Epidemiology of Gentamicin-Resistant, Gram-Negative Bacillary Colonization in a Spinal Cord Injury Unit

DAVID M. SHLAES,^{1,2,3*} CHARLOTTE A. CURRIE,³ GRACE ROTTER,⁴ MABEL EANES,⁴ AND RACHEL FLOYD³

Laboratory,^{1*} Medical,² Nursing,⁴ and Research³ Services, Veterans Administration Medical Center, Case Western Reserve University School of Medicine, Cleveland, Ohio 44106

Received 5 November 1982/Accepted 31 January 1983

A prospective epidemiological survey of a spinal cord injury unit for gentamicin-resistant, gram-negative bacilli was undertaken. The initial survey of the unit suggested a low level of cross-infection involving *Pseudomonas aeruginosa* and *Providencia stuartii*. However, a longitudinal study of new admissions revealed that only 13 of 52 nosocomial acquisitions could be considered to be due to cross colonization. Comparison of data on antibiotic use did not suggest selective pressure for resistant endogenous flora. Nosocomial acquisition was directly related to the length of the hospital stay. Antibiotic susceptibility testing of gentamicin-resistant, gram-negative bacilli showed only minor differences between nosocomial isolates and those present during the initial survey. Of the usual antimicrobial agents, amikacin, carbenicillin, and cefoxitin were the most active against all organisms, with the exception of *Serratia* spp. Of the new β -lactams, ceftazidime and imipenem (*N*-formimidoyl thienamycin) were most active.

Infection remains a major cause of morbidity and mortality among patients with spinal cord injuries (1, 5). Because of the prolonged hospital stay required for some of these patients, their associated injuries, and the need for urinary drainage, nosocomial infections account for a high proportion of acquired infection in these patients. Outbreaks of infection due to multiply resistant, gram-negative bacilli have been reported from a variety of hospital areas, including neurology wards (2, 7, 14). Although cross-infection has been implicated in some outbreaks, the ultimate source of infection remains unknown in others (2, 7, 14).

A high proportion of nosocomial gram-negative bacilli colonizing patients in our spinal cord injury unit (SCIU) are resistant to gentamicin. In this study we report a prospective evaluation of this endemic problem and attempt to establish the mode of spread of these organisms among patients and to determine the contribution of factors such as length of stay and antibiotic use to this spread. We also identify antibiotics likely to be useful in the empiric therapy of serious nosocomial infections in these patients.

MATERIALS AND METHODS

Specimen collection, processing, identification, and susceptibility testing. Fresh, clean voided urine or material obtained from intermittent catheterization or from tubing of patients with indwelling or condom catheters after disinfection of the sampling port was inoculated with a 0.001-ml loop. Rectal, wound,

throat, and environmental cultures were obtained using moist swabs (Culturette; Marion Scientific Corp., Rockford, Ill.). Hand cultures of personnel were done by a fingerprint method on agar plates. All samples were inoculated on MacConkey agar (Difco Laboratories, Detroit, Mich.) containing 20 μ g of gentamicin per ml. This concentration was chosen empirically to eliminate any organisms of intermediate susceptibility to gentamicin. Isolates were identified with API 20E strips (Analytab Products, Inc., Plainview, N.Y.) and supplemental tests as recommended by the manufacturer. Minimum inhibitory concentrations (MICs) for all commonly used antibiotics were determined using broth microdilution plates (Sensititer; GIBCO Laboratories, Grand Island, N.Y.). Gentamicin resistance was defined by an MIC of ≥ 8 μ g/ml. Despite plating on gentamicin-containing agar, nine isolates demonstrated gentamicin sensitivity, with MICs of 4 μ g/ml or less and were eliminated. An additional 20 organisms were eliminated because of slow growth or our inability to identify them by our techniques (one such isolate was later determined to be Centers for Disease Control group IVe, for example).

Susceptibility testing with new β -lactam antibiotics. All isolates were maintained frozen at -20 to -70°C . For this study, frozen stocks were subcultured on MacConkey agar containing 20 μ g of gentamicin per ml. Eighty-eight of the original strains were recovered. Frozen microtiter trays were prepared using plastic 96-well trays (Dynatech Laboratories, Inc., Alexandria, Va.) and a Quick-Spense II dispenser (Bellco Glass, Inc., Vineland, N.J.) pouring 100 μ l per well. Antibiotic powders of known potency were obtained from the following: cefotaxime, Hoechst-Roussel Pharmaceuticals, Somerville, N.J.; moxalactam, Eli Lilly & Co., Indianapolis, Ind.; cefoperazone, J. B. Roerig, New

York, N.Y.; imipemide (*N*-formimidoyl thienamycin), Merck Sharp & Dohme, Rahway, N.J.; ceftazidime, Glaxo Laboratories Ltd., Greenford, Middlesex, England; ceftriaxone, Hoffmann-La Roche Inc., Nutley, N.J.; cefsulodin, Abbott Laboratories, North Chicago, Ill.; piperacillin, Lederle Laboratories, Pearl River, N.Y.; mezlocillin, Miles Laboratories, Inc., Elkhart, Ind.; and ticarcillin, Beecham Laboratories, Bristol, Tenn. Powders were dissolved according to the instructions of the manufacturer and diluted in cation-supplemented (calcium, 50 mg/liter; magnesium, 25 mg/liter) Mueller-Hinton broth. Organisms were grown overnight in Mueller-Hinton broth at 35°C and diluted so that microtiter plates were inoculated with a final concentration of 10^5 CFU/ml. The trays were incubated overnight at 35°C and read for growth endpoints with transillumination.

Patient population and study protocol. The Cleveland Veterans Administration Medical Center has an 80-bed SCIU that occupies one half of the second floor of the hospital and is divided into two sections (wards 22 and 24 [Fig. 1]). There is no cross coverage by nursing staff of wards 22 and 24, although the SCIU physicians care for patients on both wards. Patients either are admitted immediately after injury or are transferred from outlying hospitals after initial stabilization. Some patients are readmitted at various times after discharge for further evaluation or treatment of complications. Initial hospitalization is often prolonged, but readmissions are usually for shorter periods of time. Although indwelling catheters may be inserted on a temporary basis for specific indications, only a few patients required an indwelling catheter during this study. Most patients either had achieved bladder automaticity or had intermittent catheterization or condom catheterization.

On day 1 of the study, 152 sites, including urine, rectums, wounds, and throats of all 48 SCIU inpatients (population A), were cultured. The hands of 22 personnel representing all shifts and 61 environmental sites were also sampled.

Fifty new admissions entering the SCIU over a 5-month period were cultured on admission as described above and once per week thereafter until discharged. This accounted for an additional 893 cultures. Patients colonized on admission were designated population B. Those acquiring nosocomial colonization or infection (≥ 7 days after admission) were classified as population C.

Analysis of data. All culture results, including API biotype numbers and MIC data, were entered into a Datatrieve program on a PDP 11/45 computer. Duplicate culture results were eliminated. A new organism was identified by species or within the species by biotype number or antibiogram. A previously isolated organism appearing at a new site was also classified as a nosocomial acquisition. An episode of cross colonization was defined as patients who were colonized by the same species of gram-negative bacilli exhibiting identical antibiograms, defined as a ≤ 1 -dilution difference of all MICs and API biotype numbers varying by one reaction at most. Frequency comparisons were performed by the chi-square test. Length of stay was compared by Student's *t* test and life table analysis with the generalized Wilcoxon test.

RESULTS

Initial survey. Twenty-eight sites on 16 patients were colonized by gentamicin-resistant, gram-negative bacilli (Tables 1 and 2). *Providencia stuartii* and *Pseudomonas aeruginosa* were

FIG. 1. Cleveland Veterans Administration Medical Center SCIU. Patients colonized with *P. stuartii* (▨) and *P. aeruginosa* (▤) and sinks from which *P. maltophilia* (▥) was isolated during the initial survey are indicated. New admissions colonized with *P. maltophilia* are also shown. E, Empty bed.

TABLE 1. Distribution of gentamicin-resistant, gram-negative bacilli by population

Organism	No. of isolates in population ^a :		
	A	B	C
<i>Acinetobacter calcoaceticus</i>	6	3	5
<i>Citrobacter amalonaticus</i>	0	1	0
<i>Enterobacter agglomerans</i>	0	0	1
<i>Escherichia coli</i>	1	3	3
<i>Klebsiella pneumoniae</i>	1	2	2
<i>Morganella morganii</i>	2	1	2
<i>Providencia alcalifaciens</i>	1	0	0
<i>Providencia rettgeri</i>	1	1	0
<i>Providencia stuartii</i>	8	1	10
<i>Pseudomonas aeruginosa</i>	8	3	14
<i>Pseudomonas cepacia</i>	0	1	0
<i>Pseudomonas maltophilia</i>	0	4	4
<i>Serratia liquefaciens</i>	0	0	1
<i>Serratia marcescens</i>	0	1	8
Others	0	4	2

^a A, Initial survey; B, colonized on admission; C, nosocomial acquisition.

isolated mainly from urine, accounted for eight isolates each, and were statistically associated with large, separate rooms on ward 22 (Fig. 1) ($P < 0.0125$ and $P < 0.005$, respectively). The *Providencia* and *Pseudomonas* strains had identical antibiograms and were geographically clustered, respectively, suggesting ongoing cross-infections at the time of the initial survey. No throat cultures were positive. Cultures of personnel were similarly unrevealing. Of the 61 environmental sites sampled, 6 of 11 sink drains or faucets yielded 13 strains of gentamicin-resistant, gram-negative rods. These included four *Pseudomonas cepacia* strains, two *Pseudomonas maltophilia* strains, and seven organisms that could not be identified by API 20E and supplemental tests. Two air vents grew mold. All other environmental cultures, including 10 settle plates from four patient areas and swabs from beds, nightstands, tables, telephones, and nursing carts, were negative.

New admissions. Fifty patients were admitted during the 5-month study period. Of these, 16 had 25 cultures positive at the time of admission (population B, Tables 1 and 2). Three colonized with *P. maltophilia* were in rooms where the organism had previously been isolated from sinks during the initial survey (Fig. 1). There was no clearly predominant organism in this group of patients; no species accounted for more than three isolates other than *P. maltophilia*, and no patients were thought to have active infections with gentamicin-resistant, gram-negative bacilli.

Twenty newly admitted patients acquired nosocomial colonization with 52 organisms during their hospitalization (population C, Tables 1 and

2). This was clearly related to their length of stay (Fig. 2). Among new admissions, the average length of stay for those acquiring nosocomial isolates was 72.8 ± 9.7 (standard error) days as compared with 39.4 ± 8.2 days for those without nosocomial acquisition ($P = 0.013$, Student's *t* test). Prior colonization with gentamicin-resistant, gram-negative bacilli did not predispose patients to acquisition of nosocomial isolates (life table analysis, generalized Wilcoxon).

The data were analyzed for evidence of cross colonization as described above. Only 13 of 52 nosocomial isolates could be considered to have been acquired by this mechanism, including the *P. maltophilia* colonization acquired by three patients, apparently from sinks in their rooms (Fig. 1). Nine instances were due to *P. aeruginosa*, although one of these is questionable since the organisms in question differed in tetracycline

TABLE 2. Distribution of gentamicin-resistant, gram-negative bacilli by site

Organism ^a	Population ^b	No. of isolates			
		Rectum	Throat	Urine	Wound
<i>A. calcoaceticus</i> ^c	A	6	0	0	0
	B	1	0	1	1
	C	2	0	2	1
<i>E. coli</i>	A	0	0	1	0
	B	3	0	0	0
	C	1	0	1	1
<i>K. pneumoniae</i>	A	1	0	0	0
	B	1	0	1	0
	C	1	0	1	0
<i>M. morganii</i>	A	1	0	1	0
	B	1	0	0	0
	C	1	0	1	0
<i>P. aeruginosa</i>	A	2	0	5	1
	B	1	0	1	1
	C	4	0	8	2
<i>P. maltophilia</i>	A	0	0	0	0
	B	3	1	0	0
	C	1	1	2	0
<i>Providencia</i> spp.	A	2	0	8	0
	B	1	0	1	0
	C	4	0	6	0
<i>Serratia</i> spp.	A	0	0	0	0
	B	0	0	1	0
	C	3	1	5	0

^a Five or more isolates.

^b A, Initial survey; B, colonized on admission; C, nosocomial acquisition.

^c *A. calcoaceticus* subsp. *anitratus*, 12 isolates; *A. calcoaceticus* subsp. *lwoffi*, 2 isolates.

FIG. 2. Acquisition of nosocomial isolates by new admissions: life table analysis showing patients remaining without nosocomial colonization by gentamicin-resistant, gram-negative bacilli.

MICs by three dilutions. The remaining episode was due to *Serratia marcescens*. Other than these episodes, geographic clustering was not observed.

Of the common species, *Acinetobacter calcoaceticus* and *P. aeruginosa* appeared to be evenly distributed among the three patient populations. *Providencia* spp. were found more frequently in the initial survey, whereas *Serratia* spp. were essentially limited to patients acquiring nosocomial colonization or infection (Table 3).

One possible explanation for this epidemiology is the selection for antibiotic resistance among endogenous patient flora because of antibiotic use. To examine the contribution of this factor, newly admitted patients acquiring nosocomial colonization were compared with those without acquisition for use of any antibiotic, specifically, use of an aminoglycoside antibiotic. Of 20 patients acquiring nosocomial colonization, 12 were treated with antibiotics, including 4 who received aminoglycosides before colonization occurred. Of the 30 patients who did not acquire nosocomial colonization, data on antibiotic use were available for 19. Eleven had received antibiotics, including two who were given aminoglycosides. The difference between these two groups in terms of antibiotic use in general or aminoglycoside use in particular was not statistically significant (Fisher's exact test).

Combined data. The most prevalent organisms seen were *P. aeruginosa* (25 isolates), *Providencia*

spp. (22 isolates), *A. calcoaceticus* (14 isolates), and *Serratia* spp. (10 isolates) (Table 1). These strains accounted for 68% of all isolates. Of the strains accounting for at least five isolates throughout the study, the number from rectums and urine were almost identical, 40 and 46, respectively (Table 2). *A. calcoaceticus*, *Escherichia coli*, *Klebsiella pneumoniae*, *Morganella morganii*, and *P. maltophilia* colonized the rectum more frequently than did *P. aeruginosa*, *Providencia* spp., or *Serratia* spp. (Table 2). Only three isolates from the pharynx were identified during the entire study, and two of these were *P. maltophilia* (Table 2). Wounds (mainly pressure sores) only accounted for seven isolates, four of which were *P. aeruginosa* (Table 2).

Antibiotic susceptibility testing (standard agents). The antibiotic susceptibility of all species represented by five or more isolates is shown in Table 4. For *A. calcoaceticus*, tobramycin, amikacin, and carbenicillin were the most active agents. For *P. aeruginosa*, amikacin was the most active drug, whereas *P. maltophilia* was somewhat susceptible to carbenicillin. The *Serratia* spp. were resistant to all agents tested. *Providencia* spp. and *M. morganii* were susceptible to carbenicillin, cefoxitin, and amikacin, whereas other members of the family *Enterobacteriaceae* were susceptible to cefoxitin and amikacin. There were only minor differences in the antibiograms of the common isolates when the initial survey and nosocomial populations were compared.

Antibiotic susceptibility testing (new β -lactams). The most active agents were imipenem and ceftazidime. Piperacillin was superior to mezlocillin against *P. aeruginosa*, but otherwise these two drugs appeared to be comparable. Both were more active than ticarcillin. Ceftriaxone and cefsulodin were both active against *P. aeruginosa*, whereas cefoperazone was less active against this organism. Cefotaxime, moxalactam, imipenem, ceftazidime, and ceftriaxone all had good activity against members of the family *Enterobacteriaceae*, whereas cefoperazone was again less active. Even *Serratia* spp., resistant to all previously tested agents, were inhibited by low concentrations of the new cephalosporins, with the exception of cefoperazone and cefsulodin.

DISCUSSION

Although most other studies of SCIUs and similar units have addressed specific outbreaks, few prospective studies of an endemic situation have been published (9, 11, 12). Because of the high endemic rate of colonization and infection with resistant gram-negative bacilli in the SCIU, we initiated such a study. An initial survey of the

TABLE 3. Distribution of gentamicin-resistant, gram-negative bacilli among patient populations

Organism	Population ^a	No. of isolates ^b		
<i>A. calcoaceticus</i>	A	6	} NS	} NS
	B	3		
	C	5		
<i>Providencia</i> spp.	A	10	} 0.017	} 0.090
	B	2		
	C	10		
<i>P. aeruginosa</i>	A	8	} NS	} NS
	B	3		
	C	14		
<i>Serratia</i> spp.	A	0	} NS	} 0.016
	B	1		
	C	9		

^a A, Initial survey; B, colonized on admission; C, nosocomial acquisitions.

^b Statistics by Fisher's exact test; NS, not significant.

two wards revealed a low level of cross-infection involving *P. stuartii* and *P. aeruginosa* in two large rooms, respectively. Despite this, cross-infection appeared to play only a minor role during the prospective study. Other than colonization possibly related to two sinks contaminated with *P. maltophilia*, only 10 of 49 nosocomial acquisitions could be accounted for by this mechanism. The possible discrepancy between observations during the initial survey and during the prospective study of new admissions may be related to greater care on the part of personnel, since the infection control nurses were frequently on the ward to collect specimens. Further, patterns of antibiotic use did not differ between patients who did and those who did not acquire nosocomial isolates. The lack of an explanation of outbreaks of infection and colonization of the urinary tract has been observed before, especially with *Providencia* spp. (6, 7). In those studies, as in ours, the length of stay appeared to be the most important factor determining nosocomial acquisition (7). Others have observed that gram-negative bacilli may become part of the endogenous flora of spinal cord injury patients, resulting in prolonged colonization and difficulty in eradicating the organisms from ordinarily non-colonized sites such as the skin (3, 12).

The epidemiology observed on our ward could be explained by the transmission of a single plasmid carrying gentamicin resistance and perhaps other markers among endogenous flora. Preliminary studies suggest an endemic plasmid carrying gentamicin resistance in many of the gram-negative bacilli derived from this study (15). The association of *Serratia* spp. with nosocomial acquisition may then be explained by the transfer of this marker to a relatively more pathogenic organism during the course of

the study. This mechanism has been proposed by Schaberg et al. (13) to explain one outbreak.

As others have observed, *Providencia* spp., *P. aeruginosa*, and *Serratia* spp. were more likely to colonize the urine than other sites, whereas *A. calcoaceticus*, *K. pneumoniae*, *E. coli*, *M. morganii*, and *P. maltophilia* were more likely to colonize the rectums in our patients. The lack of pharyngeal colonization observed in our study is of interest and may suggest differences between spinal cord injury patients and other populations, such as nursing home or intensive care unit patients (8, 16) in whom pharyngeal colonization with gram-negative bacilli occurs more frequently. An alternative explanation is that gentamicin-resistant organisms are less likely to colonize the upper respiratory tract than are susceptible strains.

Of the usual antibiotics tested, amikacin, carbenicillin, and ceftazidime appeared to be the most active against our resistant strains. However, none of these would be acceptable as a single agent used in expectant therapy of a potentially serious nosocomial infection in one of our SCIU patients. Our *Serratia* spp. were generally resistant to all agents tested, including amikacin. Our susceptibility studies with new β -lactams against gentamicin-resistant strains were, in general, consistent with the results of others (4, 10). Ceftazidime and imipenem were the most active agents tested. The in vitro performance of ceftazidime against these multiply resistant organisms was disappointing. These new β -lactams offer hope for single-agent therapy of potentially serious nosocomial infections in our SCIU. Clinical studies will be required to confirm this impression.

Further studies of SCIUs in other institutions and the study of other units at the Cleveland

TABLE 4. Antibiotic susceptibility of gentamicin-resistant, gram-negative bacilli

Organism ^a	No. of isolates	Drug	MIC (μ g/ml) ^b	
			50%	90%
<i>A. calcoaceticus</i>	14	Ampicillin	16	>32
		Carbenicillin	16	>512
		Cephalothin	>128	>128
		Cefoxitin	64	>128
		Cefamandole	64	128
		Amikacin	8	>32
		Tobramycin	4	>16
		Kanamycin	32	>64
		Chloramphenicol	64	>64
		Tetracycline	16	>32
	10	Cefotaxime	4	16
		Moxalactam	16	32
		Cefoperazone	8	32
		Imipemide	≤ 0.5	1.0
		Ceftazidime	2	4
		Ceftriaxone	8	16
		Cefsulodin	32	64
		Piperacillin	8	8
		Mezlocillin	8	16
		Ticarcillin	8	32
<i>E. coli</i>	7	Ampicillin	>32	>32
		Carbenicillin	>512	>512
		Cephalothin	32	>128
		Cefoxitin	4	8
		Cefamandole	8	>128
		Amikacin	2	32
		Tobramycin	>16	>16
		Kanamycin	>64	>64
		Chloramphenicol	>64	>64
		Tetracycline	32	>32
	7	Cefotaxime	≤ 0.5	≤ 0.5
		Moxalactam	≤ 0.5	≤ 0.5
		Cefoperazone	4	>64
		Imipemide	≤ 0.5	≤ 0.5
		Ceftazidime	≤ 0.5	2
		Ceftriaxone	≤ 0.5	≤ 0.5
		Cefsulodin	64	>128
		Piperacillin	256	>256
		Mezlocillin	256	>256
		Ticarcillin	>256	>256
<i>K. pneumoniae</i>	5	Ampicillin	>32	>32
		Carbenicillin	>512	>512
		Cephalothin	16	>128
		Cefoxitin	8	16
		Cefamandole	4	128
		Amikacin	2	2
		Tobramycin	8	16
		Kanamycin	64	>64
		Chloramphenicol	8	>64
		Tetracycline	4	>32
	4	Cefotaxime	≤ 0.5	32
		Moxalactam	≤ 0.5	4
		Cefoperazone	1	>64
		Imipemide	≤ 0.5	>64
		Ceftazidime	≤ 0.5	4

TABLE 4—Continued

Organism ^a	No. of isolates	Drug	MIC (μg/ml) ^b	
			50%	90%
<i>M. morganii</i>		Ceftriaxone	≤0.5	>64
		Cefsulodin	128	>128
		Piperacillin	8	>256
		Mezlocillin	16	>256
		Ticarcillin	>256	>256
	5	Ampicillin	>32	>32
		Carbenicillin	4	256
		Cephalothin	>128	>128
		Cefoxitin	8	64
		Cefamandole	32	128
		Amikacin	2	16
		Tobramycin	16	16
		Kanamycin	64	>64
		Chloramphenicol	>64	>64
		Tetracycline	>32	>32
	5	Cefotaxime	≤0.5	≤0.5
		Moxalactam	≤0.5	≤0.5
		Cefoperazone	≤0.5	2
		Imipemide	2	2
		Ceftazidime	≤0.5	≤0.5
		Ceftriaxone	≤0.5	≤0.5
		Cefsulodin	128	>128
		Piperacillin	2	8
		Mezlocillin	2	4
		Ticarcillin	8	256
<i>Providencia</i> spp.	22	Ampicillin	32	>32
		Carbenicillin	8	>512
		Cephalothin	128	>128
		Cefoxitin	4	128
		Cefamandole	8	>128
		Amikacin	4	32
		Tobramycin	16	>16
		Kanamycin	4	32
		Chloramphenicol	32	>64
		Tetracycline	>32	>32
	21	Cefotaxime	≤0.5	1
		Moxalactam	≤0.5	≤0.5
		Cefoperazone	1	16
		Imipemide	1	2
		Ceftazidime	≤0.5	1
		Ceftriaxone	≤0.5	1
		Cefsulodin	64	64
		Piperacillin	2	128
		Mezlocillin	4	128
		Ticarcillin	2	>256
<i>P. aeruginosa</i>	25	Ampicillin	>32	>32
		Carbenicillin	512	>512
		Cephalothin	>128	>128
		Cefoxitin	>128	>128
		Cefamandole	>128	>128
		Amikacin	1	8
		Tobramycin	16	>16
		Kanamycin	>64	>64
		Chloramphenicol	>64	>64
		Tetracycline	>32	>32

TABLE 4—Continued

Organism ^a	No. of isolates	Drug	MIC (μg/ml) ^b	
			50%	90%
	23	Cefotaxime	16	64
		Moxalactam	16	64
		Cefoperazone	32	>64
		Imipemide	1	4
		Ceftazidime	4	8
		Ceftriaxone	8	64
		Cefsulodin	2	64
		Piperacillin	32	128
		Mezlocillin	128	>256
		Ticarcillin	256	>256
<i>P. maltophilia</i>	8	Ampicillin	>32	>32
		Carbenicillin	32	512
		Cephalothin	>128	>128
		Cefoxitin	>128	>128
		Cefamandole	>128	>128
		Amikacin	>32	>32
		Tobramycin	>16	>16
		Kanamycin	>64	>64
		Chloramphenicol	32	>64
		Tetracycline	16	>32
	9	Cefotaxime	32	64
		Moxalactam	2	8
		Cefoperazone	4	32
		Imipemide	>64	>64
		Ceftazidime	2	64
		Ceftriaxone	64	>64
		Cefsulodin	16	>128
		Piperacillin	32	256
		Mezlocillin	16	256
		Ticarcillin	32	>256
<i>Serratia</i> spp.	10	Ampicillin	>32	>32
		Carbenicillin	>512	>512
		Cephalothin	>128	>128
		Cefoxitin	>128	>128
		Cefamandole	>128	>128
		Amikacin	>32	>32
		Tobramycin	>16	>16
		Kanamycin	>64	>64
		Chloramphenicol	>64	>64
		Tetracycline	>32	>32
	9	Cefotaxime	2	8
		Moxalactam	4	32
		Cefoperazone	32	>64
		Imipemide	≤0.5	2
		Ceftazidime	1	4
		Ceftriaxone	2	8
		Cefsulodin	>128	>128
		Piperacillin	>256	>256
		Mezlocillin	>256	>256
		Ticarcillin	>256	>256

^a Organisms not listed: *Citrobacter amalonaticus* (one isolate), *Enterobacter agglomerans* (one isolate), *Proteus mirabilis* (two isolates), *P. cepacia* (one isolate), and others (four isolates).

^b 50% and 90%, MIC at which 50 and 90% of the isolates were inhibited, respectively.

Veterans Administration Medical Center will be required for greater understanding of the spread of resistant gram-negative bacilli among patients. Plasmid analysis of our strains may also provide additional useful epidemiological information.

ACKNOWLEDGMENTS

This work was supported by a Veterans Administration grant.

We are grateful to G. Hall of the Cleveland Clinic Foundation for preparing the frozen broth microdilution trays used in this study. We thank Philip J. Spagnuolo for thoughtful review of the manuscript and Elizabeth Williams for excellent secretarial assistance. We are especially grateful to the staff of the SCIU for their cooperation.

LITERATURE CITED

- Breithaup, D. J., A. T. Jousse, and M. Wynn-Jones. 1961. Late causes of death and life expectancy in paraplegia. *Can. Med. Assoc. J.* 85:73-77.
- Flerer, J., and M. Ekstrom. 1981. An outbreak of *Providencia stuartii* urinary tract infections. Patients with condom catheters are a reservoir of the bacteria. *J. Am. Med. Assoc.* 245:1553-1555.
- Gilmore, D. S., G. D. Aeilts, B. A. Alldis, S. K. Bruce, E. M. Jimenez, D. G. Schick, J. W. Morrow, and J. Z. Montgomerie. 1981. Effects of bathing on *Pseudomonas* and *Klebsiella* colonization in patients with spinal cord injuries. *J. Clin. Microbiol.* 14:404-407.
- Hall, W. H., B. J. Opfer, and D. N. Gerding. 1980. Comparative activities of the oxa- β -lactam LY127935, cefotaxime, cefoperazone, cefamandole, and ticarcillin against multiply resistant gram-negative bacilli. *Antimicrob. Agents Chemother.* 17:273-279.
- Jousse, A. T., M. Wynn-Jones, and D. J. Breithaup. 1968. A follow-up study of life expectancy and mortality in traumatic transverse myelitis. *Can. Med. Assoc. J.* 98:770-773.
- Kaslow, R. A., J. O. Lindsey, A. L. Bisno, and A. Price. 1976. Nosocomial infection with highly resistant *Proteus rettgeri*. Report of an epidemic. *Am. J. Epidemiol.* 104:278-286.
- Klustersky, J., A. M. Bogaerts, J. Noterman, E. Van Laer, D. Daneua, and E. Mouawad. 1974. Infections caused by *Providencia* bacilli. *Scand. J. Infect. Dis.* 6:153-160.
- Mackowiak, P. A., R. M. Martin, S. R. Jones, and J. W. Smith. 1978. Pharyngeal colonization by gram-negative bacilli in aspiration-prone persons. *Arch. Intern. Med.* 138:1224-1227.
- Maizels, M., and A. J. Schaeffer. 1980. Decreased incidence of bacteriuria associated with periodic instillations of hydrogen peroxide into the urethral catheter drainage bag. *J. Urol.* 123:841-845.
- Michael, P. R., R. H. Alford, and Z. A. McGee. 1981. Superior activity of *N*-formimidoyl thienamycin against gentamicin-resistant *Pseudomonas aeruginosa*. *Antimicrob. Agents Chemother.* 20:702-704.
- Moloney, P. J., A. A. Doyle, B. L. Robinson, H. Fenster, and M. G. McLoughlin. 1981. Pathogenesis of urinary infection in patients with acute spinal cord injury on intermittent catheterization. *J. Urol.* 125:672-673.
- Montgomerie, J. Z., and J. W. Morrow. 1980. Long-term *Pseudomonas* colonization in spinal cord injury patients. *Am. J. Epidemiol.* 112:508-517.
- Schaberg, D. R., C. E. Rubens, R. H. Alford, W. E. Farrar, W. Schaffner, and Z. A. McGee. 1981. Evolution of antimicrobial resistance and nosocomial infection. Lessons from the Vanderbilt experience. *Am. J. Med.* 70:445-448.
- Schaberg, D. R., R. A. Weinstein, and W. E. Stamm. 1976. Epidemics of nosocomial urinary tract infection caused by multiply resistant gram-negative bacilli: epidemiology and control. *J. Infect. Dis.* 133:363-366.
- Shlaes, D. M., and C. A. Currie. 1983. Endemic gentamicin resistance R factors on a spinal cord injury unit. *J. Clin. Microbiol.* 18:236-241.
- Valenti, W. M., R. G. Trudell, and D. W. Bentley. 1978. Factors predisposing to oropharyngeal colonization with gram-negative bacilli in the aged. *N. Engl. J. Med.* 298:1108-1111.