prestigious journal such as CHEST that a third party has been asked to police medical professionals and interfere in the doctor-patient relationship. This sets a dangerous precedent in which a document does not just give us the science about a somewhat controversial scenario but is asking the insurance industry and the US Food and Drug Administration to trump physician judgment in an area that is not black and white.

In conclusion, we believe that this clinical commentary only creates more problems for the practicing clinician who is in the trenches and has only muddied up the trench for those of us who are in the field.

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Dr. Reddy has participated in an advisory board for CSL Behring and dinners (in Philadelphia, PA, at the CHEST conference), and in a Champions for Alpha-1 Testing (CAT) study sponsored by CSL Behring. Ms. Zaremba has participated in a round table discussion for the University of Michigan and dinners (one in Detroit, MI, with Dr. D. Kyle Hogarth, of the University of Chicago; and one in Philadelphia, PA, at the CHEST conference), and in a CAT study sponsored by CSL Behring. Reproduction of this article is prohibited without written permission from the American College of Chest Physicians (www.chestjournal.org/misc/reprints.shtml).

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DOI: 10.1378/chest.08-2580

REFERENCE


Response

To the Editor:

We appreciate the opportunity to respond to the comments of Dr. Reddy and Ms. Zaremba criticizing the perception of our “trumping” physician judgment.1 By way of clarity, we wish to emphasize that our intent is in no way to undermine physician judgment (which we believe we are also exercising in drafting this commentary), but to point out that the cornerstone of good judgment (which we believe we are also exercising in drafting this commentary), but to point out that the cornerstone of good judgment, namely evidence, is lacking with regard to the justification of augmentation therapy for individuals with PI*MZ α1-antitrypsin deficiency.

We lack clarity from reading Dr. Reddy’s letter as to how our clinical commentary1 undermines patients’ access to specialists any more than the current economic adversity may, but appreciate and agree that access to the best medical information and clinicians is essential for the optimal care of individuals with α1-antitrypsin deficiency. We suspect that in our current economic climate, patients’ precious dollars should be spent first on a visit to a well-informed physician before prescribing for them a treatment the efficacy of which in the specific setting of PI*MZ α1-antitrypsin deficiency lacks support and could contribute significant expense to the frail health-care system funding in our country. We contend that, in summarizing existing knowledge and its important gaps, our position in the clinical commentary1 facilitates informed judgment, and we applaud the leadership of CHEST for its careful and critical review of the material.

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All authors are or have been members of the Alpha-1 Foundation Medical and Scientific Advisory Committee. Dr. Silverman is also a member of the Alpha-1 Foundation Board of Director. Dr. Sandhaus serves as the clinical director of the Alpha-1 Foundation. Dr. Sandhaus has presented talks relating to α1-antitrypsin deficiency (AAT) deficiency at events sponsored by Talecris Biotherapeutics, CSL Behring, Baxter Healthcare, and Dey Pharmaceuticals, and has been a principal investigator for therapeutic clinical trials in AAT deficiency sponsored by Talecris Biotherapeutics, CSL Behring, and Kamada Pharmaceuticals. Ms. Everett has severe AAT deficiency, receives augmentation therapy, and has been a member of the voluntary leadership of the Alpha-1 Foundation for the past 12 years. Dr. Turino has no conflicts of interest to disclose. Dr. Trapnell has no conflicts of interest to disclose. Dr. Silverman received an honorarium for a talk on COPD genetics in 2006, grant support and consulting fees from GlaxoSmithKline for two studies of COPD genetics, an honorarium from Bayer Biologicals for a symposium at the 2005 European Respiratory Society Meeting, an honorarium for a talk at the Lund Symposium in 2007, and consulting fees from AstraZeneca. Dr. Stoller has served as a consultant to Talecris Biotherapeutics; has given lectures that have been supported by Talecris Biotherapeutics, Baxter Healthcare, Grifols, and CSL Behring; and has served as a member of a data monitoring and safety committee for Kamada Pharmaceuticals. In addition to individual disclosures, the authors wish to disclose the following potential conflicts of interest: the Alpha-1 Foundation relies entirely on donations for its operating budget. Included among the major donors to the Alpha-1 Foundation are all the companies that produce the augmentation therapy products mentioned in this commentary.

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DOI: 10.1378/chest.08-2533

REFERENCE


Diffusing Capacity and Alveolar Volume

To the Editor:

In discussing the diffusing capacity of the lung for carbon monoxide (Dlco), Dr. Plummer1 points out the difficulty in
“normalizing” the value with the alveolar volume (VA) because the relation between the two is nonlinear. There is another difficulty with the DLCO/VA ratio. Lung volumes determined by plethysmography or nitrogen washout are usually done along with the diffusing capacity. Frequently, I have observed that the VA derived from helium or methane dilution is considerably smaller (ie, upwards of 1 L) than the total lung capacity derived from plethysmography, even in patients whose dead space volume would be expected to be normal. In such patients, the VA should equal the total lung capacity minus an assumed dead space volume (roughly 2 mL/kg). In patients whose DLCO is low, the use of a VA derived from dilution may result in a “normal” DLCO/VA. In such cases, I calculate DLCO/VA using the total lung capacity and an assumed dead space volume, to see whether this confirms a normal DLCO/VA, which may guide interpretation of the test.

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The author has no conflict of interest to disclose.
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Daco and Kaco

How To Adjust DLCO and Kco for Lung Volume

To the Editor:

In an article in the “Topics in Practice Management” section in a recent issue of CHEST (September 2008) entitled “The Carbon Monoxide Diffusing Capacity,” Plummer1 correctly pointed out that lung diffusion capacity corrected for alveolar ventilation (Kco or ratio of diffusing capacity of the lung for carbon monoxide [DLCO] to alveolar volume [VA]) is not constant as VA changes. In fact, DLCO and Kco change with VA, as would be expected with membrane conduction varying linearly with surface area (VA2/3) or with VA, and blood conduction not changing.2 Thus, one can “volume correct” DLCO (Daco) and Kco (Kaco). The predicted Daco = predicted DLCO (0.58 + 0.42 VA/VAatl) and predicted Kaco = predicted Kco (0.42 + 0.58 VA/VAatl), where VA is the measured VA, and VAatl is the predicted VA at total lung capacity (ie, the predicted total lung capacity – predicted dead space).2 The percent predicted Daco equals the percent predicted Kaco and provides a good indication of the diffusion capacity of the lung corrected for lung volume.

Just as the predicted DLCO and Kco are adjusted for hemoglobin, the predicted DLCO and Kco should also be adjusted for lung volume. There are specific patterns of the percent predicted DLCO, Kco, VA, and Daco (or Kaco) among lung diseases.2 While patients with interstitial lung disease often have a DLCO < 80% predicted and a Kco > 80% predicted, DACO and Kaco are low. While patients with extrapulmonary restrictive but otherwise normal lungs often have low DLCO and elevated Kco levels, DACO and Kaco levels are normal. Patients with emphysema have low DLCO, Kco, DACO, and Kaco.

DLCO studies should go beyond reporting measured, predicted, and percent predicted DLCO, Kco, and VA. Predicted and percent predicted DLCO adjusted for lung volume (ie, DACO) and Kco adjusted for lung volume (ie, Kaco) should also be reported.

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The author has reported to the ACCP that no significant conflicts of interest exist with any companies/organizations whose products or services may be discussed in this article.

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REFERENCES


2 Johnson DC. Importance of adjusting carbon monoxide diffusing capacity (DLCO) and carbon monoxide transfer coefficient (Kco) for alveolar volume. Respir Med 2000; 94:28–37

Response

To the Editor:

The comments on my article in CHEST (September 2008)1 about the diffusing capacity of the lung for carbon monoxide (DLCO) by Drs. Frank and Johnson are appreciated. Both point out that the alveolar volume (VA) measurement used to calculate the DLCO/VA ratio tends to underestimate VA, thereby falsely elevating the ratio. Dr. Frank suggests that one way to avoid this is to use a calculation consisting of a separately measured total lung capacity minus the anatomic dead space to substitute for the VA measurement. This maneuver helps him to interpret the ratio. Dr. Johnson has developed equations from his study2 on the effect of lung volume changes on the DLCO in 24 healthy subjects that appear to improve the volume correction for the DLCO. He also advocates that the DLCO/VA ratio should be volume corrected as well. These adjustments improve his ability to interpret diffusion. Further studies on the use of Dr. Johnson’s equations would be helpful to determine whether these adjustments of the DLCO, VA, and DLCO/VA ratio would improve their clinical utility.

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DOI: 10.1378/chest.08-2915