the common and continuing problem of trying to develop a rational basis to discontinue isolation.

Inge Gurevich, RN, MA, Eceylin Jacobsen, RN, MPH, Antonio Ortega, MD, and Burke A. Cunha, MD, FCCP; Infection Control Section, Infectious Disease Division, Winthrop-University Hospital, Mineola, New York, and State University of New York, School of Medicine, Stony Brook

Reprint requests: Dr. Cunha, Infectious Disease Division, Winthrop-University Hospital, Mineola, NY 11501

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That’s No Lady

To the Editor:

To reprise Henny Youngman’s famous joke, I would take exception with the designation of the woman with Mycobacterium avium complex (MAC) disease involving her lingula and right middle lobe as suffering from “Lady Windermere syndrome” (CHEST 1995; 108:1156-57). Certainly, the portrait of a demure damsel who refuses to cough because it is not genteel is highly poetic imagery. But, it is extremely improbable to me that volitional suppression of the powerful cough reflex could explain this pathologic complex. Certainly, as indicated, the lingula and right middle lobe suffer from some degree of anatomical vulnerability to poor drainage, and this may well play a role in the frequent involvement of these regions with pulmonary MAC disease. Indeed, we have also observed predilection for lingular/right middle lobe MAC disease in women. However, we find that it occurs as well in those who cough with vigorous abandon.

I therefore submit an alternative hypothesis for this condition. MAC is distributed widely in the environment including potable water. Mycobacteria therefore are presumably deposited regularly in our airways, for instance, as we shower. Failure to clear the microbes selectively from the lingula/right middle lobe due to suppressed cough does not seem logical. Rather, I believe it can be more plausibly related to diminished tensile strength of the airways/air spaces in these women, probably associated with a subtle deficit of the connective tissue matrix. Evidence in support of this are associated thoracic anomalies seen among our series of women with MAC including pectus excavatum, straight-back syndrome, scoliosis, and mitral valve prolapse. But if there truly is an underlying disorder of the connective tissue, why should there be excessive disease localized to the lingula/right middle lobe? Rather than voluntary cough suppression, I believe the answer lies with the relationship of these lung regions to the pulsating heart. Among our patients, CT scans clearly showed the earliest and most severe abnormalities to lie in the medial segment of the right middle lobe and the inferior segment of the lingula, areas juxtaposed to the ventricles (also seen with Byrd’s patient). Often before inflammatory changes are seen in these areas, high resolution CT imaging suggests rarefaction and distortion of the parenychyma in these regions. Potential mechanisms for diminished defenses might include distorted tissue organization or disrupted mucociliary clearance secondary to the repetitive mechanical trauma.

To paraphrase Mr. Youngman, “That’s no Lady (Windermere), that’s bad fibrillin.”

Michael D. Iseman, MD, FCCP, Clinical Mycobacteriology Service, Division of Infectious Diseases, University of Colorado School of Medicine, Denver

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Magnesium Saga

To the Editor:

We read with interest the article by Bloch and colleagues (CHEST; 107:1576-81) about adjunct use of IV magnesium sulfate in the treatment of acute asthma. At first glance, the results look promising and represent a relative risk reduction of 58%, and even more reassuring, an absolute risk reduction of 0.45. The number needed to treat (NNT) to prevent one hospitalization is 2.2.1 However, we believe that at least three areas relative to methodology of the study are worthy of discussion.

First, the hypothesis of this study was to test whether IV magnesium sulfate, compared with placebo, would improve pulmonary function and decrease hospital admissions in all asthmatic patients, and not solely in severe asthmatic patients. Subgroup analysis is always problematic in such a small study. We reanalyzed their data by moving one patient from the placebo group to the magnesium group and using a two tailed t test, wherein the p value changed dramatically from an impressive 0.009 to a meager 0.055.

Second, the total number of patients in the severe group was only 35. For an event rate as high as 0.78 (hospitalizations in placebo group) at least a total of 87 patients would have been required to detect a clinically significant difference of 25% risk reduction.1

Third, our concerns are strengthened by recent examples of the LIMIT-2 trial2 that revealed significant benefit of IV magnesium in patients suspected of acute myocardial infarction. When the same therapy was tested in a considerably larger trial (ISIS-4),2 however, the beneficial effects of the LIMIT-2 were not observed. In the ISIS-4 trial, IV magnesium therapy resulted in significant untoward effects in patients suspected of acute myocardial infarction.

Considering these facts, we want to express a note of caution about use of IV magnesium sulfate as adjunct therapy in any form of asthma. Evidence of benefit at present is inconclusive. Until more

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