that DLco is likely to be an important and relevant prognosis factor in patients with PAH-SSc. ILD, PH due to ILD, and PAH are not the sole causes of DLco impairment in patients with SSc. Nevertheless, for clinical practice, it seems important to highlight the fact that a markedly impaired DLco <35% to 40% of predicted value in patients with PAH-SSc may be associated with a worst-outcome scenario.

Affiliations: From the Department of Internal Medicine (Drs Hachulla and Launay), National Reference Center for Scleroderma, Hôpital Claude Huriez, University of Lille 2; Organétrie Biostatistiques (Dr Clerson); and the Respiratory Department (Dr Humbert), National Reference Center for Pulmonary Hypertension, Hôpital Antoine Béclère, Assistance Publique Hôpitaux de Paris, Université Paris-Sud 11.

Financial/nonfinancial disclosures: The authors have reported to CHEST that no potential conflicts of interest exist with any companies/organizations whose products or services may be discussed in this article.

Correspondence to: Eric Hachulla, MD, PhD, Department of Internal Medicine, Hôpital Claude Huriez, 59037 Lille Cedex, France; e-mail: e.hachulla@chu-lille.fr

© 2010 American College of Chest Physicians. Reproduction of this article is prohibited without written permission from the American College of Chest Physicians (www.chestpubs.org/site/misc/reprints.xhtml).

DOI: 10.1378/chest.10-0799

REFERENCES


Effect of Continuous Positive Airway Pressure on Inflammatory Markers

To the Editor:

Wu and colleagues1 are to be congratulated on their interesting research recently published in CHEST (February 2010) that adds to our understanding of the mechanisms involved in the systemic inflammatory cascade associated with obstructive sleep apnea. The authors further demonstrated the salutary effects of continuous positive airway pressure (CPAP) in reversing the inflammatory response.

However, it was disappointing that the therapeutic intervention with CPAP was not described. How did the authors determine the therapeutic pressure setting? What was the mode of CPAP used? Equally important, was compliance with CPAP measured, and if so, was there a correlation between compliance and decrease in inflammatory mediators? It would have been especially interesting to see a graph of the dose-response curve, specifically the degree of CPAP compliance (hours on CPAP/hours of asleep) vs levels of serum high mobility group box-1 protein. Do those data exist, and could they be made available online?2

Affiliations: From the Pulmonary, Critical Care, and Sleep Medicine, Walter Reed Army Medical Center.

Financial/nonfinancial disclosures: The author has reported to CHEST that no potential conflicts of interest exist with any companies/organizations whose products or services may be discussed in this article.

Correspondence to: Arn Eliasson, MD, FCCP, Walter Reed Army Medical Center; 6900 Georgia Ave NW, Washington, DC 20307; e-mail: abeliasson@aol.com

© 2010 American College of Chest Physicians. Reproduction of this article is prohibited without written permission from the American College of Chest Physicians (www.chestpubs.org/site/misc/reprints.xhtml).

DOI: 10.1378/chest.10-0610

REFERENCES


Response

To the Editor:

We would like to thank Dr Eliasson for his thoughtful and insightful comments regarding our article in CHEST (February 2010).1 The therapeutic pressure setting for continuous positive airway pressure (CPAP) was determined in the following manner. All patients underwent a standard attended polysomnographic study for CPAP titration with the use of a Sullivan machine (ResMed SS Escape; Sydney, NSW, Australia) and a comfortably fitted mask. Initial pressure in the mask was set at 4 cm water and was increased overnight by 1-cm water increments based on the presence of apnea, hypopnea, respiratory effort-related arousals, or snoring associated with arousals. We sought to determine the lowest pressure at which most respiratory events were ablated or the point at which a maximum pressure of 15 cm water was reached with the patient in the supine position and in a rapid-eye-movement sleep period.2

All patients were educated regarding obstructive sleep apnea syndrome (OSAS) and the proper use of CPAP. A fixed-pressure CPAP machine was used following the titration. Neither auto-adjusting CPAP nor bilevel positive airway pressure was used in this study. The use of CPAP with heated humidification at the effective fixed-pressure mode every night was strongly encouraged, and most patients applied the heated humidification as suggested.

In our study, the correlation between compliance with CPAP use and decrease in inflammatory mediators was not taken into consideration. All of the patients had received recent diagnoses of moderately severe or severe OSAS and were naive to nasal CPAP treatment. They had to tolerate nasal CPAP therapy with two complete 4-week follow-up periods. Their usage diaries indicated that most had a good adherence to nasal CPAP (using CPAP >4 h/d and >5 days/week). For some patients, information on the hours of use per day and percentage of days of use could be obtained when a downloadable data card was inserted into the CPAP machine. For others, the patients self-reported CPAP use during clinic follow-up. Therefore, we have incomplete data to generate the dose-response curve of hours on CPAP vs levels of serum high mobility group box-1 protein.

www.chestpubs.org