Communications to the Editor

Communications for this section will be published as space and priorities permit. The comments should not exceed 350 words in length, with a maximum of five references; one figure or table can be included. Exceptions may occur under particular circumstances. Contributions may include comments on articles published in this periodical, or they may be reports of unique educational character. Specific permission to publish should be cited in a covering letter or appended as a postscript.

Tidal Volume Reduction in ARDS

To the Editor:

In the May 1991 issue of Chest, Leatherman and associates demonstrated the beneficial effect on cardiac output and overall oxygenation of modest tidal volume reduction (200 ml) within the 10 to 15 ml/kg range in patients with severe ARDS who require high-level positive end-expiratory pressure (15 cm H2O). In seven of 20 patients the PaO2 did actually rise after reduction of tidal volume, whereas the PaO2 fell in 13, five of whom had a fall of more than 20 mm Hg. As shown in that article, the modest correlation between change in cardiac output and total respiratory system compliance (Cst) precludes the use of a noninvasive parameter like Cst for screening out the seven patients who benefited most due to reduction in tidal volume. Overdistention of normal alveoli, especially on the plateau portion of the oxygen dissociation curve, is harmful in terms of barotrauma and systemic hypotension without much gain in terms of capillary oxygenation in the alveoli concerned. As Leatherman et al discussed, this must be one of the important causes of improved oxygenation following tidal volume reduction. Capillary compression and elongation leading to pulmonary hypertension and longer tidal volume times can also cause sluggish systemic circulation and extensive venous desaturation.

If this inhomogeneity in lung involvement could be screened by some means, then a clinical algorithm might be defined, which in this case would screen out these seven patients, as well as probably a few in the borderline group (with a minimal fall in PaO2 after tidal volume reduction) for trial and subsequent treatment with tidal volume reduction. Overdistention and hyperinflation of selected alveoli cause a widening of the alveoloarterial or end-tidal to arterial CO2 gradient. It would be interesting to know in this experiment, a cutoff point exists for the P(A-a)CO2 above which PaO2 predictably improves with reduction of tidal volume. An otherwise good correlation between Cst and cardiac output in these particular alveoli can get muffled when the whole lung, with its wide spectrum of ventilation-perfusion inequalities, is considered as a unit, but the CO2 gradient should still remain sensitive to differential CO2 elimination between alveoli.

Another important cause of worsening PaO2 in alveolar distention is surfactant depletion due to hysteresis, motion of the fluid film being greater in inspiration than in expiration. Hence, it would also be interesting to know whether the inspiratory-expiratory ratios had any significant correlation with outcome. Support of this theory would be provided by worsening PaO2 in the distended alveoli, which have an inspiratory time much shorter than the expiratory time. If the inspiratory-expiratory ratios and end-tidal CO2 were taken into consideration, we might have a better correlation between cardiac output and Cst in the subgroups thus created. We would like to know whether this would help in forming a clinical algorithm in the interesting and common situation described.

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REFERENCES

5. Faridy EE. Effect of ventilation on movement of surfactant in airways. Respir Physiol 1976; 27:323-34

Role of Transbronchial Biopsy in the Diagnosis of Cavitary Peripheral Pulmonary Lesions

To the Editor:

We reviewed the records of 109 consecutive nonimmunocompromised patients who underwent transbronchial biopsy (TBB) during fluoroscopically guided fiberoptic bronchoscopy for a cavitary peripheral pulmonary lesion (CPPL). In our series, unlike the findings in patients with dense nodules, more patients had nonmalignant lesions, and cancer was detected as frequently as pulmonary tuberculosis. A positive biopsy specimen with diagnostic histologic findings was obtained in 85 (78 percent) patients (Table 1).

We believe that such a high diagnostic yield of TBB in CPPL may be attributed to the fact that in these lesions there is a relationship

<table>
<thead>
<tr>
<th>Table 1 — Results of 85 Diagnostic Transbronchial Biopsies</th>
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<tbody>
<tr>
<td>Histologic Diagnosis</td>
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<tr>
<td>--------------------------------</td>
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<tr>
<td>Malignant (n = 29 [34%])</td>
</tr>
<tr>
<td>Squamous</td>
</tr>
<tr>
<td>Adenocarcinoma</td>
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<td>Large cell</td>
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<tr>
<td>Small cell</td>
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<tr>
<td>Metastatic</td>
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<tr>
<td>Nonmalignant (n = 56 [66%])</td>
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<tr>
<td>Tuberculosis</td>
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<tr>
<td>Mycetoma</td>
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<tr>
<td>Abscess</td>
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<tr>
<td>Radiation pneumonitis</td>
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<td>Wegener's granulomatosis</td>
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between the cavity and the bronchus. Peripheral dense nodules, especially nonmalignant ones, are not frequently in direct communication with airways, and the success of TBB depends on the possibility of reaching a bronchus that will lead forward or into the lesion.  

We conclude that TBB during fiberoptic bronchoscopy appears to be an effective method for investigation of CPPL.

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REFERENCES

Utility of Admission Chest Radiography in Management of Acute Asthma in Adults

To the Editor:

In the July 1991 issue of Chest, White et al describe the utility of admission chest radiography in the management of adults admitted to the hospital with acute asthma. They conclude their article with a recommendation for routine chest radiography in this patient group.

As a radiologist, I am concerned with several aspects of this report. Two radiographic findings that were significantly associated with subsequent antibiotic use were described as "focal opacity" and "increased interstitial markings." In a subset of patients, the presence of these opacities appeared to prompt antibiotic prescription, even in the absence of clinical indicators of infection. I do not know what pathologic findings these descriptors are intended to indicate. With respect to physiologic abnormalities characteristic of asthma, focal opacities may represent areas of focal atelectasis and/or bronchial mucus plugs. I believe that neither of these entities, as isolated radiographic findings, necessitates antibiotic therapy. Bacterial pneumonia appears on a radiograph as air-space consolidation, typically segmental or lobar in distribution. Apparently, this finding was not described in any patient in the authors' series. Parenchymal infiltrative disease that may result in abnormal "interstitial" opacities is not a feature of asthma.

I accept the fact that some patients with these opacities were subsequently treated with antibiotics. However, we do not know which patients actually had bacterial pneumonia, if any. Some may have had acute bacterial bronchitis, but this disorder does not produce parenchymal opacities. As physicians, we should attempt to determine which diagnostic and management strategies actually improve clinical outcome before recommending "routine" diagnostic tests. Although this was obviously not the specific intent of the authors' investigation, we should view their recommendation with appropriate skepticism.

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REFERENCE
1 White CS, Cole RP, Lubetsky HW, Austin JHM. Acute asthma: admission chest radiography in hospitalized adult patients. Chest 1991; 100:14-6

To the Editor:

In Dr Mann's letter, he takes issue with our use of the terms "focal opacity" and "increased interstitial markings." The descriptor "focal opacity" was intended to be inclusive of both lobar and segmental air-space disease. We cannot exclude the possibility that a component of atelectasis may have been present. None of the opacities was typical of mucus plug, which has a characteristic branching pattern. The descriptor "increased interstitial markings" was intended to be inclusive of a reticular infiltrate, a pattern that is well described in Mycoplasma pneumonia. Many of these patients were treated with erythromycin.

Ideally, it would be useful to know which patients actually had bacterial pneumonia. However, in the emergency ward setting, this information is rarely available. The intent of our study was to demonstrate the effect of the radiographic findings available at the time on the immediate management of the patient.

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REFERENCE

Pneumothorax Due to Pleural Perforation of a Pseudocavity Containing Aspergillomas in a Patient with Allergic Bronchopulmonary Aspergillosis

To the Editor:

A 22-year-old man was hospitalized for treatment of a cavitary lesion complicated by pneumothorax. He had a history of childhood asthma, which remitted during adolescence. One year previously he developed a cough, which produced purplish phlegm sometimes accompanied by plugs of dark material. A chest radiograph revealed tram lines and tubular shadows in the left upper lobe. Fiberoptic bronchoscopy and serologic tests confirmed a diagnosis of allergic bronchopulmonary aspergillosis (ABPA), for which the patient received corticosteroid therapy.

A cavitary lesion that evolved in the left upper lobe exhibited two unusual features: the lesion lacked continuity in its inferomedial aspect, and excrescences protruded from its inner wall. A computed tomographic (CT) scan, performed during a hospitalization for symptomatic pneumothorax, revealed central bronchiectasis and a cavity that contained three discrete masses (Fig 1).

The patient underwent an uncomplicated lobectomy while receiving perioperative amphotericin B therapy to a total dose of 145 mg. The pathologic findings were those of ABPA; the "cavity," which had perforated the pleural surface, was an aneurysmal expansion of an ectatic central bronchus that contained several aspergillomas.

Aspergilloma complicating ABPA has been reported. The putative mechanism is focal dilatation of the ectatic bronchi, consequent to the accretion of materials produced by the immune response to