Response

To the Editor:

We thank Drs. Netzer and Omron for their interest in our article. We agree that observational data such as we used are tricky and cannot consider all potential confounders. Confounding by indication is always the weakness of this kind of work. However, we tried to avoid this, and could not identify plausible causes for this kind of confounding. Dr. Netzer points out that we did not look at markers of cardiovascular disease, such as cardiac drugs, and she is right. However, in another study we have looked at cardiovascular morbidity as a function of respiratory drugs and found that inhaled steroids tended to be protective, but this effect was independent of interaction with cardiac drugs. Dr. Omron is not convinced that users of inhaled steroids were as sick as people who did not receive these drugs. We would argue that the evidence favors them being at least as sick; we regard frequent physician visits and multiple drugs as evidence of perceived severity, not the reverse. Further, we studied dispensed drugs only; we had no data regarding drugs that were prescribed but not dispensed. The fact that the steroid effect was most notable soon after dispensation we take as evidence favoring drug use.

Nicholas R. Anthonisen, MD
University of Manitoba
Winnipeg, MB, Canada

The author has no conflict of interest to disclose.

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Correspondence to: Nicholas R. Anthonisen, MD, University of Manitoba, RS 319 Health Sciences Centre, 810 Sherbrook St, Winnipeg, MB, Canada R3A 1R8; e-mail: nanthonisen@exchange.hsc.mb.ca

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Reflex and Autoimmunity

Common Links Among Patients With Pulmonary Fibrosis?

To the Editor:

I read with interest the article by Fischer and colleagues (October 2006), wherein they describe the characteristics of systemic sclerosis (SSc)-associated interstitial lung disease in patients who presented with an initial diagnosis of idiopathic interstitial pneumonia. Symptomatic gastroesophageal reflux (GER) was one of the clinical features that was identified by the authors as being indicative of the presence of SSc-associated interstitial lung disease. However, GER is very common among patients with idiopathic pulmonary fibrosis (IPF), the most commonly encountered idiopathic interstitial pneumonia. Raghu and colleagues recently demonstrated with the use of 24-h pH monitoring and esophageal manometry that GER was present in majority (57%) of patients with IPF, almost half of whom were symptomatic. Furthermore, the same group reported stabilization and even improvement in pulmonary function test results over a period of 2 to 6 years in a small series of patients with IPF who were treated with anti-GER medications alone. Similarly, the presence of autoimmunity in IPF is well known and was highlighted in a review. Anti-nuclear antibodies (ANAs) are demonstrable in serum in as many as 10 to 20% of patients with IPF. Presence of anti-topoisomerase antibodies (that produce a nucleolar pattern on immunofluorescence testing) among patients with IPF has also been reported. The authors of the current study had, in fact, recently demonstrated by means of a retrospective analysis that ANA positivity (with a nucleolar staining pattern) occurred in 25 patients with IPF (8.8%), more than half of whom also had presence of anti-Th/To antibodies. The small number of patients with ANA positivity, absence of cutaneous features of SSc in all the patients, and lack of differences (in clinical features, pulmonary physiologic-cum-gas exchange parameters and survival) between patients with ANA positivity and those without, as well as between patients with anti-Th/To antibody positivity and those without makes it difficult to substantiate the authors’ conclusions of attributing the pulmonary fibrosis as being related to SSc. The fact is that pulmonary fibrosis (occurring in the setting of both IPF and SSc) is commonly associated with the presence of GER as well as autoimmunity, and differentiating idiopathic from nonidiopathic entities often remains a challenging issue for treating clinicians.

Natveet Singh, MD
Postgraduate Institute of Medical Education and Research Chandigarh, India

The author has no conflicts of interest to disclose.

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Correspondence to: Natveet Singh, MD, Department of Pulmonary Medicine, Postgraduate Institute of Medical Education and Research, Sector 32, Chandigarh 160012, India; e-mail: natveetdal@yahoo.com

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Response

To the Editor:

We appreciate Dr. Singh’s interest in our article. We agree that it can sometimes be challenging to differentiate nonidiopathic forms of interstitial lung disease (ILD) from idiopathic forms. It was in this light that we identified the unique features of systemic sclerosis sine scleroderma (ssSSc)-ILD in patients who otherwise would have been categorized as having idiopathic pulmonary fibrosis (IPF).

As stated in our article, the diagnosis of ssSSc was made only if the patient had at least three or more manifestations that were typical of systemic sclerosis (SSc). We chose these manifestations based on the proposed diagnostic criteria for ssSSc.
Consequently, a patient with ILD, gastroesophageal reflux disease, and a positive test result for antinuclear antibodies (ANAs) would not have been considered to have ssSSc. ANA positivity can occur in IPF patients, and thus we did not solely rely on this in our analysis. Rather, we highlighted the specificity of nucleolar staining for ANAs. In our previously described cohort of IPF subjects who were anti-Th/To-positive, we argued that, on retrospective analysis, those with ANAs revealed by nucleolar staining, and particularly those with Th/To antibodies, appeared to have had ssSSc-ILD.

Dr. Singh states that antitopoisomerase antibodies have been reported in patients with IPF and suggests this could produce a nucleolar staining ANA. A closer review of the referenced article shows that 18 of 41 IPF patients (44%) had antibodies to DNA topoisomerase II. However, only 3 of the 18 patients had a positive ANA finding obtained by Hep2 cell substrate, and the pattern of immunofluorescence was not reported. Furthermore, to our knowledge, the presence of antitopoisomerase II antibodies in IPF patients has not been confirmed by another group, although it has been reported in Japanese patients with SSc-ILD. Additionally, antitopoisomerase II antibodies should not be confused with antitopoisomerase I (anti-Scl-70) antibodies, which are highly specific for diffuse SSc and give a nucleolar pattern on ANA testing. Notably in our study, while all six patients had ANAs revealed by nucleolar staining, only one patient showed antitopoisomerase I (anti-Scl-70) antibodies. We believe that our case definition for ssSSc-ILD accurately differentiates those patients with ssSSc from those with IPF, and that this distinction is clinically useful.

Arye Fischler, MD
Richard Meekan, MD
Kevin Brown, MD
National Jewish Medical and Research Center
Denver, CO

Carol Feghali-Bostwick, PhD
University of Pittsburgh
Pittsburgh, PA

Sterling West, MD
University of Colorado
Denver, CO

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Correspondence to: Arye Fischler, MD, National Jewish Medical and Research Center, Rheumatology/Medicine, 1400 Jackson St, Denver, CO 80206; e-mail: fischera@njc.org

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Oxygen Administration and the Protection of Health-Care Workers From Infections

To the Editor:

Hui et al provide a much needed reminder to the medical community that the very oxygen mask that is used to relieve the hypoxia may contribute to the wide dispersal of infected aerosolized particles, and thereby increases the risk of transmission of airborne infection to health-care workers. However, I believe the authors do a disservice by unequivocally declaring that their data allows the demarcation of “a zone of potential aerosol infection with an extra margin of safety.” They would do well to temper this conclusion based on theoretical arguments from a mechanical model with those based on published in vivo observations in humans that clearly demonstrate aerosolized particles traveling, not 30 or 40, but hundreds of centimeters.

The authors conclude that potential infectious patients “should, ideally, be managed in a single, isolation room, under negative pressure…” This type of conclusion simply does not follow from the type of study performed. Furthermore, it is hard to see how managing a contagious patient in a negative-pressure room would provide any protection to a health-care worker. On the other hand, preventing the patients from spraying infectious particles on health-care workers while being administered oxygen, as we have advocated, would provide protection to other patients and health-care workers alike.

Joseph A. Fisher, MD
Toronto, ON, Canada

The author is the co-developer of masks described in references 3 and 4 that have been licensed to Viasys Healthcare Inc. Reproduction of this article is prohibited without written permission from the American College of Chest Physicians (www.chestjournal.org/misc/reprints.shtml).

Correspondence to: Joseph A. Fisher, MD, Toronto General Hospital, 7EN-242, 200 Elizabeth St, Toronto, ON, Canada M5G 2C4; e-mail: joe.fisher@utoronto.ca

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Response

To the Editor:

We appreciate the comments by Dr. Fisher on our study, which showed a smoke particle dispersion distance of approximately 0.4 m during application of 4 L/min of oxygen via a