Response

To the Editor:

We thank Drs Dhillon and Berim for their interest in our article published in CHEST 1 on the release of metal particles from Vizishot needles (Olympus Ltd) used for endobronchial ultrasound-guided transbronchial needle aspiration. We believe it is important for users to report their experiences. Most bronchoscopists similarly describe a progressive increase in resistance encountered during the insertion and removal of the stylet into the target during endobronchial ultrasound-guided transbronchial needle aspiration procedures with Vizishot needles. The report by Dr Dhillon and Dr Berim is very interesting because they observed, by the naked eye, dark material on wet gauze used to clean the stylet, even before the stylet had been used. This observation strengthens the hypothesis that there is friction between the stylet and the channel in the needle. The use of smaller stylets may help avoid this friction. For such medical equipment, specifications regarding quality control on the potential release of metal particles would be necessary before the company would be granted approval from the authorities. However, to the best of our knowledge, this is not as yet a requirement.

Valérie Gounant, MD
Jocelyne Fleury-Feith, MD, PHD
Paris, France

Affiliations: From the Service de Pneumologie et Réanimation (Dr Gounant), the Service de Chirurgie Thoracique (Dr Gounant), and the Service d’Histologie et Biologie Tumorale (Dr Fleury-Feith), Hôpital Tenon, Assistance Publique-Hôpitaux de Paris, Faculté de Médecine, Université Pierre et Marie Curie.

Financial/nonfinancial disclosures: The authors have reported to CHEST that no potential conflicts of interest exist with any companies/organizations whose products or services may be discussed in this article.

Correspondence to: Valérie Gounant, MD, Hôpital Tenon 4, rue de la Chine, 75020, Paris, France; e-mail: valerie.gounant@tnn.aphp.fr

© 2012 American College of Chest Physicians. Reproduction of this article is prohibited without written permission from the American College of Chest Physicians (http://www.chestpubs.org/site/misc/reprints.xhtml).

DOI: 10.1378/chest.11-2316

Reference


Obesity

Another Good Reason to Question the Fixed FEV1/FVC Ratio When Diagnosing COPD

For every complex problem, there is a simple solution that is wrong.

G.B. Shaw

To the Editor:

The obesity epidemic does not pass by our (also growing) population of COPD patients. In their recent article in CHEST (August 2011), O’Donnell and coworkers1 analyzed the lung volumes of adult subjects who visited their lung function laboratory and who suffered from airflow obstruction based on the GOLD (Global Initiative for Chronic Obstructive Lung Disease) criterion (postbronchodilator FEV1/FVC < 0.7). One of their main conclusions was that the FEV1/FVC ratio is strongly influenced by the lung-volume-reducing effect of increasing weight. We have found similar results in earlier published data.2

So, again, we may have a problem with the use of the FEV1/FVC ratio to diagnose COPD. Not only will the use of the fixed (ie, 0.7) value of the FEV1/FVC ratio overdiagnose COPD in elderly subjects,3 it may also underdiagnose the presence of airflow obstruction in obese individuals. This is an important finding, considering the fact that 29% of O’Donnell’s study population was obese.

However, the true magnitude of this problem is likely to be underestimated in the study. The study population consisted of adults who had a postbronchodilator FEV1/FVC < 0.7. If increasing weight raises the FEV1/FVC ratio, obese subjects are more likely to be wrongly classified as having no airflow obstruction and may thus have been underrepresented in the study. This is an important issue for further research.

For clinical practice, the assessment of COPD in obese patients is further complicated by the fact that dyspnea (the key symptom of COPD) may also be caused by carrying excessive body mass.4 The combination of a higher FEV1/FVC ratio and dyspnea that may also be related to obesity may easily result in underdiagnosis of COPD in obese individuals. This could lead to inadequate weight reduction treatment decisions because, according to the obesity paradox,5 patients with severe COPD may even benefit from their excessive body mass in terms of survival. Diagnosing COPD is a complex process that is further complicated by comorbid conditions like obesity, and, again, a simplified method of defining airflow obstruction based on a fixed ratio (ie, FEV1/FVC) seems inadequate.

Lisette van den Bent, PhD
Tjard Schermer, PhD
Nijmegen, The Netherlands

Affiliations: From the Department of Primary and Community Care, Centre for Family Medicine, Geriatric Care and Public Health, Radboud University Nijmegen Medical Centre.

Financial/nonfinancial disclosures: The authors have reported to CHEST that no potential conflicts of interest exist with any companies/organizations whose products or services may be discussed in this article.

Correspondence to: Lisette van den Bent, PhD, Department of Primary and Community Care, Centre for Family Medicine, Geriatric Care and Public Health, Radboud University Nijmegen Medical Centre (Route 117), PO Box 9101, 6500 HB Nijmegen, The Netherlands; e-mail: L.vandenbent@elg.umcn.nl

© 2012 American College of Chest Physicians. Reproduction of this article is prohibited without written permission from the American College of Chest Physicians (http://www.chestpubs.org/site/misc/reprints.xhtml).

DOI: 10.1378/chest.11-2056

References

Response

To the Editor:

We thank Drs van den Bemt and Schermer for their thoughtful comments on our recent article in CHEST® on the confounding influence of increased body mass on the diagnosis of COPD. Given the dramatic global increases in the prevalence of both obesity and COPD over the past decades, we can no longer neglect the impact of this combination on common pulmonary function measurements and their clinical interpretation. The interaction of COPD and obesity is complex and poorly understood, given the vast pathophysiologic heterogeneity of both conditions.

COPD essentially remains a clinical diagnosis based on the triad of smoking (or other noxious gas) exposure, the presence of persistent respiratory symptoms, and the objective demonstration of airflow obstruction that is not fully reversible. The definition of airflow obstruction based on postbronchodilator fixed FEV<sub>1</sub>/FVC ratio < 0.7 has been criticized because of the risk of underdiagnosis in the elderly and underdiagnosis in the young. Less attention has been given to the risk of underdiagnosis of COPD in obese smokers by fixed ratio criteria, given the documented exponential decline in thoracic gas volumes (the denominator) with increasing BMI.<sup>1,4</sup>

Diagnosis of COPD is further confounded in overweight individuals by uncertainty concerning the specific origin of their respiratory symptoms. Thus, activity-related dyspnea in the obese smoker could be explained by factors other than airflow obstruction: higher ventilatory demands as a result of higher metabolic requirements of the physical task, skeletal muscle deconditioning due to decreased activity, or increased mechanical loading of the respiratory muscles due to decreased respiratory system compliance.<sup>3</sup>

The solution to this complex diagnostic dilemma is not simple. Reliance on a lower limit of normal criteria for FEV<sub>1</sub>/FVC based on population norms (instead of fixed ratio criteria) may not be the answer because the prevalence of obesity may be underestimated in the existing reference populations from which normative data were derived. Moreover, predictive equations for plethysmographic lung volumes have accounted for the influence of age, height, and sex but not of BMI per se. Future prospective population studies are urgently required to better clarify how the presence of obesity affects the diagnosis of COPD.

Denis E. O’Donnell, MD, FCCP
Katherine A. Webb, MSc
Jordan A. Guenette, PhD
Kingston, ON, Canada

© 2012 American College of Chest Physicians. Reproduction of this article is prohibited without written permission from the American College of Chest Physicians (http://www.chestpubs.org/site/misc/reprints.xhtml).

DOI: 10.1378/chest.11-2904

Macrolides in Asthma

To the Editor:

The study by Peters et al<sup>1</sup> (August 2011) demonstrating Mycoplasma pneumoniae community-acquired respiratory distress toxin in subjects with refractory asthma, provides a stronger rationale for use of macrolide in this setting, although it was not possible to eradicate this in all subjects. Could this toxin’s presence and its sometimes failed eradication potentially explain the observed, yet often unexplained and unpredictable, benefits of prolonged macrolide therapy in cases of refractory asthma when there is no apparent reflux or bronchiolitis? This would add further weight to the proposed mechanism of treatment of atypical infection<sup>2</sup> in addition to the armamentarium of well-known prokinetic<sup>1</sup> and antiinflammatory action<sup>2</sup> of these drugs.

Andrew R. L. Medford, MBChB, BSc(Hons), DM, FCCP
Westbury-on-Trym, Bristol, England

Affiliations: From the North Bristol Lung Centre, Southmead Hospital.
Financial/nonfinancial disclosures: The author has reported to CHEST that no potential conflicts of interest exist with any companies/organizations whose products or services may be discussed in this article.
Correspondence to: Andrew R. L. Medford, MBChB, BSc(Hons), DM, FCCP, North Bristol Lung Centre, Southmead Hospital, Westbury-on-Trym, Bristol, BS10 5NB, England; e-mail: andrewmedford@hotmail.com

© 2012 American College of Chest Physicians. Reproduction of this article is prohibited without written permission from the American College of Chest Physicians (http://www.chestpubs.org/site/misc/reprints.xhtml).

DOI: 10.1378/chest.11-2364

REFERENCES