Ecology of *Pseudomonas aeruginosa* in the intensive care unit and the evolving role of water outlets as a reservoir of the organism

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In spite of the significant changes in the spectrum of organisms causing intensive care unit (ICU)-associated infections, *Pseudomonas aeruginosa* has held a nearly unchanged position in the rank order of pathogens causing ICU-related infections during the last 4 decades. Horizontal transmissions between patients have long been considered the most frequent source of *P aeruginosa* colonizations/infections. The application of molecular typing methods made it possible, during the last ~7 years, to identify ICU tap water as a significant source of exogenous *P aeruginosa* isolates. A review of prospective studies published between 1998 and 2005 showed that between 9.7% and 68.1% of randomly taken tap water samples on different types of ICUs were positive for *P aeruginosa*, and between 14.2% and 50% of infection/colonization episodes in patients were due to genotypes found in ICU water. Faucets are easily accessable for preventive measures, and the installation of single-use filters on ICU water outlets appears to be an effective concept to reduce water-to-patient transmissions of this important nosocomial pathogen. (Am J Infect Control 2005;33:S41-9.)

Hospital-acquired infections constitute a major fraction of the adverse events complicating hospital treatment.[1] Patients treated in intensive care units (ICUs) are at increased risk to acquire such infections because the invasive devices often used in these patients create ports of entry for opportunistic bacteria and fungi. Evidence-based prevention strategies targeting specific pathogens should be based on a thorough knowledge of their epidemiology, reservoirs in the ICU, and modes of transmission. This article describes the current status of research concerning the ecology and reservoirs of *Pseudomonas aeruginosa*, one of the most important gram-negative pathogens causing infections in ICUs.

THE CHANGING MICROBIAL SPECTRUM OF INFECTIONS IN INTENSIVE CARE

The last 2 decades have witnessed significant changes in the spectrum of microorganisms causing nosocomial infections. Gram-negative enterobacteria, which in the 1970s and 1980s accounted for 30% to 50% of all disease-associated isolates in ICUs,[2,3] have been to a large extent replaced by gram-positive microorganisms such as staphylococci, enterocci, and corynebacteria.[4,5] The relative decrease of infections because of enterobacteria has been achieved by the successful implementation, in most ICUs, of clinical prevention strategies aimed to interrupt the transmission of these organisms from their intestinal reservoir to potential sites of infection. Techniques such as early enteral feeding,[6] use of closed suctioning systems for aspiration of tracheal secretions during mechanical ventilation,[7] and the concept of semiupright positioning of ventilated patients[8] have contributed to create functional or physical barriers between gastrointestinal flora and the respiratory tract. Currently, intensive research is ongoing to develop similarly effective prevention strategies for the gram-positive organisms and fungi that have filled the gap left by the enterobacteriaceae.[9,10]

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0196-6553/$30.00

doi:10.1016/j.ajic.2005.03.006

ROLE OF *PSEUDOMONAS AERUGINOSA*

In spite of these significant changes, *P aeruginosa* has held a nearly unchanged position in the rank order of pathogens causing ICU-related infections for more than 4 decades. In the newest US National Nosocomial Infections Surveillance (NNIS) system report, *P aeruginosa* continues to represent the third most frequent organism associated with wound or pulmonary infections, the

fourth most frequent organism causing urinary tract infection, and the fifth most frequent organism isolated from blood cultures in cases of septicemia (Fig 1).[11,12] Studies performed in individual ICUs revealed a significant role of *P aeruginosa* as a cause of ventilator-associated pneumonia (VAP).[13,14] In primary septicemia, US and European data show a relatively constant proportion of 4% of cases being caused by *P aeruginosa*.[15,16] Both pulmonary and bloodstream infections caused by the microorganism are associated with significant morbidity and with mortality rates of up to 40% to 50%.[17,18]

ANTIBIOTIC RESISTANCE OF *P AERUGINOSA* AND TREATMENT COSTS

Of particular concern is the increasing antibiotic resistance of *P aeruginosa* isolated from ICUs. NNIS data show a rise of resistance rates against commonly used antibiotics such as imipenem, ciprofloxacin, and ceftazidime by 15%, 9%, and 20%, respectively, between the periods 1998-2002 and 2003.[11] Data from a single center in Denver, Colorado, showed a dramatic increase of resistance by more than 30% for fluoroquinolones, tobramycin, and imipenem.[19] Multiresistance, defined as resistance to >3 drugs, reached a percentage of 32% in 2002.[19] Antibiotic resistance in *P aeruginosa* does not only predict a poor clinical outcome but represents a significant economic burden for the health system. An economic analysis by Carmeli et al[20] demonstrated that development of resistance during treatment of *P aeruginosa* infection was associated with increases in treatment duration, mortality, and costs. Specifically, the authors calculated a mean increase of 5.7 days in length of hospital stay and a 2.9-fold increase of mortality attributable to development of resistance. Total hospital charges increased by $11,981 in patients in whom resistance developed, compared with patients carrying sensitive strains.[20]

EPIDEMIOLOGIC TYPING OF *P AERUGINOSA*

Given the fact that *P aeruginosa* was discovered more than 100 years ago and that a multitude of scientific studies have dealt with the clinical manifestations, antibiotic resistance, and virulence factors of the organism, it is astonishing that relatively little insight into its reservoirs and transmission pathways has been gained. In contrast to gram-negative enterobacteria, no broadly applicable concept for the prevention of *P aeruginosa* infections has yet been developed.

One of the reasons for this apparent deficit lies in the inconsistency of epidemiologic data gained with conventional typing methods. When using phenotypic techniques such as phage typing, pyocin typing, and O-antigen serotyping, investigators were often unable to establish relationships between environmental and patient isolates.[21-23] It was therefore postulated that environmental *P aeruginosa* strains, although present in many hospital locations, were a separate population of organisms that was, in general, not linked to strains causing clinical disease.[24,25]

The advent of molecular typing techniques stimulated novel attempts to elucidate the ecology of *P aeruginosa* in the ICU setting. Studies comparing the classical typing methods with genotyping confirmed that O-antigen serology,[26-30] pyocin typing,[31] phage typing,[30,31] and reverse phage typing[31] were, in fact, of low discriminatory power and yielded variable results. The reproducibility was as low as 50% to 60% in some studies.[30,31] Today, it is accepted that the expression of O-antigens (which forms the basis for the serologic typing schemes) is apparently influenced by environmental factors modifying lipopolysaccharide production such as exposure to antibiotics or availability of nutrients.[32] Although the exact basis for the variability of other phenotypic traits is not known, similar mechanisms may apply.[22] Thus, only genotyping appears to be a suitable method to study relationships between strains. Methods that have been used successfully include pulsed-field gel electrophoresis (PFGE),[33-36] amplified fragment-length polymorphism (AFLP) analysis,[37] random amplified polymorphic DNA polymerase chain reaction (RAPD-PCR),[27,38] and exotoxin A[26] or pilin gene[39] probing of enzyme-digested DNA fragments.

RESERVOIRS AND MODES OF TRANSMISSION

Using molecular methods, horizontal strain transmissions were proven to be one of the most frequent causes of acquisition of *P aeruginosa* in the ICU. A series of carefully performed studies examining the epidemiology of *P aeruginosa* came from a group of researchers from The Netherlands. In their first study, Bergmans et al described the sequence of colonization in 10 ICU patients later diagnosed with VAP.[40] Sequential samples taken from the oropharynx, trachea, stomach, and rectum were genotyped by PFGE. In 9 of these 10 patients, the clone causing infection was first identified in the oropharynx. By contrast, rectal isolates obtained before the occurrence of VAP were clonally related to subsequent respiratory isolates in only 2 instances.[40] These observations indicated that oropharyngeal colonization was the first step in *P aeruginosa* infections of ICU patients and that such colonization did in most cases not result from endogenous infection. Because, in 5 of 10 patients, strains causing VAP were clonally related to strains from

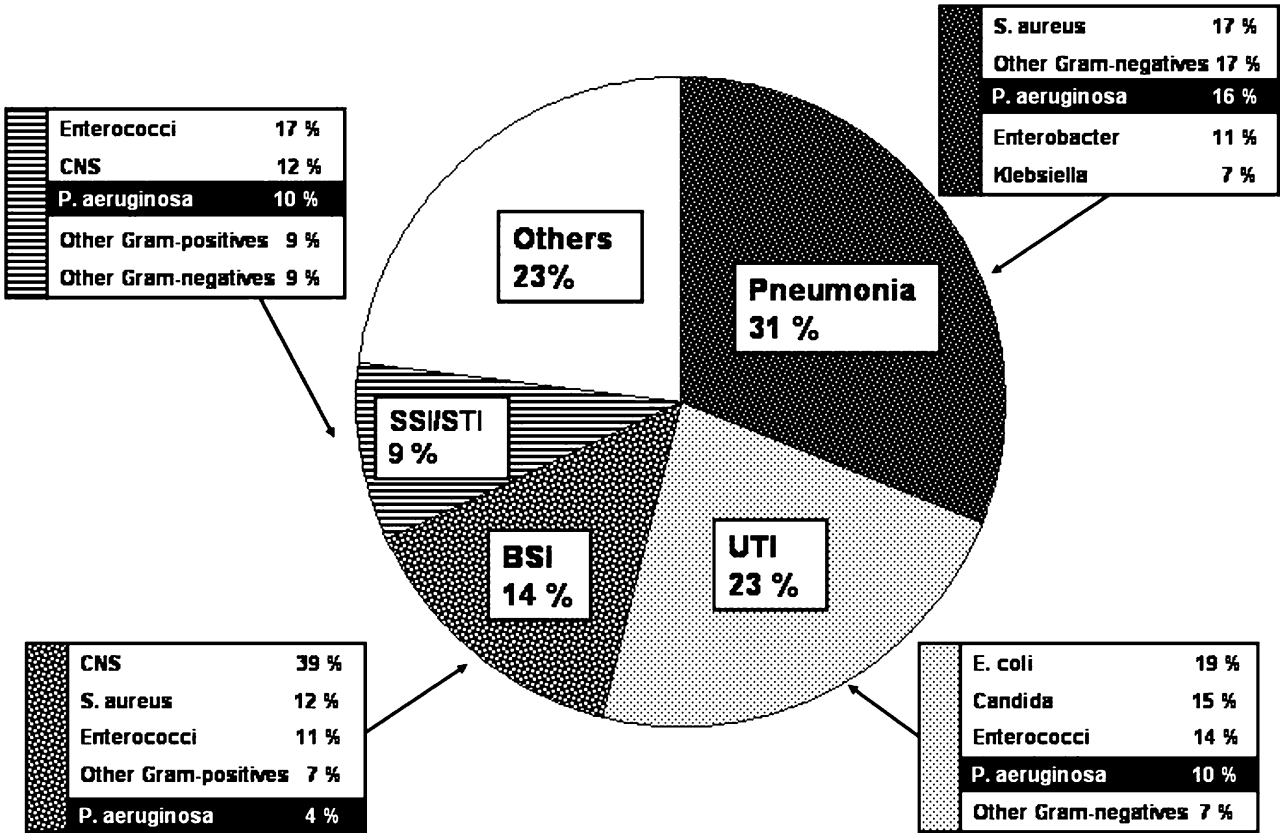

Fig 1. Relative role of *P aeruginosa* according to NNIS data. The organism ranks among the 5 top pathogens causing infections at various sites. UTI, urinary tract infections; BSI, bloodstream infections; SSI/STI, surgical site infections/soft tissue infections; CNS, coagulase-negative staphylococci. Courtesy of Dr. G.A. Ortolano, Pall Corporation, and adapted from Richards et al.[12]

neighboring patients, the authors concluded that horizontal strain transmissions were a significant mode of acquisition of oropharyngeal and respiratory strains. Although hospital personnel were not examined in this study, the authors speculated that caregivers manipulating gastric tubes or respiratory equipment might have been a vector of these strains.[40]

The significant role of cross transmissions, novel acquisitions from unknown sources, and imports of *P aeruginosa* into the ICU was confirmed in subsequent studies of these authors, whereas endogenous infections from the intestinal tract appeared to play a minor role.[41,42] These findings were in accordance with those of a study performed by Talon et al, who examined 17 patients in a medical ICU in France and found that only 2 *P aeruginosa*–infected patients had prior rectal colonization, with only 1 rectal isolate being clonally related to the disease-associated respiratory isolate.[36]

A similar study performed by Bertrand et al[43] in 4 French ICUs included 473 patients harboring *P aeruginosa*. In their study, 26% of the patients carried the microorganism on admission, whereas 74% became positive during hospitalization. Genotyping of 208 nonreplicate isolates revealed 101 major DNA patterns, and approximately 50% of *P aeruginosa* strains associated with carriage or infection were acquired via cross transmission.[43] Although cross transmissions are due to breaks in appropriate hygienic techniques, which are independent of patient status, the susceptibility of potential ''receptor'' patients to colonization by *P aeruginosa* and other nosocomial pathogens may be enhanced by broad antibiotic coverage. A recent study revealed a significantly elevated risk of infection not only by antibiotic-resistant but also sensitive *P aeruginosa* strains for patients exposed to broad-spectrum antimicrobials or vancomycin.[44] It may be speculated that elimination of competing flora facilitates the attachment of *P aeruginosa* to oral and respiratory epithelia. The implementation of prudent concepts of antibiotic use may be followed by a significant reduction of such infections on both ICUs and peripheral wards.[45]

In summary, these studies showed that approximately one fifth to one third of *P aeruginosa* strains

detected by screening cultures in the ICU were present on admission. The remaining fraction was apparently acquired newly in the ICU, and cross transmissions were identified in between 8% and 50% of these newly acquired colonizations or infections. Hospital staff was speculated to be a vector in these cases, and, in fact, several other authors were able to culture *P aeruginosa* from the hands of hospital personnel.[46,47] However, in a large fraction of approximately 30% to 60% of the cases, the mode of acquisiton of *P aeruginosa* remained unexplained in the above cited studies.

OCCURRENCE OF *P AERUGINOSA* IN MOIST HABITATS

P aeruginosa is known to thrive well in moist and humid habitats in which the organism can multiply to large numbers, even in the presence of minimal amounts of nutrients. The organism is also a common constituent of polymicrobial biofilms in plumbing systems and drain pipes. In the era of classical typing methods, numerous attempts have been made to identify relationships between disease-associated and waterborne strains.[21,23-25] However, both because of the variability of classical typing methods and the failure to perform longitudinal studies on water outlets, the relationship between these 2 populations of strains remained largely obscure. Nevertheless, in a few instances, the role of tap water as a source of disease-causing strains was established (eg, in the studies by Grieble et al[48] and Kolmos et al,[49] who evaluated the environmental sources of pulmonary and burn wound infections, respectively). Also, Martino et al were able to show that an outbreak of nosocomial septicemia in a hospital for hematologic malignancies was apparently related to a waterborne strain of the same serotype. These authors were also the first to show that measures to reduce the contamination of water taps (faucets) resulted in a significant decrease of clinical infections.[50]

MOLECULAR TYPING DATA POINTING TO TAP WATER AS A SOURCE OF *P AERUGINOSA*

Studies using molecular typing techniques and comparing water and patient isolates were first initiated in the early 1990s. Tredget et al[39] studied an outbreak of wound, respiratory, and bloodstream infections causing significant morbidity in a burn unit in Canada. As demonstrated by pilin gene typing, the strains infecting 59 patients were found to belong to a single genotye that was also detected in hydrotherapy tanks used for immersion of patients. After cessation of hydrotherapy, the strain was virtually eliminated, and the rate of *P aeruginosa* infections of burn wounds fell significantly.[39]

In a similar study initiated after the occurrence of several cases of severe *P aeruginosa* infections in newborns, Grundmann et al made several important observations regarding the colonization of water systems.[26] First, these authors showed that water taps on a neonatal ICU were often colonized with *P aeruginosa* over prolonged periods, each harboring an individual clone over many weeks. This colonization was apparently unrelated to the water from the supplying pipes because samples taken from the mains proved to be free of *P aeruginosa* on various occasions. Rather, the organisms appeared to reside deeply in the bushings and niches of peripheral taps and mixing valves. Clinical *P aeruginosa* strains cultured from 3 infected newborns were clonally related to strains isolated from water taps in their immediate neighborhoods.[26]

Peripheral colonization of water taps was also observed by Bukholm et al[51] during an outbreak of *P aeruginosa* infections on a mixed surgical and medical ICU. In their study, a multidrug-resistant clone infected a total of 19 patients. The clone was found repeatedly in washbasins as well as in and on water taps of the patient's room. Although other prevention measures were unsuccessful, regular thermal disinfection of taps and propagation of the use of sterile water for patient care activities finally stopped the outbreak.[51]

Our own work focussed on the role of water taps in surgical and medical ICUs of university and teaching hospitals. Infections because of *P aeruginosa* occurred endemically on these wards without apparent clustering. In a first pilot study, we examined 5 patient-related tap water outlets of a 16-bed surgical ICU over a period of 7 months.[27] Forty-nine of 72 cold water samples (68.1%) taken at 2-week intervals were positive for *P aeruginosa*, and all taps harbored an individual clone over prolonged time periods. Five of 17 patients (29.4%) acquiring *P aeruginosa* during the study period were infected with strains originating from a nearby water site.[27] We then expanded the study on this ward and included more patients and water samples during another 9-month observation period. Also, we followed the patients when transferred from the ICU to peripheral wards in the department of surgery.[38] In this study, 150 of 259 tap water samples (58%) taken at 2-week intervals were positive for *P aeruginosa*, and 45 episodes of clinical *P aeruginosa* infection occurred. Overall, a *P aeruginosa* strain clonally related to a previously isolated tap water strain was the cause of the infection in 13 of 31 ICU patients (42%) and in 5 of 14 (36%) patients on peripheral surgical wards. Conversely, 5 patients (11%) apparently contaminated the taps of their rooms because their individual isolates were cultured later from the water site. Thus, strain transfers occurred in both directions, but transfers

Table 1. Studies comparing genotypes of endemic *P aeruginosa* (PA) strains isolated from patients and tap water outlets in ICUs and peripheral wards

Authors, year (reference)	Study period	Setting	Ward(s)	Genotyping method	No. of positive tap water samples/no. tested (%)	No. of patients harboring a clone previously isolated from water taps/total no. of patients harboring PA	Percentage
Ferroni et al, 1998*(28)	1994	Pediatric hospital, Paris, France	Pediatric surgical ward	PFGE	21/118 (18.0)	3/14	21.4
Berthelot et al, 2001†(33)	1995-1996	University Hospital, St. Etienne, France	2 Mixed ICUs	AP-PCR, PFGE	34/n.i. (ICU 1) 34/n.i. (ICU 2)	3/12 (ICU 1) 2/14 (ICU 2)	25 14.2
Trautmann et al, 2000 (27)	1996-1997	University Hospital, Ulm, Germany	16-Bed surgical ICU	RAPD-PCR	49/72 (68.1)	5/17	29.4
Reuter et al, 2002 (38)	1998-1999	University Hospital, Ulm, Germany	16-Bed surgical ICU, peripheral surgical wards	RAPD-PCR	150/259 (58.0)	13/31 (ICU) 5/14 (peripheral wards)	42 35.7
Vallés et al, 2004 (34)	1996-1999	Teaching hospital, Barcelona, Spain	16-Bed mixed ICU	PFGE	93/149 (62.4)	3/8‡ 13/31§	37.5 42
Blanc et al, 2004#(35)	1998	University Hospital, Lausanne, Switzerland	5 ICUs of different specialities	PFGE	21/216 (9.7)#	36/132	27.3
Trautmann et al, 2005¶	2001	University Hospital, Ulm, Germany	12-Bed medical ICU	RAPD-PCR	60/143 (42.0)	8/16	50

n.i., not indicated.
*Smoldering outbreak of UTI infections (2.8% of infants positive for PA). Water sites were sampled during the outbreak.
†In this study, water was sampled from sink traps.
‡Patients positive at first PA culture on admission.
§Patients acquiring PA later during mechanical ventilation.
#Only swabs from the inner parts of the faucets were positive for PA in this study, whereas running water was uniformly negative (see text).
¶Study submitted for publication (see text).

from colonized water sites to susceptible ICU patients predominated. As in the study of Grundmann et al,[26] the supplying mains were always negative for the organism.[38] By comparison with the strains saved from the preceding study, we confirmed that the taps on our ICU harbored their individual clones over periods of up to 144 weeks.[38]

Recently, we studied the ecology of *P aeruginosa* in a 12-bed medical ICU of the same university hospital over a period of 6 months. In this study, the epidemiology of the organism was more complex, and water taps sometimes changed their resident clone. Again, 60 of 143 water samples (41.9%) were positive for *P aeruginosa*, and 8 of 16 patients (50%) became colonized or infected with waterborne clones. It remained unclear, however, to what extent horizontal transmissions between patients contributed to the epidemiology on this ward. An important indicative finding, however, was the observation that a single room whose water tap was free of *P aeruginosa* over the entire study period also never harbored a patient that acquired *P aeruginosa* [M. Trautmann et al, manuscript submitted].

Our findings were recently corroborated by other researchers studying the interrelationships between waterborne and clinical *P aeruginosa* strains (Table 1). Vallés et al[34] performed a longitudinal study on a 16-bed mixed ICU of a teaching hospital over a period of 3 years. Fifty-four percent of ventilated patients became colonized with *P aeruginosa*. Based on PFGE results, 83% of the colonizing strains were classified as exogenous, and roughly half of these were cultured from tap water.[34] Similarly, Blanc et al, performing a study on 5 ICUs in Lausanne, Switzerland, found a strain identical to one isolated from water faucets in 42% of their patients.[35] Six genotypes were recovered in the faucets before being isolated from 46 of a total of 132 patients (35%). In that study, only swabs taken from the inner parts of the faucets were positive for the organism, whereas tap water samples were uniformly negative. However, small volumes of only 0.1 mL water were cultured in this study, pointing to the need for appropriate sampling techniques to detect *P aeruginosa* in running tap water. An interesting approach in this study was the grouping of cases into those that occurred sporadically and those occurring in clusters. This analysis showed that both horizontal transmissions between patients and direct transmissions from water sites occurred, and both water-related and nonwater related clones appeared to have spread by horizontal transmission in approximately

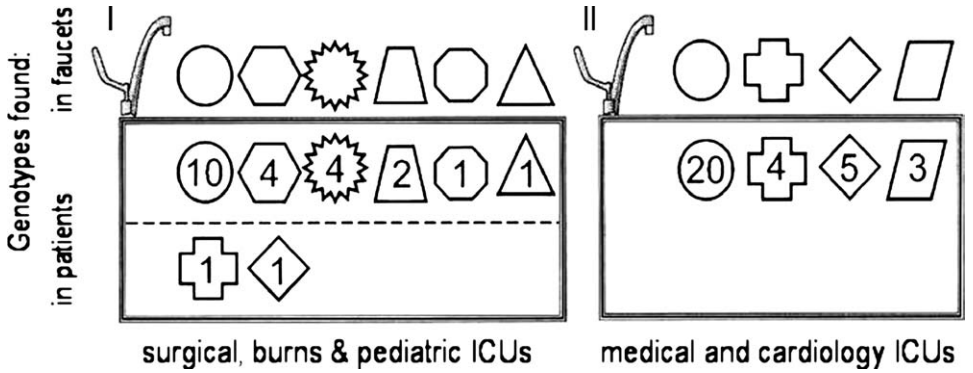

Fig 2. Schematic representation of tap water (upper part of figure) and patient clones of *P aeruginosa* in ICUs served by 2 separate water distribution systems in the study of Blanc et al.[35] Each genotype is represented by a different shape; the number of cases harboring each genotype is indicated in the shape. When genotypes were recovered from the faucets of only 1 of the 2 water distribution systems, the cases harboring these genotypes were observed almost exclusively in the ICUs supplied by this system. This strongly suggested that the faucets were the ultimate reservoir for a substantial proportion of cases colonized/infected with *P aeruginosa* (Figure adapted from Berthelot et al[33]).

Fig 3. Corroded faucet aerators create a milieu for the formation of biofilms and growth of *P aeruginosa*.

half of the instances. Approximately one fourth of all cases was neither horizontally acquired nor transmitted via tap water, a figure corresponding well to the above-mentioned fraction of patients importing their own strains into the ICU.[35] Although a single genotype occurring in both water systems supplying these ICUs accounted for 30 of 132 patient episodes (22.7%), a variety of other clones occurred that were also related to water isolates (Fig 2).

METHODOLOGIC ASPECTS OF TESTING FOR *P AERUGINOSA*

For future studies of this kind, it will be essential to use a standardized and sensitive technique for the investigation of taps and running water. The study by Blanc et al shows that examining small volumes of water is less sensitive than performing swabs from the inside of outlets.[35] We recommend the following procedure for sampling water that proved to be very sensitive in our studies: (1) take water from tap to be examined during a period of less intense use, eg, at 7 AM on Monday mornings before activities such as patient washing have started, (2) leave aerator in place, (3) collect a 100-mL sample of the first flush of cold water, (4) filtrate the whole sample volume through a disk-shaped 0.45-μm membrane filter (eg, Nalgene; Millipore, Molsheim, France), (5) place filter on cetrimide agar plate and incubate at 37°C for 24 hours. Pigmented oxidase-positive colonies growing at 42°C on subculture can be assumed to represent *P aeruginosa* and should be selected for further biochemical analysis. Additionally, taps may be examined by swabbing the inside of the outlet part after removal of the aerator or by swabbing the inner parts (bushing, grid) of the aerator. Studies comparing these techniques in a prospective manner have not been performed but are clearly warranted. The sensitivity to detect clonal relationships between water and patient isolates may be enhanced by picking 3 or more colonies from 1 plate for molecular typing.[34]

TECHNIQUES OF WATER TAP DISINFECTION

Mechanical cleaning of taps and aerators

Uniformly, the above-mentioned studies showed that *P aeruginosa* colonization of water taps was not a matter of microbial contamination of drinking water supplied by the mains. The microorganism probably gains access to the taps by retrograde contamination,

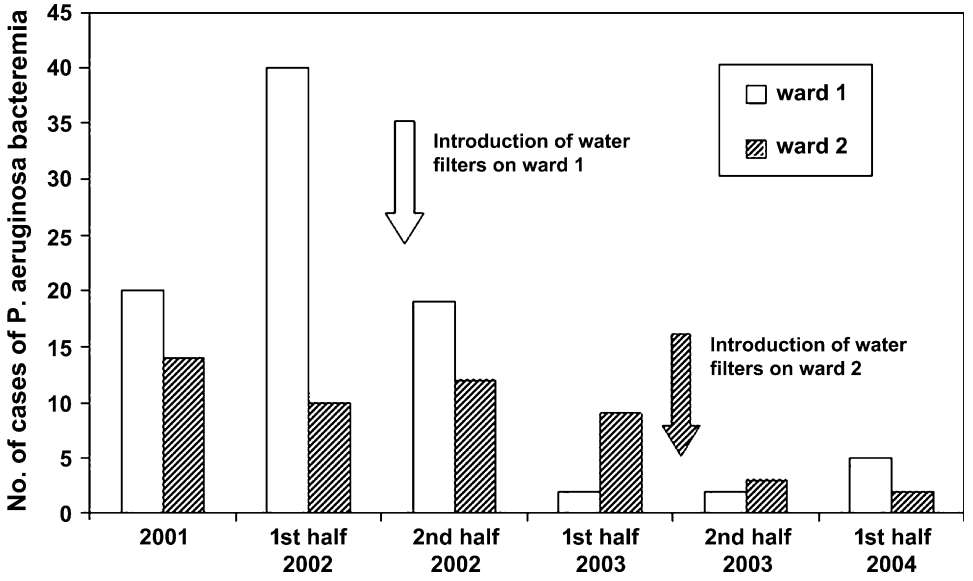

Fig 4. Effect of point-of-use water filtration in 2 hematological wards in Italy (adapted from Ricci et al[56]).

thereafter establishing itself deeply in the bushings of the aerator and mixing valves and in niches and threads of fittings and fixtures. Strategies to keep the organism at low densities should always include careful cleaning and regular descaling of outlets and aerators. Observations in hospitals show that aerators are frequently corroded, thus exhibiting irregular surfaces (Fig 3). However, in our experience, even programs of regular exchange and descaling of aerators did not result in complete eradication of *P aeruginosa* from outlets (data not shown). Similar experiences were made by Bert et al,[52] who, after unsuccessful attempts to eradicate the organism by descaling and repairing the pipes and outlets, finally closed their ward and replaced all sinks and outlets. Thereafter, a prolonged outbreak related to waterborne *P aeruginosa* subsided.[52] However, as a routine measure, removal and replacement of taps and fittings is very costly and does not preclude retrograde recontamination.

Chemical disinfection

Hyperchlorination of cold water systems is another theoretic approach to water disinfection, but, apart from the disturbance of the taste of the water, the often sparse use of some outlets will result in suboptimal chlorine treatment. The latter has recently been shown to lead to selection of multiple antibiotic-resistant strains.[53] Addition of other chemicals is prohibited by the Safe Drinking Water Act and similar legal regulations in other countries.

Thermal disinfection

Regular thermal disinfection of taps (weekly heating to 75°C for 60 minutes) was an effective measure in the study by Bukholm et al; however, the authors did not describe their technique in detail.[51] Earlier, Schoenen et al found that heating the exterior surface of the taps with a flexible electric ribbon was ineffective, but flushing the interior parts of the fittings and fixtures with boiling water pumped from an electric water bath placed in the sink resulted in eradication of *P aeruginosa*.[54] However, in face of the cutback of resources and the ever increasing workload of both technical and nursing personnel associated with the recent restructuring of many hospitals, it remains doubtful whether techniques requiring regular, time-consuming procedures to be performed will be accepted in everyday ICU practice. For the same reasons, restrictions of the use of washbasins (eg, forbidding to dump used wash water into sinks) will probably not be accepted for longer periods of time.

THE CONCEPT OF POINT-OF-USE WATER FILTRATION

In our own study of the surgical ICU of a teaching hospital, we observed approximately 5 to 10 cases of *P aeruginosa* infection or colonization per month, along with uniformly positive tap water cultures. On this ward, we decided to start a program of point-of-use water filtration using disposable tap-mounted filters used for 7 days. Filtered water proved to be always sterile after mounting the filters. Thereafter, the number of infections and colonizations fell constantly, reaching a level of 1 to 2 cases per month.[55] A detailed epidemiologic analysis of these data is ongoing.

Similar observations were recently reported by Ricci et al from an Italian hospital for oncologic patients. After observing an increase in bacteremia episodes

because of *P aeruginosa*, the authors installed disposable, point-of-use water filters on taps and showerheads on 2 wards. Thereafter, an impressive decrease of bacteremia episodes occurred (Fig 4). The authors also made a rough calculation of the cost-effectiveness of water filtration and found the additional costs to be widely counterbalanced by the bacteremia episodes prevented.[56] Similarly, Hall et al[57] compared the costs of point-of-use water filtration with costs for the use of sterile bottled water for drinking and showering in immunocompromized patients. These authors calculated that point-of-use water filters caused only 12% of the costs when compared with the expenses for sterile bottled water.[57]

In conclusion, a number of studies from the last ~7 years have provided clear evidence that tap water is a significant source of *P aeruginosa* colonizations and infections in the ICU setting. Although the exact modes of transmission have not been addressed in these studies, it is obvious that patient care activities such as face and body washing, tooth brushing, oral care, or rinsing of dental prostheses with tap water are occasions at which strains from faucets can be carried over to patients. Faucets are not only a well-documented source of *P aeruginosa* but are also easily accessable for interventions. Given the promising results obtained with point-of-use water filtration, it is certainly time to apply this concept in controlled, prospective studies on various types of ICUs.

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DOI: https://doi.org/10.1016/j.ajic.2008.01.001

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Correction

In the article entitled ''Ecology of *Pseudomonas aeruginosa* in the intensive care unit and the evolving role of water outlets as a reservoir of the organism'' (Am J Infect Control 2005;33(Suppl):S41-S49), the source of Figure 2 was incorrectly identified. The legend should read ''Copied from Blanc DS, Nahimana I, Petignat I, Wenger A, Bille J, Francioli P. Faucets as a reservoir of endemic *Pseudomonas aeruginosa* colonization/infections in intensive care units. Intensive Care Med 2004;30:1964-8.''