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# The increasing isolation of *Serratia* species from clinical specimens

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SUMMARY

A 14-month survey was undertaken in a diagnostic bacteriology laboratory to determine the incidence of *Serratia* spp. in routine clinical specimens. Gram-negative organisms with enterobacteria-like colonies were tested by a simple screening procedure. Fifty-eight strains of *S. marcescens* and two strains of *S. liquefaciens* were isolated from 59 patients. The strains were usually non-pigmented and exhibited multiple antibiotic resistance. Serotyping and determination of bacteriocine sensitivity patterns revealed that the majority of infections were sporadic, although episodes of cross-infection did occur.

*S. marcescens* was considered to contribute significantly to morbidity and mortality in 53% of patients and appears to be of increasing importance in hospital-acquired infections.

## INTRODUCTION

*Serratia marcescens* is well established as an important cause of hospital acquired infection and cross-infection in certain parts of the world, particularly the U.S.A. (Clayton & von Graevenitz, 1966; Wilfert, Barrett & Kass, 1968; Davis, Foltz & Blakemore, 1970) and Canada (Black *et al.* 1973). It is generally accepted that this organism is a rare pathogen in Britain and is occasionally responsible for outbreaks of cross-infection (Whitby, Blair & Rampling, 1972; Taylor & Keane, 1962) and isolated cases of incidental infection (Black, Pollock & Batchelor, 1967; McCracken & Lipscomb, 1965). There is, however, evidence that *S. marcescens* is becoming increasingly important in this country (*Lancet*, 1977, *B.M.J.* 1977). During a 4-month period Black & Hodgson (1971) isolated 10 strains of *S. marcescens* from clinical specimens examined in a routine bacteriology laboratory, Ball, McGhie & Geddes (1977) have reported 14 cases of infection with *S. marcescens* over an 8-month period and Tabaqchali, Chambers & Brooks (1977) in a preliminary report demonstrated a higher incidence. In contrast, Hedges, Rodrigues-Lemoine & Datta (1975) recorded only one or two *Serratia* isolations per annum in a laboratory handling about 60000 clinical specimens each year. It is possible that this low incidence and the general scarcity of reports in British literature on the occurrence of *Serratia* in clinical specimens reflect, at least in part, the difficulties encountered in separating members of the *Klebsiella–Enterobacter–Serratia* group.

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Table 1. *The incidence of* Serratia *amongst enterobacteria-like organisms (ENT) isolated from clinical specimens*

| Clinical specimen | No. of (ENT) tested* | Serratia strains isolated | |
|---|---|---|---|
| | | No. | % |
| Urine | 2552 | 30 | 1·2 |
| Swab | 1465 | 12 | 0·8 |
| Sputum | 635 | 12 | 1·9 |
| Blood | 48 | 2 | 4·2 |
| Others | 435 | 4 | 0·9 |

* These figures also represent the number of specimens and number of patients from whom (ENT) were isolated.

We report here the results of a survey undertaken to investigate the incidence of *Serratia* spp. in clinical specimens and the contribution of such organisms to morbidity and mortality in our hospital, because these organisms like *Pseudomonas* spp. are opportunistic pathogens, invariably resistant, and treatment of infections is difficult.

## MATERIALS AND METHODS

### *Specimens*

Approximately 85000 clinical specimens are examined annually in the diagnostic laboratory at St Bartholomew's Hospital (Hospital 1) and these include specimens from St Leonard's Hospital (Hospital 2) and from two other smaller hospitals. The total bed complement of the four hospitals is 1456. All clinical specimens were screened for the presence of Enterobacteria-like organisms (ENT). The number and type of specimens with (ENT) are shown in Table 1.

### *Screening for* Serratia

A total of 5135 cultures of lactose-fermenting and lactose non-fermenting gram-negative bacilli isolated over a 14-month period, March 1974 to April 1975, were tested for DNase production by the method of Schreier (1969). These also included some unpigmented *Pseudomonas* spp. and *Acinetobacter* spp. which have enterobacteria-like colonies. DNase-positive strains were selected for further examination by the Hugh and Leifson, phenylalanine deaminase and oxidase tests. All strains that were DNase positive, oxidase negative, fermentative in Hugh and Leifson's medium and did not produce phenylpyruvic acid (PPA) from phenylalanine were tentatively placed in the genus *Serratia*. These were confirmed as *Serratia* and identified at species level by their carbohydrate fermentation and decarboxylase reactions (Center for Disease Control, 1972).

### *Antibiotic sensitivity testing*

Resistance patterns were determined by the disk method on DST agar containing lysed blood (Oxoid) using a standardized lawn inoculum of $10^2$–$10^3$ organisms per plate. The following antibiotics were tested (abbreviations used in the tables

Table 2. *Antibiotic resistance of* 60 *strains of* Serratia *isolated from clinical specimens*

| Antibiotic and (μg/disk) | Resistant strains | | Antibiotic and (μg/disk) | Resistant strains | |
|---|---|---|---|---|---|
| | No. | % | | No. | % |
| Ak — | 0 | 0 | N (10) | 37 | 62 |
| G (10) | 0 | 0 | S (25) | 39 | 65 |
| To (10) | 0 | 0 | Ca (100) | 42 | 70 |
| Tri (2·5) | 2 | 3 | T (30) | 48 | 80 |
| C (25) | 7 | 12 | P (100) | 53 | 88 |
| NA (30) | 8 | 13 | A (25) | 54 | 90 |
| K (30) | 35 | 58 | Nit (200) | 56 | 93 |
| Su (50) | 37 | 62 | Cep (30) | 60 | 100 |

are given in brackets): gentamicin (G), tobramycin (To), trimethoprim (Tri), chloramphenicol (C), nalidixic acid (NA), kanamycin (K), sulphamethoxazole (Su), neomycin (N), streptomycin (S), carbenicillin (Ca), tetracycline (T), ampicillin (A), polymyxin (P), nitrofurantoin (Nit), cephalothin, cephalexin, cephaloridine (Cep).

Minimum inhibitory concentrations (MICs) of amikacin were determined by a plate dilution method. Doubling dilutions of amikacin were incorporated into DST agar plates so that the final concentrations ranged from 0·5 to 256 μg/ml. Plates were inoculated with 1/1000 dilutions of overnight broth cultures using an automatic, multi-point inoculator which delivered 20 spot inocula per plate. After incubation overnight at 37 °C they were examined for inhibition of growth.

*Serological and bacteriocine typing*

Strains of *S. marcescens* were serologically typed against 15 O-antisera by the method of Edwards & Ewing (1972). Sensitivities to the bacteriocines of a standard set of 13 strains of *S. marcescens* were determined by the method of Anderhub *et al.* (1977). Both procedures were carried out by the Cross-Infection Laboratory, Colindale, London.

## RESULTS

Over the 14-month survey period *S. marcescens* was isolated from 57 patients and *S. liquefaciens* from 2. The same strain of *Serratia* was repeatedly isolated from the same site in some patients. To avoid overestimation of the incidence only 1 strain per patient has been included in the results, except in one case where 2 different serotypes of *S. marcescens* were isolated from a single wound. Thus a total of 60 strains are considered in the results. Isolation rates from various clinical specimens are shown in Table 1; half the *Serratia* strains were isolated from urines.

Five strains initially identified as *Serratia* on the results of DNase, Hugh and Leifson, phenylalanine deaminase and oxidase tests proved not to belong to this genus on further testing.

Five strains of *S. marcescens* developed a red pigment on storage at room temperature.

Table 3. *Common antibiograms of* Serratia *isolated from clinical specimens*

| Resistance pattern | No. of strains |
|---|---|
| NA K Su N S Ca T A P Nit Cep | 6 |
| C K Su N S Ca T A P Nit Cep | 5 |
| K Su N S Ca T A P Nit Cep | 21 |
| T A P Nit Cep | 8 |

Table 4. *Distribution of* Serratia marcescens *O serotypes*

| | Number of strains isolated from: | | | | |
|---|---|---|---|---|---|
| Serotype | Urine | Wound Swab | Sputum | Others | Total |
| O3 | — | — | — | 1 | 1 |
| O4 | — | — | 1 | 1 | 2 |
| O5 | — | 1 | — | 2 | 3 |
| O6 | 3 | 1 | 1 | 1 | 6 |
| O8 | 19 | 3 | — | 1 | 23 |
| O12 | — | — | 2 | — | 2 |
| O14 | 7 | 4 | 4 | 3 | 18 |
| Non-typable | 1 | — | 2 | — | 3 |

Total number of strains typed was 55, +3 untypable strains.

*Antibiotic resistance*

The incidence of antibiotic resistance is shown in Table 2. All strains were sensitive to G and To; only 2 were resistant to Tri, 7 to C and 8 to NA. Resistance to K, Su, N, S, Ca, T, A, P, Nit and Cep ranged from 58 % to 100 % of strains and all were multiply resistant. The commonest antibiograms are given in Table 3. The pigmented strains were resistant to Nit, P and Cep; 2 were additionally resistant to T, 1 to A and T and 1 to A, T and C.

Amikacin was active against all 60 strains of Serratia. Fifty-eight had MICs of 4–8 $\mu$g/ml and the remaining two were inhibited by 16 $\mu$g/ml.

*Serological and bacteriocine typing*

Seven O serological types were represented and only three strains were non-typable. The distribution of serotypes in various clinical specimens is shown in Table 4. O8 was the commonest serotype associated with urinary tract infection but O14 was more evenly distributed amongst the different strains. Types O8 and O14 accounted for 68 % of all *S. marcescens* isolated, whilst more than one third of all the strains were type O8. No strain of this type was isolated from sputum specimens.

The *S. marcescens* strains comprised a wide variety of O types and bacteriocine sensitivity pattern types the majority of which showed more or less random distribution. In two surgical wards, P and G at Hospitals 1 and 2 respectively, the incidence of *Serratia* isolation was relatively high and an attempt was made to detect episodes of cross-infection using three criteria – O serotype, bacteriocine sensitivity pattern and antibiotic resistance (antibiogram) – to distinguish different strains (details are shown in Table 5). Type O8 strains were the most

Table 5. *Serotypes, bacteriocine sensitivity patterns and antibiograms of total number of* Serratia *spp. isolated from two wards*

| Hospital and ward | Patients' initials | Date serratia isolated | Species | Specimen | O-serotype | Bacteriocine sensitivity pattern* | Antibiogram† |
|---|---|---|---|---|---|---|---|
| Hospital (2), ward G | F.S. | 7. 3. 1974 | *S. marcescens* | Urine | 8 | 1 | A |
| | | 12. 3. 1974 | | Urine | 8 | 1 | A |
| | G.W. | 12. 3. 1974 | | Urine | 8 | 1 | A |
| | M.F. | 12. 3. 1974 | | Urine | 8 | 1 | B |
| | | 1. 4. 1974 | | Urine | 8 | 1 | B |
| | F.G. | 1. 4. 1974 | | Urine | 8 | 1 | A |
| | R.B. | 4. 5. 1974 | | Urine | 8 | 1 | A |
| | J.M. | 14. 10. 1974 | | Dialysate | 5 | — | — |
| | T.W. | 12. 2. 1975 | | Urine | 8 | 1 | A |
| Hospital (1), ward P | J.M. | 1. 1. 1974 | *S. marcescens* | Urine | 8 | 1 | C |
| | H.S. | 28. 1. 1974 | *S. marcescens* | Wound swab | 8 | 1 | C |
| | F.O. | 31. 1. 1974 | *S. marcescens* | Urine | 8 | 1 | A |
| | G.S. | 2. 10. 1974 | *S. marcescens* | Urine | 8 | 1 | B |
| | C.S. | 11. 11. 1974 | *S. marcescens* | Wound swab | 8 | 1 | A |
| | B.F. | 14. 11. 1974 | *S. liquefaciens* | Sputum | — | — | — |
| | C.S. | 25. 11. 1974 | *S. marcescens* | Wound swab | 6 | — | — |
| | I.M. | 11. 2. 1975 | *S. marcescens* | Wound swab | 8 | 1 | A |
| | B.R. | 15. 3. 1975 | *S. marcescens* | Urine | 8 | 1 | A |

* Bacteriocine sensitivity pattern 1 denotes sensitivity to the bacteriocines of 6 of the 12 'producer' strains (No. 7, 40, 45, 47, 53, 84) of Anderhub *et al.* (1977).

† Antibiogram A denotes resistance to: Su, A, Nit, P, Ca, K, Cep, T, S, N. Antibiogram B denotes resistance to: A, Nit, P, Cep, T. Antibiogram C denotes resistance to: Su, A, Nit, P, Ca, K, Cep, T, S, C, N.

Table 6. *Clinical data on patients from whom* Serratia *spp. were isolated*

| Source of *Serratia* spp. | No. of patients | Age: years (mean) | Predisposing factors to infection with *Serratia* | No. of patients in whom there were pre-disposing factors | Type of infections | No. of patients |
|---|---|---|---|---|---|---|
| Urine | 25 | 22–82 (63) | Cytoscopy, catheterization and bladder washouts | 19 | Urinary tract infection | 11 |
| Sputum or bronchial secretion | 10 | 49–85 (72) | Tracheostomy, mechanical ventilation and suction | 6 | Respiratory tract infection | 4 |
| Swabs | 8 | 28–74 (58) | Irrigation and repeated dressings of chronic ulcers and wounds | 8 | Wound infections and trophic ulcers* | 6 |
| Bone aspirate | 2 | 42, 44 | (1) Open heart surgery | 1 | (1) Osteomyelitis sternum | 1 |
| | | | (2) Traumatic injury | | (2) Osteomyelitis terminal phalanges | 1 |
| Blood | 2 | 44, 54 | Open-heart surgery intra-venous feeding C.V.P. canulation | 2 | Septicaemia endocarditis | 2 |
| Peritoneal dialysis fluid | 2 | 41, 50 | Long-term peritoneal dialysis | 2 | Peritonitis | 1 |

* Patients with diabetes and/or peripheral vascular disease.

prevalent in both wards; they exhibited the same bacteriocine sensitivity pattern, denoted by '1', but belonged to three different antibiogram types (A, B and C). It is evident that a variety of strains were isolated from the two wards but there was some evidence of cross-infection (Table 4). In ward G, *S. marcescens* O type 8, bacteriocine type 1 and antibiogram A (O8/1/A) was isolated from the urinary tract of four patients over a period of 2 months. During this time an O8/1 strain with a different antibiogram, B, was also isolated from the urinary tract of a fifth patient. In ward P there was one instance of possible cross-infection but this involved two patients only. In February 1975, *S. marcescens* O8/1/A was isolated from a wound swab from 1 patient and 4 days later from the urine of a second patient.

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*Clinical data*

The relevant clinical information obtained from 49 patients – age, factors predisposing to infection, types of infection and clinical significance of *Serratia* isolations – are reported in Table 6. Ages ranged from 22 to 85 years but 5 patients were over 50 years of age. A wide variety of underlying conditions was recognized and these were generally of a chronic debilitating nature. It is notable that urinary tract infection was associated with instrumentation in 19 out of 25 cases. *Serratia* isolation was preceded by antibiotic therapy in 34 cases, ampicillin being the most popular choice. Nineteen patients received this drug alone or in combination with other antibiotics and 11 patients were treated with 2 or more antibiotics simultaneously.

Based on the outcome of infection, response to treatment and the presence or absence of other organisms at the site of infection, *S. marcescens* was considered to contribute significantly to morbidity and mortality in 26 patients requiring specific therapy. The death of 2 patients with endocarditis was directly attributable to *S. marcescens* which was isolated in pure culture from the blood of both patients throughout the course of infection. The 2 strains of *S. liquefaciens* were isolated from sputum specimens and were not considered to be of significance. It is of interest that 2 of the 5 pigmented strains contributed considerably to morbidity. Both were isolated in pure culture, one from a retropharyngeal abscess, the other from a case of osteomyelitis of the terminal phalanges following a traumatic injury.

Bacteriological investigations were carried out on 40 patients soon after admission and *S. marcescens* was isolated from 4. The remainder of strains were isolated after a longer period in hospital.

## DISCUSSION

The generally held view that *Serratia* spp. are rare nosocomial pathogens in Britain was not confirmed by the present study. Fifty-eight strains of *S. marcescens* and 2 strains of *S. liquefaciens* were isolated from 59 patients over a period of 14 months. The isolation rate was low compared with hospitals in the U.S.A. but was approximately twice that reported by Black & Hodgson (1971), who conducted a similar survey in Scotland, whether isolations per month or per specimen were compared. This difference may be fortuitous, but the possibility that there has been a recent, true increase in the occurrence of *Serratia* in British hospitals cannot be excluded. *Serratia* was cultured from specimens submitted to the laboratory by all 4 hospitals in the group, suggesting this organism is widespread.

It is also possible that *Serratia* has been present in the hospital community for some time but has not been recognized. Apart from pigmented variants, *Serratia* has no special morphological characteristics and identification by the limited number of biochemical tests commonly in use in diagnostic laboratories may not be possible. Four tests were found to be of value in screening large numbers of cultures. These were DNase, oxidase and phenylalanine deaminase production and the oxidation/fermentation test of Hugh and Leifson. Using these tests for the purpose of preliminary identification, only 5 strains were initially

misplaced in the genus *Serratia*. A small proportion of strains, 3·3% of *S. marcescens* and 11·7% of *S. liquefaciens*, do not produce DNase (Center for Disease Control, 1972) and would not be identified as *Serratia* using our techniques. The expensive, time-consuming alternative of subjecting over 5000 cultures to a large battery of biochemical tests was now, however, considered justified.

In general agreement with American experience (Gale & Sonnenwirth, 1962; Wilfert *et al.* 1968; Schaefler *et al.* 1971) our strains exhibited a high degree of antibiotic resistance and were always multiply-resistant, but they were sensitive to the aminoglycosides gentamicin, tobramycin and amikacin. Wilfert *et al.* (1968) and Meltz & Grieco (1973) considered gentamicin to be the drug of first choice in the treatment of *Serratia* infections but in a recent study Meyer *et al.* (1976) reported resistance to gentamicin, tobramycin and sisomicin in 50% of *Serratia* strains isolated over a one-year period in a Los Angeles hospital. The majority of our strains were sensitive to trimethoprim, chloramphenicol and nalidixic acid, yet a high incidence of resistance to both chloramphenicol and nalidixic acid was reported by Dodson (1968) and Schaefler *et al.* (1971). Amikacin is active against strains resistant to other aminoglycosides, but amikacin-resistant *Serratia* do occasionally occur (Price, DeFuria & Pursiano, 1976). Antibiotic resistance transfer has been demonstrated in only a small proportion of *Serratia* strains but some of the R factors detected are unusually widespread (Hedges *et al.* 1975). Clearly, the selective pressure of liberal antibiotic use may well encourage the emergence and proliferation of strains resistant to all known antibiotics.

Almost all our strains were resistant to cephaloridine and nitrofurantoin. This appears to be a special feature (Maki *et al.* 1973; Wilfert *et al.* 1968) and may be helpful in the identification of *Serratia* spp. in the routine laboratory.

The wide variety of sero-bacteriocine types indicates that the *S. marcescens* strains did not originate from a single source. Episodes of cross-infection did occur although there was no evidence to suggest that this was a major mode of spread. The sources of *Serratia* spp. in hospitals are, at present, a matter for speculation. Previous investigations have indicated that rectal and pharyngeal carriage are negligible but contaminated irrigating fluids, breathing machines, catheters and suction tubing have been implicated in outbreaks of *Serratia* infection (McCormack & Kunin, 1966; Wilkowske *et al.* 1970; Whitby *et al.* 1972). There is strong evidence that *Serratia* can be transmitted passively on the hands of hospital personnel (Traub, 1972; Maki *et al.* 1973). However, some authors have been unable to locate the source of outbreaks of *Serratia* infection or to demonstrate the presence of this organism on inanimate objects and hospital personnel (Taylor & Keane, 1962; Cardos *et al.* 1973).

*Serratia* was considered to contribute to morbidity and mortality in 53% of patients from whom the organism was isolated. It was responsible for urinary tract, respiratory tract, wound and peritoneal infections, as well as osteomyelitis and septicaemia. Acquisition of *Serratia* occurred after admission in most patients and the possibility that the four patients presenting with *Serratia* infections on admission had acquired them during previous admissions to hospital could not be excluded. In common with other opportunistic pathogens, patients with chronic

debilitating disease appeared to be especially susceptible to infection. In agreement with other reports (Davis *et al.* 1970; Lancaster, 1962; Wilfert *et al.* 1968) we found a strong association between prior antibiotic therapy, instrumentation and colonization with *Serratia*. Instrumentation of the urinary tract was an important predisposing factor and the evidence for carriage on the hands of hospital personnel suggests *Serratia* may be introduced into the bladder during catheterization. Taylor & Keane (1962) attributed an outbreak of urinary tract infection in a urological ward to faulty aseptic technique and frequent opening of closed drainage systems.

Although the incidence of *Serratia* in clinical specimens was relatively low (1 % of enterobacteria-like strains) these organisms are usually highly resistant to antibiotics and are certainly capable of causing severe infection. That *Serratia* has become an increasing problem in the U.S.A. and has now been isolated in significant numbers in this country is sufficient cause for concern about the future of hospital-acquired infections.

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Requests for reprints should be addressed to S.T.

## REFERENCES

Anderhub, B., Pitt, T. L., Erdman, Y. J. & Willcox, W. R. (1977). Comparison of typing methods for *Serratia marcescens*. *Journal of Hygiene* **79**, 89–102.

Ball, A. P., McGhie, D. & Geddes, A. M. (1977). *Serratia marcescens* in a general hospital. *Quarterly Journal of Medicine* **40**, 63–71.

Black, W. A., Hatch, L. A., Binnie, P. & Newberry, J. (1973). *Serratia macescens* infection in a general hospital. *Journal of Clinical Pathology* **26**, 984–5.

Black, W. A. & Hodgson, R. (1971). Search for *Serratia*. *Journal of Clinical Pathology* **24**, 444–8.

Black, W. A., Pollock, A. & Batchelor, E. L. (1967). Fatal transfusion reaction due to *Serratia marcescens*. *Journal of Clinical Pathology* **20**, 883–6.

British Medical Journal – editorial (1977). *Serratia marcescens* in general hospitals. 7th May, pp. 1177–8.

Cardos, S. F., Florman, A. L., Simberkoff, M. S. & Lanier, L. (1973). *Serratia marcescens*: Use of detailed characterisation of strains to evaluate an increase of isolates in an intensive care unit. *American Journal of the Medical Sciences* **366**, 447–52.

Center for Disease Control – Guide to Proficiency Testing (Dec. 1st, 1972). *Serratia marcescens*, pp. 21–3.

Clayton, E. & Von Graevenitz, A. (1966). Non-pigmented *Serratia marcescens*. *Journal of the American Medical Association* **197**, 1059–64.

Davis, J. T., Foltz, E. & Blakemore, W. S. (1970). *Serratia marcescens*: a pathogen of increasing importance. *Journal of the American Medical Association* **214**, 2190–92.

Dodson, W. H. (1968). *Serratia marcescens* septicemia. *Archives of Internal Medicine* **121**, 145–50.

Edwards, P. R. & Ewing, W. H. (1972). In *Identification of Enterobacteriaceae*, 3rd ed., pp. 311–316. Minneapolis, Minnesota: Burgess.

Gale, D. & Sonnenwirth, A. C. (1962). Frequent human isolation of *Serratia marcescens*. *Archives of Internal Medicine* **109**, 414–21.

Hedges, R. W., Rodrigues-Lemoine, V. & Datta, N. (1975). R factors from *Serratia marcescens*. *Journal of General Microbiology* **86**, 88–92.

LANCASTER, L. J. (1962). Role of *Serratia* species in urinary tract infection. *Archives of Internal Medicine* **109**, 536–9.

LANCET – editorial (1977). The importance of *S. marcescens*, i, 636.

MAKI, D. G., HENNEKENS, C. G., PHILLIPS, C. W., SHAW, W. V. & BENNETT, J. V. (1973). Nosocomial urinary tract infection with *Serratia marcescens*: An epidemiologic study. *Journal of Infectious Diseases* **128**, 579–87.

MCCORMACK, R. C. & KUNIN, C. M. (1966). Control of a single source nursery epidemic due to *Serratia marcescens*. *Pediatrics* **37**, 750–55.

MCCRACKEN, A. W. & LIPSCOMB, F. E. (1965). *Serratia marcescens* infection complicating peritoneal dialysis. *British Medical Journal* i, 1536–7.

MELTZ, D. J. & GRIECO, M. H. (1973). Characteristics of *Serratia marcescens* pneumonia. *Archives of Internal Medicine* **132**, 359–64.

MEYER, R. D., HALTER, J., LEWIS, R. P. & WHITE, M. (1976). Gentamicin-resistant *Pseudomonas aeruginosa* and *Serratia marcescens* in a general hospital. *Lancet* i, 580–583.

PRICE, K. E., DEFURIA, M. D. & PURSIANO, T. A. (1976). Amikacin, an aminoglycoside with marked activity against antibiotic-resistant clinical isolates. *Journal of Infectious Diseases* **134**, S249–61.

SCHAEFLER, S., WINTER, J., CATELLI, A., GREENE, J. & TOHARSKI, B. (1971). Specific distribution of R factors in *Serratia marcescens* isolated from hospital infections. *Applied Microbiology* **22**, 339–43.

SCHREIER, J. B. (1969). Modification of deoxyribonuclease test medium for rapid identification of *Serratia marcescens*. *American Journal of Clinical Pathology* **51**, 711–16.

TABAQCHALI, S., CHAMBERS, T. J. & BROOKS, H. J. L. (1977). *Serratia marcescens* in hospital. *Practice* **1**, 306–7.

TAYLOR, G. & KEANE, P. M. (1962). Cross-infection with *Serratia marcescens*. *Journal of Clinical Pathology* **15**, 145–7.

TRAUB, W. H. (1972). Continued surveillance of *Serratia marcescens* infections by bacteriocin typing: Investigation of two outbreaks of cross-infection in an intensive care unit. *Applied Microbiology* **23**, 982–3.

WHITBY, J. L., BLAIR, J. N. & RAMPLING. A. (1972) A cross-infection with *Serratia marcescens* in an intensive-therapy unit. *Lancet* ii, 127–8.

WILFERT J. N., BARRETT, F. F. & KASS, E. H. (1968). Bacteraemia due to *Serratia marcescens*. *New England Journal of Medicine* **279**, 286–9.

WILKOWSKE, C. J., WASHINGTON, J. A., MARTIN, W. J. & RITTS, R. E. (1970). *Serratia marcescens*. Biochemical characteristics, antibiotic susceptibility patterns and clinical significance. *Journal of the American Medical Association* **214**, 2157–62.