with armodafinil.\textsuperscript{3} In this study, we screened all patients for OSA and only included in the study those patients with an apnea/hypopnea index < 6/h. All patients then underwent an overnight sleep study followed by a multiple sleep-lateness test before and after each treatment arm.

Despite the absence of OSA, we found one-half of the patients with sarcoidosis had a sleep onset latency of < 8 min, indicative of hypersomnolence. Furthermore, despite using two fatigue instruments, there was no difference in the fatigue severity for those patients with or without a shortened sleep-onset latency. In addition, armodafinil significantly improved fatigue in these patients, including those with objective evidence of hypersomnolence.

Fatigue can be due to several factors in patients with sarcoidosis, including not only sleepiness but also depression, medications, and ongoing inflammation from the disease.\textsuperscript{4} However, patients with sarcoidosis may still have marked fatigue without either depression or sleep apnea. For these patients, treatment with stimulants\textsuperscript{3,5} may be useful.

**References**


**Response**

To the Editor:

We thank Dr Lower and colleagues for their comments and their interest in our study.\textsuperscript{1} In their study, they demonstrated that treatment with the stimulant armodafinil was associated with a reduction in fatigue in patients with sarcoidosis.\textsuperscript{3} A subset of their patients had coexisting fatigue and hypersomnolence, although improvements in fatigue were observed in patients with and without hypersomnolence.

In our study of patients referred for polysomnogram testing, we found that excessive daytime sleepiness, or hypersomnolence, was more common in patients with sarcoidosis compared with control subjects.\textsuperscript{1} As we did not measure fatigue, we were not positioned to comment on the relationship between fatigue and sleepiness in our cohort. We agree, though, that these are distinct clinical conditions. Even as they may coexist or have overlapping features, distinguishing between fatigue and excessive daytime sleepiness is clinically relevant. While treatment with stimulants may be beneficial in both, patients with fatigue also warrant an evaluation for comorbidities, which may be targeted for other primary interventions.\textsuperscript{2,4}

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**REFERENCES**


**Airways Disease Presenting as Restrictive Impairment**

A Variant in Asthma, a Defining Feature in World Trade Center Lung Disorder

To the Editor:

The recent article by Berger et al\textsuperscript{1} in CHEST (July 2013) has refocused the interest of readers of CHEST to airway disease...
preventing as restrictive dysfunction. As cited in the article, this syndrome was noted in individual patients and then by the senior correspondent of this letter in a large series of patients with asthma, in whom it was estimated to characterize at least 5% of patients presenting with asthma. The decrease in expiratory reserve volume was especially notable in the days of precomputerized spirometry, when tracings of tidal breathing were followed by a forced exhalation. "Airway disease presenting as restrictive dysfunction" has come to characterize a large number of patients at the World Trade Center (WTC) on September 11, 2011, with typical airway symptoms of cough, dyspea, and wheezing.\(^1,2\)

The findings of Berger et al.\(^1\) including normal pulmonary compliance, elastic recoil, and lung parenchyma on CT scan, support the attribution to an airways disorder. Another strong indication, bronchial hyperreactivity, was noted both in known patients with asthma\(^3\) and in WTC patients with restrictive dysfunction.\(^2\) Early in the experience with WTC lung disorder, Prezant et al.\(^4\) reported "nearly equal" reductions (from pre–September 11, 2001, values) in FVC and FEV\(_1\), of at least 0.5 L in > 50% of exposed firefighters. Response to a bronchodilator was seen in 63%, and bronchial hyperreactivity was noted in 24% of these patients. These rates are notable because selection criteria for firefighters rigidly exclude asthma. Bronchodilatation in airways disease presenting as restriction often results in equivalent increases in FVC and FEV\(_1\) with little change in FEV\(_1\)/FVC ratio, and bronchoprovocation brings about equivalent decreases. It is, therefore, surprising that Berger et al.\(^1\) noted no change in postbronchodilator spirometry and that impulse oscillometry, presumably a more sensitive, if less specific, measure of airways dysfunction, showed only small changes with bronchodilator, leaving persistent abnormality in the majority.

Pseudorestrictive dysfunction due to airways obstruction was known previously in patients with emphysema and severe asthma associated with massive air trapping, who have functional residual capacity values ≥ 130 and residual volume (RV) values ≥ 200% predicted. Increases in RV parallel decreases in FVC. The air trapping seen in WTC airways disorder as localized changes on high-resolution CT scans is decidedly different. Berger et al.\(^1\) report reduced FRC and unimpressive RV values (eg, an RV/total lung capacity ratio of 0.35 at a mean age of 47 years) in their resident-exposed subjects.\(^1\) Weiden et al.\(^5\) reported median FRC 96% predicted and RV 123% predicted in their firefighters. The highest RV, 131% predicted, was in the least exposed group.

The authors report a reduced mean diffusing capacity but do not describe the number of patients below the lower limit of normal, nor do they characterize those with truly low values. A low diffusing capacity of the lung may point to a specific abnormality originating in the small airways that results from the complex WTC inhalational exposure, namely constrictive or oblitative bronchiolitis. These correspondents reported a biopsy-proven case with onset shortly after WTC exposure and response to azithromycin; this was also cited by Berger et al.,\(^1\) who also noted pathologic bronchiolitis reported by Caplan-Shaw et al.\(^6\) Additionally, two cases were reported by Mount Sinai pathologists.\(^7\) The description by Berger et al.\(^1\) and the dialogue it elicits contribute to the understanding of the pathophysiology of WTC (and other) airways disorders and of the need to continue surveillance of these patients.

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**Response**

To the Editor:

We thank Drs Miller and Mann for their letter regarding our article\(^1\) and agree with their perspective. Our findings\(^2\) expand on a prior report by Miller and Palecki\(^3\) in a population of patients with asthma and isolated case reports by other authors as cited in our article. These studies indicate that a restrictive pattern on plethysmography with normal FEV\(_1\)/vital capacity may be found in patients with airway disease more often than previously appreciated.

Our study and prior lung biopsy findings\(^4\) provide a pathophysiologic mechanism for the restriction related to injury to the distal lung unit. As Drs Miller and Mann indicated, our spirometry and oscillometry findings were minimally responsive to bronchodilator. We believe that this is in accord with the histologic evidence for bronchiolitis, small airway fibrosis, and emphysema noted in subjects exposed to World Trade Center (WTC) dust and fumes.\(^5\) In addition, our article provides clues from the standard testing modalities to identify this phenotype: (1) reduction in vital capacity due to reduced expiratory reserve volume with normal inspiratory capacity (ie, restriction from expiratory impairment), (2) relative preservation of diffusing capacity for carbon monoxide suggesting normal alveolar-capillary interface, and (3) reduced alveolar volume to total lung capacity ratio indicating nonuniform airflow.