Severe obstructive disease,
  • Chest wall deformities, severe hypothyroidism, neuromuscular diseases, central hypoventilation syndrome such as congenital hypoventilation syndrome or Arnold-Chiari type II malformations.

Why should we care about OHS

• Clinical Presentation
  • OHS is established >80% in hospital
  • In the 5\textsuperscript{th} or 6\textsuperscript{th} decade of life (mean 52 years (42 - 61))

• 3 most common presentations
  - Acute on chronic respiratory acidosis admitted to the ICU
  - Misdiagnosed acute on chronic decompensated heart failure that declines (↑\text{CO}_2) with diuresis
  - Misdiagnosed of acute on chronic exacerbation of chronic obstructive pulmonary disease

The average number of patient visits to the doctor for each group is plotted against each year of the study. The average number of physician visits for OHS patients was significantly higher.

The large arrow indicates the time of diagnosis and the institution of treatment.

Adverse Events during Hospitalization, Length of Stay, and Discharge Status among Patients with Simple Obesity or Obesity-Associated Hypoventilation (n=150)

Consecutive admissions to internal medicine services were screened over a 6-months hospital course and mortality at 18 months.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Simple Obesity (n = 103)</th>
<th>Obesity-Associated Hypoventilation (n = 47)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intensive care</td>
<td>27 (26%)</td>
<td>19 (40%)</td>
<td>0.08</td>
</tr>
<tr>
<td>Mechanical ventilation</td>
<td>0</td>
<td>3 (6)</td>
<td>0.01</td>
</tr>
<tr>
<td>Thromboembolism</td>
<td>3 (3%)</td>
<td>2 (4%)</td>
<td>0.67</td>
</tr>
<tr>
<td>Length of stay (days)</td>
<td>6 ± 9</td>
<td>8 ± 11</td>
<td>0.16</td>
</tr>
<tr>
<td>Discharge to long-term care facility</td>
<td>2 (2%)</td>
<td>9 (19%)</td>
<td>0.01</td>
</tr>
</tbody>
</table>

Survival curves for patients with obesity-associated hypoventilation or simple obesity after discharge from hospital, with adjustment for age, sex, body mass index, electrolyte abnormalities, renal insufficiency, history of thromboembolism, and history of hypothyroidism. (n=150)

At 18 months, mortality was 9%

Hazard ratio for mortality of 4.0 (95% confidence interval [CI]: 1.5 to 10.4).

Most of the deaths associated with obesity-associated hypoventilation occurred in the first 3 months following hospital discharge.

Only 13% (6/47) were discharged with a recommendation for long-term treatment for hypoventilation.
The Clinical Characteristics and Hospital and Post-hospital survival of patients with OHS

- Methods:
  - EMR of patient with unequivocal OHS admitted (index)
  - 5 years retrospective chart review
  - 600 enrolled
    - Mean Age 58 ± 15
    - Mean BMI 48.2± 8.3 kg/m2
    - ♂ 64%, ♀ 46%
    - DM Type II 37%
    - 43% misdiagnosed - COPD

Died 90 (15%) during index hosp.

Survivors follow for 3.2 ± 1.3 years

The clinical characteristics and hospital and post hospital survival of patient with OHS

Survivors follow for 3.2 ± 1.3 years

<table>
<thead>
<tr>
<th>Variable</th>
<th>All (n=600)</th>
<th>Survivors (n=510)</th>
<th>Died (n=90)</th>
<th>p values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>58.4 ± 15.2</td>
<td>57.2 ± 14.9</td>
<td>65.6 ± 14.7</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Cr (mg/dL)</td>
<td>1.59 ± 1.73</td>
<td>1.3 ± 1.5</td>
<td>2.8 ± 2.1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Respiratory failure</td>
<td>376 (63%)</td>
<td>300 (59%)</td>
<td>76 (84%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>12.4 (3.5%)</td>
<td>91 (18%)</td>
<td>33 (37%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>ICU admission (n, %)</td>
<td>370 (61%)</td>
<td>296 (58%)</td>
<td>74 (82%)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

In follow-up 98 of 510 (19%) died = Cumulative mortality of 31.3%

Airline Perks We'd Like to See...

In the event of severe snoring, a CPAP mask will drop from the compartment above...
## Predictors of hypercapnia in patients with OHS

<table>
<thead>
<tr>
<th>Variable</th>
<th>Odds Ratio (95% CI)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI &gt; 40</td>
<td>1.8 (0.9 – 3.5)</td>
<td>0.1</td>
</tr>
<tr>
<td>AHI &gt; 50</td>
<td>2.2 (1.1 – 4.4)</td>
<td>0.02</td>
</tr>
<tr>
<td>Oxygen desaturation nadir &lt; 60% during polysomnography</td>
<td>4 (2 - 8)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Moderate to severe restriction on pulmonary function testing</td>
<td>10 (5 - 24)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

*Predictors of hypercapnia in patients with OHS syndrome using a logistic regression model*
Serum bicarbonate level

Sleep Apnea
BMI ≥ 30 kg/m²

Serum HCO₃ ≤ 27 mEq/L
n = 257
3% with OHS

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

Lowest SpO₂ (<60%) during sleep
or AHI >100
n = 79
76% with OHS

Serum bicarbonate level

Sleep apnea referral
Obese > 30 kg/m²
n = 329

Serum HCO₃⁻ ≥ 27 mEq/L

Yes 170 (38%) with OHS

Respiratory restriction or Severe OSA

forced vital capacity of less than 60% of predicted

AHI > 30/hour

Both 84 (56%)

Either 65 (25%)

Neither 21 (10%)

No 159 (0%) with OHS

Sleep Apnea Testing/Diagnosis

• Dissatisfied with sleep then…

• Screening Tests
  ( All have low quality of evidence )
  • Epworth Sleepiness Scale
  • Berlin Questionnaire (PCP)
  • STOP-BANG (Pre-Op)
  • Sleep Quality Index

Screening tool for OSA: STOP-Bang

- Does the patient snore loudly (louder than talking or loud enough to be heard through closed doors)? Y/N
- Does the patient often feel tired, fatigued, or sleepy during the day? Y/N
- Has anyone observed the patient stop breathing during their sleep? Y/N
- Does the patient have, or is the patient being treated for, high blood pressure? Y/N
- Does the patient have a BMI of more than 35? Y/N
- Age. Is the patient older than 50? Y/N
- Is the patient’s neck circumference greater than 40cm? Y/N
- Gender. Is the patient male? Y/N

Scoring: 
Y ≥ 3 = high risk of OSA
Y < 3 = low risk of OSA

**Suggested algorithm for preoperative evaluation**

1. **Suspected OHS Pre Op Assessment**
   - Screen STOP-Bang questionnaire
   - Measure SpO2 & Serum HCO3 level

2. **STOP-Bang ≥ 3**
   - SpO2 < 90%
   - Serum HCO3 level ≥ 27 mEq
   - **Perioperative OHS Precautions**
     - Potential difficult airway
     - Opioid – induced ventilator impairment
     - Post-extubation PAP therapy

3. **STOP-Bang < 3**
   - SpO2 > 90%
   - Serum HCO3 level ≤ 27 mEq
   - **Sleep medicine consult**
     - Polysomnography for PAP therapy
     - Assessment of PaCO2, RV function & PAH.

4. **Routine management**

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Common pitfalls of management in obesity hypoventilation syndrome.

- Excessive administration of oxygen
  - ↑ CO₂ production, reduced minute ventilation

- Excessive diuresis contraction alkalosis
  - ↑ serum bicarbonate & alkalemia

- Excessive psychotropic/sedatives/opiates
  - ↑ upper airway collapsibility

Pitfalls in management lead to:
- Blunted ventilator response
- Worsening hypercapnia

The Effect of Supplemental Oxygen on Hypercapnia in Subjects With Obesity-Associated Hypoventilation

Double blinding, randomized, controlled, crossover trail  
\( n = 24 \) outpatients with OHS  
100% oxygen for 20 mins on 2 separate days

Pt\( \text{CO}_2 \) increased by 5.0 mm Hg (95% CI, 3.1–6.8; \( P < .001 \))

Minute ventilation decreased by 1.4 L/min  
(95% CI, 0.11–2.6 L/min; \( P = .03 \))

Dead space to tidal volume ratio increased by 0.067 (95% CI, 0.035–0.10; \( P < .001 \)) with oxygen compared with room air

Treatment options for OHS

- Treatment has traditionally followed three modalities:
  - Reversal of sleep disorder breathing.
    - CPAP / NIV
  - Weigh reduction.
    - lifestyle modification,
    - bariatric surgery
  - Pharmacotherapy.
    - Weight loss medications,
    - Respiratory stimulants

Treatment options for OHS

• Reversal of sleep disorder breathing.
• PAP therapy was first described in 1982
• CPAP verses NIV
  • Prospective study of outpatients with severe OHS, based on the severity of obesity and OSA and the degree of hypercapnia,
  • 57% of patients were titrated successfully with 13.9 cmH$_2$0.

Efficacy of Different Treatment Alternatives for Obesity Hypoventilation Syndrome. Pickwick Study

- Lifestyle modification: • 1,000 calorie diet; • maintenance of sleep hygiene, (avoid supine sleep, maintain a regular sleep time and exercise); • no use of sedative, stimulants, or alcohol; • no smoking; • avoiding heavy meals within 4 hours of sleep.

Control
n = 70

CPAP
n = 80

NIV
n = 71

Lifestyle modification: + • Fixed PAP during sleep period > 4 hours

Data collected Start, 1 month, 2 month follow up

Parameters collected: +
Primary Outcome: = PaCO\(_2\)
Secondary Outcomes:
• Anthropometric data;
• Clinical symptoms;
• Medical Research Counsel dyspnea scale,
• Epworth sleepiness score,
• Health related quality of life testing,
• Short form 36 (SF-36),
• Visual analogical well being score,
• PSG,
• Spirometry for lung function;
• 6 minute walk test.

OHS
n = 221

Spanish study
4 years, 15 – 80 years of age, 16 centers,
Strict OHS enrollment criteria

Efficacy of Different Treatment Alternatives for Obesity Hypoventilation Syndrome. Pickwick Study

Intergroup PaCO\textsubscript{2} changes (means and 95% confidential intervals), adjusted according to basic adjustments (PaCO\textsubscript{2}, age, sex, body mass index, and apnea–hypopnea baseline values), weight change, and continuous positive airway pressure (CPAP)/noninvasive ventilation (NIV) use (more or less than 4 h/night). NS = not significant.

Spanish study
4 years, 15 – 80 years of age, 16 centers,
Strict OHS enrollment criteria

PaCO$_2$ (Δ = −3.5 mm Hg; 95% CI, −6.2 to −0.8)

HCO$_3^−$ (Δ = −2.0 mmol/L; 95% CI, −0.44 to −3.65)

**P < .001.

Effectiveness of NIV in OHS compared with control therapy.

OHS
n = 37

Randomized

Group A
lifestyle
n = 18

Group B
NIV
n = 19

1 month

ABGs
ESS

1 month

Metabolic parameter
(hsCRP, leptin, RANTES, Interleukins)

PART I

- OHS not tolerate to CPAP
- CPAP = 8.9 ± 1.0

PART II

- 10 patients (mean age, 53.5 ± 11.7 years; mean BMI, 41.6 ± 12.1 kg/m²; mean FEV1/FVC ratio, 79.4 ± 6.5%; mean transcutaneous [PtcCO2], 58 ± 12 mm Hg)

VENTILATION PATTERN, GAS EXCHANGE, SLEEP QUALITY, HEALTH-RELATED QUALITY OF LIFE (HRQL) SEVERE RESPIRATORY INSUFFICIENCY QUESTIONNAIRE (SRI)

In OHS patients who did not respond to therapy with continuous positive airway pressure, the effects of BPV with the spontaneous/timed (S/T) ventilation mode with and without AVAPS over 6 weeks

Effect of liraglutide 3.0mg in individuals with obesity and severe OSA: The SCALE Sleep Apnea randomized clinical trial

Change in AHI and body weight over 32 weeks of treatment and the relationship between weight loss and change in AHI.

Data are mean ±s.e. Weight category represents the change in body weight (%) from baseline after 32 weeks of treatment. PSG assessments were performed at weeks (baseline), 12 and 32.

From: A Randomized, Double-Blind, Placebo-Controlled Study of an Oral, Extended-Release Formulation of Phentermine/Topiramate for the Treatment of Obstructive Sleep Apnea in Obese Adults

Pharmacotherapy for OHS

• Few drugs known for their respiratory stimulant effects:
  • progesterone,
  • acetazolamide,
  • Almitrine,
  • Aminophylline (never studies in OHS)

• All have been tried in patients with sleep apnea syndromes; however, the two most widely quoted drugs when dealing with OHS patients are medroxyprogesterone and acetazolamide.
Progesterone therapy


<table>
<thead>
<tr>
<th></th>
<th>N = 15</th>
<th>Mean</th>
<th>SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td></td>
<td>67.5</td>
<td>6.0</td>
<td>56 – 76 years</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td></td>
<td>26.9</td>
<td>4.9</td>
<td>15.4 – 35.2</td>
</tr>
<tr>
<td>Smoking (pack-year)</td>
<td></td>
<td>8.8</td>
<td>13.9</td>
<td>0 – 50</td>
</tr>
<tr>
<td>FEV₁ (L)</td>
<td></td>
<td>0.76</td>
<td>0.3</td>
<td>0.44 – 1.80</td>
</tr>
<tr>
<td>FEV₁ (%)</td>
<td></td>
<td>34%</td>
<td>12.4</td>
<td>15 – 63</td>
</tr>
<tr>
<td>Arterial pH</td>
<td></td>
<td>7.38</td>
<td>0.05</td>
<td>7.25 – 7.45</td>
</tr>
<tr>
<td>PaCO₂ (Torr)</td>
<td></td>
<td>45</td>
<td>8.2</td>
<td>41.2 – 74.2</td>
</tr>
<tr>
<td>PaO₂ (Torr)</td>
<td></td>
<td>67</td>
<td>9.0</td>
<td>42 – 94.5</td>
</tr>
</tbody>
</table>

Randomized

Group A
Post-menopausal ♂ n = 7

Group B
Post-menopausal ♂ n = 7

MPA 60 mg day 2 weeks
Placebo 2 weeks
MPA 60 mg day 2 weeks
Placebo 2 weeks
washout 6 weeks

Cross over
Tx + 2 weeks
Changes in fasting serum leptin concentration versus changes in the carbon dioxide pressure in arterial blood (Pa,CO₂) in postmenopausal females with moderate-to-severe chronic respiratory impairment showing a positive correlation (r=0.60, p=0.031).

Ten patients with the Pickwickian syndrome, MPA 20 mg Q8 sublingual.

**Figure 1.** Improvement in arterial blood gases in 10 patients after institution of medroxyprogesterone acetate therapy. $\text{PaO}_2$ reached $62 \pm 2.3$ mm Hg (Denver normal = $69 \pm 1.2$ mm Hg). $\text{PaCO}_2$ showed a complementary decrease to $38 \pm 1.2$ Hg (Denver normal = $36 \pm 0.6$ mm Hg). These improvements were sustained throughout treatment. Bars represent standard error of the mean.

**Figure 3.** During 1 month withdrawal of medroxyprogesterone acetate therapy, significant decreases in $\text{PaO}_2$ and increases in $\text{PaCO}_2$ were observed. These values reverted to previous treatment levels 1 month after reinstitution of therapy. Bars represent standard error of the mean.
Acetazolamide therapy

Health Subjects
n = 9
Randomized

Acetazolamide 250 Q8
n = 9
Placebo
n = 9

3 days
2 weeks
3 days

Examples of individual DEF runs in one subject after placebo and acetazolamide intake, respectively. Upper traces are end-tidal PCO₂. Breath-by-breath ventilatory data are represented by small open circles. The solid line through these actual ventilatory data is the model output. V'c and V'p are the calculated contributions of the central and peripheral chemoreflex loops, respectively. CO₂ sensitivities of both chemoreflex loops are similar after placebo and acetazolamide. In both the placebo and acetazolamide condition, 2 to 3 DEF runs were performed in all subjects.

Hypercapnic respiratory failure in obesity-hypoventilation syndrome: CO₂ response and acetazolamide treatment effects

• 25 obesity-hypoventilation-syndrome patients mechanically ventilated for hypercapnic respiratory failure ready for weaning.

• Weaning = SBT F₁O₂ ≤ 0.5; PEEP ≤ 8 cmH₂O; and Pa/F₁O₂ > 150 mmHg.

• ABG prior to Mechanical Ventilation:
  • pH 7.18 ± 0.08
  • CO₂ 102 ± 25 mmHg
  • SAPS II score 35 ± 10 (mortality = 2.9% - 8.9%)
  • Ventilator days 6.7 ± 5.3 d (mean 5 d)

• Acetazolamide was NG for 4 days dose 500 - 2,750 mg.

Hypercapnic respiratory failure in obesity-hypoventilation syndrome: CO₂ response and acetazolamide treatment effects.

HCO₃ reduced by 8.4 ± 3.0 mmol/l (p = 0.01)

CO₂ drive response increase by 0.14 ± 0.16 cmH₂O/mmHg (p =0.02)

CO₂ response was 5.9 ± 6.9

Ventilator days 6.7 ± 5.3 d (mean 5 d) verses 3.3 ± 2.1 d (mean d 2)

Death = 3 patients (no explanation)

Airway-occlusion pressure 0.1 s after the start of inspiratory flow (P₀.₁) versus PₐCO₂, and minute volume (Vₑ) versus PₐCO₂ increase in 8 patients, before (solid lines) and after (dashed lines) acetazolamide treatment.

Conclusions

• In summary, obesity is a major health problem and has detrimental effects on the health-care systems at many levels.

• OHS is a far more serious problem, frequently missed and improperly treated.

• Better recognition of OHS may lead to better outcomes.

• Currently, the best available options for treating OHS patients are weight reduction and non invasive ventilation > positive airway pressure ventilation.