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Gentamicin- and silver-resistant pseudomonas in a burns unit

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Summary and conclusions

In 1977-8 gentamicin-resistant strains of *Pseudomonas aeruginosa* became very common in a burns unit, over 90% being resistant at the peak of the outbreak. Some strains were also resistant to silver nitrate, though silver resistance was not found in any other strains of *Ps aeruginosa* isolated. Unlike the gentamicin resistance, the silver resistance was unstable, and strains became sensitive on repeated subculture. All the gentamicin-resistant strains of *Ps aeruginosa* were of the same serotype (O:11, H:2,5). Though gentamicin resistance could be transferred in vitro from resistant strains of *Ps aeruginosa* to one sensitive strain of *Ps aeruginosa*, there was no evidence of in-vivo transfer of gentamicin resistance between strains of pseudomonas in the patients' burns, nor was there evidence of transfer of gentamicin resistance between *Ps aeruginosa* and enterobacteria. Carbenicillin-resistant and gentamicin-resistant *Ps aeruginosa* were sometimes found in the same burns, but no gentamicin-carbenicillin (doubly) resistant strains were found among the 986 strains tested during the outbreak.

The outbreak of gentamicin-resistant *Ps aeruginosa* from burns was not reduced by stopping treatment with gentamicin and its analogues but only by segregating all patients with *Ps aeruginosa* in one of the two wards of the unit and admitting new patients only to the other ward.

Introduction

Systemic gentamicin and topical silver preparations have been used in this unit since the mid-1960s. For most of that period few strains of *Pseudomonas aeruginosa* isolated from the patients'

burns have been resistant to gentamicin and none have been resistant to silver.¹⁻³ In 1977, however, gentamicin resistance became very common, and the resistant strains were often also resistant to silver nitrate. Since gentamicin (and its analogues) and silver preparations are probably the most effective of the few agents available for, respectively, systemic treatment and topical prophylaxis against pseudomonal infections, the emergence of these resistant variants posed a serious threat to controlling infection in the unit.

We describe here the emergence of these resistant variants and our measures to eliminate them. We also describe in-vitro tests for transfer of resistance.

Incidence and control of resistance

METHODS

Burns were examined for bacterial flora when patients were admitted to the unit, at all changes of dressings, at operations, and daily if the burns were exposed, by methods described elsewhere.² *Ps aeruginosa*, *Escherichia coli*, *Klebsiella* spp, *Enterobacter* spp, *Proteus* spp, and other Gram-negative bacilli were identified by standard methods.⁴ All isolates of Gram-negative bacilli were tested for sensitivity to gentamicin, amikacin, carbenicillin, neomycin, trimethoprim, and sulphadiazine by a ditch-plate diffusion test.⁵ Tube dilution tests were also made on some of the strains, using nutrient broth as the culture medium, with 1 drop (0.02 ml) of a 1/1000 dilution of an overnight broth culture as the inoculum. In the ditch-plate test for gentamicin sensitivity two dilutions were used, one containing 50 mg/l and one containing 10 mg/l of gentamicin sulphate in the nutrient agar of the ditch. Strains were considered sensitive if the minimal inhibitory concentration (MIC) of gentamicin was 8 mg/l or less. All strains which were gentamicin-sensitive by the tube dilution test showed zones of inhibition in the ditch-plate diffusion test which were closely similar to that obtained with the sensitive control strain (MIC 2 mg/l) tested on the same plate; resistant strains, which had MICs of 16-32 mg/l, grew across the agar of the ditch containing 10 mg/l gentamicin.

Tests for silver nitrate resistance were performed as described by Cason *et al*,⁶ using a plate dilution method with serial dilutions of silver nitrate in nutrient agar.

Results

Gentamicin-resistant *Ps aeruginosa*

Table I shows the proportion of gentamicin-resistant strains of *Ps aeruginosa* isolated from burns from June 1977 to September 1978. Each patient had one or more burns, and each burn colonised by

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TABLE I—Gentamicin-resistant *Ps aeruginosa* isolated from burns in 1977-8

	No of patients with <i>Ps aeruginosa</i> in burns	No (%) of patients with gentamicin-resistant <i>Ps aeruginosa</i> in burns	No of strains of <i>Ps aeruginosa</i> isolated	No (%) of strains of <i>Ps aeruginosa</i> resistant to gentamicin
1977				
June	9	0	16	0
July	8	0	25	0
August	19	8 (42)	76	38 (50)
September	21	12 (57)	106	70 (66)
October	12	5 (42)	84	44 (52)
November	11	4 (36)	54	15 (28)
December	7	6 (86)	49	45 (92)
1978				
January	14	6 (43)	152	122 (80)
February	19	15 (79)	181	142 (78)
March	22	13 (59)	190	120 (63)
(Gentamicin stopped)				
April	24	13 (54)	276	131 (48)
May	26	17 (65)	236	140 (59)
June	18	11 (61)	151	111 (74)
July	16	11 (69)	210	167 (80)
(Patients segregated)				
August	12	4 (33)	99	41 (41)
September*	8	3 (38)	221	143 (65)
October 1-7	5	1 (20)	16	5 (31)

*Increased isolations in September followed treatment of one patient with gentamicin.

TABLE II—Gentamicin-resistant "coliform bacilli"* isolated from burns in the period when gentamicin-resistance in *Ps aeruginosa* was common

	No of strains tested*	No (%) of strains resistant to gentamicin	% of <i>Klebsiella aerogenes</i> resistant to gentamicin
1977			
January	65	1 (1.5)	0
February	58	1 (1.7)	8
March	37	2 (5.4)	33
April	30	0	—
May	67	2 (3.0)	25
June	31	2 (6.5)	0
July	47	3 (6.4)	30
August	93	9 (9.7)	23
September	76	8 (10.5)	30
October	73	17 (23.3)	58
November	55	2 (3.6)	8
December	42	2 (4.8)	0
1978			
January	43	1 (2.3)	0
February	52	2 (2.5)	0
March	78	0	0
(Gentamicin stopped)			
April	97	0	0
May	84	0	0
June	93	0	0
July	114	0	0
(Patients segregated)			
August	76	0	0
September	60	0	0

*Strains tested (and No and % resistant to gentamicin): *Klebsiella aerogenes* 210 (39; 19%), *Proteus* spp 385 (0), *A. anitratum* 278 (9; 3%), others 498 (4; 0.8%).

Ps aeruginosa yielded the organism on one or more successive swab samplings. The incidence of gentamicin-resistant isolates rose rapidly to 50%, and then to over 90% in December 1977 and remained high for about a year.

Some gentamicin-resistant and gentamicin-sensitive strains of *Ps aeruginosa* were phage-typed and serotyped at the Central Public Health Laboratory, Colindale. All the gentamicin-resistant strains were of one serotype (O:11, H:2,5) and untypable by phages; sensitive strains were all of other serotypes, the commonest being type O:16, H:4.

We tried to eliminate the gentamicin-resistant strains from the unit by discontinuing gentamicin and tobramycin treatment in patients with severe pseudomonal infection on 13 March 1978 and using amikacin, which was fully active against all the gentamicin-resistant strains, instead. This did not reduce the incidence of gentamicin-resistant *Ps aeruginosa*. On 15 July 1978 we segregated all patients with gentamicin-resistant pseudomonal infections or infections with *Ps aeruginosa* of unknown sensitivity in one of the two wards of the burns unit, and closed that ward to further admissions until all the patients in it with resistant pseudomonads had been discharged. The two wards had separate nursing staffs, so that *Ps aeruginosa* was not likely to be transferred from one to the other. The numbers of patients carrying gentamicin-resistant (and other) *Ps aeruginosa* in the unit fell

progressively after segregation (table I). The increase in the proportion of gentamicin-resistant isolates in September 1978, when they were thought to have been eliminated, was due to the continued presence of two patients with extensive burns, one of whom had sensitive *Ps aeruginosa* in his burns which were replaced by the resistant strain of *Ps aeruginosa* when he was treated with gentamicin; this infection was transferred to the other extensively burnt patient by cross-infection.

Table II shows the proportion of gentamicin-resistant isolates of enterobacteria (predominantly *Klebsiella* spp, *Escherichia coli*, *Proteus* spp, and *Acinetobacter anitratum*) in a sampling of one strain per patient per week from burns, and the proportion of patients with these organisms in whom the strains were resistant to gentamicin from January 1977 to September 1978. There were few gentamicin-resistant coliform bacilli and no gentamicin-resistant *Proteus* spp. *Klebsiella* spp showed some resistance, with a temporary increase in October 1977. The frequency of isolation of gentamicin-resistant enterobacteria fell several weeks before the use of gentamicin and tobramycin in the ward was stopped; their presence was apparently unrelated to the presence of gentamicin-resistant *Ps aeruginosa*, which persisted for long after the gentamicin-resistant enterobacteria had become scarce. Gentamicin-resistance in *Ps aeruginosa* rose to its highest in December 1977, when gentamicin-resistant *Klebsiella* had virtually disappeared from the unit.

Silver-resistant *Ps aeruginosa*

Strains of *Ps aeruginosa* isolated in this unit have been tested for sensitivity to silver nitrate since 1965, but no resistant strains were found until August 1977. Only strains of serotype O:11, H:2,5 (which were also resistant to gentamicin) were resistant to silver, but they lost their silver resistance on repeated subculture, while retaining their gentamicin resistance.

Carbenicillin-resistant *Ps aeruginosa*

After *Ps aeruginosa* carrying plasmids determining high-level resistance to carbenicillin had been eliminated from the unit in 1972,⁷ such strains were not seen again until October 1977, when highly resistant strains producing diffusible "carbenicillinase" reappeared. These carbenicillin-resistant strains were all of serotypes other than type O:11, H:2,5. On several occasions and in several patients two strains of *Ps aeruginosa*, one resistant to gentamicin and one highly resistant to carbenicillin, were present simultaneously in the same burn, but no strains resistant to both gentamicin and carbenicillin were found in these burns or among 986 strains of *Ps aeruginosa* tested while both gentamicin and carbenicillin resistant strains were present in the unit.

Experiments on resistance transfer

Several studies were made to discover whether the gentamicin and carbenicillin resistance of the strains of *Ps aeruginosa* and of enterobacteria could be transferred on resistance plasmids to other bacteria of the same or different species.

METHODS

We used the same technique of testing for resistance transfer as in earlier studies on sulphonamide resistance,⁸ though we modified it to suit the sensitivity and resistance patterns of the donor and recipient strains under investigation. One-ml amounts of 24-hour nutrient broth culture of donor and recipient strains were added to 2 ml of nutrient broth, and the mixed culture was incubated at 37°C for 24 hours. It was then centrifuged at 3000 rpm for 15 minutes, and the deposit was resuspended in 0.02 ml of nutrient broth. This was plated on nutrient agar containing appropriate concentrations of antibiotics to inhibit the growth of the donor strain (see below). After incubation at 37°C for 24 hours the plates were examined and the colonies were picked, identified by routine methods, tested for sensitivity to gentamicin and carbenicillin by methods described above, and tested for serotype and phage type at the Central Public Health Laboratory, Colindale.

We performed a test to see whether gentamicin resistance could be transferred from *Ps aeruginosa* to a culture of *E coli* K12 which was

resistant to 3000 mg/l of streptomycin. The selective medium contained 3000 mg/l streptomycin and 12 mg/l gentamicin. The *E coli*, if resistant to gentamicin, would grow well on this medium, and the *Ps aeruginosa* appeared as very small colonies. Tests were made on six isolates of gentamicin-resistant *Ps aeruginosa*. As an alternative method tests (on eight isolates of gentamicin-resistant *Ps aeruginosa*) were done with nutrient agar plates containing 12 mg/l gentamicin and using anaerobic culture to inhibit the growth of the pseudomonas. Twelve tests were also set up on plates of MacConkey's medium containing 12 mg/l gentamicin.

For testing the transfer of resistance plasmids from gentamicin-resistant to gentamicin-sensitive strains of *Ps aeruginosa*, recipient strains resistant to carbenicillin (MIC > 4000 mg/l) were used, and the selective medium of the plates contained 300 mg/l carbenicillin and 16 mg/l gentamicin. This inhibited both donor and recipient strains but would allow growth of either a recipient that had acquired gentamicin resistance by transfer of a plasmid from the donor or of a presumptive donor of gentamicin resistance that had acquired carbenicillin resistance from the presumptive recipient of gentamicin resistance. Thirty-four tests were made, including tests using different proportions of donor to recipient strains (1:2, 1:5, and 1:10).

The transfer of gentamicin resistance from a resistant *Klebsiella aerogenes* to a sensitive strain of *Ps aeruginosa* (Ellsworth) was also tested; the klebsiella was isolated from a burn wound which also contained gentamicin-resistant *Ps aeruginosa*. Five donor isolates were tested for transfer of resistance by plating the mixture of donor and recipient strains on a selective medium containing 16 mg/l gentamicin and 200 mg/l neomycin (which would inhibit the klebsiella but not a gentamicin-resistant pseudomonas). Tests were also made to detect possible transfer of gentamicin resistance from a resistant *Ps aeruginosa* to a sensitive klebsiella strain isolated from the same patient.

Tests for transfer of carbenicillin resistance from *Ps aeruginosa* to *E coli* K12 were made in the manner described by Roe *et al*⁹ but with selective plates containing 3000 mg/l streptomycin and 200 mg/l carbenicillin; the higher concentration of streptomycin was used because the carbenicillin-resistant *Ps aeruginosa* was moderately resistant to streptomycin. As an alternative technique, plates containing 200 mg/l carbenicillin were incubated anaerobically. Seven isolates of carbenicillin-resistant *Ps aeruginosa* were tested.

RESULTS

Tests for transfer of gentamicin-resistance from 26 donor strains of *Ps aeruginosa* to *E coli* K12 gave negative results. The results of transfer testing were assessed by aerobic culture on streptomycin-gentamicin agar plates for six of the donor strains, by anaerobic culture on gentamicin agar for eight, and by aerobic culture on gentamicin-MacConkey plates for 12.

Tests for transfer of gentamicin resistance from one strain of *Ps aeruginosa* to another of the same species were negative with 26 donor strains, using six gentamicin-sensitive recipient strains, four of them mated each with two donor strains, one (1686) with eight donor strains, and one (7188B) with 10 donor strains. With the gentamicin-sensitive strain 8005 (serotype O:16, H:4) five of the donor strains (6005, 6189, 6320, 6369, and 6332) which had failed to transfer gentamicin resistance to recipient strain 1686 transferred gentamicin resistance. Growth of these organisms on the selective medium may have been due either to the transfer of gentamicin-resistance from the donor strains or to transfer, in the opposite direction, of carbenicillin-resistance from the supposed recipient strain. To clarify this point the strains were serotyped. All five of the doubly resistant strains growing on the selective medium were found to be of serotype O:16, H:4. We therefore concluded that gentamicin resistance had been transferred from the presumptive donor of gentamicin-resistance to strain 8005. Carbenicillin resistance was not transferred from *Ps aeruginosa* to *E coli* K12 or to other strains of *Ps aeruginosa*.

Discussion

The emergence and subsequent predominance of gentamicin-resistant *Ps aeruginosa* in many burn wounds has been described by Stone,¹⁰ and resistant strains were often isolated in some centres.^{11,12} Witchitz and Chabbert¹³ and Kontomichalou *et al*¹² have described the intraspecies transfer of gentamicin resistance plasmids between different strains of *Ps aeruginosa* and also intergeneric transfer of such plasmids.

Our studies have shown the rapid emergence and continued presence of a high incidence of gentamicin-resistant *Ps aeruginosa* in 1977-8. All the gentamicin-resistant strains were of one serotype (O:11, H:2,5), though sensitive strains of other serotypes were present in the unit and were several times found in the same burn. During the same period a larger proportion of gentamicin-resistant strains of enterobacteria (mainly *Klebsiella* spp) briefly emerged, but the resistance disappeared long before the introduction of measures that reduced the incidence of gentamicin-resistant *Ps aeruginosa* in the wards. Such results contrasted with our findings when transferable carbenicillin-resistance became predominant among the strains of *Ps aeruginosa* isolated in this unit.¹ At that time several different phage or serotypes of *Ps aeruginosa* showed plasmid-determined carbenicillin-resistance. Plasmid-determined resistance of enterobacteria to tetracycline, ampicillin, kanamycin, and cephaloridine increased in parallel and persisted until all the relevant antibiotics were discontinued.^{7,14} By contrast, the gentamicin resistance of *Ps aeruginosa* which predominated in 1977-8 was apparently not transferred in the burns either to sensitive strains of *Ps aeruginosa* or to enterobacteria, though there was evidence of in-vitro transfer of gentamicin-resistance from the endemic strain to one of the recipient strains used in transfer experiments. Unlike the carbenicillin-resistant strains of *Ps aeruginosa* which were predominant in 1969-72, and which quickly disappeared from the unit when the use of carbenicillin was stopped, gentamicin-resistant *Ps aeruginosa* persisted after withdrawal of gentamicin and were eliminated only by segregating patients carrying such strains. Measures that eliminate one pattern of resistance will not necessarily eliminate another pattern. Even a multiresistance plasmid may sometimes be eliminated from a ward by withdrawing only one of the antibiotics for which the plasmid codes resistance.⁸

In-vitro tests showed that gentamicin-resistance was not transferred from *Ps aeruginosa* to *E coli* K12 and not usually transferred from one strain of *Ps aeruginosa* to another. A gentamicin-resistant klebsiella, present in burns which were also colonised by a gentamicin-resistant *Ps aeruginosa*, also did not transfer this resistance in vitro to a gentamicin-sensitive *Ps aeruginosa*. Only one of the gentamicin-sensitive strains of *Ps aeruginosa* tested as recipients accepted the plasmid (becoming doubly resistant to carbenicillin and gentamicin and retaining the serotype of the carbenicillin-resistant recipient).

Silver nitrate resistance was associated with gentamicin resistance in several strains and not found in any gentamicin-sensitive strains of *Ps aeruginosa*. But the gentamicin resistance persisted, while silver resistance was readily lost on storage or subculture of these strains, showing that there was no genetic link between these resistance characters. The relevance of silver resistance in relation to topical prophylaxis by silver nitrate and other silver preparations is uncertain, since most bacterial cells in cultures of *Ps aeruginosa* described as silver-resistant are fully sensitive. Experiments in progress, to be reported elsewhere, suggest that dressings soaked in 0.5% silver nitrate may act as a barrier against contamination of the burn by silver-resistant as well as by silver-sensitive strains.

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Alternative postmeniscectomy regimen

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Summary and conclusions

Eighty patients who had undergone routine meniscectomy were divided into two groups for postoperative management. The first 40 patients were treated by the traditional method of a compression bandage with a backsplint, followed by a 10-day stay in hospital. In the other 40 patients a compression bandage was applied, reinforced by a plaster cylinder. Weight-bearing was allowed with the aid of crutches, and patients were discharged after four days in hospital. Adequate splintage and early ambulation was more convenient and comfortable for postoperative management, and no complications ensued. Less time was needed for physiotherapy, and earlier return to work and resumption of normal activities were made easier.

Introduction

Meniscectomy is one of the commonest orthopaedic operations, and much has been published on the mechanism of the injury

and the diagnosis of a tear as well as the operative technique. Little attention, however, has been paid to postoperative management. Many orthopaedic surgeons use the traditional method, as outlined by Watson Jones¹—namely, a compression bandage with or without a backsplint and early and intensive quadriceps exercises followed by weight-bearing when the sutures are removed on the 10th day. Other workers^{2,3} have advised early mobilisation in a compression bandage, and O'Donoghue⁴ recommended weight-bearing on the second or third day. Early movement, however, is often painful, producing a large effusion and wasting of the quadriceps muscles.⁵ If movement could be prevented by adequate splintage discomfort would be lessened and the patient become ambulant sooner, thus helping to reduce quadriceps wasting. To test this theory we compared postoperative progress and function in two groups of patients. The first were treated by traditional methods and the second by immobilisation and early weight-bearing.

Patients and methods

Eighty consecutive patients who had undergone routine meniscectomy for a demonstrable lesion of the meniscus were divided into two groups for postoperative management. All operations were performed by, or under the immediate supervision of, one of us. Patients with other conditions affecting the knee—for example, ligamentous ruptures or bone pathology—were excluded. Group 1—Forty of the 80 patients were treated by compression bandage and backsplint, and quadriceps exercises were begun on the second postoperative day. Weight-bearing was not permitted until the sutures had been removed. All of these patients remained in hospital for a minimum of 10 days. Group 2—In the second group of 40 patients a compression

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TABLE I—Details of patients who had undergone meniscectomy and received either traditional postoperative treatment or were treated by adequate splintage and early mobilisation

	No	Average age	Sex		Occupation		Sporting activities			Duration of symptoms			Side of injury	
			M	F	Light	Heavy	Professional	Amateur	None	<4 wks	4 wks-3 mths	>3 mths	Medial	Lateral
Traditional management	40	28.2	30	10	36	4	3	26	11	4	16	20	26	14
Splintage and early mobilisation	40	29.9	32	8	35	5	5	25	10	6	16	18	29	11

TABLE II—Operative findings in patients who underwent meniscectomy and were managed postoperatively either traditionally or by adequate splintage and early mobilisation. Figures are numbers of patients

Type of lesion:	Posterior	Anterior	Bucket-handle	Degenerative	Cystic
Traditional management	11	10	14	2	3
Splintage and early mobilisation	10	11	15	3	1