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Risk factors for Nosocomial Colonization with Multiresistant *Acinetobacter baumannii*

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A six-month prospective survey was carried out in a university hospital to assess the incidence of *Acinetobacter baumannii* cross-contamination and to identify risk factors for colonization. Clinical isolates obtained during the study period were biotyped and genotyped by pulsed-field gel electrophoresis after *Apa*I macrorestriction of total DNA. Case-control univariate and multivariate analyses were performed to identify risk factors for *Acinetobacter baumannii* colonization. One hundred forty-seven patients hospitalized in 36 units were colonized or infected, of whom 52 were in three intensive care units. The urinary (29 %) and bronchopulmonary tracts (26 %) were the most frequently colonized sites. Nine major restriction patterns were identified: two were exhibited by epidemic multiresistant strains of biotype 9 which were isolated from 65 patients hospitalized in ten units. Multivariate analysis showed that case-patients were (a) more likely than non-infected controls to be male, to have been previously hospitalized in another unit and to have had longer stays in the unit before colonization and hyperalimentation; and (b) more likely than controls colonized with other gram-negative bacilli to be male, to have had longer hospitalization, to have received treatment with third-generation cephalosporins and to have had a urinary catheter. The high incidence of colonization with *Acinetobacter baumannii* can thus be attributed to frequent cross-contamination and the use of broad-spectrum antibiotics. Colonized patients appear to be the major source of cross-contamination as epidemic strains spread throughout the hospital.

Acinetobacter is widespread in nature: strains can be isolated from soil and water samples as well as from dairy products, poultry and frozen foods (1). Epidemiological surveys have suggested that as many as 25 % of healthy males may harbor the organisms on skin, and up to 7 % of healthy people may carry the organisms in their throat (2, 3). The digestive tract of hospitalized patients has been found to be frequently colonized with *Acinetobacter baumannii* (4). During the last decade, *Acinetobacter baumannii* and *Acinetobacter* spp., particularly genospecies 3, have emerged as significant hospital pathogens (5). Although most clinical isolates represent colonization rather than infection, *Acinetobacter baumannii* has been reported with increasing frequency as the cause of various serious infections such as septicemia, pneumonia, meningitis and urinary tract infections (6–9). Occasional community-acquired infections have been described (10, 11), but *Acinetobacter baumannii* has been mainly implicated in hospital outbreaks, especially in intensive care units (6, 12). Various factors predisposing to these infections have been identified, such as malignancy, burns, major surgery (13), enteral hyperalimentation, mechanical ventilation, tracheostomy or endotracheal tubes and presence of intravenous and urinary catheters (6, 14, 15).

It is thus important to recognize sources and routes of transmission to be able to prevent the hospital acquisition of *Acinetobacter baumannii*. Several typing schemes have therefore been developed: antibiogram analysis (16), serotyping (17), biotype and bacteriophage type determinations (18) and cell envelope protein patterns (19). With the advent of molecular biology technology, several highly discriminatory techniques have

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been developed using DNA polymorphism as a marker for strain comparison for epidemiological purposes: plasmid profiles (20), ribotyping (21), analysis of chromosomal DNA by pulsed-field gel electrophoresis (22, 23) and fingerprinting by arbitrary primed polymerase chain reaction (24). The incidence of cross-contamination can now be evaluated accurately by genotyping methods.

We report herein the results of a six-month prospective survey of colonization of patients hospitalized in the university hospital of Besançon, France. The aims of this work were to estimate the incidence of cross-contamination within and between wards and to highlight risk factors for *Acinetobacter baumannii* colonization among patients hospitalized in intensive care, surgical and medical units by a case control study.

Materials and Methods

Background. The university hospital in Besançon, France, has 1,300 beds in 60 care units (surgical, medical and intensive care units) in two sites. An average of 2,200 patients per month are admitted for more than 48 h.

Study Design. Clinical cultures of all patients present between 1 June 1993 and 1 December 1993 were examined to identify possible cases of *Acinetobacter baumannii* colonization/infection. Strains were biotyped, fingerprinted by pulsed-field gel electrophoresis and tested for antibiotic sensitivities and plasmids. Patients with positive cultures were compared to two separate control groups.

Bacterial Strains. Acinetobacter baumannii was identified using the API20 NE system (bioMérieux, France). Six carbon sources (levulinate, citraconate, L-phenylalanine, phenylacetate, 4-hydroxybenzoate and L-tartrate) were used for biotyping according to Bouvet et al. (18). Antibiotic sensitivities were determined by a disk diffusion method (Diagnostics Pasteur, France) on Mueller-Hinton agar medium.

Epidemiologic Genotyping. Plasmid profiles were determined according to Hartstein et al. (20). Unsheared DNA was prepared by the method of Prévost et al. (25), digested with the restriction endonuclease *Apa*I according to the manufacturer's instructions and subjected to pulsed-field gel electrophoresis using a contour-clamped homogeneous electric field (Chef-DRIII system, Bio-Rad, USA) (pulse times of 20 sec for 12 h and then 5 to 15 sec for 17 h at 150 V and 14°C). Gels were stained with ethidium bromide (0.1 %) for 30 min.

Analysis of DNA Relatedness. The electrophoretic restriction patterns (number and size of fragments) were analyzed by scanning photographic negatives with an LKB 2222–020 Ultrascan laser densitometer (LKB Pharmacia, Sweden) as described by Prévost et al. (25). The restriction pattern of each strain was compared to the profile of all other strains. The DNA fingerprint of each isolate was scored for the presence or absence of individual bands (negative character: absence of a band; positive character: presence of a band). A similarity index was determined for each pair of strains by the Jaccard-Sneath formula: $(S(i,j) = N_a/(N_a + N_b))$ (26), where N_a is the number of characters shared by i and j and N_b the number of different characters. We compared inter-gel restriction fragment length polymorphisms by including an internal reference strain in each gel. Major restriction genotypes were defined according to Struelens et al. (27) (common restriction patterns differed by 3 or fewer fragments and showed a similarity coefficient of > 85 %). Major genotypes are designated with numerals, and each of their variant subtypes are indicated by a letter suffix.

Case-Control Study. Cases were identified prospectively during the study period; a case was defined as any patient with a positive culture for *Acinetobacter baumannii* without evidence of tissue invasion. Only one isolate from each patient was included (only the first *Acinetobacter baumannii* positive specimen). Two separate control groups were used. Both were randomly selected from patients hospitalized in the same unit and during the same period (± 21 days) as that of the matched case. For the first control group (A) the same type of specimen (blood, superficial swabs, urinary tract specimens, tracheal aspirates or genital tract specimens) as that from the matched case had to be negative for *Acinetobacter baumannii*, hence this group was used to identify risk factors for *Acinetobacter baumannii* colonization. For the second control group (B) the same type of specimen had to be positive for another gram-negative bacteria: this group was then used to identify specific risk factors for *Acinetobacter baumannii* colonization among gram-negative bacteria.

The following data were extracted from medical records: age, sex, previous hospitalization, major underlying diseases, duration of stay in the unit, surgical and nursing procedures and medication (antibiotics, steroids and antacids).

Statistical Analysis. Univariate conditional logistic regressions were performed to identify risk factors. Variables significantly associated with colonization, at least for one of the two control groups, are the only ones displayed in the tables. Odds ratios were estimated by exponentiation of regression coefficients and their 95 % confidence interval (CIs) reported. To adjust for confounding factors, variables with a p value below the 10 % significance level in univariate analysis were entered in multiple conditional logistic regression models. Two sets of analyses were carried out, one for each control group. Egret software (SERC, USA) was used for all statistical calculations.

Results

During the study period, 147 patients (102 males and 45 females) were colonized or infected with isolates identified by the API20 NE system as presumptive *Acinetobacter baumannii*. Their median age was 57 years (range, 0–94 years). Fifty-six patients were hospitalized in a medical unit, 39

Table 1: Phenotypic and genotypic characteristics of initial isolates of *Acinetobacter baumannii*.

Unit	Source	No. of isolates	Antibiotype*	Biotype	Plasmid profile	Major DNA pattern
Hospital St. Jacques						
Medical unit A	urinary tract	1	Ipm	9	A	2
Medical unit A	urinary tract	1	Ipm	9	–	3
Medical unit B	urinary tract	1	Ipm	9	A	2
Medical unit C	superficial swab	1	Tic, TicAc, Ipm, G, T, Ak	2	–	5
Medical unit D	vaginal swab	1	Tic, TicAc, Ipm, G, T, Ak	6	–	4
Urology	urinary tract	1	Ipm	9	A	2
Urology	urinary tract	1	Ipm	9	–	3
Urology	urinary tract	1	Ipm, Ak	9	–	6
Hospital Jean Minjoz						
MICU	urinary tract	1	Ipm	9	A	2
MICU	respiratory tract	2	Ipm	9	A	2
MICU	urinary tract	6	Ipm	9	–	3
MICU	respiratory tract	7	Ipm	9	–	3
MICU	blood catheter	2	Ipm	9	–	3
MICU	blood	1	Ipm	9	–	3
MICU	urinary tract	2	Ipm, Ak	11	–	4
SICU 1	urinary tract	5	Ipm	9	A	2
SICU 1	respiratory tract	4	Ipm	9	A	2
SICU 1	superficial swab	1	Ipm	9	A	2
SICU 1	urinary tract	5	Ipm	9	–	3
SICU 1	respiratory tract	4	Ipm	9	–	3
SICU 1	superfcial swab	2	Ipm	9	–	3
SICU 1	urinary tract	1	Ipm, Ak	11	–	4
SICU 1	respiratory tract	2	Ipm, Ak	11	–	4
SICU 2	urinary tract	1	Ipm	9	A	2
SICU 2	respiratory tract	3	Ipm	9	A	2
SICU 2	urinary tract	3	Ipm	9	–	3
SICU 2	respiratory tract	2	Ipm	9	–	3
SICU 2	vaginal swab	1	Ipm	9	–	3
SICU 2	blood catheter	1	Ipm	9	–	3
Radiotherapy	urinary tract	1	Tic, TicAc, Ipm, G, T, Ak	2	–	1
Medical unit (2AN)	superficial swab	1	Ipm	9	A	2
Medical unit (2AN)	urinary tract	1	Ipm, Ak	11	–	9
Medical unit (3AN)	superfcial swab	1	Ipm	9	A	2
Haematologic unit	urinary tract	1	Ipm	9	A	2
Haematologic unit	urinary tract	1	Tic, TicAc, Ipm, G, T, Ak	7	–	7
Medical unit (5AO)	urinary tract	1	Ipm	9	–	3
Medical unit (6AO)	urinary tract	1	Ipm	9	–	3
Surgical unit (4AO)	superficial swab	1	Ipm	9	A	2
Surgical unit (4AO)	superficial swab	1	Ipm	9	–	3
Surgical unit (4AO)	superficial swab	1	Ipm	9	A	2
Surgical unit (7AN)	superficial swab	1	Ipm	9	A	2

* Antibiotype expressed as susceptibility profile based on testing the following antimicrobial agents: imipenem (Ipm), ticarcillin (Tic), ticarcillin-clavulanate (TicAc), gentamicin (G), tobramycin (T) and amikacin (Ak).
MICU: medical intensive care unit; SICU: surgical intensive care unit.

in a surgical unit and 52 in an intensive care unit. Sixty patients (41 %) had isolates cultured from superficial swabs (12 blood catheters, 22 surgical wounds and 26 eschares), 42 (29 %) from urinary tract specimens, 38 (26 %) from broncho-pulmonary tract specimens, 4 (3 %) from blood specimens and 3 from vaginal swabs. Among these 147 patients, 51 had one or more other specimens positive for *Acinetobacter baumannii*.

Epidemiological Investigations. Ninety-seven *Acinetobacter baumannii* clinical isolates were typed. They were collected from 76 patients hospitalized in 16 units at the two hospital sites. Results of different typing methods for initial isolates are reported in Table 1. There were 15 different DNA patterns among the corresponding initial *Acinetobacter baumannii* isolates (Figure 1). Five related patterns (types 2a, 2b, 2c, 2d and 2e) were clustered in a clonal group of patterns, accounting for 26 isolates. One other major pattern could be subdivided into three types (types 3a, 3b and 3c) and accounted for 39 isolates. A third major pattern (type 4) was demonstrated in five patients, and the remaining six patterns were each demonstrated in only one patient. The two major epidemic patterns (types 2 and 3) were exhibited by strains belonging to bio-

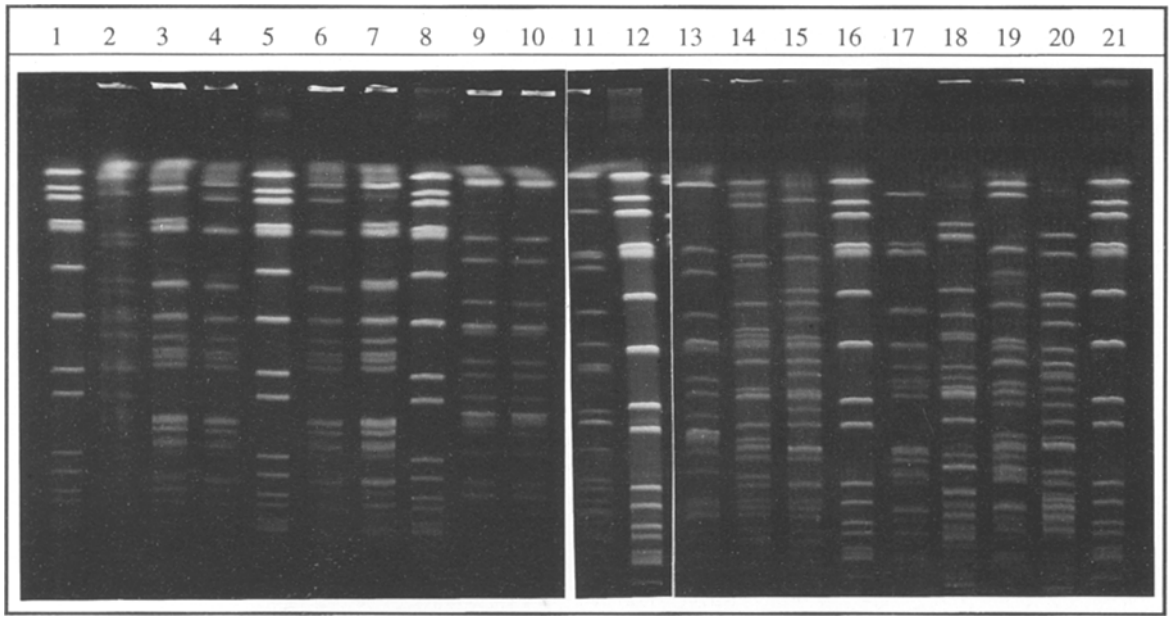

Figure 1: Pulsed-field gel electrophoresis of *ApaI* digested DNA from *Acinetobacter baumannii* isolates. Lanes 1, 5, 8, 12, 16 and 21: *Staphylococcus aureus* NCTC 8325; lane 2: pattern 1; lane 3: 2a; lane 4: 2b; lanes 6, 17: 2c; lane 7: 2d; lane 9: 3a; lane 10: 3b; lane 11: 4; lane 13: 3c; lane 14: 5; lane 15: 6; lane 18: 7; lane 19: 8; lane 20: 9.

type 9; these strains were resistant to all antibiotics tested except imipenem. All strains exhibiting pattern 2 harbored a 6.6 kb plasmid. Strains that displayed pattern 3 were not typeable by plasmid profile analysis.

The 65 strains exhibiting these two epidemic patterns were isolated from patients hospitalized in ten units: 51 patients in intensive care units, eight in medical units and six in surgical units. The colonized patients hospitalized in medical and surgical units had not been transferred from intensive care units. The incidence of these two patterns both peaked in August. The different colonization sites were similarly distributed for the two epidemic strains (i.e. mainly from urinary tract specimens, or less often from broncho-pulmonary tract specimens), except blood catheters, which were colonized only by strains with pattern 3. Strains displaying the six sporadic patterns were isolated from among 20 patients who had a specimen positive for *Acinetobacter baumannii* during the first 48 h of hospitalization (3 patients in medical units and 3 in surgical units). In 17 of 76 patients, two strains were isolated from two different sites: in 15 of the 17 cases the two strains displayed the same pattern. Four repetitive strains (same specimens from same patients) were isolated at intervals of 15 days to two months: in one case, one variation was observed between the patterns of the initial and the subsequent isolate.

Case-Control Study. The characteristics of the patient population included in the case-control study are given in Table 2. One hundred twenty-one patients with negative specimens fulfilling matching criteria were eligible as controls (group A). Twenty-six cases without a matched control were excluded by conditional logistic regression (leaving 121 matched pairs). Table 3 lists the variables significantly associated with colonization for group A controls. Cases were more likely than controls to be male (OR = 2.06), to have hypertension (OR = 2.75) and to have been previously hospitalized in another unit (OR = 2.09). Biological data were similar in cases and A controls, except that the blood albumin level was lower among cases (OR = 0.90). A clear association was found between *Acinetobacter baumannii* colonization and the use of antibiotic therapy within the two days before culture, with a relative risk factor of 4.3. Aminoglycosides were found to be a protective factor for *Acinetobacter baumannii* colonization (OR = 0.37). Nasogastric tube and its duration, enteral hyperalimentation and its duration, and urinary catheter and its duration greatly increased the relative risk of *Acinetobacter baumannii* colonization. Case-patients were significantly more likely than controls to

Table 2: Characteristics of cases and controls.

Variable	No. (%) ± SD		
	Cases (n = 147)	A controls (n = 121)	B controls (n = 116)
Males	102 (69)	70 (58)	62 (53)
Hypertension	40 (27)	21 (17)	21 (18)
Diabetes	19 (13)	13 (11)	8 (7)
Antibiotic therapy	76 (52)	57 (47)	44 (38)
Third-generation cephalosporins	25 (17)	15 (12)	5 (4)
Ureidopenicillin	2 (1)	2 (2)	7 (6)
Aminoglycosides	11 (7)	19 (16)	10 (9)
Nasogastric tube	90 (61)	61 (50)	57 (49)
Days with nasogastric tube	15 ± 14	4 ± 3	16 ± 27
Enteral hyperalimentation	68 (46)	23 (19)	34 (29)
Days of enteral hyperalimentation	13 ± 8	4 ± 4	16 ± 26
Tracheostomy	37 (25)	3 (2)	18 (16)
Days of mechanical ventilation	12 ± 13	3 ± 3	10 ± 24
Blood catheter	135 (92)	101 (83)	101 (87)
Days with CVC	16 ± 10	5 ± 5	13 ± 25
Days with arterial catheter	12 ± 9	4 ± 4	7 ± 6
Days with peripheral i.v. catheter	13 ± 11	6 ± 6	10 ± 20
Urinary catheter	103 (70)	73 (60)	59 (51)
Days with urinary catheter	15 ± 14	4 ± 4	12 ± 24
Days of hospitalization before colonization*	15 ± 13	4 ± 4	9 ± 16
Days in unit	29 ± 20	13 ± 10	20 ± 20
Previous hospitalization in another unit	49 (33)	24 (20)	27 (23)
Albumin concentration (g/l)	34 (8)	37 (6)	35 (6)

*Before colonization for cases and B controls, or before negative specimen for A controls.

have had an indwelling blood catheter (OR = 2.66). The duration of a central i.v. catheter, an arterial catheter and a peripheral i.v. catheter was significantly longer for cases than for controls. Tracheostomy was found to be a risk factor (OR = 28.00), and duration of mechanical ventilation also increased the risk of *Acinetobacter baumannii* colonization (OR = 1.30). The total duration of stay in the unit was longer for cases than for controls (OR = 1.09), as was the duration of stay in the unit before a positive specimen for cases or before a negative matched specimen for controls. Multiple conditional logistic regression analysis identified five independent risk factors: sex, duration of stay before specimen collection, enteral hyperalimentation, previous hospitalization in another unit and aminoglycoside therapy (Table 4).

One hundred sixteen patients with a specimen that matched that of the cases and was positive for gram-negative bacteria other than *Acinetobacter baumannii* were eligible as group B controls. Thirty-one cases without a matched control were excluded from the analysis (leaving 116 matched pairs). Table 3 lists the variables significantly associated with colonization for control group B. In common with the analysis using group A controls, male sex, hypertension, antibiotic therapy, nasogastric tube and its duration, enteral hyperalimentation, urinary catheter, duration of catheterization with a central intravenous or an arterial catheter, duration of mechanical ventilation, total length of the stay in the unit and length of the stay in the unit before colonization were identified as risk factors. In addition, diabetes predisposed to *Acinetobacter baumannii* colonization (OR: 3.66). Treatment with third-generation cephalosporins appeared as a specific risk factor (OR: 5.33) for *Acinetobacter baumannii* colonization, whereas ureidopenicillin had a protective role (OR: 0.14). Multivariate analysis identified four independent risk factors (Table 4): sex, duration of stay in the unit, administration of third-generation cephalosporins and the presence of a urinary catheter.

Discussion

The genus *Acinetobacter* contains 19 known genospecies (28), of which most can be separated by phenotypic tests (29) and among which *Acinetobacter baumannii* is becoming frequently associated with nosocomial infection outbreaks. However, the identification of these organisms should usually be reported as presumptive because tests to determine assimilation of carbon sources are difficult to interpret (30). To control the incidence of hospital-acquired infection and

Table 3: Univariate analysis of risk factors.

Risk factor	Cases vs. A control group		Cases vs. B control group	
	Odds ratio	95 % CI	Odds ratio	95 % CI
Males	2.06[a]	1.16 – 3.70	2.27[b]	1.23 – 4.16
Hypertension	2.75[b]	1.22 – 6.17	2.25[a]	1.14 – 4.44
Diabetes	NS	–	3.66[a]	1.02 – 13.14
Previous hospitalization in another unit	2.59[a]	1.14 – 5.91	NS	–
Albumin concentration (g/l)	0.90[b]	0.84 – 0.96	NS	–
Antibiotic therapy	4.3[b]	1.23 – 15.21	1.42[a]	1.04 – 1.95
Aminoglycosides	0.37[a]	0.14 – 0.95	NS	–
Ureidopenicillin	NS	–	0.14[a]	0.017 – 1.00
Third-generation cephalosporins	NS	–	5.33[b]	1.55 – 18.3
Nasogastric tube	4.00[b]	1.50 – 10.66	2.37[a]	1.04 – 5.42
Days with nasogastric tube	1.22[b]	1.09 – 1.36	1.05[b]	1.01 – 1.10
Enteral hyperalimentation	7.6[b]	2.99 – 19.31	2.9[b]	1.41 – 5.95
Days with enteral hyperalimentation	1.35[a]	1.00 – 1.98	NS	–
Urinary catheter	5.3[b]	1.55 – 18.3	11.5[b]	2.71 – 48.78
Days with urinary catheter	1.17[b]	1.08 – 1.27	NS	–
Blood catheter	2.66[a]	1.04 – 6.81	NS	–
Days with CVC	1.20[b]	1.09 – 1.34	NS	–
Days with arterial catheter	1.31[b]	1.08 – 1.58	1.07[a]	1.00 – 1.14
Days with peripheral i.v. catheter	1.15[b]	1.06 – 1.24	1.07[b]	1.02 – 1.12
Tracheostomy	28.00[b]	3.8 – 205.8	–	–
Days of mechanical ventilation	1.30[b]	1.11 – 1.52	1.05[a]	1.00 – 1.11
Days in unit	1.09[b]	1.05 – 1.13	1.02[a]	1.01 – 1.04
Days of hospitalization before colonization[c]	1.22[b]	1.13 – 1.32	1.03[b]	1.00 – 1.05

[a] $p < 0.05$.
[b] $p < 0.01$.
[c] Before colonization for cases and B controls, or before negative specimen for A controls.
NS = odds ratio not significant.

Table 4: Adjusted risk factors for *Acinetobacter baumannii* colonization.

Risk factor	Cases vs. A control group		Cases vs. B control group	
	Odds ratio	95 % CI	Odds ratio	95 % CI
Males	2.95[a]	1.00 – 8.96	2.12[a]	1.01 – 4.46
Duration of hospitalization before colonization[c]	1.23[b]	1.12 – 1.35	NS	–
Enteral hyperalimentation	7.47[a]	1.36 – 40.95	NS	–
Urinary catheter	NS	–	10.17[b]	2.19 – 47.13
Third-generation cephalosporins	NS	–	6.89[b]	1.71 – 27.64
Aminoglycosides	0.06[a]	0.007 – 0.63	NS	–
Previous hospitalization in another unit	9.1[a]	1.31 – 63.20	NS	–
Days in unit	NS	–	1.02[a]	1.01 – 1.04

[a] $p < 0.05$.
[b] $p < 0.01$
[c] Before colonization for cases and B controls, or before negative specimen for A controls.
NS = odds ratio non significant.

to identify outbreak risk factors, epidemic strains must be differentiated from control strains from patients with sporadic infections. Biotyping by carbon source assimilation patterns discriminates 17 types within the species *Acinetobacter baumannii* (17). Biotypes 1, 2, 6 and 9 appear to be the most prevalent in hospitals (22). *Acinetobacter baumannii* hospital strains are frequently multi-resistant to antibiotics, and Seifert et al. (23) identified only one antibiotype among 21 unrelated strains. Other conventional epidemiological analysis techniques are appropriate only for reference laboratories and not for moderate-sized laboratories that do not possess all the media, phages and antisera required. Plasmid profile analysis is simple and has proved useful for the study of outbreaks (6, 12, 31) but is less discriminatory than analysis of genomic DNA by pulsed-field gel electrophoresis (23). Struelens et al. (32) and Seifert et al. (23) confirmed that considerable DNA polymorphism within an *Acinetobacter baumannii* biotype can be detected by

macrorestriction analysis. The incidence of cross-colonization was thus accurately evaluated in several intensive care unit outbreaks (23, 32, 33).

In this investigation we used biotype, antibiotype, plasmid profile and genome fingerprinting by macrorestriction analysis to follow transmission of *Acinetobacter baumannii* within and between hospital units. We observed concurrent transmission of two epidemic strains that belonged to the same biotype and had the same antibiotic susceptibility pattern but displayed different genotypic characteristics. These two strains were isolated during a period of high incidence in the summer (34) and are less frequent in the autumn. By another way, epidemic patterns were present throughout the study, so that cross-colonization during and outside the outbreak period appeared as the major risk for acquisition of *Acinetobacter baumannii.*

Several risk factors for nosocomial acquisition of *Acinetobacter baumannii* identified in previous studies were confirmed: male sex (11, 34), prolonged stay in hospital (15), enteral hyperalimentation (6, 14) and urinary catheter (34). However, in our study, only male sex and urinary catheter were specific risk factors for *Acinetobacter baumannii* among gram-negative bacteria colonization. The length of stay in the unit appeared to be a risk factor, but it could be, in part, the consequence of *Acinetobacter baumannii* colonization. In several studies the use of broad-spectrum antibiotics [aminoglycosides (34) and broad-spectrum penicillins (32)] was identified as a risk factor. In our study the protective role of aminoglycosides in a univariate analysis was surprising and was not confirmed either in the multivariate analysis or in the literature. Among β-lactam antibiotics our study identified third-generation cephalosporins as a specific risk factor for colonization by *Acinetobacter baumannii* among gram-negative bacteria.

During outbreaks, potential environmental sources were linked to *Acinetobacter baumannii* infections: pulmonary arterial catheters and blood infections (6); ventilators and bronchopulmonary tract and infections (7, 15); and enteral nutrition solutions and pneumonia (14). Hand carriage of the epidemic strain by hospital personnel has been demonstrated (33, 35). Strains displaying the two epidemic patterns were isolated from various sites of colonization from patients hospitalized in all units at the two hospital sites. Intensive care units are possible environmental reservoirs and hand carriage was a probable vehicle for cross-colonization within these units. However, colonized patients and carriers could be reservoirs for inter-unit dissemination of *Acinetobacter baumannii* in the hospital, as suggested by the risk factor of previous hospitalization in another unit.

The comparison of strains from patients hospitalized in different units and settings gave an overview of the hospital epidemiology of *Acinetobacter baumannii* colonization. Despite the peak of cases in August, *Acinetobacter baumannii* appeared to be endemic in the hospital because the same patterns were present throughout the six-month prospective study and in different wards. Studies limited to one unit, often an intensive care unit, highlight the role of environmental carriage and hand carriage in cross-colonization but do not underscore the role of patients as vehicles, as suggested by our study. The frequent presence of risk factors in intensive care units presumably explains the high incidence of *Acinetobacter baumannii* colonization in these units, and the transfer of patients to other units may explain the widespread distribution of epidemic patterns throughout the hospital. Various measures have been proposed to control nosocomial *Acinetobacter* infections, including body-substance isolation and intensive environmental disinfection. These measures are probably useful. However, the early detection of *Acinetobacter baumannii* carriers on admission, especially after transfer from other units, should be effective to control this dangerous nosocomial pathogen.

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