Communications by both cardiac Doppler examination and catheterization might have been misleading because of the low cardiac output. The correct diagnosis of critical aortic valve stenosis was possible only on the basis of estimation of the valve area by Doppler study, confirmed by cardiac catheterization and later at necropsy.

Our case underscores the fact that the only way to establish a proper diagnosis in patients with suspected aortic valve stenosis and a clinical picture characterized by congestive heart failure, low cardiac output, and absence of thickened left ventricular walls is to calculate valve area by noninvasive or invasive methods.

A multicenter prospective study would be worthwhile to reliably estimate the clinical relevance of this particular subgroup of patients.

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To the Editor:

We appreciate the positive and supporting comments of Cavalli, Volpi and Maggioni on the malignant subgroup of patients with critical aortic valve stenosis who fail to develop adequate compensatory left ventricular hypertrophy.

There are two major points that we would like to re-emphasize from our article. Patients who present with physical examination and echocardiographic findings of aortic stenosis should be evaluated with the following caution. A patient with critical aortic stenosis and a markedly reduced injection fraction may lack adequate compensatory left ventricular reserve and fail to demonstrate left ventricular hypertrophy. Furthermore, a cardiac Doppler finding of a small peak gradient is not uncommon. It has been our experience that many patients with these findings are thought to have a primary myopathy and are medically treated, only to expire. We certainly agree that, at the least, a noninvasive valve area should be calculated and, in most, cardiac catheterization should be undertaken. Since the mortality in this subgroup of patients with medical management is 100 percent, the only recourse is surgical intervention. Unfortunately, the cardiac surgical results are anecdotal from local community to university. These varying surgical results may be due to aortic stenosis with a mixture of reversible and irreversible myocardial dysfunction. For this reason, we are in agreement with the authors that a multicenter prospective surgical study might help clarify the factors which enter into the true surgical mortality of this interesting subset of patients.

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Therapeutic Failure due to Branhamella catarrhalis In Pneumococcal Pneumonia

To the Editor:

According to Wallace and Musher (Chest 1986; 90:447-50), the role of Branhamella catarrhalis (Bc) may be uncertain when recovered from sputum in association with other pathogens. We would like to report a case of pneumococcal pneumonia with poor response to penicillin due to mixed infection with a beta-lactamase-producing Branhamella catarrhalis (B-LPBC).

CASE REPORT

A 74-year-old man was admitted to hospital because of sudden onset of pleuritic chest pain, shortness of breath, fever and cough. Clinical examination and chest roentgenographic film revealed a right lower lobe pneumonia.

No bronchial secretions could be obtained on admission. The patient was treated with intravenous penicillin G (6 million units per day). Two days later, a good quality sputum sample yielded a pure culture of B-LPBC which was considered clinically nonsignificant as one blood culture drawn on admission grew a pure culture of penicillin-sensitive Streptococcus pneumoniae.

In spite of penicillin therapy, fever persisted with no clinical improvement.

On day 6, gram stain of a second sputum sample again showed numerous polymorphonuclear leukocytes and gram-negative cocci. Therapy was shifted to cotrimoxazole (SMX 400 mg), two tablets twice a day, and dramatic clinical improvement consequently occurred within 48 hours.

DISCUSSION

As Bc is an oropharyngeal commensal, its clinical significance in sputum sometimes remains questionable. However, mixed bronchopulmonary infections with either Hemophilus influenzae or Streptococcus pneumoniae are described in up to 30 percent of clinical infections in which Bc seems clearly implicated.1 In fact, recent literature reports suggest that, beside its own pathogenicity, B-LPBC can act as an indirect pathogen through production of β-lactamase.2,3 Both mechanisms could be advocated in the present case to explain the poor response to penicillin G and the subsequent dramatic response when cotrimoxazole was substituted. This suggests that, even in the case of well-documented pneumococcal pneumonia, the recovery of Bc from good quality sputum sample could no longer be disregarded.

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1 Slevin NJ, Aitken J, Thornley PE. Clinical and microbiological features of Branhamella catarrhalis bronchopulmonary infection. Lancet 1984; 2:782-83
4 Wardle JK. Branhamella catarrhalis as an indirect pathogen. Drugs 1986; 31:93-96
5 Brook I. Direct and indirect pathogenicity of Branhamella catarrhalis. Drugs 1986; 31:97-102

To the Editor:

Motte et al have raised an important point that has concerned a number of clinicians, and not just with regard to Branhamella catarrhalis. Failure to clear penicillin-susceptible pathogens with penicillin in mixed infections (which include a beta-lactamase-producing species such as bacteroides) is well recognized with intra-
abdominal abscesses and has been suggested for some cases of anaerobic lung abscess. That treatment failure with penicillin in this situation does not always occur is supported by the high degree of success of penicillin in lung abscess, including many of the 10 to 20 percent of cases in which *Bacteroides fragilis* has been proven to be present by transstrachael aspiration. Determination of which cases of mixed infections (which include both beta-lactamase-producing and non-producing species) will fail and which can be successfully treated with penicillin alone may be determined by a number of factors that may include the absolute numbers of each of the species, their individual virulence, the role of spontaneous or surgical drainage, and the natural history of the disease.

Similar penicillin failures have been described with penicillin-susceptible organisms in which the beta-lactamase-producing organism may not be producing disease, but is providing a safe environment for the real pathogen—the concept of "indirect pathogenicity." The best example of this is the high incidence of *Staphylococcus* and beta-lactamase-producing bacteroides species in the pharynx of patients with presumed streptococcal pharyngitis in whom cultures remain persistently positive, despite penicillin therapy. In the case described by Motte et al, these same kinds of concerns are being raised. Since *B. catarrhalis* is clearly present (by spumon examination) in the normally sterile lower respiratory tract, its importance could be both as a direct and an indirect pathogen. I and others have experienced clinical as well as microbiologic failures when penicillin or ampicillin has been used to treat acute bronchitis due to a mixed infection that included beta-lactamase-producing *B. catarrhalis*. In many of these cases, the penicillin-susceptible pathogen persisted, as did the *B. catarrhalis*. Although the number of cases of pneumonic disease with treatment failure that have been documented is smaller, the current case is an important reminder that they do occur. I suspect that some cases of mixed infection involving beta-lactamase-producing *B. catarrhalis* and the pneumococcus will respond to penicillin therapy also. However, since I cannot pick out those cases of co-pathogenicity or indirect pathogenicity where treatment failure will occur, it is probably most prudent to treat all potential pathogens recovered from the lower respiratory tract regardless of whether I believe one of the pathogens (eg, *St. pneumoniae*) is more likely the pathogen, or more pathogenic than *B. catarrhalis*.

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1 Panwalker AP. Failure of penicillin in anaerobic necrotizing pneumonia. Chest 1982; 82:500-01

Table 1—Frequency of Tuberculosis in AIDS Patients in Florida, by Race, Country of Origin, and Age

<table>
<thead>
<tr>
<th>Age group (yrs)</th>
<th>White, non-Hispanic (percent)</th>
<th>Black, US born (percent)</th>
<th>Haitian (percent)</th>
<th>Hispanic (percent)</th>
<th>Other (percent)</th>
<th>Total (percent)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-24</td>
<td>3 (1/36)*</td>
<td>21 (4/19)</td>
<td>40 (8/20)</td>
<td>0 (0/5)</td>
<td>0 (0/32)</td>
<td>12 (13/112)</td>
</tr>
<tr>
<td>25-44</td>
<td>2 (6/365)</td>
<td>13 (22/165)</td>
<td>29 (45/157)</td>
<td>7 (7/105)</td>
<td>7 (2/24)</td>
<td>10 (82/16)</td>
</tr>
<tr>
<td>45+</td>
<td>5 (5/106)</td>
<td>22 (5/23)</td>
<td>15 (2/13)</td>
<td>10 (2/21)</td>
<td>0 (0/3)</td>
<td>8 (14/166)</td>
</tr>
<tr>
<td>Total</td>
<td>2 (12/507)</td>
<td>15 (31/207)</td>
<td>29 (55/190)</td>
<td>7 (9/131)</td>
<td>3 (2/59)</td>
<td>10 (109/1094)</td>
</tr>
</tbody>
</table>

*AIDS patients with tuberculosis/all AIDS patients.

3 Brook I. The role of beta-lactamase producing bacteria in the persistence of streptococcal tonsillar infection. Rev Infect Dis 1984; 6:601-07
5 Wardle JK. *Branhamella catarrhalis* as an indirect pathogen. Drugs 1986; 31:93-96

Pulmonary Tuberculosis and Acquired Immunodeficiency Syndrome

To the Editor:

I read with interest the editorial report by Drs. Reider and Snider (Chest 1986; 90:469-70) discussing tuberculosis and acquired immunodeficiency syndrome (AIDS). I would like to comment on the relationship between these two diseases in Zambian, Central Africa.

Our limited experience shows that 40 percent of patients with AIDS have pulmonary tuberculosis at the same time; this represents 2.9 percent of all pulmonary tuberculosis cases. This figure is between that reported by Drs. Louis, Rice and Holzaman in non-Haitian patients (Chest 1986; 90:542-45) and that of Pitchenik et al in Haitian patients (Ann Intern Med 1984; 101:641-45). All these patients were heterosexual males aged between 25 and 50 years and the diagnosis of pulmonary tuberculosis was established before that of AIDS and all the patients died within a year after the diagnosis of AIDS.

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To the Editor:

Dr. Syabbalo reemphasizes the importance of tuberculosis in patients with the acquired immunodeficiency syndrome (AIDS). In some parts of Africa, tuberculosis has been recognized to be associated frequently with human immunodeficiency virus (HIV) infection. In Kinshasa, Zaire, a seroprevalence of HIV infection of 33 percent (53 of 159) in patients with smear-positive pulmonary tuberculosis has been reported. In Lusaka, Zambia, 24 percent (17 of 71) of tuberculosis patients were seropositive for HIV. It is biologically and epidemiologically plausible to hypothesize that the tuberculosis incidence among AIDS patients is directly correlated to the prevalence of latent tuberculosis infection in the population from...