bacterial pneumonia who needed to be hospitalized in Singapore yielded strikingly similar results, with notable exceptions.2

From March to September 1991, consecutive patients who were admitted to National University Hospital with community-acquired pneumonia were studied. A total of 95 patients were recruited, and no organism was identified in 42 percent. The results that were strikingly similar to the findings of Chan et al are as follows: (a) Streptococcus pneumoniae accounted for 12 percent of our cases, compared with 12 percent in the study by Chan et al. (b) Mycobacterium tuberculosis was more common than expected (21 percent of cases in our study, compared with 12 percent in their study). (c) As in the study by Chan et al, no cases of pneumonia due to Legionella pneumophila were found. A notable difference from the findings in the study by Chan et al, as well as those in other series, was the higher incidence (10 percent) of Gram-negative organisms (excluding Haemophilus influenzae). In addition, M tuberculosis was the organism most commonly isolated, and patients with pulmonary tuberculosis had higher platelet counts than patients infected with other groups of organisms (377 × 10^9/L vs 253 × 10^9/L, p = 0.001).

Our studies thus support the concept of differences in local flora pattern in the etiology of community-acquired pneumonia. The importance of acute pneumonia due to M tuberculosis cannot be overemphasized in our local context. The higher incidence of Gram-negative organisms (excluding H influenzae) poses a difficult problem, as Gram-negative organisms were more resistant to antibiotics and thus more costly antibiotic therapy was necessary.

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REFERENCES
2 Hui KP, Chin NK, Chow K, Yeo TC, Kumaraisinghe G, Tan WC. Pattern of community-acquired pneumonia at National University Hospital, Singapore. Presented at the First Combined Scientific Meeting of the Singapore and Malaysian Thoracic Societies, Singapore, December 7-8, 1991

To the Editor:

We thank Dr. Hui and colleagues for their comments on our study. As they said, the incidence of pneumococcal pneumonia of 12 percent is remarkably low in Hong Kong and Singapore. We are not sure whether this is a true incidence or whether it was due to difficulties in diagnosis despite our use of the pneumococcal antigen assay. We believe that previous use of antibiotics was at least partially responsible for the lower incidence as compared to Western countries. Dr. Hui and colleagues did not mention what diagnostic methods they used in their study, and we would be interested in this. As expected, tuberculosis (TB) was common, but the incidence in Singapore seems to be higher than in Hong Kong. In our study, we rigorously excluded all cases in which TB was suspected, and our incidence of 12 percent consisted of TB cases that were clinically indistinguishable from cases of acute pneumonia due to other causes. Perhaps this might explain our lower incidence of TB. Nevertheless, the conclusion that TB often masquerades as acute pneumonia in the Far East seems inescapable. Although we did not find any cases of Legionnaires' disease, atypical pneumonia mainly due to Chlamydia species accounted for 9 percent of our cases. Is this the experience in Singapore also?
The higher incidence of Gram-negative organisms (10 percent) in Singapore is interesting. We have previously found a high prevalence of Gram-negative organisms in the lungs of patients with bronchiectasis.1 Is it possible that the relatively high incidence of Gram-negative organisms in the Singapore study may be due to acute pneumonia exacerbations in patients with bronchiectasis? If so, costly antibiotics directed at Gram-negative organisms should then be reserved for patients with acute pneumonia who also have evidence of coexisting bronchiectasis. We are against the use of extended antibiotic coverage for community-acquired pneumonia in the absence of other coexisting lung diseases.

In conclusion, we are encouraged by the study by Dr. Hui and colleagues supporting our main findings in community-acquired pneumonia in the Far East. The differences are interesting but seem to be minor. We look forward to reading their report in full.

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Fatal Diffuse Alveolar Injury Following Use of Intrapleural Bleomycin

To the Editor:

Bleomycin is an agent commonly used to cause pleurodesis as treatment for malignant and nonmalignant pleural effusions. Serious side effects have been described following systemic, but not intrapleural, administration.1 We present a case of fatal diffuse alveolar injury following its use for the induction of pleurodesis as therapy for a nonmalignant pleural effusion.

A 62-year-old woman with chronic renal failure developed a symptomatic right pleural effusion, which persisted despite hemofiltration and multiple thoracocenteses. She was admitted for elective chemical pleurodesis. Her initial chest radiograph confirmed the presence of a large right pleural effusion. Significant laboratory data included the following: blood urea nitrogen, 35 mg/dl; serum creatinine, 10.1 mg/dl; hemoglobin, 6.5 g/dl.

A 32F chest tube was placed in the right pleural space, draining 1,500 ml of clear yellow exudate (total protein, 5.4 g/100 ml; WBC, 220/ml; lactate dehydrogenase (LDH), 634 IU/L; fluid-serum protein ratio, 3.1; fluid-serum protein ratio, 0.7).

After 36 h of drainage, pleurodesis was attempted on each of 2 successive days with 60 units of bleomycin, using a previously described protocol.1 Twelve hours after the second instillation, the patient developed a fever of 38.7°C and leukocytosis (WBC, 10,600/ ml). Sputum and pleural fluid Gram stains showed no bacteria. Urine and blood cultures drawn then and subsequently were sterile.

Over the next 7 days, the patient became progressively hypoxic, ultimately requiring endotracheal intubation and mechanical ventilation. A diagnosis of diffuse alveolar injury was made on the basis of decreasing lung compliance, an increasing alveolar-arterial oxygen pressure gradient, and worsening bilateral diffuse alveolar infiltrates on chest radiographs despite continued aggressive dialysis. Doppler ultrasonography of both lower extremities revealed no evidence for deep venous thrombosis. Serologic examination for anti-nuclear and anti-basement membrane antibodies and Legg-ellit titters were all negative. Methylprednisolone for possible hypersensitivity pneumonitis and empiric broad-spectrum antibiotics were used; both modes of treatment were ineffective. The patient died on day 14 of her hospital stay. Her family did not consent to autopsy.

The association of intravenous bleomycin and acute respiratory distress syndrome is well documented.2 Although applied "topically" within the pleural cavity, up to 45 percent is absorbed systemically.3 Furthermore, being principally cleared by the renal route, its half-life is prolonged by decreased creatinine clearance.4 Our patient's renal failure increased its half-life and may have potentiated its toxicity,5 although the total dose instilled was corrected as recommended for renal failure.6 Other potential causes of lung injury, such as pulmonary emboli, drug reaction, infections, and autoimmune diseases, were excluded after extensive diagnostic studies. Bleomycin should be used with caution as a pleural sclerosing agent in patients with underlying renal disease.

5 Siegel RD, Schiffman FJ. Systemic toxicity following intracavitary administration of bleomycin. Chest 1990; 98:507

Thrombolytic Therapy for Pulmonary Embolism

To the Editor:

I read with interest the supplement to the April 1992 issue of Chest, in which the tone advocates the use of thrombolytic therapy for the treatment of pulmonary embolism.1 However, after 25 years of study, the major fundamental question has yet to be answered: does thrombolytic therapy decrease morbidity and/or mortality compared with heparin therapy in the management of pulmonary embolism? The Urokinase Pulmonary Embolism Trial of the 1970s does not support this.4 This study showed that although there was initially a more rapid rate of thrombus resolution, the degree of resolution was the same within 2 weeks, and thrombolytic therapy offered no mortality reduction. Recently, Dr. Goldhaber cited an abstract of data on 23 patients as evidence that thrombolytic therapy reduces morbidity, but an issue this important demands a more solid answer than these data provide.5 There is evidence that thrombolytic therapy improves right-sided hemodynamics, but it would be unwise to extrapolate this to mean clinical benefit for the patient.

Until thrombolytic therapy has been shown to offer true benefit