


**Response**

To the Editor:

We have read with great interest the comments of Drs Siakalas and Tzortzaki regarding our article on small airway disease in asthma and COPD. They suggest excluding the term “abnormal inflammatory response to cigarette smoke” from the definition currently used for COPD.

Although smoking is the most important risk factor for COPD, only approximately 20% to 30% of smokers will ultimately develop COPD. A key unanswered question is why smoking causes irreversible and progressive obstructive changes in these susceptible subjects and how they differ in their response to smoking from nonsusceptible subjects.

To clarify the abnormal response to smoking in susceptible individuals, several possible mechanisms have been put forward with respect to airway inflammation, oxidative stress, protease-antiprotease imbalance, tissue injury, and repair and remodeling. Thus far in research, patients with established COPD have been compared with control subjects without COPD. Therefore, any observed difference between these groups will as likely reflect a cause as a consequence of COPD. For this reason, it is presently unknown whether susceptible smokers exhibit a different or abnormal inflammatory response when compared with nonsusceptible smokers who will not develop COPD over time.

In this context, the findings of Silverman et al are of interest. They demonstrated that susceptible subjects can be identified based on family history. When first-degree relatives of patients with severe early-onset COPD (defined by lung function, ie, FEV1 < 40% predicted and age < 53 years) smoked, their FEV1 was significantly lower than in subjects who smoked and were not first-degree relatives.

A study is now needed that investigates if there are differences in the response to smoking between young healthy subjects who are susceptible according to the criteria of Silverman et al and young healthy subjects who are nonsusceptible. Such a study will certainly generate highly valuable new insights in the mechanisms that contribute to COPD pathogenesis. Only then can we decide to definitively exclude the possibility that a different or abnormal response to cigarette smoking contributes to the development of COPD.

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**References**


**IV Immunoglobulin Might Be Considered as a First-line Treatment of Severe Interstitial Lung Disease Associated With Polymyositis**

To the Editor:

We read with interest the article by Bakewell and Raghu in a recent issue of *CHEST* (February 2011), in which the authors relate the case of a patient with severe interstitial lung disease associated with polymyositis/dermatopolymyositis (ILD-PM/DM). The patient, remarkably, improved after three monthly doses of 2 g/kg IV immunoglobulin (Ig) without any other immuno-suppressive agent, with sustained clinical remission after > 2 years. According to the authors, IV Ig has not yet been tested as a first-line treatment of this disease. Our recent observation of a patient who did not respond to steroids and worsened after one infusion of cyclophosphamide but did improve dramatically after IV Ig is consistent with Bakewell and Raghu, who suggest that IV Ig should be considered as a potential first-line therapy for ILD-PM/DM.

Our patient was a 47-year-old man with ILD-PM/DM in the context of dyspnea associated with severe weakness. Creatine kinase value was increased to 1,110 units/L, and antinuclear auto-antibodies were positive without anti-Jo-1. The results of an electromyogram suggested a myopathy, and the results of a deltoid biopsy confirmed the diagnosis of polymyositis. Chest radiograph and CT scans revealed consolidation, with air bronchograms and ground-glass opacities in both lungs with nodular opacities. Total lung capacity (TLC) was 52% of predicted values, and vital capacity (VC) was 55%. Diffusion capacity of the lung for carbon monoxide (DLCO) was 61%. The results of BAL revealed mixed
alveolitis with 22% lymphocytes and 10% neutrophils. Transbronchial biopsies were not performed because of poor tolerance for BAL. Prednisone was given at 80 mg/d (1 mg/kg) with little response in terms of muscle weakness and no respiratory improvement after 15 days. A bolus of 0.6 mg/m² of cyclophosphamide was administered, as currently recommended for refractory cases. Reassessment before the second scheduled cyclophosphamide infusion revealed a dramatic deterioration of pulmonary function (TLC, 41%; VC, 51%; and DLco, 32%). Cyclophosphamide treatment was stopped, and IV Ig at a dosage of 2 mg/kg was begun.

A marked clinical and biologic improvement (normalization of creatine kinase) was observed 21 days after the first infusion. After three monthly courses of IV Ig, the patient recovered normal muscle function and showed a marked improvement on pulmonary function test (TLC, 59%; VC, 63%; and DLco, 51%) and a regression of fibrotic changes on CT scans. This observation lends support to the suggestion that IV Ig treatment might be considered as a treatment of ILD-PM/DM, especially for critical cases.

To the Editor:

I am writing in regard to the article by Christopher and Schwartz on transtracheal oxygen therapy (TTO) in a recent issue of CHEST (February 2011). Long-term oxygen therapy (LTOT) by the transtracheal route has not been given enough credit by the medical community. I speak from experience as a retired board-certified general surgeon with severe COPD who is receiving LTOT.

I required nocturnal oxygen using a nasal cannula for 8 years but suffered with recurrent epistaxis due to the nasal cannula. One year ago, after an acute exacerbation, I required continuous oxygen, and this presented a dilemma due to the ongoing epistaxis. My pulmonologist did not offer TTO as an alternate method of LTOT. I researched oxygen therapy on the Internet and found Transtracheal Systems. I contacted John Goodman, BRT, and after I explained my situation, he referred me to Dr Michael Schwartz at National Jewish Health in Denver, Colorado. Dr Schwartz spent a great deal of time with me on the telephone, but I felt Denver’s altitude might be problematic for me. I sought established transtracheal programs in Texas, New Mexico, and Arizona without success.

I then located an otolaryngologist in Phoenix, Arizona, who had experience performing the (preferred) Lipkin surgical procedure, so I went to Phoenix. The procedure went very well, and I returned to my home in El Paso, Texas. After 10 days, I started changing my catheter per protocol and was able to do just fine. I am back now to most normal activities, including golf twice a week, pulmonary rehabilitation twice weekly, and more.

Before TTO, my hemoglobin level was 17.5, and my hematocrit value was 51%; today, my hemoglobin level is 14.9, and my hematocrit value is 40.9%. I require a setting of 2 while sedentary and 3 when ambulatory or exercising using a liquid oxygen portable delivery system on intermittent flow. I use continuous flow at 2.5 L/min for sleep.

TTO requires teamwork, and the pulmonologist is the linchpin. It is the pulmonologist who must know that such a procedure exists and that it is a viable and often a preferable modality. Patient selection is extremely important, and pulmonologists should know that TTO is not a therapy intended only for the patient with refractory hypoxemia. Finally, the patient must be highly motivated, have some manual skill, and have a great deal of confidence in his or her pulmonologist. Again, I thank CHEST for publishing this excellent article and the authors for their fine work.

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Transtracheal Oxygen Therapy Success

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