TABLE III—Antibiotic resistance pattern of strains of Staph aureus isolated from patients who had received an antibiotic. Number of patients in parentheses

<table>
<thead>
<tr>
<th>Antibiotic</th>
<th>No of patients</th>
<th>Resistance patterns*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Penicillin</td>
<td>10</td>
<td>P (9); P T (1)</td>
</tr>
<tr>
<td>Erythromycin</td>
<td>5</td>
<td>P S (1); P (3); P E (1)</td>
</tr>
<tr>
<td>Tetracycline</td>
<td>1</td>
<td>P T (1)</td>
</tr>
</tbody>
</table>

*See footnote to table II for abbreviations.

Discussion

Previous reports from this casualty department showed that the percentage of strains of Staph aureus resistant to penicillin had gradually increased from 6% to 50%. This study shows that the trend has continued, 81.5% of strains now being resistant to penicillin. This result approximates to the incidence of penicillin resistance found in strains isolated from inpatients. None of the 200 strains of Staph aureus were resistant to methicillin, chloramphenicol, or gentamicin. Chloramphenicol resistance has not increased since the surveys in 1952 and 1955. Resistance to tetracycline has increased, however, results of reports in 1955 showing no resistant strains, while in 1968 6.5% of strains were resistant. The present series shows a slight increase to 8%. Only a few strains were resistant to erythromycin, clindamycin, and sodium fusidate. Most of the strains were resistant only to one antibiotic, 12.5%, were resistant to two drugs, and only one strain was resistant to more than three antibiotics.

Patients infected with strains resistant only to penicillin still respond to treatment with this drug, possibly because such strains produce only a small amount of penicillinase. Our findings therefore suggest that penicillin is still suitable antibiotic treatment in outpatients, and erythromycin is a suitable alternative for those allergic to penicillin.

References


(Accepted 16 June 1978)

SHORT REPORTS

Chronic bronchitis: is bacteriological examination of sputum necessary?

This study was undertaken to assess the common hospital practice of routine bacteriological examination of specimens of purulent sputum is necessary for the efficient management of infective exacerbations of chronic bronchitis.

Patients, methods, and results

Sixty patients admitted to hospital with exacerbations of chronic bronchitis associated with purulent sputum were included and randomly allocated into two groups. Group A was managed in the usual way, with regular reviews of the bacteriological reports of sputum culture. Group B had sputum examined, but the reports were sent to the supervising physician unless formally requested. There were 30 patients in each group. The average ages were similar (group A: 62.4 years; group B: 67.1 years). Eleven patients in group A and 12 patients in group B had ventilatory failure on admission. Inflammatory changes were present on a chest x-ray film of 11 patients in each group. The approximate time of sputum purulence before admission for both groups was 11 days, and 10 patients in group A compared with 12 in group B had had previous antibiotic treatment. Treatment with antibacterial drugs was selected according to usual practice and sputum was examined bacteriologically immediately after admission and after five or seven days' treatment. Sputum appearance was noted daily. Chest x-ray films were taken at appropriate intervals, and treatment changes were documented.

In group A the sputum of 25 patients became mucoid in 7-68 days (range 1-16). The sputum of the other five patients remained purulent. One of these patients had bronchiectasis. In group B 28 patients produced mucoid sputum in 6-08 days (range 2-12). One of the two patients with persistently purulent sputum had bronchiectasis and the other died from a cerebrovascular accident while his sputum was still purulent. The table shows that the results of sputum culture were similar in the two groups. Details of chemotherapy given to both groups are shown in the table. Twenty-four patients in group A, compared with 26 in group B, required a single course of antibiotic. In six cases from group A a change of treatment was necessary but in only two was mucoid sputum achieved. In group B antibiotic treatment was changed in four cases and in only one patient was there failure of conversion of sputum. In group B one change of treatment was made because of bacteriological findings but this patient's sputum remained purulent. In group B it was felt necessary to examine bacteriological results in three patients because of poor clinical response, but in none of these did the bacteriology findings affect the outcome. The average times to the clearing of the inflammatory changes in the chest x-ray film in both groups were similar (group A: 8-5 days; group B: 7-5 days).

Comment

After completion of the trial a review of results in group B indicated that no changes of treatment would have been made had the bacteriological results been immediately available. Indeed, these reports might have been misleading in three patients since there was satisfactory sputum clearance with antibiotics to which in-vitro resistance had been reported.
Search for late-onset side effects of practolol

Side effects of practolol, such as the oculomucocutaneous syndrome, are well recognised. Many occur during treatment but pulmonary complications may appear 20 months after stopping practolol.1 We have therefore reviewed for late-onset side effects all patients at the Glasgow Blood Pressure Clinic ever given practolol.

Patients, methods, and results

The patients were all those treated at the clinic. Clinical and therapeutic details were documented on prorfoms and stored in a KDF9 digital computer.2 By using an extract programme, we identified patients who had taken practolol for more than one month for review. Investigations included: clinical and ophthalmological examination, full blood count and ESR; urea and electrolyte concentrations; liver function tests, chest x-ray film; electrocardiograph; antinuclear factor (ANF) and indirect immunofluorescence studies. Ophthalmological assessment included visual acuity, slit-lamp examination, Schirmer's test, and Rose Bengal dye test. Interstitial antibody was searched for by the Amos3 technique, using baboon oesophagus as substrate.

Of the 59 patients identified, six had died, 17 had received practolol for less than one month, and five were untraceable. Thirty-one, 15 women and 16 men, were reviewed (see table). Three had died of myocardial infarction and three of stroke. Postmortem confirmation was available in four. There was no suggestion of late-onset toxicity in their records. Examination of the survivors showed no pericarditis, scarring peritonitis, or systemic lupus syndrome.

Three patients had histories of probable practolol toxicity rashes. Case 1—a 44-year-old man had persisting eczema on his hands which had appeared during practolol treatment. Case 2—a 54-year-old woman had developed severe nummular eczema while taking practolol. This had cleared after stopping it. Case 3—a 40-year-old man had developed a persisting psoriatic rash on his thighs during treatment with practolol.

Case 4—Serious otitis media developed after two years in a 66-year-old woman treated from May 1969 to November 1974. This persists. Three patients had ophthalmological complaints but none of these could be related to practolol.

| Date and duration (mean and range) of practolol treatment in 31 hypertensive patients who were reviewed |
|-----------------|---------|---------|
| Patients         | 16 men  | 15 women|
| Age (years)      |         |         |
| Dosage of practolol (mg) |         |         |
| Time on practolol (months) |         |         |
| Time off practolol (months) |         |         |

Case 5—a 53-year-old man had recurrent corneal erosions, starting three years after stopping practolol. Case 6—a 54-year-old man had grittiness of both eyes starting four years after stopping practolol. The result of examination was normal.

Case 7—a 58-year-old man with rheumatoid arthritis had signs of keratoconjunctivitis sicca.

The results of all routine investigations were normal other than in one patient with active rheumatoid arthritis who had a raised ESR and anaemia. ANF was detected in three women and two men, the titres being 1/16 in one, 1/64 in one, and 1/256 in two. Interstitial antibody was not detected in any of the 31 patients.

Comment

Because of the serious nature of practolol toxicity we decided to review all exposed patients and to look particularly for late-onset consequences. Computerised records were essential for this study, providing prompt and accurate identification of the patients at risk. The study identified three patients known to have probable practolol toxicity and one other patient with chronic serous otitis media in whom the link with practolol treatment had been unrecognised. Amos4 has suggested that interstitial antibody is a sensitive index of practolol toxicity, but it was not found in this study. ANF was present in low titres in 16 of the patients and none of these showed any evidence of toxicity. This frequency of detectable ANF was little different from the 10-14%, expected in healthy subjects.5

In conclusion, this review of patients who received practolol showed no late-onset toxicity. Though this result is reassuring, continued vigilance may be required.

We thank Dr J J Brown, Dr G J Addis, Dr M Ramsay, and Mrs E Adams for their valuable help. Requests for reprints should be addressed to JB.

1 Wright, P, British Medical Journal, 1975, 1, 595.

(Received 15 May 1978)

Legionnaires' disease and acute renal failure

Legionnaires' disease was named after an outbreak of 182 cases at the American Legion conference in 1976.6 It is now apparent that the infecting organism had been responsible for previous outbreaks of pneumonia.7 One of these affected holidaymakers from Glasgow who visited Benidorm in 1973.8 A television broadcast on this episode interested one of our patients; he had contracted pneumonia while in the same Spanish hotel four years later and drew the fact to our attention.

Case report

A 66-year-old man had been treated for myxoedema due to autoimmune thyroiditis in 1973 but had stopped taking his thyroxine in 1976. He smoked 20 cigarettes a day and had three "cold"s on the chest" each year with wheezing but no dyspnoea or cough between attacks. In May 1977 he took two weeks'