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Oropharyngeal and Fecal Carriage of *Pseudomonas aeruginosa* in Hospital Patients

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This prospective study was designed to determine the incidence of rectal and/or oropharyngeal colonization rates of patients with *Pseudomonas aeruginosa* upon admission to a general hospital and the risk of becoming colonized while hospitalized. Consecutive 186 admissions (180 patients) to one medical ward, one surgical ward, and the intensive care unit were studied over a period of 5 months. Rectal and oropharyngeal swabs for *P. aeruginosa* were obtained on admission, weekly thereafter, and/or upon discharge. Forty-two patients (22.6%) were colonized on admission, and 20 patients (10.8%) acquired *P. aeruginosa* during hospitalization. Colonization on admission was observed twice as frequently on the surgical ward and in the intensive care unit as on the medical ward. Positive rectal cultures were more frequent than oropharyngeal cultures throughout the study ($P < 0.01$). For patients admitted culture positive or culture negative, the probabilities of remaining culture positive or culture negative, respectively, remained at 44 and 72% after 35 days of hospitalization. The most common *P. aeruginosa* serotypes were 1, 6, and 10, and pyocin types 1, 3, and 10 were predominant. There was no statistical difference in the serotypes or pyocin types detected on admission or acquired during hospitalization. Except for two hospital-acquired first isolates which were resistant to moxalactam, all first isolates were susceptible to the four antibiotics tested. During the study, one isolate became resistant to azlocillin, gentamicin, and tobramycin, while two isolates became resistant to moxalactam. A statistical analysis was performed for 13 risk factors for all colonized and noncolonized patients. Colonization detected at the time of admission was positively associated with age (>65 years), previous surgery of the gastrointestinal tract for neoplasm, and anemia ($P < 0.05$). In contrast, for patients who entered the study culture negative, none of the analyzed 13 risk factors was associated with an increased probability for colonization. This observation included the administration of antimicrobial agents singly or in combination or both.

The incidence of serious *Pseudomonas aeruginosa* infections in hospitalized patients has continued to increase during the last three decades (1–3, 10, 14, 16). Most epidemiologic studies concerned with *P. aeruginosa* have included sources and mechanisms of transmission to patients from the hospital environment. These sources frequently included contaminated solutions of disinfectants, aspirators, incubators, and respiratory equipment (4, 19, 26, 30, 33). In addition, it has been demonstrated that fresh vegetables, salads, and feeding formulas may be responsible for colonization of the gastrointestinal (GI) tract of many hospitalized patients (11, 21). It is well known that systemic *P. aeruginosa* infections may result from prior colonization of patients (1, 3, 6, 10, 16, 26, 28, 29).

Most studies describing the carriage rate of *P. aeruginosa* in the GI tract have been concerned with healthy adults or patients with hematologic malignancies or burns (6, 7, 9, 13, 25, 28, 29, 31–35). Little information is available about the incidence rates of *P. aeruginosa* colonization of the patient population in a general hospital. This prospective study was designed to detect the incidence of rectal and oropharyngeal colonization rates of patients with *P. aeruginosa* on admission, during hospitalization, and at discharge. A correlation of these findings is made with the clinical and demographic data of the patients as well as with serotype, pyocin type, and antimicrobial susceptibilities of the isolates.

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MATERIALS AND METHODS

Patient population. Consecutive new admissions to one general medical 40-bed ward and one general surgical 40-bed ward as well as admissions to a 16-bed intensive care unit (ICU) of the 800-bed Albany Veterans Administration Medical Center between 1 June 1982 and 13 September 1982 were included in this study. Of the 186 patient admissions for 180 patients (6 patients had 2 admissions), 80 admissions (1 June to 23 August) were on the medical ward, 74 (8 July to 13 September) were on the surgical ward, and 32 (20 July to 13 September) were into the ICU (only 8 of the ICU admissions were direct; 24 were transfers from other hospital wards). The records of all 180 patients were reviewed for an analysis of the demographic and clinical data, which included the following: patient age, sex, and race, length of hospital stay, diabetes mellitus, anemia (hematocrit of <30%), history of GI or other malignancy, history of GI tract or other surgery, colostomy, ileostomy, previous admission to the hospital within 3 to 6 months, administration of corticosteroids or cytotoxic agents, radiation therapy, and administration of oral or parenteral antimicrobial agents or both, singly or in combination.

Culturing technique and isolation of *P. aeruginosa*. Swab specimens (Culturette; Marion Scientific, Div. Marion Laboratories, Inc., Kansas City, Mo.) from the oropharynx and rectum were obtained from patients on admission, weekly for 8 weeks, and/or on discharge. The specimens were first inoculated onto plates containing MacConkey agar, heart infusion agar with 0.09% cetrimide, and Pseudosel agar (BBL Microbiology Systems, Cockeysville, Md.) and then

placed into nutrient broth containing 0.03% cetrimide (20, 22, 24, 25, 28). All culture plates were incubated for 18 to 24 h at 37°C. The broth cultures were subcultured in 24 h by swabbing onto cetrimide agar plates. The primary (direct culture) and secondary (broth subculture) plates were kept at room temperature for an additional 24 h before examination for presence of *P. aeruginosa* colonies. Blue-green- or red-pigmented colonies giving a rapid oxidase-positive reaction were accepted as *P. aeruginosa*. Non-lactose-fermenting colonies from MacConkey agar and nonpigmented colonies from Pseudosel and cetrimide plates were examined for oxidase reaction. The growth was quantitated from 1+ to 4+, with 4+ representing confluent growth and 3+, 2+, and 1+ representing >30 colonies, 5 to 30 colonies, and <5 colonies, respectively. All nonpigmented oxidase-positive isolates were further characterized by biochemical reactions in the API 20E system (Analytab Products, Div. of Ayerst Laboratories, Plainview, N.Y.), supplemented by additional tests for nitrate reduction, motility, oxidation or fermentation of glucose in oxidation-fermentation basal medium (BBL), and growth at 42°C. *P. aeruginosa* isolates recovered within 24 h of hospital admission were designated as "present on admission," and those isolates recovered following the initial week of hospitalization were designated as "hospital acquired."

***P. aeruginosa* typing techniques.** Serotyping of all *P. aeruginosa* isolates was done by utilizing commercially prepared antisera (Difco Laboratories, Detroit, Mich.) to the 17 somatic O antigens as determined by the International Antigenic Typing Scheme (15, 27). This slide agglutination technique utilized heat-killed bacterial cells. Agglutination reactions of >25% (>2+) were considered positive. Pyocin typing was performed by the method of Gillies and Govan, using eight indicator strains (17, 18). All tests were performed in duplicate on the same day, and four known control strains were included in each experimental run.

Antibiotic susceptibility testing. All *P. aeruginosa* isolates were tested for susceptibility to tobramycin, gentamicin, azlocillin, and moxalactam. Gentamicin, azlocillin, and moxalactam were supplied as dry powders by the Schering Corp., Kenilworth, N.J.; Miles Laboratories, Inc., Elkart, Ind.; and Eli Lilly & Co., Indianapolis, Ind., respectively. Tobramycin sulfate (Eli Lilly) was supplied as a solution containing 1,000 μg/ml. All antimicrobial agents were reconstituted immediately before use. The agar dilution method, utilizing a Steers replicator, was used (36). One microliter of a 4 to 6 h bacterial culture containing 10^6 CFU/ml was applied to the plate. The MIC, in micrograms per milliliter, was read as the lowest concentration of antimicrobial agent at which complete inhibition, a barely visible haze, or a single colony was found.

Statistical analysis. Interrelationships among the variables were investigated by using the methodology of log linear hierarchical models (5) and a priori rationale. Maximum-likelihood estimates of elementary cells were obtained by using iterative proportional fitting of the sufficient configurations. The likelihood ratio statistic was used to summarize and test for goodness of fit. The actuarial method was used to summarize the follow-up data with respect to patient-days at risk of acquiring *P. aeruginosa* and the probabilities of remaining *P. aeruginosa* culture positive or negative (12). Null hypotheses were tested at the 0.05 level of significance.

RESULTS

A total of 180 patients contributed 186 admissions to the study (6 patients had 2 admissions during the study period) (Table 1). Two patients were female. Forty-two (67.7%) of the colonized patients were colonized on admission, and 20 patients (32.3%) acquired *P. aeruginosa* during hospitalization. Of the patients admitted to the medical ward, 38% entered the study in June and none entered after 23 August 1982, while 38% of patients entering the surgical ward were admitted in late August and early September and none entered in June. On first admission to the study, the proportion of patients admitted to the surgical ward culture positive was statistically significantly greater ($P < 0.01$) than the corresponding proportion admitted to the medical ward (33.8 versus 13.2%). The difference was not a function of date of entry: for patients on both the medical and surgical wards, the proportions of patients entering the study culture positive did not vary significantly over the 4-week periods of patient entry. The most frequently colonized site was the rectum; oropharyngeal colonization alone was detected in three patients only, and ten patients had colonization in both sites. Of the 80 patients admitted to the medical ward, 10 patients (12.5%) were culture positive on admission and 9 patients (12.3%) acquired the organism during hospitalization. *P. aeruginosa* was isolated from the oropharynx of only one patient on the medical ward, and he acquired the organism after the first week of hospitalization. In contrast, of 74 patients admitted to the surgical ward, 24 (32.4%) were colonized on admission and 8 patients (10.8%) acquired the organism during hospitalization. Of these 32 culture-positive patients, 2 had *P. aeruginosa* in the oropharynx only, and 5 patients had the organism in the rectal and oropharyngeal cultures.

TABLE 1. Number of patients with positive cultures for *P. aeruginosa* in the oropharynx or rectum or both[a]

Ward (no. of patients)	No. of patients culture positive		% Positive[b]
	Upon admission	Acquired	
Medicine (80)	10	9	23.8
Surgery (74)	24	8	43.2
ICU (32)	8	3	34.4

[a] Rectum, 49 patients; oropharynx, 3 patients; rectum and oropharynx, 10 patients.
[b] Total percent positive, 33.3%.

Of the 32 patients studied in ICU (8 patients admitted directly and 24 admitted from other wards of the hospital), 11 patients (34.4%) were culture positive: 8 (72.7%) were positive on admission (from other hospital wards) and 3 (27.3%) acquired the organism after admission to ICU. Of the 11 culture-positive patients, 5 were colonized in both the rectum and oropharynx.

Overall, the percentage of positive rectal cultures was significantly higher ($P < 0.01$) than that of positive oropharyngeal cultures for those culture-positive patients at time of entry into the study (20 versus 3%) and for those colonized during the study (14 versus 3%). For patients who were admitted with positive cultures, there was a positive association between the occurrences of positive cultures in both areas ($P < 0.05$). For patients admitted with negative cultures but who subsequently acquired *P. aeruginosa*, no association between positive rectal and oropharyngeal cultures was demonstrated.

For patients culture positive on admission and remaining hospitalized and in the study for more than 24 h, the observed probability of remaining positive following admis-

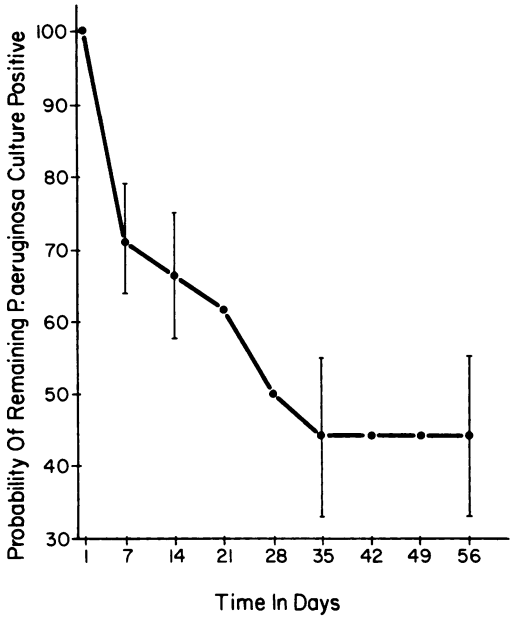

FIG. 1. Probability of remaining *P. aeruginosa* culture positive in the GI tract after admission to the study. Vertical bar represents 2 standard errors of the estimated probability of remaining culture positive.

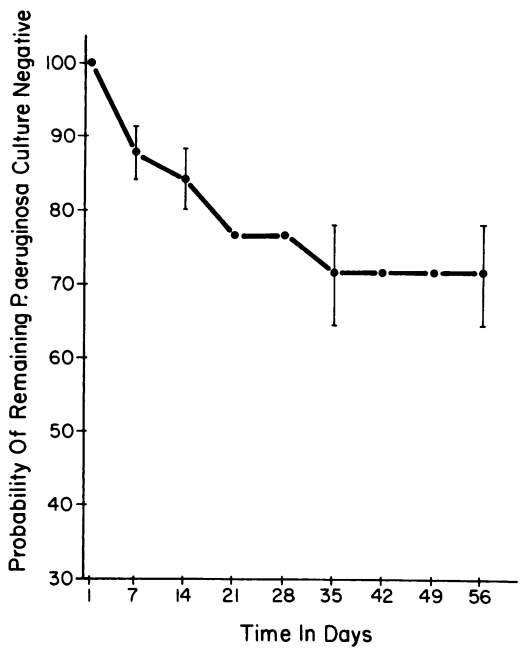

FIG. 2. Probability of remaining *P. aeruginosa* culture negative in the GI tract after admission to the hospital. Vertical bar represents 2 standard errors of the estimated probability of remaining culture negative.

sion to the study is illustrated in Fig. 1. Medical, surgical, and ICU patients were combined for this analysis. A steep decline from 100 to 70% occurred by 1 week, followed by a slower decline during the subsequent 2 to 8 weeks. From day 35 to the end of the study on day 56, 44% of the patients remained positive. Except for one surgical patient, only patients in ICU maintained positive cultures during the last 3 weeks of the study.

For patients culture negative on admission and remaining hospitalized and in the study for more than 24 h, the observed probability of remaining negative following admission from the community is illustrated in Fig. 2. Medical and surgical patients were combined, but the ICU patients were excluded from this analysis. A decline from 100 to 88% occurred by week 1, followed by a gradual decline to 72% by day 35, after which no further acquisition of *P. aeruginosa* was detected. The corresponding curves calculated for the medical and surgical patients separately were in close agreement. Collectively, the eight patients admitted to the ICU from the community spent a total of 107 days in the study; none became colonized.

For the 121 patients who entered the study culture negative on the first admission and for whom subsequent cultures were done, the total number of days spent in the hospital free of *P. aeruginosa* was 1,760. Approximately 1 (1.1 days) of every 100 days, on the average, yielded a positive culture. This risk of acquiring *P. aeruginosa* was constant for the three services and did not vary significantly over the 5 months of the study.

Table 2 depicts the pyocin types of 54 *P. aeruginosa* isolates obtained from patients during this study. Eight isolates obtained from patients entering ICU culture positive from other hospital wards were excluded from the analysis. The overall typability of strains producing pyocin was 85%. Only 7 of 35 known pyocin types were represented in this study. Of the 22 nonclassifiable strains, 81.8% were inhibited by *P. aeruginosa* marker strains 1, 2, 3, 5, 7, and 8, indicating a particular stable pyocin pattern among our isolates. Among the typable strains of *P. aeruginosa*, pyocin types 1, 3, and 10 were observed most frequently. More than one pyocin type of *P. aeruginosa* was isolated from 11 patients. Of the 10 patients culture positive in both rectal and oropharyngeal sites, 7 patients had the same pyocin type and the remaining 3 had different types in the two areas. Pyocin typing of the isolates obtained from eight culture-positive patients entering ICU were types 1 (three patients), 3, 10, and 31 (1 patient each), and two isolates were nontypable. There was no statistical difference in the types observed on admission from the community or types acquired in the hospital.

Serotyping data of the 54 *P. aeruginosa* isolates are depicted in Table 3. The typability was 93%, with 25.9% of

TABLE 2. Pyocin types of *P. aeruginosa* islates from the rectum and oropharynx

Pyocin type	No. of *P. aeruginosa* isolates		%
	Upon admission[a]	Acquired	
1	6	2	14.8
3	1	4	9.3
5	1	0	1.9
10	4	1	9.3
22	2	0	3.7
28	1	0	1.9
31	0	2	3.7
Nonclassifiable[b]	13	9	40.7
No inhibition[c]	6	2	14.8

[a] Eight patients admitted to ICU from other hospital areas are excluded.
[b] Nonclassifiable within the known 35 types (81.8% inhibited markers 1, 2, 3, 5, 7, and 8).
[c] Non-pyocin-producing strains.

TABLE 3. Serotypes of *P. aeruginosa* isolates from the rectum and oropharynx

Serotype	No. of *P. aeruginosa* isolates		%
	Upon admission[a]	Acquired	
1	5	3	14.8
3	2	0	3.7
5	2	1	5.6
6	5	3	14.8
10	4	1	9.3
11	3	2	9.3
12	0	2	3.7
14	0	1	1.9
16	1	1	3.7
Polyagglutination[b]	10	4	25.9
No agglutination	2	2	7.4

[a] Eight patients admitted to ICU from other hospital areas are excluded.
[b] Agglutination with more than one serotype.

the strains showing polyagglutination. Only 9 of the 17 known serotypes are represented. Of the typable strains, serotypes 1, 6, 10, and 11 were observed most frequently. More than one serotype was isolated from nine patients. Of the nine culture-positive patients in both rectal and oropharyngeal areas, six patients had the same serotype, and in the remaining three patients different serotypes were detected. Serotypes of *P. aeruginosa* isolates obtained from eight culture-positive patients upon entering ICU revealed serotypes 6 (two patients), 11, 12, and 16 (1 patient each), and three isolates showed polyagglutination. There was no statistical difference in the *P. aeruginosa* serotypes isolated from patients on admission or serotypes acquired during hospitalization.

Antimicrobial susceptibilities for the 54 *P. aeruginosa* isolates (34 obtained on admission from the community and 20 acquired during hospitalization) were determined for four antibiotics. Except for two hospital-acquired first isolates which were resistant to moxalactam (MIC, >128 μg/ml), all first isolates were susceptible to the four antibiotics tested, including the eight strains from patients entering the ICU from other areas of the hospital. Of the 44 follow-up isolates obtained from patients enrolled in the study, 1 isolate became moderately resistant to azlocillin (MIC, 128 μg/ml), gentamicin (8 μg/ml), and tobramycin (8 μg/ml), while two isolates became resistant to moxalactam (MIC, >128 μg/ml). The serotypes and pyocin types of these isolates remained unchanged from the original type.

Two of the 62 culture-positive patients developed bacteremia during the study period. The isolates from one patient in ICU showed the following: blood—serotype 1, pyocin type 31; rectum (two isolates)—serotype 16 and polyagglutinating, pyocin types nonclassifiable and no inhibition; pharynx (two isolates)—serotype 16 and polyagglutinating, pyocin types nonclassifiable and no inhibition. The isolates from the second patient showed the following: blood and rectum—serotype 11, pyocin type 10 (pharyngeal culture was negative). This patient was in ICU but became bacteremic while on a medical ward 3 weeks later.

P. aeruginosa colonization at the time of the first admission of the medical and surgical patients from the community to the study is associated with service. Table 4 presents the incidence (per 100 patients) of each clinical factor at the time of first admission from the community to the study, specific for service and for colonization status upon admission. The eight patients admitted to the ICU from the community are excluded. Colonization at time of admission is statistically ($P < 0.05$) associated with anemia, age, and previous surgery for malignancy of the GI tract. For both medical and surgical services, a significantly ($P < 0.05$) higher proportion of the colonized patients had anemia (70.0 versus 42.6% and 45.8 versus 27.7%, respectively). Among surgical patients colonized at time of admission, a significantly ($P < 0.05$) higher percent were age 65 or older (58.3 versus 22.7 to 30.0%) and a significantly ($P < 0.05$) higher percent had had previous surgery for malignancy of the GI tract (25.0 versus 0.0 to 6.4%). For the surgical service, the presence of previous surgery for malignancy of the GI tract had a strong positive association with three of the other clinical factors investigated: colostomy or ileostomy, malignancy of the GI tract, and older age ($P < 0.01$).

TABLE 4. Incidence of designated clinical factors per 100 patients, specific for colonization status on first admission to the study from the community (medical and surgical services)

Clinical factor	Incidence in patients:			
	Colonized		Not colonized	
	Medical (10)[a]	Surgical (24)	Medical (66)	Surgical (47)
Age ≥65 yr	30.0	58.3[b]	22.7	29.2
Hospital stay, ≥15 days	40.0	50.0	45.5	44.7
Previous hospitalization (within 3–6 mo)	50.0	58.3	43.9	36.2
Colostomy or ileostomy (previous or new)	0.0	16.7	1.5	8.5
Previous surgery				
Malignancy				
GI tract	0.0	25.0[b]	3.0	6.4
Other	10.0	4.2	0.0	10.6
No malignancy				
GI tract	0.0	12.5	18.2	19.1
Other	30.0	4.2	21.2	17.0
Malignancy				
GI tract	0.0	25.0	12.1	8.5
Other	10.0	8.3	7.6	27.7
Immunosuppressives	0.0	4.2	10.6	4.3
Anemia (hematocrit, <30%)[c]	70.0[d]	45.8[d]	42.6[d]	27.7[d]
Diabetes mellitus	10.0	8.3	12.1	6.4

[a] Total number of patients in subgroup.
[b] Statistically significantly higher for colonized patients on surgical service ($P < 0.05$).
[c] Presence of anemia unknown for five culture-negative patients admitted to medical service.
[d] Statistically significantly higher for colonized patients ($P < 0.05$).

For the patients who entered the study culture negative for *P. aeruginosa*, none of the clinical factors was significantly associated with an increased risk of colonization during the observed hospital stay. The clinical factor-specific risks of nosocomial colonization ranged from 0.0 (patients becoming colonized per 100 patient-days at risk of colonization) to 2.1. The risk of 0.0% was observed among patients who had had previous surgery for malignancy of the non-GI tract, and the

risk of 2.1% was observed in the group of patients who had had previous surgery for malignancy of the GI tract. In no case was the observed risk statistically significantly different from the corresponding risk among patients with the clinical factor absent.

Antibiotic therapy was administered to a statistically significantly ($P < 0.01$) higher proportion of ICU patients (89.7%) than medical (38.2%) and surgical (43.7%) patients. For those given antibiotics, the percentage of patients receiving beta-lactam and an aminoglycoside only was similar for the three services: 11.5 (ICU), 12.9 (surgery), and 20.7% (medicine). On the three services, the predominant (76.9 to 80.6%) method of administration was parenteral alone.

For those entering the study *P. aeruginosa* culture negative, the risk of nosocomial colonization was the same for patients receiving and not receiving antibiotics: 1.1 and 1.2 patients colonized per 100 patient-days at risk of colonization. Risk of nosocomial colonization was the same for those receiving drugs parenterally only (1.2%) and for those receiving drugs orally alone or orally and parenterally (1.1%). Among patients receiving beta-lactam and an aminoglycoside only, the observed risk of nosocomial colonization was 3.1% (patients colonized per 100 patient-days at risk); for patients receiving all other antibiotic therapies, the risk was 0.9%. This difference was not statistically significant ($P = 0.052$).

DISCUSSION

The observations of our prospective epidemiologic study done in a general hospital setting over a 5-month period and including a medical ward, surgical ward, and ICU indicate that 62 (33.3%) of 186 hospital admissions (180 patients) were colonized with *P. aeruginosa* in the GI tract. Of particular interest was our finding that the admission colonization rate was more than twice as common in the surgical patients as in the medical patients. Only 17% of the culture-negative patients (11% of total admissions) on the medical and surgical wards acquired *P. aeruginosa* in the hospital, and this colonization rate was similar for both services. When the various lengths of hospital stays among culture-negative admissions are taken into account, the cumulative percentage of acquired *P. aeruginosa* patients rose to an estimated 28% by the end of 8 weeks (Fig. 2). The analysis of the colonization rate in the ICU patients was performed separately because only 8 of 32 patients were admitted to the ICU directly (25.0% were colonized at the time of admission, and an additional 9% became colonized during hospitalization). Overall, 11% (20 of 186) of the patients admitted into the study acquired *P. aeruginosa* in the hospital. Earlier epidemiologic studies performed in oncology institutions indicated a higher colonization rate (50%) in hospitalized patients (6). Lower colonization rates were described in healthy adults (11.9%) by Sutter et al. (34), in hospitalized patients (19%) by Grogan (19), and in burn unit patients (20%) by Lowbury and Fox (25). These four studies included hospitalized patients studied in a single time period in contrast to our study which was a prospective study including all patients on admission to 3 different wards with a follow-up period of up to 8 weeks while in the hospital.

In contrast to the low *P. aeruginosa* acquisition rates reported in our study (11%), higher acquisition rates in earlier studies by Shooter et al. (31), reaching 17%, and by Bodey (6), as high as 28%, were observed. As clearly demonstrated in Fig. 1 and 2, no change in the *P. aeruginosa* carriage rate was detected after day 35 of hospitalization. This colonization remained constant at 44% in patients who entered culture positive, while for patients who entered culture negative the rate remained constant at 28%.

As described earlier by several investigators (13, 32), the rectum was the most commonly observed colonization site for *P. aeruginosa*. Our study indicated that oropharyngeal colonization alone was observed in only 3 of 62 colonized patients. Furthermore, only one of these three patients entered the hospital with *P. aeruginosa* in the oropharynx. In all cases in which colonization was observed in both rectal and oropharyngeal areas, colonization of the rectum occurred first. The oropharyngeal strains were of the same pyocin type and serotype in 70% of the patients who were culture positive in both sites.

The most common pyocin type and serotype of *P. aeruginosa* resembled those we described previously in blood culture isolates (8). Although 35.5% of the strains were nonclassifiable among the known 35 pyocin types, 81.8% of these strains had a constant pyocin inhibition pattern, indicating one predominant strain in this nonclassifiable group of *P. aeruginosa*. In contrast to an 8% incidence of polyagglutinable strains in blood culture isolates (8), 27.4% of the GI tract strains in this study were polyagglutinable.

All admission *P. aeruginosa* strains were susceptible to azlocillin, tobramycin, gentamicin, and moxalactam, while two hospital-acquired isolates were resistant to moxalactam on first culture. Weekly or discharge cultures or both indicated that the susceptibility of all strains remained unchanged except for three isolates: two became resistant to moxalactam, and one became resistant to tobramycin, gentamicin, and azlocillin. These strains demonstrated evidence of acquired resistance since their serotypes and pyocin types remained unchanged.

It has been noted that previous colonization of the GI tract may predispose patients with hematologic malignancies to become bacteremic (5, 13, 28, 29). None of our patients had hematologic neoplasias. However, 2 of the 62 patients colonized with *P. aeruginosa* in the GI tract developed bacteremia during the study period. Both of these patients were colonized in multiple sites. In one patient, the pyocin type and serotype of the blood culture were the same as for the rectal isolate, while in the other patient a different strain of *P. aeruginosa* from that detected in the rectal culture was isolated.

Because this was a prospective study, we included an analysis of 13 possible risk factors for patients who entered the hospital culture positive or who acquired *P. aeruginosa* while in the hospital. These data were also analyzed for the culture-negative patients. Colonization detected at the time of admission was associated with age, previous surgery for malignancy of the GI tract, and anemia ($P < 0.05$). Furthermore, for the surgical ward the previous history of surgery for GI tract neoplasm was strongly associated with colostomy or ileostomy, malignancy of the GI tract, and advanced age of the patient ($P < 0.01$). In contrast, for patients who entered the study culture negative, none of the analyzed 13 clinical factors was statistically significantly associated with an increased risk for colonization. Of special interest was our observation that the risk of nosocomial GI tract colonization with *P. aeruginosa* was the same for patients who received or did not receive antibiotics. Administration of adrenocorticosteroids, radiation therapy, presence of diabetes mellitus, length of hospital stay, or previous recent admission was unassociated with higher risks for colonization with *P. aeruginosa*.

In summary, this prospective epidemiologic study per-

formed in a general hospital setting for acquisition of *P. aeruginosa* in the GI tract indicates that about one-third of the patients were found to be colonized upon admission to the surgical ward and ICU and only 13% were colonized upon admission to the medical ward. The risk of becoming colonized with *P. aeruginosa* did not change after 35 days of hospitalization. Similarly, the possibility of losing *P. aeruginosa* from the colonized GI tract ceased after 35 days of hospitalization. Analysis of clinical and demographic data demonstrated that older age, history of surgery for neoplastic disease of the GI tract (especially the presence of colostomy or ileostomy), and anemia are statistically significantly associated with colonization of the GI tract with *P. aeruginosa*. In this study, we were unable to demonstrate that the administration of antibiotics is associated with colonization of the GI tract with *P. aeruginosa*.

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