Treatment of Bronchial Asthma in a Developing African Country

To the Editor:

I read with interest the special supplement of Chest containing a symposium titled, "International scope of asthma" (1986; 90:385-845). I would like to add to this information on the treatment of asthma in a developing African country, which was not tackled in this detailed publication.

Treatment of asthma in developing African countries (Table 1) and indeed in other third world countries is influenced by: the price and availability of drugs, customs and cultural beliefs; and the seasonal and diurnal variation in the frequency and severity of asthmatic attacks.

In Zambia, for instance, inhaled beta-agonist and steroid drugs, cromoglycate, anticholinergic and allergic extracts for immunotherapy are very scarce, especially in rural health centers. When inhalation devices are available, patients are afraid to use them because they think they cause sudden death, and of those who use them only 70 percent do so correctly. Aminophylline suppositories are also unacceptable to the majority of patients, especially women; some of the patients feel they cause diarrhea or hemorrhoids.

Asthmatic attacks in tropical and sub-tropical Africa occur most often during the hot, humid and rainy season (December through March) and usually at night. For this reason most patients who do not improve after initial parental aminophylline therapy receive parental steroids, and many patients need oral steroid maintenance therapy during this period.

Ketotifen—with either oral theophylline, oral beta-agonist or inhaled beta-agonist drugs—is often used to decrease the frequency and severity of nocturnal asthma. This drug is also useful in asthmatic subjects with allergic rinitis, and in treating analgesic-induced asthma (AlA).

Like physicians elsewhere, we recommend either oral/inhaled beta-agonist or inhaled cromoglycate therapy to prevent exercise-induced asthma; unlike our Japanese counterparts, we have little experience in the use of gold for treatment of asthma.

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Table 1—Treatment of Asthma in Africa

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<th>Rural health centers</th>
<th>Urban health centers</th>
<th>Pediatric patients</th>
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<td>A) Acute episodes</td>
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<td>B) Maintenance</td>
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<td>alone or with IBA</td>
<td>Oral theophylline</td>
<td>Oral beta-agonist</td>
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<td>Inhaled beta-agonist</td>
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<td>alone or with K or IC</td>
<td>Inhaled cromoglycate</td>
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<td>or IC</td>
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<td>IBA—inhaled beta-agonist; IC—inhaled cromoglycate; K-ketotifen; IA—inhaled anticholinergic</td>
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Normal Echocardiographic Left Ventricular Wall Thickness in Severe Aortic Stenosis

To the Editor:

We read with great interest the paper, "Implications of normal left ventricular wall thickness in critical aortic stenosis" by G. A. Bergeron and N. B. Schiller (Chest 1986; 90:380-82).

We have encountered a similar case of critical aortic stenosis, without echocardiographic evidence of increased left ventricular wall thickness.

CASE REPORT

A 55-year-old man was admitted to our Cardiac Department because of congestive heart failure (CHF). Physical examination disclosed grade 3/6 systolic ejection murmur. Chest x-ray film revealed moderate cardiomegaly with signs of interstitial pulmonary edema. ECG showed sinus tachycardia, left atrial abnormality and left ventricular hypertrophy. M-mode echocardiographic examination disclosed interventricular septum and left ventricular posterior wall thickness in the upper normal limits (12 mm and 11 mm respectively), and shortening fraction severely depressed (nine percent), with no definite increase in left ventricular end-diastolic and endsystolic diameters (54 mm and 49 mm respectively). The left atrium was moderately enlarged (47 mm). Two-dimensional tomographic study revealed a globular, severely hypokinetic left ventricle and a thickened, hypomobile aortic valve.

The absence of echocardiographic evidence of ventricular wall hypertrophy and of upstroke delay in the carotid pulse tracing suggested that the left ventricular dysfunction was not secondary to aortic valve disease and therefore cardiac catheterization was not planned. The patient was discharged with standard treatment. Twelve months later he experienced another episode of CHF. A new echocardiographic study confirmed previous data. Since carotid pulse tracing showed a mildly delayed upstroke (T-time 55 msec), a cardiac Doppler flow study was made. It indicated low cardiac output (2.42 L/min) and an estimated peak systolic gradient of 37 mm Hg (mean 17 mm Hg). Valve area was estimated to be 0.54 cm².

Cardiac catheterization revealed depressed cardiac output (2.1 L/min), cardiac index (1.3 L/min) and ejection fraction (20 percent); severe pulmonary hypertension (70/26 mm Hg, mean 40), and a peak-to-peak systolic gradient of 30 mm Hg across the aortic valve. The calculated aortic valve area was 0.4 cm². Left ventriculographic study confirmed severe global hypokinesia. Coronary arteries were free of significant lesions.

The possibility of surgical aortic valve replacement was rejected by the patient because of the high perioperative risk. Clinical conditions progressively deteriorated and the patient died in low output syndrome secondary to pulmonary embolism four months later.

Cardiac postmortem examination confirmed the existence of severe calcific aortic stenosis without significant left ventricular wall hypertrophy. The coronary arteries did not show critical stenosis. Histologically, a picture of interstitial myocardial fibrosis was present.

DISCUSSION

Our case further backs Bergeron and Schiller's suggestion to take into account the presence of this malignant subgroup of patients with critical aortic valve stenosis who fail to develop an adequate hypertrophic response to the elevated systolic wall stress of aortic stenosis.

It is worth recalling that the low transvalvular gradient estimated
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 underlines 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 Maggoni 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 secretion 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. In fact, recent literature reports suggest that, beside its own pathogenicity, B-LPBC can act as an indirect pathogen through production of β-lactamase. 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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REFERENCES

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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-