but this needs to be informed by the “science” of medicine. I agree with Dr Cunha that we must not lose the “art” of medicine, and an exacerbation of asthma. We, therefore, wholeheartedly support his view that the results of our study in light of the pretest probability. Indeed, in the absence of microbiological and serologic evidence that sputum eosinophilia helps to identify patients who will respond to corticosteroids and targeted anti-IL-5 therapy.12 Likewise in asthma, there is increasing evidence that sputum eosinophilia helps to identify patients who will respond to corticosteroids and targeted anti-IL-5 therapy.3 In our study of the patients admitted to the hospital with pneumonia or exacerbations of asthma or COPD,1 we found that in addition to those patients with pneumonia, a large proportion of patients with asthma or COPD received antibiotics. Current guidelines do not advocate antibiotics for asthma exacerbations, although there is evidence to suggest that macrolide antibiotics hasten the rate of recovery.4 Most hospitalized patients with exacerbations of COPD fulfill current clinical guidelines to receive antibiotic therapy, but the benefit is relatively small and likely to be limited to a subgroup within those with more severe exacerbations. This is demonstrated in studies that have successfully reduced antibiotic usage in an acute setting by using procalcitonin levels to direct clinical decision making without increased adverse events in those patients not treated with antibiotics.5 The application of biomarkers such as procalcitonin or C-reactive protein add value beyond standard clinical care, and in our study,4 the modified early warning score, which is a composite of clinical assessment, was a poor discriminator between patients with pneumonia and an exacerbation of asthma. We, therefore, wholeheartedly agree with Dr Cunha that we must not lose the “art” of medicine, but this needs to be informed by the “science” of medicine.

REFERENCES


Response

To the Editor:

We thank Dr Cunha for his interest in our study on the potential role of procalcitonin and C-reactive protein in the management of exacerbations of airways disease.1 We agree with Dr Cunha that biomarkers should complement rather than replace good clinical practice and that results from biomarkers should be considered in light of the pretest probability. Indeed, in the absence of good clinical judgment, biomarkers may impair rather than facilitate decision making. Biomarkers are particularly valuable in helping to stratify risk, where the management strategy is uncertain or controversial, and to direct therapy. For example, in the management of chronic heart failure, directing therapy based on peripheral blood levels of brain natriuretic hormone reduces all-cause mortality.2 Likewise in asthma, there is increasing evidence that sputum eosinophilia helps to identify patients who will respond to corticosteroids and targeted anti-IL-5 therapy.3 In our study of the patients admitted to the hospital with pneumonia or exacerbations of asthma or COPD,1 we found that in addition to those patients with pneumonia, a large proportion of patients with asthma or COPD received antibiotics. Current guidelines do not advocate antibiotics for asthma exacerbations, although there is evidence to suggest that macrolide antibiotics hasten the rate of recovery.4 Most hospitalized patients with exacerbations of COPD fulfill current clinical guidelines to receive antibiotic therapy, but the benefit is relatively small and likely to be limited to a subgroup within those with more severe exacerbations. This is demonstrated in studies that have successfully reduced antibiotic usage in an acute setting by using procalcitonin to direct clinical decision making without increased adverse events in those patients not treated with antibiotics.5 The application of biomarkers such as procalcitonin or C-reactive protein add value beyond standard clinical care, and in our study,4 the modified early warning score, which is a composite of clinical assessment, was a poor discriminator between patients with pneumonia and an exacerbation of asthma. We, therefore, wholeheartedly agree with Dr Cunha that we must not lose the “art” of medicine, but this needs to be informed by the “science” of medicine.

REFERENCES


Conventional Ventilation vs Protective Strategies for Thoracic Surgery

The Results May Be Too Good to Be True

To the Editor:

In a recent issue of CHEST (March 2011), Yang et al demonstrated a surprisingly clear benefit of protective-ventilation strategy compared with conventional ventilation (CV) for thoracic surgical procedures in a small sample of patients. With 50 patients in each study arm, they demonstrated a 22% rate of pulmonary dysfunction in the conventional group vs 4% in the protective-ventilation cohort. Yet, serious problems with randomization and study design cast some doubts over the suggestive results. In the CV group, the majority of the procedures (>50%) were performed by one surgeon. In addition, in the same group, <40% of the patients (vs 60% in the protective ventilation group) had an epidural for postoperative pain management, making the two groups almost incomparable.1 Furthermore, in 30% of the patients in the CV group, a change in ventilation mode was necessary for

Correspondence

Affiliations: From the Institute for Lung Health, University of Leicester, and Department of Infection, Immunity and Inflammation, Glenfield Hospital.

Financial/nonfinancial disclosures: The authors have reported to CHEST the following conflicts of interest: Dr Brightling has received consultancy fees from MedImmune, AstraZeneca, GlaxoSmithKline, and Roche, and research grants from AstraZeneca, MedImmune, and GlaxoSmithKline. Dr Bafadhel has reported that no potential conflicts of interest exist with any companies/organizations whose products or services may be discussed in this article.

Correspondence to: Christopher E. Brightling, PhD. Institute for Lung Health, University of Leicester, Department of Infection, Immunity and Inflammation, Clinical Sciences Wing, Glenfield Hospital, Groby Rd, Leicester LE3 9QE, England; e-mail: ceb17@le.ac.uk

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DOI: 10.1378/chest.11-2492

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the continuation of the procedures. Too many variables, such as FiO₂, positive end-expiratory pressure, mode of ventilation, postoperative pain management, open vs video-assisted operation, and so forth, make a statement about the advantage of one strategy over the other nearly impossible. Although Yang et al. intended to show some evidence in favor of a protective strategy, the jury on this issue is still out.

Alimorad Djalali, MD, PhD
Stanford, CA

Affiliations: From the Department of Anesthesia, Stanford University.

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: Alimorad Djalali, MD, PhD, Department of Anesthesia, Stanford University, 300 Pasteur Dr, Room H3687, MC 3640, Stanford, CA 94305; e-mail: adjalali@stanford.edu

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DOI: 10.1378/chest.11-1791

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Response

To the Editor:

We thank Dr Djalali for his interest in our recent article1 and would like to respond to his questions. First, the sample size is small (n = 50 in each group): There have been no reports, to our knowledge, comparing the incidences of PAO₂/FiO₂ < 300 mm Hg and pulmonary complications between the two ventilation strategies; the sample size was calculated based on previous data, which showed a difference in postoperative PAO₂/FiO₂ between the conventional strategy and protective strategy (PV) groups; and 47 subjects in each group were required.

Second, as to the comment about having too many variables (different FiO₂, tidal volume, positive end-expiratory pressure, mode of ventilation), there are already a number of reports that used a single element or two elements of PV strategy to see the effect of each element in relation to lung injury.1,2,3 We applied most of the known elements of PV strategy (small tidal volume, low airway pressure and FiO₂, application of positive end-expiratory pressure) to see the total effect of PV strategy. Therefore, including many variables was essential for our study.

Third, as to the problem in randomization (differences in surgeons, postoperative pain control methods, operation methods), those variables were not statistically different between the groups. However, we agree that all these factors may have affected the results to some degree. More strict control of these variables is ideal, and we will do that in future studies.

Finally, as to the question about changing ventilation mode to pressure control in 30% of patients of the conventional strategy group: To keep the peak inspiratory pressure (PIP) < 30 mm H₂O, which was our protocol, we changed ventilation mode to pressure control in those patients who exceeded this limit. Even though these patients got the advantage of reduced PIP compared with their original values, the benefit of PV was still apparent in our study. These patients were included to show the benefit of pressure control mode in PIP. We hope our answers to the questions posed are helpful.

Alimorad Djalali, MD, PhD
Hyun Joo Ahn, MD, PhD
Seoul, Republic of Korea

Affiliations: From the Samsung Medical Center, Sungkyunkwan University, School of Medicine-Anesthesiology and Pain Medicine.

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: Hyun Joo Ahn, MD, PhD, Samsung Medical Center, Sungkyunkwan University, School of Medicine-Anesthesiology and Pain Medicine, 50, Ilwon-Dong, Kangnam-Gu, Seoul 135-710, Republic of Korea; e-mail: hyunjooahn@scknu.ac

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DOI: 10.1378/chest.11-1943

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Diagnostic Performance of Percutaneous Core-Needle Lung Biopsy Under CT Scan Fluoroscopic Guidance for Pulmonary Lesions Measuring ≤ 10 mm

To the Editor:

We know of two previous reports that have focused on the diagnostic performance of CT scan-guided fine-needle aspiration biopsy of pulmonary lesions measuring ≤ 10 mm.1,2 To our knowledge, however, the diagnostic accuracy of CT scan fluoroscopy-guided core-needle biopsy (Fig 1) for pulmonary lesions measuring ≤ 10 mm has not been evaluated.

We retrospectively identified 73 patients who underwent percutaneous core-needle lung biopsy under CT scan fluoroscopic guidance for pulmonary lesions measuring ≤ 10 mm between October 2002 and June 2009. The biopsy specimen results as well as the final diagnoses were available in 50 of these patients, and the results were compared (one lesion per patient). The diagnostic performance was also compared according to the lesion size

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