Heliox Therapy in Acute Severe Asthma*

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**Study objective:** To assess how patients with respiratory acidosis from acute severe asthma respond to helium-oxygen (heliox) mixtures.

**Design:** Consecutive case series.

**Setting:** Urban community teaching hospital.

**Patients:** Over a 2-year period, 12 asthmatics (mean age, 33.8 ± 11.3 years) presented to the emergency department with acute respiratory acidosis (pH < 7.35 and PaCO₂ ≥ 45 mm Hg). All 12 patients were treated with heliox (60 to 70%, helium/30 to 40% oxygen). Five patients received heliox through a ventilator and seven received heliox via face mask.

**Results:** Arterial blood gases (ABGs) were drawn immediately before and at a mean of 49.2 ± 25.2 min after beginning heliox therapy. No therapeutic interventions were made between ABGs. For the entire group, the mean PaCO₂ decreased from 57.9 to 47.5 mm Hg (p < 0.005) and the arterial pH increased from 7.23 to 7.32 (p < 0.001). In an attempt to find characteristics that might predict the response to heliox, a clinically significant response to heliox was defined as a drop in PaCO₂ (to normal or by ≥15%) coupled with a rise in pH by ≥ 0.05. Using this definition, there were eight responders (67%) and four nonresponders (33%). The responders had a shorter duration of symptoms (17.8 vs 78.0 h, p < 0.05) and a lower preheliox pH (7.20 vs 7.30, p < 0.05). All of the responders presented within 24 h of symptom onset. Three of the four nonresponders reported prolonged (≥96 h) duration of symptoms, and two eventually required intubation.

**Conclusion:** Heliox can rapidly improve ventilation in patients presenting to an emergency department with acute severe asthma with respiratory acidosis and a short duration of symptoms.  

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**Key words:** asthma; heliox; helium; respiratory acidosis; status asthmaticus

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**ABGs=arterial blood gases; heliox=helium-oxygen**

**Methods**

Over a 2-year period (1988 to 1990) there were 312 patients, 179 women and 133 men, admitted to Mount Sinai Hospital in Hartford Conn, with acute asthma. Twelve (3.8%) of these patients had an acute respiratory acidosis as defined by a pH of < 7.35 and a PaCO₂ ≥ 45 mm Hg. All 12 were treated with a heliox mixture containing 60 to 70% helium and 30 to 40% oxygen. There were nine men and three women. The mean age was 33.8 ± 11.3 years. They had asthma for 23.4 ± 9.6 years. Four of the patients had been intubated in the past. Eleven of the 12 patients were receiving maintenance β₂-agonist metered-dose inhaler therapy. Eight of the 12 were taking oral theophylline. One was using ipratropium bromide by metered-dose inhaler. None of the patients was being treated with inhaled or oral corticosteroids. All 12 were either lifetime nonsmokers or had a smoking history of less than half pack per day for < 5 years. None had smoked at all within a year of hospital admission.

Prior to the institution of heliox therapy, all patients received three to five treatments of nebulized albuterol, 2.5 mg per treat-
ment, and 125 mg of methylprednisolone given intravenously. Nine patients also received 0.25 to 0.5 mg of subcutaneous terbutaline. Eight patients were treated with intravenous aminophylline at rates of 0.3 to 0.6 mg/kg/h starting 160±106 min prior to the administration of heliox.

Five of the patients were intubated and mechanically ventilated shortly (24.0±26.4 min) after admission to the emergency department. These patients received heliox through a ventilator (Bear 1, Bear Medical Systems Incorporated; Riverside, Calif) by connecting its air intake to a helium tank and blending this with oxygen. Because ventilator blenders are calibrated for oxygen/air, while it is being used for helium/oxygen, a separate oxygen analyzer (VTI Oxygen Analyzer/Monitor, Vascular Technology Incorporated; Chelmsford, Mass) was used to determine $F_{\text{IO}_2}$. The remaining seven patients were given heliox via a nonrebreathing face mask by blending 100% oxygen with 100% helium through a blender (Bird Oxygen Blender, Bird Corporation; Palm Springs, Calif) with a monitor (VTI Oxygen Analyzer/Monitor). Heliox was started a mean of 165.0±106.5 min after admission to the emergency department. The patients received heliox for a total of 16.8±6.3 h.

Arterial blood gases (ABGs) were drawn immediately before and at a mean of 49.2±25.2 min after beginning the heliox. No $\beta_2$-agonist or other new medication was given during the interval between baseline and heliox ABGs. For the patients receiving mechanical ventilation, no settings were changed over that interval.

Statistics

Two-tailed Student’s $t$ tests were used to analyze continuous variables between groups, while a Fisher’s exact test was used to analyze noncontinuous variables.

RESULTS

For the entire group ($n=12$), institution of heliox was associated with a drop in PaCO$_2$ from 57.9±8.3 mm Hg to 47.5±4.3 mm Hg ($p<0.005$) (Fig 1) and

![Figure 1. The effect of heliox on PaCO$_2$. Patients with a <24-h duration of symptoms (open boxes) are contrasted with patients who had symptoms for >96 h (solid boxes).](image1)

![Figure 2. The effect of heliox on pH. Patients with a <24-h duration of symptoms (open boxes) are contrasted with patients who had symptoms for >96 h (solid boxes).](image2)
a rise in arterial pH from 7.23 ± 0.07 to 7.32 ± 0.04 (p<0.005) (Fig 2). There was no significant change in HCO₃⁻ (24.1 mmol/L ± 2.7 to 23.8 mmol/L ± 1.8, p=0.674), or alveolar to arterial oxygen ratio (3.27 ± 1.98 to 3.61 ± 1.07, p=0.554).

A clinically significant response to heliox was defined prior to data analysis as either normalization of PaCO₂ (defined as a PaCO₂ <45 mm Hg) or a ≥15% drop in PaCO₂, either of which had to be coupled with an increase in pH of ≥0.05. By this definition, there were eight responders, seven of whom had a ≥15% decrease in PaCO₂, and one of whom had a normalization of the PaCO₂ to 41 mm Hg. There were four nonresponders. None of the nonresponders had more than a 10% drop in PaCO₂ and none lowered the PaCO₂ to <45 mm Hg.

Characteristics of the responders and the nonresponders were examined. The responders had a significantly lower preheliox pH (7.20 ± 0.06 vs 7.30 ± 0.03, p<0.05) and a significantly shorter duration of symptoms at time of presentation (17.8 ± 8.9 h vs 78 ± 36 h, p<0.05) (Table 1). The small sample size prevented logistic regression analysis to determine whether initial pH or duration of symptoms was a more important predictor of a response to heliox. All responders (eight of eight) presented with an acute (<24 h) exacerbation of symptoms. Three of the four nonresponders presented ≥96 h after onset of symptoms (Figs 1 and 2). There were no significant differences (p>0.1) between the groups in the number of nebulized albuterol treatments or the number of patients who had received subcutaneous terbutaline, maintenance oral theophylline, or intravenous aminophylline. Although length of stay in the intensive care unit was not significantly different between the two groups (responders, 1.3 ± 1.1 days, vs nonresponders, 3.1 ± 3.3 days; p=0.359), the responders had a significantly shorter hospital length of stay (3.8 ± 1.9 days vs 7.3 ± 3.2 days, p<0.05).

Three of the seven patients who started heliox via face mask had symptoms for >96 h, and two of them ended up being intubated. The other four had symptoms for <24 h, and none required intubation. In the present study, the conditions of all of the patients were sufficiently improved after 24 h for heliox therapy to be discontinued without clinical deterioration.

### DISCUSSION

This study showed that heliox can cause an acute improvement in respiratory acidosis in patients with severe asthma. The rapidity of improvement with heliox inhalation strongly suggests a direct beneficial relationship, although the lack of a control group raises the possibility that this improvement was due to the delayed effects of previously administered therapies. Patients with a shorter duration of symptoms were much more likely to respond to heliox therapy. In this subset of patients, heliox improves respiratory acidosis by decreasing airway resistance and consequently the work of breathing. In those who respond to helium given via mask, intubation and mechanical ventilation may be prevented. Heliox thus serves as a therapeutic bridge for the 6- to 12-h interval from patient arrival in the emergency department until corticosteroid impact. Conversely, failure of asthmatics with hypercapnia and respiratory acidosis to respond to heliox therapy given via mask might herald a failure to respond to aggressive treatment and thus indicate an increased risk for intubation and mechanical ventilation.

Asthmatics with hypercapnia and respiratory acidosis unresponsive to medical therapy may require mechanical ventilation. Although the initiation of mechanical ventilation in patients with intractable severe asthma may save lives, it is associated with increased morbidity. Intubation and mechanical ventilation may actually increase the risk of death in some patients with severe asthma. Heliox may sometimes eliminate the need for intubation and mechanical ventilation, obviating this dilemma. Heliox benefits the intubated asthmatic, as its use allows adequate ventilation with reduced pressures, thereby decreasing the risk of barotrauma.

In conclusion, heliox rapidly improves ventilation in patients presenting to an emergency department with acute severe asthma with respiratory acidosis and a short duration of symptoms.

### REFERENCES

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**Table 1— Differences Between Heliox Responders and Nonresponders**

<table>
<thead>
<tr>
<th>Group (n)</th>
<th>No. of Hours of Symptoms</th>
<th>Preheliox PaCO₂</th>
<th>Heliox PaCO₂</th>
<th>Preheliox pH</th>
<th>Heliox pH</th>
<th>Preheliox PAO₂/PAO₂</th>
<th>Heliox PAO₂/PAO₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>Responders (8)</td>
<td>17.8 ± 8.9</td>
<td>60.9 ± 8.9</td>
<td>46.9 ± 5.2</td>
<td>7.20 ± 0.06</td>
<td>7.32 ± 0.05</td>
<td>3.24 ± 2.12</td>
<td>3.63 ± 1.15</td>
</tr>
<tr>
<td>Nonresponders (4)</td>
<td>78.0 ± 36.0</td>
<td>52.0 ± 5.0</td>
<td>48.8 ± 3.0</td>
<td>7.30 ± 0.03</td>
<td>7.32 ± 0.04</td>
<td>3.35 ± 0.52</td>
<td>3.57 ± 1.77</td>
</tr>
<tr>
<td>p</td>
<td>&lt;0.05</td>
<td>0.095</td>
<td>0.526</td>
<td>&lt;0.05</td>
<td>0.919</td>
<td>0.885</td>
<td>0.946</td>
</tr>
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