Letters to the Editor

Bacteriuria with *Escherichia coli* Resistant to Ciprofloxacin in Patients With Spinal-Cord Injury

To the Editor:

We have observed a growing number of isolates of ciproflaxicin-resistant *Escherichia coli* (CREC) at our rehabilitation hospital. In initial studies,[1-3] we observed that almost all (98%) of the isolates were obtained from the urine; 86% of the patients had spinal-cord injury, and 80% were outpatients. Because the outbreak was limited mainly to the spinal-cord–injury outpatients, a retrospective case-control study was conducted to identify risk factors for the spread of the CREC in outpatients with spinal-cord injuries from January to December 1993. Control subjects were spinal-cord–injury outpatients with ciprofloxacin-susceptible *E coli* bacteriuria who immediately followed each patient with CREC. The variables studied are shown in the Table. The only significant factors that we could identify were previous use of fluoroquinolones and the use of intermittent catheterization. The control group, who used more self-catheterization, may have had less direct contact with medical personnel to obtain urine samples, resulting in less chance of transmission of the resistant strain by the personnel. Transmission occurred equally in the two main clinics.

We were surprised at the lack of supportive evidence of spread of the CREC. At a minimum, we had expected to find that patients acquired CREC at an earlier visit to the clinic on a day attended by patients colonized or infected with the CREC. Only one patient with newly recognized CREC bacteriuria had attended a prior clinic on the same day as a colonized patient. We still assume that transfer was occurring between patients at the two main clinics, because that was their only important common ground. The CREC most likely spread via unrecognized breaks in techniques and unrecognized carriers of the resistant strains. Spread also may have occurred through contamination of wheelchairs, clothing, or the environment. On three occasions, cultures of floors, beds, wheelchairs (wheels, grips, brake handles), and hands of the patients colonized with CREC did not reveal any gram-negative bacilli, suggesting that the environment was not contaminated heavily. Others, however, have found gram-negative bacilli on wheelchairs.[4]

A possible explanation for our inability to observe transfer in the clinics may be the complexity of the regulation of the resistance genes for fluoroquinolones. One of the features of bacterial resistance to fluoroquinolones has been the ability to accumulate several mutations affecting both DNA gyrase and bacterial permeability.[5] Some mutants with low level of resistance may remain unde-

TABLE
CASE-CONTROL STUDY OF CIPROFLOXACIN-RESISTANT *ESCHERICHIA COLI* BACTERIURIA AMONG PATIENTS WITH SPINAL-CORD INJURY

Clinical Feature	Cases (N=30)	Controls (N=31)	P*
Age (mean)	35	33	.5
External catheter	12	13	1.0
Intermittent catheter	10	21	.04
Indwelling urethral catheter	4	1	.3
Urinary diversion (Koch pouch)	0	2	
Clinic			
Urology	18	20	.5
Walk-in	9	10	
Other	3	1	
Prior clinic visit			
Urology	16	15	.6
Walk-in	7	10	
Other	6	4	
Duration of spinal-cord injury (y)	7	6	.5
Previous manipulation (within 3 mo)	5	8	.4
Prior urinary tract infection (within 6 mo)	18	18	.8
Antibiotic use (within 6 mo)	18/23†	21/29†	.8
Regimens of antibiotics (some patients received more than one antibiotic)			
Fluoroquinolone	18	13	.004
Trimethoprim/oxazole sulfameth	4	10	.2
Macrodantin	6	3	.3
Ampicillin	3	8	.2
Aminoglycoside	3	0	.09
Other	7	4	.3

* Student's *t* test and Fisher's Exact Test (two-tailed) were used where appropriate.
† Number of patients in whom the information was available.

tectable by routine testing in the clinical laboratory.[5] Our speculation is that such strains may spread among patients and become recognized only when the patients receive fluoroquinolones, the most significant factor associated with CREC colonization. That we may have had such strains in our patients was suggested from in vitro studies of these CREC, in which a wide range of minimum inhibitory concentrations to ciprofloxacin was observed.[3] If low-level resistance occurred and was not detected by the clinical laboratory, these patients could have been colonized for longer periods than we had thought. It would appear that the epidemiology of ciprofloxacin resistance in *E coli* is complex, and further studies focusing on the nature of the resistance of the *E coli,* specific sites of acquisition, and colonization might be useful to determine exact reservoirs and mechanisms of spread of resistant strains.

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Patient Versus Healthcare Worker Risks in Needleless Infusion Systems

To the Editor:

In the August 1997 issue of *Infection Control and Hospital Epidemiology,* Voss, Verweij, L'Ecuyer, and Fraser[1] posed vital questions: Are needleless intravenous (IV) systems safe for patients? Needless? Efficient? Cost-effective?

At the Seventh Annual Meeting of the Society for Healthcare Epidemiology of America, McDonald et al[2] reported a comparison of central venous catheter-associated bloodstream infections (BSI) in patients in a hospital where the Baxter InterLink (Baxter Health Care Corp, Deerfield, IL) needleless IV system was changed to an intravenous access (IVAC; IVAC Medical Systems, San Diego, CA) needleless IV system. They found a threefold increase of BSI in patients infused via the IVAC system. In 1996, L'Ecuyer et al[3] reported that the use of needleless IV systems reduced, but did not entirely eliminate, accidental needlesticks in healthcare workers (HCWs). In 1995, Danzig et al[4] compared prior use of standard infusion systems with use of Baxter's InterLink needleless IV system in home healthcare settings wherein total parenteral nutrition was indicated. She reported a 10-fold increase in BSI in patients infused via the Baxter needleless IV system. She obtained cultures of bacteria from the infusion side of the slit latex infusion port cap and theorized that the slit in the cap provides a recess, albeit small, away from the mainstream wherein bacteria might proliferate between port injections via a blunt cannula. Cogent to these questions and reports, I observed the following:

1. The IVAC SmartSite (B. Braun Medical Inc, Bethlehem, PA) needleless IV system infuses ports via a blunt cannula or via the nozzle on a Luer-Lok syringe inserted into a recessed space containing a collapsing slit silicone port cap. The slit cap dribbles infusion fluid back into the recessed space each time the blunt cannula or nozzle is withdrawn from an infusion port more than 30 cm below the water level in the infusion source. Infusion fluid squirts through in a stream whenever more than 100 cm of hydrostatic pressure is exerted in the infusion system when the SmartSite cap is not screwed on. Fluid remains in the capped recess until the next blunt cannula or syringe nozzle is inserted.

2. The Braun SAFSITE-Y (B. Braun Medical Inc) needleless IV system depends on a line valve that opens with insertion of a standard syringe nozzle or a Tubex Blunt Pointe (Wyeth Laboratories Inc, Philadelphia PA) into a recess in the open side of an infusion access port. During the withdrawal of the nozzle, while the line valve is still partly open and hydrostatic pressure in the infusion system exceeds that in syringe or the cartridge used for injecting soluble fluid, fluid from the infusion leaks back into the recess and remains there or evaporates. With the next injection, some residual recessed fluid may enter the line.

3. Use of the CLAVE (McGaw Inc, Irvine, CA) system depends on a tapered needle with a compressible silicone cap, both located in a recess on the open side. The CLAVE has external threads for attachment of a Luer-Lok syringe. When the filled syringe is advanced and locked onto the CLAVE, the nozzle progressively presses against the silicone cap, which com-