Using the rate of clearance of sputum purulence and rate of improvement of x-ray inflammatory change as an index of clinical response, the results of this study show that routine bacteriological examination of sputum is of no value. Almost all patients responded to a single course of antibiotic treatment which was chosen using clinical criteria only. The vast majority of patients with exacerbations of chronic bronchitis can and should be managed without routine bacteriological control. Sputum need be examined in the laboratory only if it remains purulent after seven days of antibiotic treatment, but even then clinically misleading results may be obtained. Exceptions to this practice should be made, of course, when, from the history, clinical findings, or chest x-ray appearances, it is strongly suspected that an unusual organism is present.

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Search for late-onset side effects of practolol

Side effects of practolol, such as the oculomucocutaneous syndrome,¹ are well recognised. Many occur during treatment but pulmonary complications may appear 20 months after stopping practolol.² 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 proformas and stored in a KDF9 digital computer.³ 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. Intercellular antibody was searched for by the Amos⁴ 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, sclerosing 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—Serous 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.

Dose and duration (mean and range) of practolol treatment in 31 hypertensive patients who were reviewed

	Patients:	16 men	15 women
Age (years)		56	42-65
Dosage of practolol (mg)		400	100-2000
Time on practolol (months)		16.5	3-66
Time off practolol (months)		44	18-92

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 two, 1/64 in one, and 1/256 in two. Intercellular 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 intercellular 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.

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Requests for reprints should be addressed to JB.

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

² Marshall, A J, *et al*, *Lancet*, 1977, **2**, 1254.

³ The Glasgow Blood Pressure Clinic, *Journal of the Royal College of Physicians of London*, 1972, **7**, 87.

⁴ Amos, H E, Brigden, W D, and McKerron, R A, *British Medical Journal*, 1975, **1**, 598.

⁵ MacSween, R N M, personal communication, 1977.

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Legionnaires' disease and acute renal failure

Legionnaires' disease was named after an outbreak of 182 cases at the American Legion conference in 1976.¹⁻³ It is now apparent that the infecting organism had been responsible for previous outbreaks of pneumonia.^{2,3} One of these affected holidaymakers from Glasgow who visited Benidorm in 1973.⁴ 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 "colds on the chest" each year with wheezing but no dyspnoea or cough between attacks. In May 1977 he took two weeks'

holiday in Spain. Soon after arrival he had an infected spot beside his nose and within a few days began to feel unwell. Malaise and dyspnoea increased but he stayed in his hotel until the return flight. On arrival home he was confused and febrile but had no chest pain, cough, or sputum. He started taking ampicillin four days after returning home and was admitted to hospital on the next day, 2 June 1977.

His fever of 38.5°C settled within three days but rose briefly to 38°C on days 7 and 11. He had an expiratory wheeze on both sides and crepitations on the right. Chest radiography (figure 1a) showed blotchy opacity on the right side. After five days of dry cough he produced green sputum, from which *Klebsiella aerogenes* and *Candida albicans* were grown (presumed to be superinfectants). Ampicillin, 500 mg 6-hourly, was continued until the 12th

Chest radiograph on second day of hospital admission (3 June 1977) (a) and four weeks later (b). Some shadowing and mediastinal spread persisted nine months later.

day after admission but the radiological signs resolved slowly (figure 1b). No diagnostic changes in antibody titres to influenza A, B, C, or psittacosis occurred over the next two weeks, and no cold agglutinins were detected. His initial WBC was 9700/mm³. All serum globulin fractions were raised, presumably due to his Hashimoto's disease.

He had +proteinuria and +haematuria on admission, which cleared in five days. He was never oliguric but his plasma concentrations of urea rose to 26.5 mmol/l (159 mg/dl) and of creatinine to 512 µmol/l (5.8 mg/dl) on the second day of admission. Both concentrations fell slowly and were still slightly abnormal nine months later: plasma urea 10.1 mmol/l (61 mg/dl), creatinine 170 µmol/l (1.9 mg/dl); he had no proteinuria but an excess of hyaline casts in the deposit. Since his plasma urea and creatinine concentrations and the results of urine analysis and microscopy had all been normal on two occasions in the four years before his pneumonia, his renal impairment appears to be a late legacy of his acute renal failure.

In March 1978 a serum sample was examined for antibodies to the organism causing legionnaires' disease; by the indirect fluorescent antibody test it had a titre of 1/256, which strongly supports the diagnosis.

Comment

In the Philadelphia outbreak of legionnaires' disease "transient impairment of renal function was frequent and usually mild."¹ Four patients developed acute renal failure requiring dialysis. In three of these, and in our patient, there was no obvious precipitant such as hypotension or respiratory failure. It appears that the infection has a deleterious effect on renal function, which may be permanent. The clinical course is more suggestive of interstitial nephritis than of tubular necrosis but we did not perform a renal biopsy.

One other patient is known to have developed legionnaires' disease after visiting the same hotel in 1977.⁴ The occurrence of two minor outbreaks four years apart in the same locality in one Spanish town should be a useful clue to the mode of spread. Contamination of a ventilation system was blamed for the outbreak at Pontiac.⁵ Our patient was predisposed to infection by age and chest disease,¹ whereas most of his fellow travellers were probably young and healthy. Nevertheless, other cases have probably been overlooked, and their discovery might throw further light on the pathogenesis.

Application for reprints to DNSK.

¹ Fraser, D W, *et al*, *New England Journal of Medicine*, 1977, **297**, 1189.

² *British Medical Journal*, 1978, **1**, 2.

³ *Lancet*, 1977, **1**, 1265.

⁴ Lawson, J H, *Scottish Medical Journal*, 1978, **23**, 121.

⁵ McDade, J E, *et al*, *New England Journal of Medicine*, 1977, **297**, 1197.

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Breakfast and Crohn's disease—I

A recent report by James¹ of a significant association between Crohn's disease and eating cornflakes for breakfast prompted us to study this association in a larger series.

Patients and controls; inquiry and results

A total of 100 men with Crohn's disease currently attending our out-patients clinic were subjected to a detailed dietary interview to elicit the same information about their breakfast eating habits as was employed by James.¹ A total of 100 controls was obtained from the visitors to the general wards of the Radcliffe Infirmary. None of them were suffering, or had ever suffered, from any major gastrointestinal disease. They were matched with the Crohn's disease group for age, sex, and social class according to the criteria employed by the Office of Population Censuses and Surveys (1970).

All the patients and the subjects in the control group were interviewed by PMR, using a questionnaire. This was designed to elicit all the items of information used by James¹ and also to obtain certain additional information. In view of the observations by Martini and Brandes² suggesting that a high intake of sucrose is associated with Crohn's disease, the use of sugar on breakfast cereals and in breakfast drinks was noted in detail. Details of the type and quantity of fluid consumed at breakfast were also recorded. In many of the patients with Crohn's disease the disease was of long standing and therefore the inquiry about their breakfast eating habits was directed at the period preceding the onset of symptoms. To make a fair comparison, the appropriate members of the control group were questioned about their breakfast eating habits at corresponding periods in their lives.

The table shows the data corresponding to those presented by James¹ in his table I. The chief finding is that in the present series there was no significant difference between the patients with Crohn's disease and the controls in respect of regular eating of cornflakes for breakfast. The two groups did not differ significantly in their consumption of other common breakfast cereals consisting of wheat, porridge, and rice. So far as bran and muesli are concerned, our results are diametrically opposed to those of James, in that regular bran and muesli eaters were confined to the control group. The difference in respect of bran is statistically significant and in respect of muesli is suggestive. Trowell³ has suggested that Crohn's disease is one of the diseases of Western society that may result from eating a diet deficient in dietary fibre, so possibly the present findings are a clue worth pursuing. Naturally, a study simply of breakfast habits does not give an adequate picture of the diet as a whole.