most rooms, and cleaning procedures strictly revised by an infection control nurse who performs weekly environmental cultures, focusing on the above-mentioned four groups of items. Furthermore, information on contamination rates and outbreak evolution are given regularly to ICU personnel. The evolution of the endemic over the next months will determine whether further drastic measures, such as a global structural redesign of our ICUs, must be carried out to control the outbreak.

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Is the European Interhospital Clonal Spread of Serotype O12 *Pseudomonas aeruginosa* Related to the Patients' Prolonged Carriage Duration?

To the Editor:

It has now been over 10 years since, officially, every French hospital has established its own Nosocomial Infection Control Committee (NICC), as well as its own Antiinfectious Chemotherapy Control Committee (AICCC). One of the main objectives of these mandatory creations was to limit the spread of infections with multidrug-resistant (MDR) bacteria. Quite simple and basic measures can help in reaching this objective: hand washing by healthcare providers before and after all patient contacts; screening, signaling, and isolating of MDR bacteria carriers, regardless of their symptoms; rational use of antibiotics; adequate sterilization of materials; etc.[1] Over this last decade, the diffusion in our hospital of an MDR clone of serotype O12 *Pseudomonas aeruginosa* (*P12*)[2] might illustrate the difficulties that our NICC and AICCC are meeting in the application of these basic measures.[3]

In the Table, I indicate some ecological characteristics pertaining to the 1,046 *P aeruginosa* isolates that have been obtained from clinical specimens in our hospital over the last 7 years (June 1991-October 1998), as recorded in our computerized epidemiological expert system (SIR, I2A, Montpellier, France). The particular ecological characteristics of *P12* in our hospital (Table) must be interpreted in light of the following facts: (1) Almost all of the *P12* isolated in our hospital (and in some other hospitals in our neighborhood)[2] are indistinguishable from the MDR European clone of *P12* that seems to have spread throughout many different,[2] but not all,[4] European hospitals. (2) In our hospital, our NICC and AICCC have not, so far, succeeded in convincing all of the wards (particularly, but not only, the long-stay wards) that the aforementioned basic measures must be systematically applied[3] (in our opinion, it cannot be excluded that this reluctance might be a consequence of a "feudal system" possibly found in certain French medical institutions),[5] and a similar situation is likely to be the case in some other hospitals in our area.[2]

Because the epidemiological mechanisms possibly responsible for the clonal European interhospital spread of MDR *P12* are not clearly understood at this time,[2,4] we advise colleagues from affected hospitals to publish their own ecological data. Such reports (easily done with the help of SIR or any computerized expert system of this sort)[3] might confirm (or not) our own ecological data, help in the designing of future intra- and interhospital epidemiological studies of *P12* infections in hospitals where *P12* has clonally spread, and thus perhaps eventually confirm (as has been suggested by others)[2] that infected or colonized patients might be the primary reservoirs of the multiresistant European clone of *P12.*

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TABLE

PSEUDOMONAS AERUGINOSA ISOLATES BY SEROTYPE: NUMBER OF LONG-STAY WARD AND SUPERFICIAL PUS ISOLATES, NUMBER OF PATIENTS, AND OVERALL CARRIAGE DURATION (CALCULATED ON THE WHOLE POPULATION OF PATIENTS)

	Isolates	LSW Isolates	SP Isolates	Patients	Carriage Duration
O12	152/1,046 (15%)	34/147 (23%)	36/146 (25%)	91/815 (11%)	68±254 d
Non-O12	894/1,046 (85%)	113/147 (77%)	110/146 (75%)	724/815 (89%)	18±119 d
P		<.001	<.001	<.0001	<.005

Abbreviations: LSW, long-stay ward; SP, superficial pus.

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Vancomycin-Resistant Enterococci in Hemodialysis Patients Is Related to Intravenous Vancomycin Use

To the Editor:

In many hospitals worldwide, there has been an increase in vancomycin-resistant enterococci (VRE) colonization and infection and in the use of intravenous vancomycin. To study the relation of parenteral vancomycin use to VRE colonization and infection, we prospectively examined our hemodialysis (HD) patient population, because vancomycin is frequently used for prophylaxis or treatment of staphylococcal infections in HD patients.[1-5]

The target population of the surveillance screening included all clinically stable HD patients dialyzed during six daytime shifts at the outpatient dialysis unit. Patients screened had been undergoing routine HD for a minimum of 3 months. Patients were excluded from analysis if their age was <18 years, if there had been a hospital admission in the prior month, if they did not consent to be studied, or if there was a known enterococcal infection in the previous year.

Rayon-tipped rectal swabs were obtained from 111 consenting HD patients immediately prior to dialysis treatment and were transported promptly to the microbiology laboratory in BBL culturette transport media (BBL Microbiology Systems, Cockeysville, MD). Specimens were planted promptly onto bile-esculin agar (BBL) and incubated at 35°C for up to 48 hours before being discarded as negative. Colonies developing a black to brown color were identified as enterococci, based on the pyrrolidonyl arylamidase disk test (PML Microbiologicals, Tualatin, OR) and tolerance to 6.5% NaCl (BBL). Enterococcal isolates were tested for susceptibility to vancomycin by the standardized disk-diffusion procedure. A suspension of organisms equivalent to a McFarland 0.5 barium sulfate turbidity standard was prepared in trypticase soy broth (BBL) and swabbed onto the surface of a Mueller-Hinton agar plate (BBL). After the application of antibiotic disks, the plates were incubated at 35°C in ambient air for 24 hours. Zone size interpretations suggested by the National Committee for Clinical Laboratory Standards (December 1993) were used to determine the susceptibility of isolates to vancomycin and teicoplanin.

Patients found to be VRE-positive at initial screening did not receive any specific treatment. Charts were reviewed for demographic information, cause of end-stage renal disease, number of hospital days, use of intravenous or oral vancomycin, and intensive-care unit (ICU) hospitalizations in the previous year. The initial cohort was followed for 1 year, during which all microbiological isolates and any evidence of relevant infection were evaluated. An investigator summarized each patient event during the year with regard to the culture results and clinical disease episodes. Isolation of VRE from a normally sterile site, in association with clinical symptoms and signs, was considered to represent clinically relevant infections.

Statistical analysis used Student's unpaired *t* test for continuous variables and Fisher's Exact Test for discrete variables. All results are reported as mean ± standard deviation. A P of <.05 was considered significant.

A total of 111 patients (63 male) with a mean age of 59.9±11.3 (range, 20-85) years had VRE. The mean duration of dialysis treatment was 23.2±10.7 months.

Enterococcus was isolated from 91 (82%) of 111 patients. Ten patients (9%) had isolates that were resistant to vancomycin; 3 of these 10 isolates also were resistant to teicoplanin. Various factors were analyzed as possible predictors of VRE colonization (Table). The only statistically significant difference between VRE-positive and VRE-negative patients was in the volume of intravenous vancomycin exposure in the year prior to study (4.4±5.9 g compared to 1.4±2.5 g, respectively; P=.003). Of note, risk factors for VRE colonization found in studies of nonuremic populations (number of hospital days, number of ICU days, and use of multiple antibiotics) were not found to be predictors in this hemodialysis population.

In the year of follow-up, clinically relevant VRE infection developed in 3 (2.7%) of the 111 patients. During the year, there were 24 deaths, 6 patients received transplants, and 3 had incomplete follow-up. In total, there were 1,144 patient-months observed, yielding an incidence rate of 0.03 cases per patient-month. VRE infections included a sacral osteomyelitis in 1 patient, bacteremia in a second, and a peritoneal dialysis-associated peritonitis in a third. Of these 3 patients, 2 initially were VRE-negative and 1 was VRE-positive. Thus, 2 (1.9%) of the 101 patients who were VRE-negative subsequently developed a VRE infection, compared to 1 (10%) of 10 patients initially VRE-positive (not significant).

In summary, we found VRE rectal carriage in 9% of stable HD patients, with prior intravenous van-

TABLE
RECTAL CARRIAGE OF ENTEROCCI IN HEMODIALYSIS PATIENTS

	VRE	No VRE	P
Subjects	10	101	—
Age (y)	61.7±12.2	59.4±11.2	NS
Male:female	3:7	60:41	NS
Days hospitalized in the past year	15.8±25.9	15.6±22.2	NS
IV vancomycin in the past year (g)	4.4±5.9	1.4±2.5	.003
Total antibiotic doses in the past year (no. of doses)	9.1±8.3	8.9±8.0	NS
ICU hospital days in the past year	1.9±7.5	1.1±2.4	NS
Vascular catheter-days in the past year	4.8±5.3	4.1±5.5	NS

Abbreviations: ICU, intensive-care unit; IV, intravenous; NS, not significant; VRE, vancomycin-resistant enterococci.