

Prospective Survey of Fecal, Urinary Tract, and Environmental Colonization by *Providencia stuartii* in Two Geriatric Wards

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A prospective survey of fecal, urinary tract, and environmental colonization by *Providencia stuartii* in two wards was undertaken over a 5-month period. Eight of 53 male patients and 2 of 89 female patients were colonized with the endemic serotype O:63. Two patterns of colonization were found on the male ward. Two patients had persistent urinary tract colonization with no detectable fecal carriage. The other patients had fecal carriage, in some cases persistent, with intermittent urinary tract colonization. The ward environment was in general not contaminated. This study demonstrates that fecal colonization of patients by *P. stuartii* may be an important and previously underestimated nosocomial reservoir.

Providencia stuartii is an established nosocomial pathogen, commonly associated with urinary tract infection (11, 12). The bacterium is often resistant to many antibiotics, including gentamicin (10). Septicemia is not common and usually occurs in elderly or compromised patients (9), but since gentamicin is often used for blind treatment of septicemic episodes, deaths have been reported (11).

Reports of outbreaks have shown the ability of *P. stuartii*, once established in a hospital, to persist and spread among susceptible patients (9, 19), but the reservoir and mode of spread have often been obscure (11, 19). Clusters of cases may occur with heavily colonized urine as the source of infection (7), but at other times occurrence of overt infection may be sporadic and the endemic nature of a particular strain may go unrecognized. Only one recent outbreak has been reported in the United Kingdom (6), and this was the only one in which the investigation was supported with serotyping data (12). Fecal colonization has not been systematically examined. One investigation with negative findings (18) involved only the examination of six fecal specimens on a single day when only one patient in the ward had urinary tract infection, and in other investigations examination for gut carriage was intermittent (8, 9).

We report here a prospective investigation, for a 5-month period, of the occurrence of *P. stuartii* in the urine and feces of an elderly hospital population in wards in which cases of

urinary tract colonization had been detected previously by routine diagnostic specimens.

MATERIALS AND METHODS

Patients. Two geriatric wards in a general hospital in Bristol were surveyed. They were mostly occupied by acute medical patients plus a small number of patients requiring chronic care. Both wards were of open plan design, and there was a single utility room for each ward. Ward 1 was for female patients (19 beds) and ward 2 was for males (15 beds). The study was conducted from February to July 1980, and 53 male and 89 female patients were surveyed during this period. This number includes all patients already on the wards at the start of the study and all subsequent admissions during the study period.

Bacterial cultures. Urine specimens and rectal swabs (cotton wool swab; Medical Wire Co., Corsham, England) were collected weekly (by P.M.H.) from all patients. Any patient known to be colonized by *P. stuartii* from the previous week's screening was subjected to a personal and immediate environmental survey, comprising collection of swabs of fingers, buttocks, perineum, and clothes-locker surface. The environment of the dirty utility room (sluice room), including such articles as urine jugs, taps, commodes, and sluice (lavatory), was examined by surface swabbing, with a swab moistened in normal saline.

Media and culture techniques. All swabs were placed in 2 ml of 2.5% infusion broth (BBL Microbiology Systems, Cockeysville, Md.) and incubated for 18 h at 37°C. The broths were subcultured onto MacConkey agar (Oxoid Ltd., Basingstoke, England) containing 10 mg of gentamicin sulfate (GM medium) per liter and onto PR agar medium. PR agar was used as a selective

medium for *Proteus-Providencia* group and was prepared as follows. To 1 liter of deionized water the following were added: 12 g of agar (Difco Laboratories, Detroit, Mich.), 10 g of infusion broth base (dehydrated) (Becton, Dickinson and Co., Cockeysville, Md.), 10 g of lactose, and 75 mg of neutral red indicator. After autoclave sterilization and cooling to 50°C, 30 mg of clindamycin base and 100 mg of colistin sulfate were added as sterile powders. Plates were incubated for up to 40 h at 37°C in air.

Urine specimens were plated directly onto GM and PR medium with a 10- μ l disposable plastic loop (Nunc Plastics, Kamstrup, Denmark) and incubated for up to 40 h at 37°C.

Both PR medium and GM medium were shown to be non-inhibitory to growth of gentamicin-resistant *P. stuartii* (O:63) by viable counting of suspensions in saline.

All bacterial colonies appearing after 40 h of incubation at 37°C on GM medium were subcultured and fully identified. All gram-negative, oxidase-negative rods were identified initially by the API 20E system, and resistance to gentamicin was confirmed by disk testing (17). All phenylalanine deaminase-positive isolates were further biotyped by the method of Penner et al. (14). The biotyping was performed by examining these isolates for indole production from tryptophan, ability to grow on citrate, hydrolysis of urea, and decarboxylation of ornithine. The production of acid from mannitol, inositol, adonitol, trehalose, maltose, and xylose was also tested for by using the Minitex system. Bacterial colonies growing on PR medium after 20 h of incubation at 37°C were Gram stained, and oxidase-negative, gram-negative rods were subjected to a short series of biochemical tests: phenylalanine deaminase (liquid malonate phenylalanine medium), urea hydrolysis (Chrisensen urea agar medium), and hydrolysis of *o*-nitrophenyl- β -D-galactopyranoside (3). All phenylalanine deaminase-positive, urease-negative isolates were biotyped as before. In this way gentamicin-sensitive *P. stuartii* strains could be isolated provided that they were urease negative, as were the majority of strains in other studies.

Serotyping and biotyping. Serotyping of isolates was by the slide agglutination reaction with O antisera (14), and biotyping was done by methods described in the previous section.

Antibiotic sensitivity testing. Antibiotics were incorporated into Diagnostic Sensitivity Test agar (Oxoid), and approximately 10^4 colony-forming units of each isolate were inoculated by the multiple inocula-replicating method of Steers et al. (16). The minimum inhibitory concentration (MIC) was recorded as the minimum concentration of antibiotic that totally inhibited growth at 20 h.

RESULTS

Biotyping and serotyping. All isolates were of the same biotype: positive for phenylalanine deaminase, indole production, and growth on citrate, and negative in the urease and ornithine decarboxylase tests. Acid was produced from inositol and trehalose, but not from mannitol, adonitol, maltose, or xylose. Isolates therefore belonged to biogroup 5 (14).

All isolates from patients and environment on wards 1 and 2 during the entire period of the study were O serotype O:63.

Survey for *P. stuartii* in two hospital wards. The results of the colonization survey on ward 2 are shown in Fig. 1 and 2. On ward 2 (male patients), 45 patients during the survey were never found to be colonized with *P. stuartii*, but 8 patients were colonized. In addition, the dirty utility room floor, dressing cart tops, hand creams, and bedpan washer door handle were all cultured repeatedly with negative results, apart from the first two sites on single occasions only.

Only 2 patients on ward 1 (female patients) were colonized with *P. stuartii*; 87 were negative. One patient (no. 9), admitted at the beginning of week 1, had positive rectal swabs in weeks 1 and 2 of the study. The isolate taken one day after admission was sensitive to gentamicin (MIC, 2 mg/liter), but the isolate in week 2 was resistant (MIC, 64 mg/liter). Personal and environmental screening in week 2 yielded a positive perineal swab (MIC, 64 mg/liter). Urine specimens taken in weeks 1 and 2 were negative, and this suggested fecal carriage only. Environmental screening of ward 1 during week 2 resulted in one commode positive for *P. stuartii* (gentamicin MIC, 8 mg/liter); this was the only environmental isolate from ward 1 during the study. This patient died at the beginning of week 3 before being screened. The second female patient colonized (no. 10) had a positive urine on week 11 and a negative rectal swab on week 10 only. She died at the end of week 11.

Staff were not tested regularly for carriage, but the possibility of hand contamination during simple nursing procedures was demonstrated in one experiment. After washing her hands with chlorhexidine hand scrub (Hibiscrub; ICI Pharmaceuticals, Macclesfield, England) a nurse turned a colonized patient (no. 6, during week 9 of the investigation) in bed. Culture of washings of her hands with nutrient broth in a disposable plastic glove (1) revealed that her hands had become colonized by *P. stuartii* during the procedure.

Antibiotic sensitivity. The results of the plate incorporation MICs are shown in Table 1. Two isolates (rectal swab of patient no. 9 and commode on ward 1) were sensitive to gentamicin. All other isolates were resistant to gentamicin (MIC, ≥ 32 mg/liter).

DISCUSSION

Recent reviews of major episodes of nosocomial infection caused by *P. stuartii* have drawn attention to the sporadic nature of outbreaks against an unknown background of occurrence of *P. stuartii* (11, 12), and these reports and others (5, 8, 18) suggest an unidentified hospital

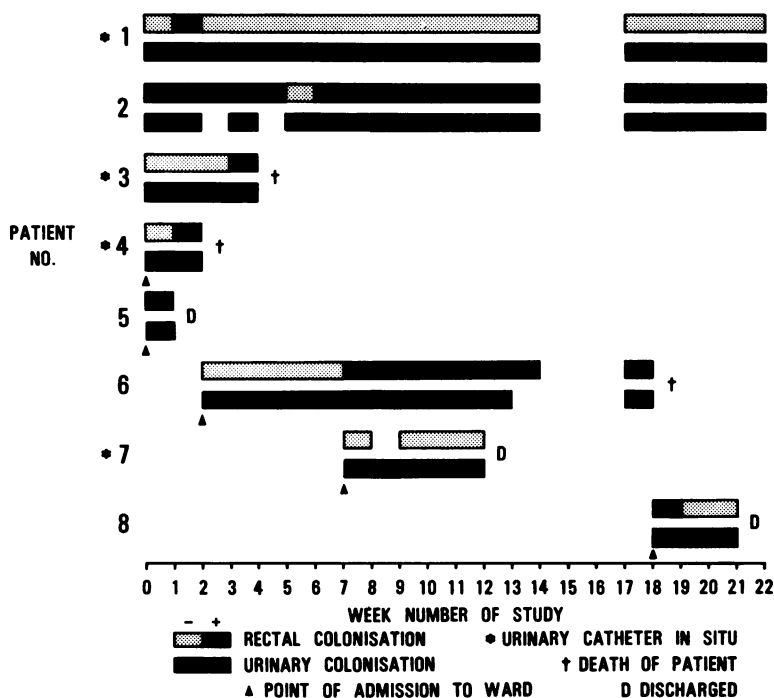


FIG. 1. Temporal relationship of rectal and urinary tract colonization with multiply resistant *P. stuartii* among eight male patients in ward 2.

environmental source. Fecal carriage of multiply resistant *Klebsiella* sp. (4) and *Proteus mirabilis* (2) is common and is an important nosocomial reservoir in outbreaks of cross-infection. *P. stuartii* has been reported to be occasionally isolated from feces (8, 9), but examination of feces in these studies was intermittent rather than part of a systematic survey. A prospective intensive study of fecal and urinary carriage of this organism in a closely observed ward population has not been previously reported.

In the present study, screening of the whole ward population at repeated intervals revealed fecal carriage in some patients. The use of nonselective enrichment before plating on selective media for rectal and environmental swabs may well have revealed low levels of fecal carriage. Numbers of *P. stuartii* as low as 50 colony-forming units per g of feces can be detected by this method. Sparse fecal carriage of this organism may explain the insidious nature of some outbreaks of infection with *P. stuartii* (12). It is possible that patients with fecal colonization subsequently develop urinary tract colonization and that the large numbers of organisms present in urine result in cross-infection and an apparently isolated outbreak.

O serotyping was used in this study because a method was required which would be independent of plasmid-mediated characteristics. Al-

though all isolates were of the same O serotype, O:63, two isolates from ward 1 in week 1 were sensitive to gentamicin. This event could represent the introduction of a nonhospital strain of a serotype known to be common in other studies (12). Alternatively, plasmid-mediated gentamicin resistance could have been acquired, but this is unlikely, as gentamicin resistance in *P. stuartii* is commonly chromosomally mediated (10).

P. stuartii of serotype O:63 seems to be commonly associated with nosocomial urinary tract infection. The one reported incident of cross-infection in the United Kingdom showed the cross-infecting strain to be O:63 (12) when serotyping was performed (6). This serotype was the most frequently isolated in Canadian hospitals (12). In an outbreak of cross-infection in a Toronto hospital the cross-infecting O:63 strain was probably introduced in the infected urine of a catheterized patient (13). However, serotype O:63 is not the only serotype that has been implicated in nosocomial urinary tract infections. Serotype O:55 was responsible for a major outbreak in Hamilton, Ontario, Canada (19).

Two patterns of colonization were seen on ward 2. Two patients (no. 1 and 7), who had long-term Foley catheters in situ, appeared to carry *P. stuartii* only in their urine. This pattern was similar to that described in an outbreak associated with urinary "condom catheters" (7),

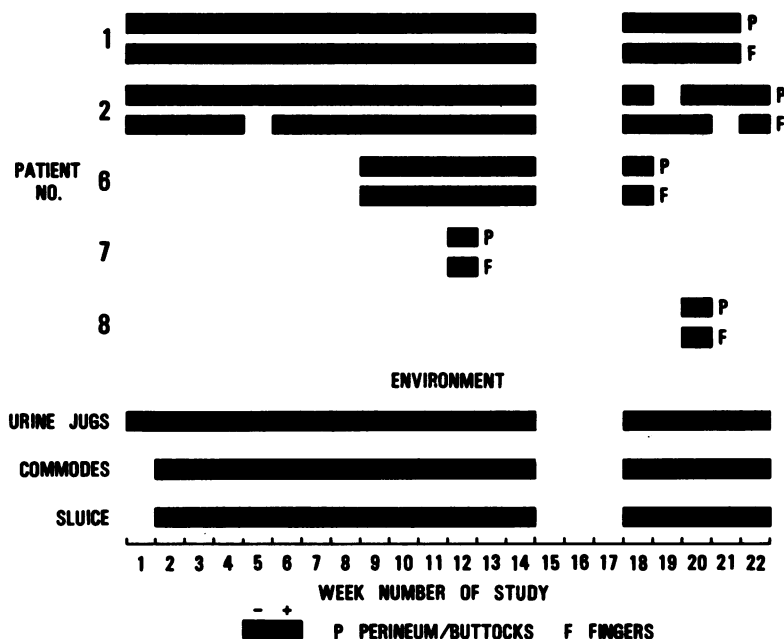


FIG. 2. Temporal relationship of skin contamination of three of the male patients shown in Fig. 1 and environmental isolation of multiply resistant *P. stuartii* in ward 2.

in which fecal carriage was not detected, and which was controlled by isolation of patients and careful disposal of urine.

The other pattern of colonization, seen in patients no. 2 and 6, was very similar to that described for multiply resistant *P. mirabilis* (2). Fecal carriage was persistent, and urinary carriage was intermittent. Some patients had fecal colonization only (no. 3, 4, 5, and 8). One patient (no. 6) had negative rectal swabs on admission and for the next 5 weeks. He then acquired persistent fecal carriage with intermittent urinary carriage. This pattern of colonization suggested that *P. stuartii* was acquired from a hospital source and that fecal colonization oc-

curred before urinary tract colonization. Patients no. 3 and 4 had negative rectal swabs for 1 and 3 weeks and then acquired *P. stuartii* in their feces only shortly before they died. The route of infection on the ward is likely to be the hands of medical and nursing staff. Handwashing with chlorhexidine-containing detergent was commonly practiced; because colonized patients were not identified, however, the staff did not always carry out this procedure when leaving the patient. We have demonstrated that a nurse's hands may become contaminated when she performs a very minor nursing procedure. This observation draws strong parallels with the mode of transmission demonstrated for *Klebsiella aerogenes* (1) and *Providencia rettgeri* (15).

Jugs were used for emptying urinary catheter bags. These collection jugs were found to be contaminated frequently and may have resulted in urinary tract colonization of catheterized patients such as no. 7. These jugs were not used for noncatheterized patients. The lack of contamination of the general ward environment suggests that colonized patients were the reservoir of infection.

At no time did patients exhibit any signs or symptoms of urinary tract infection, and no cases of bacteremia were noted. For this reason no patients were isolated or treated with appropriate antibiotics.

The pattern of antibiotic resistance was very similar to that described in a recent study (10). It

TABLE 1. Concentrations of antibiotics inhibiting 50% (MIC₅₀) and 90% (MIC₉₀) of 149 isolates of *P. stuartii*

Antimicrobial agent	Range of MICs (mg/liter)	MIC ₅₀ (mg/liter)	MIC ₉₀ (mg/liter)
Streptomycin	>256	>256	>256
Gentamicin	2-128	64	64
Kanamycin	2-16	8	16
Tetracycline	8-32	16	32
Chloramphenicol	8-16	16	16
Ampicillin	32-128	64	128
Carbenicillin	0.5-4	1	2
Trimethoprim	0.5-4	1	2
Sulfamethoxazole	>256	>256	>256
Cephaloridine	>128	>128	>128

has been suggested that maintenance of high levels of gentamicin resistance in *P. stuartii* is related to frequent use of that antibiotic (10). On ward 2, gentamicin had been used for 5 days on one occasion in one patient 10 months previously and at no time subsequently. Multiply resistant *P. stuartii* is therefore able to spread and to colonize patients in the absence of direct selective pressure from the use of aminoglycoside antibiotics. A moderate amount of ampicillin and co-trimoxazole was prescribed on the wards for urinary tract and respiratory infections, and these may well have helped maintain *P. stuartii* in the ward.

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