Extrathoracic Obstruction and Hypoxemia Occurring During Exercise in a Competitive Female Cyclist*

Hans Haverkamp, MS; Jordan Miller, MS; Joshua Rodman, PhD; Lee Rower, PhD; David Pegelow, BS; Marcus Santana, MD; and Jerome Dempsey, PhD

A 22-year-old competitive female cyclist complained of cough, chest tightness, and wheeze during high-intensity exercise that had previously been diagnosed as exercise-induced bronchospasm (EIB). A loud stridor, a sensation of her “throat closing,” and severe dyspnea developed during maximal cycling exercise with concomitant reductions in both inspiratory and expiratory flow rates. A decrease of 25 L/min (26%) in minute ventilation and arterial hypoxemia (Pao2 decrease, 93 to 76.5 mm Hg) resulted from this obstruction. Spontaneous tidal flow-volume loops (FVLs) during exercise exhibited a sawtooth pattern during inspiration, and substantial drops in flow rates after the stridor developed. However, maximal FVLs were unchanged from baseline following exercise, indicating that the obstruction was not EIB. We suggest that the continuous measurement of spontaneous breath-by-breath tidal FVLs may be a useful diagnostic tool for the identification of exercise-induced extrathoracic obstruction. Additionally, extrathoracic obstruction should be considered as an uncommon but potential cause of inadequate ventilation and arterial hypoxemia during exercise.

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Key words: exercise-induced arterial hypoxemia; exercise-induced bronchospasm; stridor; upper airway dysfunction

Abbreviations: EIAH = exercise-induced arterial hypoxemia; EIB = exercise-induced bronchospasm; FVL = flow-volume loop; V02 = oxygen uptake

*From the Department of Population Health Sciences, John Rankin Laboratory of Pulmonary Medicine, University of Wisconsin-Madison, Madison, WI.

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Correspondence to: Hans C. Haverkamp, MS, 504 N Walnut St, Madison, WI 53726; e-mail: hchaverkamp@students.wisc.edu

Airway resistance during exercise is normally maintained at or below resting levels because the intrathoracic airways undergo bronchodilation and the abductor muscles of the upper airway provide maximum glottal aperture during both inspiration and expiration. Several reports have documented the presence of an extrathoracic obstruction following the termination of an exercise bout that previously had been misdiagnosed as exercise-induced bronchospasm (EIB). Indeed, EIB is known to occur after exercise cessation, although a small amount of bronchospasm has been shown to develop during the exercise period. In this case report, spontaneous tidal flow-volume loops (FVLs) and arterial blood gas levels during exercise are reported for a female cyclist who developed an apparent extrathoracic obstruction that had been misdiagnosed as EIB.

Case Report

A 22-year-old competitive female cyclist attended the laboratory in association with an asthma study. Her major complaints were wheeze, severe dyspnea, and cough during heavy exercise. She had been diagnosed with asthma, EIB, and allergic rhinitis, and was atopic to several common allergens, as indicated by the results of skin prick testing. An induced sputum analysis revealed a high macrophage count with no airway eosinophilia or neutrophilia. At the time of study, the patient was using a nasal corticosteroid spray (fluticasone propionate; GlaxoSmithKline: Research Triangle Park, NC), a combination bronchodilator/corticosteroid inhalation powder (Advair Diskus; GlaxoSmithKline) daily, and a fast-acting β-agonist when she felt it necessary. Her baseline FEV1/FVC ratio was 78% and was unchanged following administration of a fast-acting bronchodilator, suggesting the presence of a mild, but fixed, obstruction. The shape of the inspiratory and expiratory limbs of the maximal volitional flow-volume envelope performed at rest were normal (ie, smooth with no truncation of flow-rates).

Cycling exercise was performed on a magnetically braked cycle ergometer after a catheter was placed in the radial artery for the collection of arterial blood. After a brief warmup (3 min each at 50% and 75% of peak oxygen uptake [V02]), the patient began exercising at a fixed workload that previously had been determined to elicit a V02 of approximately 90% peak V02. Exercise was performed until exhaustion (total time, 13 min). Thirty minutes later, and after another brief warmup (3 min at 90% V02 peak), exercise was recommenced at her previously determined maximal workload (ie, 100% peak V02) and was performed to exhaustion (total time, 1.25 min). Pulmonary function tests were performed at 5, 10, and 20 min after both exercise bouts.

Spontaneous tidal exercise FVLs obtained during exercise at 50%, 75%, and 90% of peak V02 are shown in Figure 1, left, A, and during maximal exercise in Figure 1, right, B. The inspiratory limbs exhibited a sawtooth pattern, which became more pronounced as
intensity increased (Fig 1, left, A). During maximal exercise (Fig 1, right, B), a loud stridor and severe dyspnea appeared at the same time that both inspiratory and expiratory flow rates were clearly reduced when compared with those values obtained only 30 s earlier at the initiation of the maximal workload. A breath-by-breath analysis of breathing mechanics during the maximal workload (Fig 2) revealed sudden and significant decreases in peak inspiratory and expiratory flows, breathing frequency, and minute ventilation at the same time the symptoms appeared. A significant CO₂ retention and moderate arterial hypoxemia occurred immediately following the onset of symptoms (Table 1). On exercise termination, the dyspnea and stridor disappeared almost immediately. As summarized in Table 2, expiratory flow rates at both high lung volumes (i.e., FEV₁) and lower lung volumes (midexpiratory range of forced expiratory flow) were not different from baseline values at all time points postexercise.

**Discussion**

The major finding in this case was a significant restriction of inspiratory and expiratory tidal flow rates during maximal intensity exercise due to an apparent extrathoracic upper airway obstruction, causing extreme dyspnea, alveolar hypoventilation, and arterial hypoxemia in an otherwise healthy endurance athlete. Our findings support those of previous investigations that prolapse of supraglottic structures into the airway³,⁴ or paradoxical inward movement of the true or false vocal cords⁶ during exercise can be misdiagnosed and treated as EIB.

An important finding in this study was that by using tidal FVLs the increase in pulmonary resistance was shown to appear during exercise while the subject was maintaining power output. Previously, maximal volitional FVLs have been used to document extrathoracic obstruction after subjects had stopped exercising.³,⁶ The first signs of obstruction appeared during submaximal exercise, at which time the inspiratory limb of the tidal FVL exhibited a sawtooth pattern, and then more obviously with a sudden reduction in flow rate on the inspiratory and expiratory limb of the tidal FVL during maximal exercise, coincident with the onset of loud stridor and severe dyspnea. In contrast to these changes during exercise, maximum volitional FVLs obtained at 5, 10, and 20 min during the recovery period showed no abnormalities. This is important in light of the fact that a reduction in postexercise maximal FVLs is the “gold standard” for the diagnosis of EIB. Thus, we think that our data support the use of continuous breath-by-breath measurement of tidal FVLs during exercise as a useful diagnostic tool for the identification of exercise-induced upper airway dysfunction. These breath-by-breath measurements also have been shown to be valuable in identifying the onset and progression of expiratory flow.

**Table 1—Results of Blood Gas Measurements for the Submaximal and Maximal Exercise Workloads⁴**

<table>
<thead>
<tr>
<th>Exercise Intensity</th>
<th>Rest</th>
<th>50%</th>
<th>75%</th>
<th>90%</th>
<th>VO₂ Peak</th>
</tr>
</thead>
<tbody>
<tr>
<td>PAO₂, mm Hg</td>
<td>102.9</td>
<td>103.5</td>
<td>102.1</td>
<td>108.4</td>
<td>99.8</td>
</tr>
<tr>
<td>Pao₂, mm Hg</td>
<td>100.0</td>
<td>93.1</td>
<td>87.0</td>
<td>90.2</td>
<td>76.5</td>
</tr>
<tr>
<td>P(A-a)O₂, mm Hg</td>
<td>3.0</td>
<td>10.4</td>
<td>15.1</td>
<td>18.2</td>
<td>23.3</td>
</tr>
<tr>
<td>PaCO₂, mm Hg</td>
<td>39.1</td>
<td>37.2</td>
<td>40.9</td>
<td>34.6</td>
<td>44.4</td>
</tr>
<tr>
<td>pH</td>
<td>7.44</td>
<td>7.45</td>
<td>7.43</td>
<td>7.36</td>
<td>7.30</td>
</tr>
<tr>
<td>SaO₂, %</td>
<td>98.3</td>
<td>97.8</td>
<td>97.2</td>
<td>95.8</td>
<td>92.5</td>
</tr>
</tbody>
</table>

*PAO₂ = alveolar partial pressure of oxygen; P(A-a)O₂ = alveolar-arterial oxygen pressure difference.*
limitation, and its effects on dynamic hyperinflation and dyspnea during exercise in healthy individuals and in patients with COPD and chronic heart failure.7–9 Our findings also demonstrate for the first time that CO\textsubscript{2} retention and arterial hypoxemia occur commensurate with the onset of an increased upper airway resistance during heavy exercise. Exercise-induced arterial hypoxemia (EIAH) has been observed in a subpopulation of otherwise healthy,
habitually active endurance athletes. It normally occurs immediately at the onset of heavy exercise and persists throughout the exercise session. EIAH is attributed primarily to a widened alveolar-to-arterial \( \text{PaO}_2 \) difference in combination with a limited hyperventilatory response, which is due, at least in part, to expiratory flow limitation.\(^\text{10}\) This case report suggests that upper airway dysfunction also should be considered as a potential cause of ventilatory constraint contributing to EIAH.

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REFERENCES


High Dose Rate Brachytherapy for Nonmalignant Airway Obstruction*

New Treatment Option

Baruch Brenner, MD; Mordechai R. Kramer, MD, FCCP; Alan Katz, MD; Rafael Feinmesser, MD; Alina Brenner-Weissmann, MD; Aaron Sulkes, MD; and Eyal Fenig, MD

Study objectives: High dose rate (HDR) endobronchial brachytherapy is widely used as a palliative treatment for symptomatic airway obstruction by primary or secondary malignant tumors. We report on a successful use of HDR brachytherapy in patients with nonmalignant airway obstruction.

Design: Case series.

Patients: Six patients received HDR brachytherapy for airway obstruction caused by granulation tissue around a metal stent placed for restoration of the airway patency for nonmalignant causes. In four patients, brachytherapy was performed following recurrent occlusion of the airway by granulation tissue formation; in two patients, it was done as a prophylactic procedure.

Intervention: HDR brachytherapy catheters were passed through the metal stents under direct fluoroscopic guidance. Simulation and computerized treatment planning were done, and a single dose of 10 Gy was administered using a brachytherapy remote afterloader with a \(^{192}\)Ir source. The dose was prescribed to a distance of 1 cm from the center of the source, with a margin of 1 cm from the proximal and distal ends of the stent.

Results: At a median follow-up of 15 months, moderate granulation tissue formation was observed in only one patient; in four others, it was categorized as minimal, 5 to 30 months from the procedure. Restoration of the lumen was complete in four patients, near complete in one patient, and partial in one patient. In one patient, previously treated by external radiotherapy, local tissue necrosis was evident.

Conclusion: HDR brachytherapy can be used safely for nonmalignant airway obstruction. Further studies including more patients and longer follow-up are needed.

Key words: airway stenting; high dose rate brachytherapy; nonmalignant airway obstruction; radiation therapy

Abbreviation: HDR = high dose rate

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Table 2—Pulmonary Function Measured at Baseline, 5 min, 10 min, and 20 min after the 90% and 100% Peak \( \text{Vo}_2 \) Exercise Bouts*

<table>
<thead>
<tr>
<th>Variables</th>
<th>Baseline</th>
<th>5-min</th>
<th>10-min</th>
<th>20-min</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \text{FVC, L} )</td>
<td>4.52</td>
<td>4.42</td>
<td>4.5</td>
<td>4.47</td>
</tr>
<tr>
<td>90% peak ( \text{Vo}_2 )</td>
<td>(116%)</td>
<td>4.33</td>
<td></td>
<td>4.31</td>
</tr>
<tr>
<td>( \text{PEF, L/s} )</td>
<td>7.57</td>
<td>7.59</td>
<td>7.33</td>
<td>7.68</td>
</tr>
<tr>
<td>90% peak ( \text{Vo}_2 )</td>
<td>(103%)</td>
<td>7.59</td>
<td></td>
<td>7.24</td>
</tr>
<tr>
<td>( \text{FEV}_1, \text{L/s} )</td>
<td>3.54</td>
<td>3.64</td>
<td>3.59</td>
<td>3.6</td>
</tr>
<tr>
<td>90% peak ( \text{Vo}_2 )</td>
<td>(104%)</td>
<td>3.57</td>
<td></td>
<td>3.45</td>
</tr>
<tr>
<td>( \text{FEF}_{25-75}, \text{L/s} )</td>
<td>2.86</td>
<td>3.13</td>
<td>2.98</td>
<td>3.07</td>
</tr>
<tr>
<td>90% peak ( \text{Vo}_2 )</td>
<td>(72%)</td>
<td>3.17</td>
<td></td>
<td>2.96</td>
</tr>
<tr>
<td>100% peak ( \text{Vo}_2 )</td>
<td>(103%)</td>
<td>4.32</td>
<td></td>
<td>4.31</td>
</tr>
</tbody>
</table>

*PEF = peak expiratory flow; FEF\(_{25-75}\) = average flow over the middle 50% of FVC.