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Yoga and Pneumothorax

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sarily be useful in elucidating these aspects of the study because virtually all smokers included in the study were already consuming cigarettes 12 months before the interview. We think that definitive answers regarding this important matter may only be obtained with the performance of longitudinal studies involving larger samples of representative populations.

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Yoga and Pneumothorax

To the Editor:

We agree with Johnson and colleagues¹ (May 2004) that the “breath of fire” yoga technique most probably induced the pneumothorax they reported, and also with their appropriate cautionary advice. However, there is a question of balance and perspective here. Nowhere in their report do they clearly define the breath of fire as an advanced technique, or “extreme yoga” technique, to be practiced only by advanced students after appropriate instruction. By implication therefore, their report appears to unjustly flame all yoga techniques. This is not appropriate for a discipline that has generally been practiced safely for not hundreds, but thousands of years.

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Dr. Sharma is the author of the patient instruction video, “Stretching Breathing Exercises: Adapted for People with COPD.” This is a project of the National Emphysema/COPD Association (NECA) [www.necacommunity.org]. Both authors are members of NECA, and Dr. Sharma is on the NECA Board. This project has been self-funded by Dr. Sharma, who will be commercializing this video. Neither author is receiving financial remuneration from NECA or any other sources. Dr. Hillsman is a volunteer medical technical advisor to Dr. Sharma as a part of the NECA project.

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Missed Opportunity To Address the Critical Care Medicine Crisis

To the Editor:

The recent white paper from the Critical Care Professional Societies¹ notes “an unprecedented, and largely unrecognized, shortage of physician intensivists in the near future” that “will deny standard critical care services for large populations of patients with serious illnesses.” To avert this crisis, the Critical Care Professional Societies offer a variety of recommendations, including steps to increase the number of fellowship-trained providers. We strongly support the need for the quality training of skilled physicians in critical care medicine.

One recommendation was to change immigration laws to permit international medical graduates increased access to critical care fellowship training. Ironically, the American Board of Internal Medicine (ABIM) recently closed access to critical care specialty training fellowships to US emergency medicine (EM) residency graduates. This decision was unrelated to any objective data suggesting suboptimal performance by EM residency graduates in critical care training programs or practice. While formal certification pathways have remained blocked to EM residency graduates by various members of the American Board of Medical Specialties (ABMS), EM residency graduates have successfully completed critical care fellowships across the country for 2 decades, performing at levels equal to their internal medicine (IM) and other colleagues.

Graduates of EM residencies are mandated to participate in more critical care training opportunities than their counterparts in categorical IM or pediatrics. In addition, EM residents deliver and teach care from the home or site of illness/injury—prehospital—through to the hospital. They offer a unique ability to understand a broad part of the continuum of care, a natural addition to a training program, complementing other graduates.

EM residents also participate in a large number of critical care activities in their daily work. Hospital crowding and the increased demand for intensive care services equate to prolonged emergency department stays for gravely ill patients (or those in jeopardy of becoming more ill), who usually remain under the care of the EM physician. In many community hospitals, the EM provider is often the sole on-site physician, and responds to all critical status changes for patients in the ICUs. Ultimately, it is obvious that we do care for the same patients.

Thus, one component to solving the impending patient care crisis in critical care medicine would be to reverse the recent ABIM ban on training EM residents. EM residents have not “usurped” critical care training positions from IM residency graduates, but rather allowed creation of broad, diverse training programs. EM residency graduates constitute a ready pool of high-quality fellowship candidates (note the National Resident Matching Program results for the last decade placing EM residencies as one of the top three most competitive to access for US medical school graduates). Since expanding the pool of candidates from within IM programs has limits, restoring the access to training of the highly skilled EM residency graduates would be a better step than seeking solutions elsewhere (including foreign-trained graduates). Separately, we encourage the ABMS to consider allowing EM graduates who have successfully completed a critical care fellowship to be eligible for certification after training. This would increase the pool of interested EM graduates and would assist in reaching the goal of expanding both

the pool and number of critical care fellowship positions and trained intensivists. Ultimately, it is in the best interests of our patients.

Carey D. Chisholm, MD
Donald M. Yealy, MD
on behalf of the Board of Directors
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Treatment Outcome in *Mycobacterium avium* Pulmonary Disease

A Correction and Comment

Huey Long (“Kingfish”), the famously corrupt, demagogic, depression-era governor of Louisiana, was interviewed by a high school senior, whose last question was, “Tell me, governor, is there a place for honesty in politics?” Long replied, reassuringly, in the affirmative, but as the student left the room, he winked at his surrounding cronies and commented, “In politics, boys, we use anything we can get!” We applied the same expediency rule in our treatment of pulmonary disease due to *Mycobacterium avium*.

Due to a misinterpretation of our data,¹ the authors of a recent review of the treatment of *M avium* pulmonary disease² conveyed a misleadingly pessimistic view. They assumed (Stephen Field, MD, FCCP; personal communication; August 29, 2004) that individuals in whom surgery was an adjunct to medical therapy and those who had responded to drug treatment, as indicated by sputum conversion and radiographic improvement, but whose information had not been completed when we collated our data, were treatment failures. They excluded from consideration two patients whose primary treatment was surgery. Consequently, they considered that only 6 of 14 patients (43%) were treated successfully.

Sixteen of our patients were judged to be suitable for aggressive management, and 2 patients underwent surgery as the primary treatment. In Table 1 of the article by Reich and Johnson,¹ case 17 was a 3-year-old child with unexplained right hilar adenopathy and middle lobe opacification. The diagnosis was established based on histology and the results of a culture of the resected middle lobe. This patient and patient 10 represent successful surgical treatment, evidenced by the absence of recurrence. Surgery was an adjunct to successful drug treatment, which was indicated by sputum conversion and radiographic improvement in cases 6, 9, 13, and 14. Each patient had localized disease, and their relative youth (mean age, 41 years) indicated a low surgical risk, which I judged to be lower than the risk of recurrence from residual disease. They had no recurrence after long-term follow-up. The title of column 8 in Table 1, “months observed since drug treatment was DC,” applied only to those individuals in whom treatment was completed. The patients in

cases 7, 8, and 11 had each received ≤ 18 months of the planned 24 months of drug therapy when the data were collated. Each patient exhibited sputum conversion and radiographic improvement, and I classified them as treatment successes. I stand by the summary of successful outcomes in the 16 persons treated with intent to cure (short-term follow-up, 15 of 16 patients [94%]; long-term follow-up, 11 of 12 patients [92%]). We attributed our high success rate to the following two factors: fewer of our patients had moderate-to-far advanced disease than in compendia drawn from tertiary care settings; and none had experienced prior treatment failure. A decision to increase the dose of ethionamide stepwise may have contributed to success by reducing drug intolerance. We could not exclude the possibility that our favorable outcome reflected infection with a population of less virulent or more drug-susceptible organisms. With the advent of the newer macrolide agents, one would hope that aggressive treatment would be even more effective.

Primary disease is an infrequently cited pulmonary manifestation of *M avium*, which, when a plausible cause is sought, appears to be ascribable to a large, avian-sourced inoculum of organisms of low virulence in an immunologically naïve host.^{3–6} For example, our 3-year-old patient resided in a home in which a number of pet birds were housed.

In contrast to *Mycobacterium tuberculosis*, to which African Americans (AAs), in comparison with whites, exhibit a marked susceptibility, a noteworthy epidemiologic feature of pulmonary disease due to *M avium*, to which the authors allude, is its rarity in AAs. That genetic factors play a key role in resistance has been highlighted by several reports of a remarkably low incidence of *M avium* bacteremia in AAs with AIDS. Fordham et al⁷ reported that disseminated *M avium* in patients with AIDS was far less common in undeveloped countries, chiefly Africa, in comparison with developed countries. Morrissey et al⁸ reported the absence of *M avium* bacteremia in AIDS patients in Uganda, despite the high prevalence of the organism in soil and water samples. It seems plausible that genetic sorting in sub-Saharan Africa has selected AAs possessing an inherent resistance to *M avium*. If this resistance resides in a propensity to develop a brisk granulomatous response, one might speculate that the increased susceptibility of AAs to sarcoidosis is a manifestation of the same propensity to develop a granulomatous response to undefined, possibly related, antigens. Evidence that this speculation is plausible is provided by the experiment of Hurley and Shelley,⁹ who reported that 5 of 50 healthy AAs developed persistent papules at the site of intradermal injections of first-strength purified protein derivative, which, at 4 weeks, demonstrated epithelioid granulomas that were indistinguishable from positive Kveim test results.

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