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 printed. 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.

Failure to Recover Chlamydia trachomatis from Open-Lung Biopsy Tissue of Adult Immunosuppressed Patients

To the Editor:

As part of a routine protocol for processing open-lung biopsy (OLB) tissue from immunosuppressed hosts, we have inoculated specimens from 245 patients (mean age 53 years, SD ±16 years, range 20-82 years) into cell cultures from March 1978 to September 1983 for the isolation of C trachomatis. We added 8 ml of nutrient broth to the OLB specimens (usually 1 cm³) and disrupted the cells of the tissue in a stomacher for 1 min. A portion of this suspension (0.2 ml) was placed in 25P transport medium and inoculated immediately into McCoy cell cultures.¹ None of the 245 specimens yielded C trachomatis in cell culture. Although a few iodine-staining inclusions were observed in one specimen, the agent could not be subpassaged. Further attempts to reisolate the organism from the original specimen extract frozen at ~70°C were unsuccessful.

Tack and associates¹ detected C trachomatis inclusions in cell cultures from sputum, bronchial brushings or lung tissue from six adults (four actively immunosuppressed) with bilateral interstitial infiltrates. Interestingly, none of these infected patients developed a serologic response to C trachomatis. More recently, C trachomatis (and cytomegalovirus) was isolated from lung biopsy tissue from a 35-year-old immunosuppressed patient with pneumonia. Although a serologic response to C trachomatis was demonstrated, the organism was not specifically characterized and identified according to serotype.²

Thus, documentation of the presence of C trachomatis from the upper respiratory tract of adults has, to date, been rare with the exception of a report of six isolates from 108 patients with par- yngitis³ and an isolate from a single sputum specimen from a bone marrow transplantation patient at the onset of pneumonia.⁵ In contrast to the well-established etiology for C trachomatis in children, we believe that definite etiologic proof for C trachomatis as a cause of respiratory infections in adults awaits the isolation, serial propagation, and specific serotypic identification of C trachomatis in this group of patients.

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REFERENCES
1 Smith TF, Brown SD, and Weed LA. Diagnosis of Chlamydia trachomatis infections by cell cultures and serology. Lab Med 1982; 13:92-100
5 Meyers JD, Hackman BC, Stamm WE. Chlamydia trachomatis infection as a cause of pneumonia after marrow transplantation. Transplantation 1983; 36:130-34

Potential False Positive Mediastinal Transbronchial Needle Aspiration in Bronchogenic Carcinoma

To the Editor:

We believe the enthusiasm demonstrated over use of the transbronchial needle aspiration (TBNA) technique developed by Dr. Ko-Pen Wang and associates should be tempered. In our opinion, surgical staging of the mediastium should be performed in most patients with non-small cell carcinoma until the specificity of positive transbronchial needle aspirates can be determined.

We are currently involved in a study utilizing computed tomography (CT)-directed TBNA in patients with presumed or documented non-small cell carcinoma of the lung. During the preliminary stages of the study, a false positive TBNA occurred in a patient with a right paratracheal mass and apparent mediastinal extension on chest CT (Fig 1). False positive is defined as a malignant TBNA and negative mediastinal surgical staging. Cervical mediastinal exploration (CME) and thoracotomy revealed no mediastinal tumor. The right upper lobe neoplasm, a poorly differentiated large cell carcinoma, "fell away" from the mediastinal pleura when the thoracotomy was performed. All surgical margins and lymph nodes sampled were free of tumor.

Normally, the right paratracheal region presents on chest radiographs as a thin stripe approximately 2-3 mm thick. This stripe may also exist more posteriorly in a retrotracheal position if lung inserts into the retrotracheal-esophageal recess which, as its lateral counterpart, is approximately 2-3 mm in thickness. TBNA procedures in the region of 3-6 o'clock (with 6 o'clock representing the posterior tracheal wall as viewed from the patient's head) may pierce the tracheal wall and mediastinal pleura, thereby sampling lung parenchyma. Dr. Wang has demonstrated the ability to diagnose malignant right paratracheal masses using the TBNA technique.⁶ Right upper lobe parenchymal masses that abut visceral pleura and factiously appear to have invaded the mediastinum on CT, may be biopsied by TBNA and may inappropriately be considered inoperable if the TBNA reveals malignant cytology.

A second clinical state in which a false positive TBNA might be obtained is with respiratory tract "contamination" by malignant cells, as demonstrated in patients with malignant sputum cytology. As Dr. Wang suggests, cellular material may spread within the airway following brushing, washing or biopsy. Accordingly, he recommends
performing TBNA prior to these procedures. In addition, Dr. Wang has stated that “the interpretation of a positive TBNA in patients who lack roentgenographic evidence of mediastinal disease represents a still more difficult problem, and further experience is necessary to clarify the significance of this finding.” We suggest that some of these patients may have false-positive TBNA secondary to “contamination” by aspirated endobronchial secretions.

We are concerned about the potential false positive TBNA, and therefore, have incorporated surgical staging (cervical or parasternal exploration, and mediastinal exploration at thoracotomy) in all patients in our study, regardless of TBNA results, to better assess the specificity of this technique. We feel it is premature to withhold surgical staging in many patients who demonstrate malignant cytology (non-small cell carcinoma) via TBNA. It is hoped that our study will better delineate the specificity of a positive TBNA.

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REFERENCES

“Stage Prop” Humor

To the Editor:

A recent commentary by Freedman et al (Chest 1984; 85:406-07), argued that screening for early stages of chronic obstructive pulmonary disease would cost this country at least $500 million and the likelihood of any benefit to large numbers of persons identified with early abnormalities would be small. Macklem’s response in the dialogue in the March issue of Chest came forward with resounding arguments that spirometry is as necessary to the practice of medicine as the sphygmomanometer and the clinical thermometer. Thus, the stage is set for laughter!

Freedman et al can’t possibly be serious! If they are serious, they have completely overlooked the fact that FEV1 abnormalities are highly predictive of premature morbidity and mortality from chronic obstructive pulmonary disease and that those with abnormality who stop smoking have a better outcome than those who continue (Peto et al. Am Rev Respir Dis 1983; 128:491-500). They must really be pulling our leg by being negative about spirometry because they completely left out the fact that the forced vital capacity was one of the best predictors of survival, including deaths from all causes in the Framingham study (Kanner et al. Tr Am Life Ins Med Dir 1980; 64:66-81). They continue to dwell on the small airways business and here they are right because no one really knows whether tests of small airways dysfunction are relevant to clinically significant COPD or not, although answers to this question will be forthcoming. They also completely omit the fact that knowledge of early pulmonary function abnormalities can be motivating factors in smoking cessation as recent studies have strongly suggested. Finally, they are excessive in their estimates of cost, since simple spirometric tests for FVC and FEV1 are commercially available for $5.00 in many parts of this country. Isn’t it worth at least $5.00 to be informed about prognosis for COPD and indeed prognosis for life?

In response, Macklem, who continues to be a mild opponent of the use of spirometry in screening, makes strong arguments for the use of spirometry in physicians’ offices for clinical purposes. Thus, an expert who is often blamed for retarding programs in the early identification and intervention of COPD (and other lung diseases) now argues eloquently for the widespread use of spirometry in clinical practice!

Thus, Freedman and colleagues become the classic “straight man” in the typical Abbott and Costello routine, “Who’s on first? What’s on second? and I don’t know on third.” An analogy to the routine in the old movie could be the present “players” in this comic routine who can be likened to patients seen in the doctor’s office. “Who is that person destined to premature morbidity and mortality from emerging chronic lung disease; who later becomes the patient requiring pulmonary rehabilitation techniques, home oxygen therapy, management of acute respiratory failure in intensive care units and/or home oxygen therapy. “What” could be likened to the 500,000 Social Security disability receipts alone, if all these advanced patients are placed on home oxygen at an approximate cost of $4,000 per year. This would cost $2 billion on oxygen services alone, to say nothing of physician’s fees, hospitalizations and intensive care unit stays. I’ll leave this argument there.

“I don’t know” is the physician sitting in his office wondering whether or not his coughing-smoking patient could be developing early states of chronic airflow limitation as a prelude to emerging chronic obstructive pulmonary disease. This same physician has 20 to 30 years available in order to intervene through smoking cessation and perhaps other pharmacologic manipulations.

Therefore, as one who has had some concerns about the problem of COPD and the lack of interest in the widespread use of spirometric testing for reasons which have never been explained, I spilled my morning cup of coffee all over my March issue of Chest from laughing so hard!

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To the Editor:

While our colleague was laughing violently, spilling coffee on our article and recalling with obvious pleasure his memories of watching Abbott and Costello routines, he apparently missed the gist of our