Heliox Therapy for Acute Vocal Cord Dysfunction

To the Editor:

At the National Jewish Center for Immunology and Respiratory Medicine, approximately 10% of the patients referred for inpatient evaluation for refractory asthma have vocal cord dysfunction without any evidence of asthma. An additional 30% are diagnosed as having both vocal cord dysfunction and asthma. Based on this data, we would have expected a significant number of patients treated by Kass and Castriotta, as described in their article published in the March 1995 issue (CHEST 1995; 107:757-60), to have had vocal cord dysfunction. While potentially of some value in the treatment of asthma, heliox is particularly efficacious in the treatment of an acute episode of vocal cord disease, which frequently masquerades as severe asthma unresponsive to standard therapy. It is thus surprising that this disease entity was not aggressively looked for by Kass and Castriotta. It is equally surprising that it was not mentioned in the follow-up editorial by Madison and Irwin (CHEST 1995; 107:597-98).

We suggest the patients presented in the study by Kass and Castriotta undergo rhinolaryngoscopy when symptomatic or that this test be performed when asymptomatic in conjunction with a challenge test looking for paradoxical motion of the vocal cords.

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REFERENCE


To the Editor:

Drs. Reisner and Borish raise the interesting possibility that some of the patients we treated with heliox might have had acute vocal cord dysfunction. They quote the experience at the National Jewish Center for Immunology and Respiratory Medicine where there is an approximate 40% incidence of vocal cord dysfunction in patients referred for refractory asthma.

Seven of our 12 patients underwent endotracheal intubation. If they had had vocal cord dysfunction, their “asthma” should have disappeared with the endotracheal intubation. In our study severe airways obstruction continued to be a problem after intubation. All of our patients also had acute hypercapnia, which has not shown to be associated with vocal cord dysfunction during an episode of wheezing and dyspnea. Although it does not preclude vocal cord dysfunction, none of our patients had stridor or predominantly anterior inspiratory wheezing, which are common physical findings in vocal cord dysfunction.

Our patients were drawn from an urban population for whom the hospital was the traditional primary care giver. The incidence of acute vocal cord dysfunction in a nonreferral urban population is not known. The high incidence of vocal cord dysfunction at the National Jewish Center cannot be extrapolated to our patient population. In addition, our patients did not have chronic refractory asthma. We suspect that this patient population is substantially different from those patients referred to the National Jewish Center for refractory asthma.

Although heliox may also be an effective treatment for vocal cord dysfunction, there was no evidence to support the diagnosis in our patients. Physical findings of diffuse expiratory wheeze without inspiratory wheeze or stridor, hypercapnia with acute respiratory acidosis, failure to improve with endotracheal intubation, and the patient profiles of the study population are indicative of acute severe asthma.

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REFERENCES


Stridor From Edema of the Arytenoids, Epiglottis, and Vocal Cords After Use of Free-Base Cocaine

To the Editor:

We have read with great interest the article by Haim and colleagues in the January 1995 issue (CHEST 1995; 107:233-40) in which the authors reviewed the various respiratory consequences of free-base cocaine inhalation. We wish to report a case of a young woman with recurrent episodes of stridor from free-base cocaine-induced upper airway edema.

A 45-year-old woman presented to our emergency department with dyspnea, dysphagia, hoarseness, and sore throat. She had previous multiple visits to the emergency department for similar problems. Each time she was treated with inhaled bronchodilators and short courses of oral corticosteroids. Because of chronic complaints, she was referred to an ear, nose, and throat physician. Her direct laryngoscopic examination less than 24 h before her current visit was normal. She had no significant medical illnesses and was taking no medications. She admitted to using injectable and nasal cocaine in the past, but denied any other drug use in the past few years.

Initial examination revealed inspiratory and expiratory stridor and the use of accessory respiratory muscles. There was minimal improvement in her symptoms in spite of nebulated bronchodilators. Chest roentgenograms showed no infiltrates. However, a subglottic stenosis was visualized on her neck radiographs.

On bronchoscopy, the arytenoids, intraarytenoid space, epiglottis, and false and true vocal cords were markedly edematous. She