In recent years, we have developed an alternative approach when teaching to our housestaff trainees thoracocentesis that does not rely on chest percussion, namely, clavicle tapping with posterior chest auscultation. Stated simply, this technique takes advantage of the sound transmission characteristics of the inflated lung and the loss of sound transmission caused by the intersection of a layer of fluid between the air-filled lung and the chest wall. A steady tapping of the clavicle anteriorly by the examiner, who is positioned posteriorly reaching over the ipsilateral shoulder, generates a repeating sound that is well-transmitted through the lung to the posterior chest wall and that is readily appreciated via a stethoscope. The stethoscope then is moved slowly inferiorly from the apex to the base with each appreciated tap to the level below which no further conducted sound can be appreciated. This place represents the level of the superior aspect of the pleural effusion.

In our experience, this technique correlates well with chest wall percussion performed by experienced examiners, is readily taught and learned, and has increased the comfort of our housestaff with thoracocentesis.

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Alveolar Hemorrhage Associated With Antiphospholipid Syndrome

To the Editor:

Santos-Ocampo et al (October 2000) reviewed seven patients with systemic lupus erythematosus (SLE) admitted to the hospital with episodes of alveolar hemorrhage (AH). Recently, we published a case report of multiple organ dysfunction with acute respiratory failure associated with AH associated with antiphospholipid antibodies in a 42-year-old woman with a medical history of antinuclear antibodies-negative SLE and antiphospholipid syndrome (APS). The term antiphospholipid syndrome was introduced to describe patients presenting with combination of recurrent arterial and venous thrombosis, recurrent fetal loss, often accompanied by thrombocytopenia, and elevations of serum antiphospholipid antibodies, e.g., lupus anticoagulant (LA) and anticardiolipin antibodies. APS may present as a primary disorder in patients without other medical history of cerebral vascular accident from APS. We are interested to know if all patients were tested for antiphospholipid antibodies, e.g., LA and anticardiolipin antibodies or anti-β2-glycoprotein I antibodies, and AH could also be the result of APS in some of the patients.

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References


Lost in the Labyrinth of End Points

To the Editor:

We feel that the attack on our article in CHEST (October 2000) by Kaas and Barter in their editorial is unjustified on the basis of the data that have been presented. If they had taken care to read the “Materials and Methods” section carefully, they would have clearly seen that the primary outcome variable was the effect on bronchial hyperresponsiveness to adenosine monophosphate challenge, which is a suitable surrogate for mast cell-mediated airway inflammation. On the basis of this end point, there was significant superiority of monotherapy with budesonide over formoterol after 4 weeks, amounting to a 2.5-fold difference. A 1 doubling dilution shift (ie, twofold) in bronchial hyperresponsiveness is usually taken as being a clinically relevant effect. Likewise, there was also a significant difference when comparing these two therapies in the suppression of exhaled nitric oxide, which is another airway inflammatory surrogate. We did not show any difference between combination therapy and budesonide monotherapy for either of these surrogates. We did not show any difference between combination therapy and budesonide monotherapy for either of these surrogates inflammatory markers, suggesting that any improvements in control with combination therapy are simply due to bronchodilatory activity. For Kaas and Barter to concentrate their editorial on end points of lung function such as FEV1 and peak expiratory flow, which are insensitive in patients with mild-to-moderate asthma, and which clearly were not chosen as the a priori primary efficacy variable, is to miss the whole rationale of the study, even though we clearly stated the emphasis on the inflammatory markers in the title and in the introduction. Indeed, it has been shown previously that the dose response for inhaled budesonide is much steeper for effects on bronchial hyperresponsiveness than on lung function in patients with mild-to-moderate asthma. To properly evaluate the effects on lung function would require hundreds of patients with more severe asthma, as has already been done in pharmaceutically sponsored multicenter studies.

It was interesting that our patients preferred the treatment regimens that contained formoterol, perhaps suggesting that they had acquired a taste for its rapid effect on airway caliber. We believe

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that this response may alert patients into a false sense of security, especially if they receive a suboptimal dose of an inhaled steroid before considering adding in the long-acting β2-agonist. It was evident from a multicenter study that optimizing the dose of budesonide alone to 800 μg/d had a significantly greater impact on severe exacerbations than did the addition of formoterol to budesonide, 200 μg/d (ie, a 49% vs 26% reduction).4

This is especially pertinent as two different fixed-dose combination therapy inhalers (fluticasone-salmeterol and budesonide-formoterol) will soon be available in the United States. These are being aggressively marketed for use in patients with all grades of asthma when there is ample evidence to show that a low-to-medium dose of an inhaled steroid will suffice in most cases of mild-to-moderate asthma. While there is convincing independent evidence to support the use of long-acting β-agonists as steroid-sparing agents in cases of more severe persistent asthma, these studies had a duration of only 6 to 12 months and have not evaluated bronchial biopsy, especially over a longer period of several years to see whether using a lower dose of inhaled steroid is associated with irreversible airway damage due to putative airway remodeling. Until such long-term data are available, it makes more sense to use long-acting β2-agonists on a as-required basis rather than on a regular basis, once the dose of inhaled steroid has been optimized, perhaps using bronchial hyper-reactiveness in addition to other control markers to titrate the dose, as suggested by the study of Sont et al.5 We never intended to make sweeping implications on the basis of the small study, but we hope it will make prescribers appraise their choices for each individual patient more carefully.

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1 Aziz I, Wilson AM, Lipworth BJ. Effects of once daily formoterol and budesonide given alone or in combination on surrogate inflammatory markers in asthmatic adults. Chest 2000; 118:1049–1053

Lung Cancer Clarifications

To the Editor:

In the article by Minami et al (December 2000),1 in Table 1, in the “Complete Resection” line, the numbers that are shown (249 [79.6] and 654 [85.2]) seem to be erroneous. I get 72.2 and 73.8. Am I missing something or can you please explain? This is a key figure in the study.

In the article by Asaph et al (December 2000),2 in Table 1, the line under “Bronchoscopy” that reads “negative” shows a figure of 15 (41); on page 1623, line 7, the following statement appears: “second neoplasm was confirmed in 15 patients (40.5%; Table 1).” If it was confirmed, it should be positive. Is the figure correct in the table or is the text worded correctly?

I appreciate your assistance.

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To the Editor:

We thank Ms Wald for her close attention on our article.1 The number she points out is not an error, but it is easily misunderstood due to lack of the words “without induction therapy.” Complete resection was estimated in 249 of 313 women and in 654 of 768 men without induction therapy. We calculated the percentage of complete resection in 249/313 (79.6%) and 654/768 (85.2%) for the patients without induction therapy, not 249/337 or 905/954.

The main finding is that T3 (malignant effusion) in lung cancer is encountered more often in women than in men. Malignancy cannot be judged correctly only from completely resected cases

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To the Editor:

Thanks to Ms. Wald for careful reading of our article.1 In fact, Table 1 of that article is correct, in that 13 positive bronchoscopies were found in 28 total bronchoscopies. This finding reflects the earlier-stage patients who have peripheral nodules and who are submitted to bronchoscopy to help separate this histology from the original. We will submit an erratum (page 325) to change the text to read “in which the second neoplasm was confirmed in 13 patients (46% of those receiving bronchoscopy).”

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