Resistance rates of *Staphylococcus aureus* in relation to patient status and type of specimen

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Objectives: The aim of this study was to assess the impact of data stratification on the resistance rates of *Staphylococcus aureus*, with emphasis on the value of blood culture-based resistance data.

Materials and methods: All *S. aureus* isolates from patients in the Vienna University Hospital (2140 beds), isolated between 1/1996 and 12/2002, were stratified by patient status (ICU patient, regular inpatient, outpatient). Four kinds of specimen [blood, respiratory tract (RT), wounds and urine] were defined for analysis. Oxacillin and eight other compounds were considered.

Results: In total, 10,575 first isolates per patient were detected, derived from ICU patients (*n* = 1464), inpatients (*n* = 4152), and outpatients (*n* = 4959). From blood, wounds, RT and urine, 610, 1464, 2716 and 3370 first isolates per patient, respectively, were available. The blood-MRSA-rate (19.93%) was similar to the MRSA-rate of RT- (OR: 0.98, 95% CI: 0.76–1.25), wound- (OR: 0.89, 95% CI: 0.71–1.12), and urine-isolates (OR: 0.91, 95% CI: 0.72–1.14). Isolates from inpatients (OR: 0.59, 95% CI: 0.47–0.74) and outpatients (OR: 0.16, 95% CI: 0.13–0.21), regardless of the specimen, showed lower MRSA-rates than blood-isolates, in contrast to isolates from ICU patients (OR: 1.12, 95% CI: 0.87–1.44). For other compounds, the resistance rates of blood-isolates were not always representative for RT- (six of eight rates similar), wound- (7/8), or urine-isolates (5/8). Most importantly, RT-, wound- and urine-isolates were significantly more often resistant to ciprofloxacin. Resistance rates of blood-isolates were more representative for isolates from inpatients (five of eight rates similar) than from ICU patients or outpatients (each 3/8).

Conclusions: The resistance rates of blood culture isolates enable a good overall assessment of the resistance of other clinically significant isolates. However, resistance data derived from selected specimens must not be equated with the overall resistance situation in the hospital.

Keywords: MRSA, *S. aureus*, blood culture isolates, antibiotic resistance, surveillance

Introduction

Resistance surveillance of nosocomial pathogens is an important issue in all healthcare settings. These data are needed for the choice of empirical therapy of suspected infections. There are no evidence-based guidelines with regard to the clinical specimens from which antimicrobial susceptibility data should be collected. The European Antimicrobial Resistance Surveillance System (EARSS) has chosen blood culture isolates.¹ In contrast, other networks like the SENTRY Antimicrobial Surveillance Program also include isolates from patients with community-acquired and nosocomial respiratory infections, wounds or skin and soft tissue infections, and urinary tract infections.² Relying exclusively on blood culture results is relatively common, because these isolates are considered to be clinically significant. However, with the exception that recent work has suggested a higher virulence of methicillin-resistant *Staphylococcus aureus* (MRSA) compared to methicillin-susceptible *S. aureus* (MSSA), there is no evidence that the in vitro antimicrobial resistance profile of an *S. aureus* isolate is linked to its virulence.³ More likely, it depends on host factors whether *S. aureus* isolates cause bloodstream infection.⁴

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The applicability of blood culture-based resistance rates to isolates from other types of specimens, which may be considered clinically significant (e.g. pus or lower respiratory tract), is poorly elucidated. In this context, the aim of this study was to determine the impact of data stratification (by specimen and by patient population) on the resistance rates of *S. aureus*.

**Materials and methods**

**Setting**

The Vienna University Hospital is a 2140 bed tertiary care teaching hospital, which has 1924 normal care beds, and 216 ICU (or intermediate care) beds. Per year, the hospital provides medical services for an average of 95,000 inpatients (of these, 4000 are ICU patients) and 430,000 outpatients. All microbiological specimens are processed at the Division for Clinical Microbiology.

**Identification and in vitro susceptibility testing of *S. aureus***

All *S. aureus* isolates obtained between January 1996 and December 2002 in patients of the Vienna University Hospital were subject to analysis. *S. aureus* was cultured and identified according to standard procedures. Antimicrobial susceptibility testing for penicillin, oxacillin, erythromycin, clindamycin, gentamicin, amikacin, trimethoprim and ciprofloxacin was routinely carried out by the Kirby–Bauer disc diffusion method on Mueller–Hinton agar trimethoprim and ciprofloxacin was routinely carried out by the Kirby–Bauer disc diffusion method on Mueller–Hinton agar according to NCCLS guidelines. For fusidic acid, NCCLS does not provide disc susceptibility breakpoints, the required diameters for susceptibility, intermediate susceptibility, and resistance were ≤14 mm, 15–21 mm, and >22 mm, respectively (10 μg disc).

**Definitions and data stratification**

Eighty different types of specimen were sent for microbiological analysis. For the purpose of this study, four classes of specimen were defined for further analysis: blood (all isolates from peripheral and central blood specimens), respiratory tract (RT; sputum and specimens collected by bronchoscopy), wounds (specimens designated as ‘wound swab’, ‘wound secretion’, or ‘pus’), and urine (both spontaneous and catheter urine). All patients were classified according to their patient status [ICU patients, regular inpatients (referred to as ‘inpatients’) and outpatients].

By this stratification procedure, seven subgroups of isolates were defined. Four subgroups comprised isolates from defined clinical specimens (blood, RT, wounds and urine), and three comprised isolates from defined patient categories (ICU, inpatients and outpatients).

In order to assess the resistance rates of *S. aureus* isolates from ICU patients, inpatients and outpatients, the first isolate from each patient within the study period (regardless of the type of specimen) was subject to analysis. With regard to the defined clinical specimens, the first isolate per patient from the defined type of specimen was considered. In addition to these subgroups of isolates, first isolates per patient within the study period (referred to as ‘first patient isolates’, including all *S. aureus* isolates regardless the type of specimen) were analysed. The resistance rates of blood culture isolates and of first patient isolates were compared with the resistance rates in the other subgroups.

In *in vitro* resistant and intermediate susceptible isolates were uniformly referred to as non-susceptible isolates.

In addition, the effect of inclusion of all *S. aureus* isolates (per specimen or patient category) instead of the first isolates per patient on the MRSA-rate was assessed. The MRSA-rate in the first patient isolates was compared to the MRSA-rate in all available isolates (all patients and all types of specimens including repeat isolates).

**Statistics**

Calculation of statistical significance (χ² test with Yates correction; a P value of <0.05 was considered significant) and odds ratios with Cornfield 95% confidence intervals (CI) was carried out using EpiInfo 2002 (CDC, Atlanta, GA, USA).

**Results**

**Number and sources of *S. aureus* isolates**

From 1/1996 to 12/2002, 29,611 isolates of *S. aureus* were detected. These isolates were derived from 10,575 patients in Vienna University Hospital (ICU: 1464; inpatients: 4152; outpatients: 4959). The number of isolates in the categories blood, RT, wounds and urine are shown in Table 1.

**Oxacillin resistance rates**

The MRSA-rates in each subgroup are shown in Table 2. The comparison of the MRSA-rate in the first patient isolates with the MRSA-rate in the other subgroups is shown in Table 3.

The blood-MRSA-rate (19.93%) was similar to the MRSA-rate of RT- (*P = 0.87; OR: 0.98, 95% CI: 0.76–1.25), wound- (*P = 0.34; OR: 0.89, 95% CI: 0.71–1.12), and urine-isolates (*P = 0.41; OR: 0.91, 95% CI: 0.72–1.14). Isolates from inpatients (*P <0.0001; OR: 0.59, 95% CI: 0.47–0.74) and outpatients (*P <0.0001; OR: 0.16, 95% CI: 0.13–0.21), regardless of the type of specimen, showed lower MRSA-rates than blood-isolates, in contrast to isolates from ICU patients (*P = 0.38; OR: 1.12, 95% CI: 0.87–1.44).

Inclusion of all *S. aureus* isolates (regardless of the type of specimen) instead of the first patient isolates gave a significantly higher MRSA-rate (*P <0.0001; OR: 4.33, 95% CI: 4.05–4.64). This was also observed for outpatients (10.4% versus 3.9%, *P <0.0001; OR: 2.82, 95% CI: 2.39–3.33), inpatients (38.8% versus 12.8%, *P <0.0001; OR: 4.31, 95% CI: 3.9–4.77), and ICU patients (51.3% versus 18.1%, *P <0.0001; OR: 4.75, 95% CI: 4.1–5.5). Analogously, the inclusion of all *S. aureus* isolates per specimen instead of the first isolate per patient and specimen resulted in a higher MRSA-rate for isolates from the RT (31.5% versus 19.6%, *P <0.0001; OR: 1.89, 95% CI: 1.63–2.2), wounds (30.7% versus 18.1%, *P <0.0001; OR: 2.0, 95% CI: 1.78–2.25), and resistance to analysis. *S. aureus* was cultured and identified according to standard procedures. Antimicrobial susceptibility testing for penicillin, oxacillin, erythromycin, clindamycin, gentamicin, amikacin, trimethoprim and ciprofloxacin was routinely carried out by the Kirby–Bauer disc diffusion method on Mueller–Hinton agar according to NCCLS guidelines. For fusidic acid, NCCLS does not provide disc susceptibility breakpoints, the required diameters for susceptibility, intermediate susceptibility, and resistance were ≤14 mm, 15–21 mm, and ≥22 mm, respectively (10 μg disc).

<table>
<thead>
<tr>
<th>Number and sources of isolates from defined types of specimens</th>
<th>Blood (1115)</th>
<th>RT (3511)</th>
<th>Wounds (4593)</th>
<th>Urine (6911)</th>
</tr>
</thead>
<tbody>
<tr>
<td>First isolates/specimen</td>
<td>610</td>
<td>1464</td>
<td>2716</td>
<td>3370</td>
</tr>
<tr>
<td>ICU</td>
<td>95</td>
<td>864</td>
<td>145</td>
<td>694</td>
</tr>
<tr>
<td>Inpatients</td>
<td>448</td>
<td>291</td>
<td>1596</td>
<td>1469</td>
</tr>
<tr>
<td>Outpatients</td>
<td>67</td>
<td>309</td>
<td>975</td>
<td>1207</td>
</tr>
</tbody>
</table>

| Of the 610 patients with *S. aureus* blood culture isolates, the blood culture isolate was the first isolate from the respective patient within the study period in 354 cases (58.0%) (RT: 1017/3511, 69.5%; wounds: 1747/4593, 64.3%; urine: 2167/6911, 64.3%).

The applicability of blood culture-based resistance rates to isolates from other types of specimens, which may be considered clinically significant (e.g. pus or lower respiratory tract), is poorly elucidated. In this context, the aim of this study was to determine the impact of data stratification (by specimen and by patient population) on the resistance rates of *S. aureus*.
Table 2. Resistance rates of *S. aureus* isolates stratified by subgroup

<table>
<thead>
<tr>
<th>Category of isolates</th>
<th>PEN S NS %NS</th>
<th>OXA S NS %NS</th>
<th>ERY S NS %NS</th>
<th>CLI S NS %NS</th>
<th>GEN S NS %NS</th>
<th>AMK S NS %NS</th>
<th>FA S NS %NS</th>
<th>TMP S NS %NS</th>
<th>CIP S NS %NS</th>
</tr>
</thead>
<tbody>
<tr>
<td>First patient isolates</td>
<td>2453 9079 78.7</td>
<td>10415 1096 9.5</td>
<td>8935 2299 20.5</td>
<td>10459 1194 10.2</td>
<td>10526 1392 11.7</td>
<td>11033 442 3.8</td>
<td>11737 202 1.7</td>
<td>4868 264 5.1</td>
<td>5190 1231 19.2</td>
</tr>
<tr>
<td>Outpatients</td>
<td>1004 3566 78.0</td>
<td>4393 180 3.9</td>
<td>3751 746 16.6</td>
<td>4396 249 5.4</td>
<td>4511 346 7.1</td>
<td>4468 107 2.3</td>
<td>4778 81 1.7</td>
<td>1748 84 4.6</td>
<td>2158 219 9.2</td>
</tr>
<tr>
<td>Inpatients</td>
<td>858 3184 78.8</td>
<td>3504 515 12.8</td>
<td>2967 897 23.2</td>
<td>3508 559 13.7</td>
<td>3460 619 15.2</td>
<td>3826 219 5.4</td>
<td>4016 80 1.9</td>
<td>1790 110 5.8</td>
<td>1842 584 24.1</td>
</tr>
<tr>
<td>ICU patients</td>
<td>273 1169 81.1</td>
<td>1182 262 18.1</td>
<td>1072 353 24.8</td>
<td>1209 233 16.2</td>
<td>1200 261 17.8</td>
<td>1392 54 3.7</td>
<td>1441 22 1.2</td>
<td>743 33 4.2</td>
<td>533 267 33.4</td>
</tr>
<tr>
<td>Blood</td>
<td>116 494 81.0</td>
<td>466 116 19.9</td>
<td>419 187 30.9</td>
<td>477 133 21.8</td>
<td>454 129 22.1</td>
<td>572 38 6.2</td>
<td>593 17 1.7</td>
<td>328 20 5.7</td>
<td>417 144 25.7</td>
</tr>
<tr>
<td>RT</td>
<td>267 1192 81.7</td>
<td>1176 286 19.6</td>
<td>1063 399 27.3</td>
<td>1198 264 18.1</td>
<td>1162 301 20.6</td>
<td>1387 76 5.2</td>
<td>1448 16 1.1</td>
<td>656 36 5.2</td>
<td>498 285 36.4</td>
</tr>
<tr>
<td>Urine</td>
<td>630 2737 81.3</td>
<td>2746 619 18.4</td>
<td>2033 851 29.5</td>
<td>2711 569 17.3</td>
<td>2715 655 19.4</td>
<td>3185 185 5.5</td>
<td>3318 50 1.4</td>
<td>1784 115 6.1</td>
<td>1191 639 34.9</td>
</tr>
<tr>
<td>Wounds</td>
<td>508 2198 81.2</td>
<td>2213 490 18.1</td>
<td>1959 741 27.4</td>
<td>2211 495 18.3</td>
<td>2140 567 20.9</td>
<td>2538 170 6.3</td>
<td>2653 55 2.0</td>
<td>1187 98 7.6</td>
<td>943 526 35.8</td>
</tr>
</tbody>
</table>

Abbreviations: S, susceptible; NS, non-susceptible; PEN, penicillin; OXA, oxacillin; ERY, erythromycin; CLI, clindamycin; GEN, gentamicin; AMK, amikacin; FA, fusidic acid; TMP, trimethoprim; CIP, ciprofloxacin.

Table 3. Resistance rates of the first patient isolates (regardless of type of specimen) compared with other subgroups of isolates

<table>
<thead>
<tr>
<th></th>
<th>Outpatients</th>
<th>Inpatients</th>
<th>ICU patients</th>
<th>Blood</th>
<th>RT</th>
<th>Wounds</th>
<th>Urine</th>
</tr>
</thead>
<tbody>
<tr>
<td>PEN</td>
<td>0.39 (0.33–0.46)</td>
<td>1.4 (1.25–1.56)</td>
<td>2.11 (1.81–2.45)</td>
<td>2.37 (1.9–2.94)</td>
<td>1.21 (1.05–1.39)</td>
<td>1.17 (1.05–1.3)</td>
<td>1.17 (1.06–1.3)</td>
</tr>
<tr>
<td>OXA</td>
<td>0.77 (0.7–0.85)</td>
<td>1.17 (1.08–1.28)</td>
<td>1.28 (1.12–1.46)</td>
<td>1.73 (1.45–2.08)</td>
<td>1.46 (1.29–1.65)</td>
<td>1.46 (1.33–1.62)</td>
<td>1.63 (1.48–1.79)</td>
</tr>
<tr>
<td>ERY</td>
<td>0.5 (0.43–0.57)</td>
<td>1.40 (1.25–1.56)</td>
<td>1.69 (1.45–1.97)</td>
<td>1.24 (1.99–3.0)</td>
<td>1.93 (1.66–2.24)</td>
<td>1.93 (1.66–2.24)</td>
<td>1.84 (1.65–2.05)</td>
</tr>
<tr>
<td>CLI</td>
<td>0.58 (0.51–0.66)</td>
<td>1.35 (1.22–1.5)</td>
<td>1.64 (1.42–1.91)</td>
<td>2.15 (1.74–2.65)</td>
<td>1.96 (1.7–2.26)</td>
<td>2.0 (1.8–2.24)</td>
<td>1.82 (1.65–2.02)</td>
</tr>
<tr>
<td>GEN</td>
<td>0.60 (0.48–0.75)</td>
<td>1.44 (1.21–1.70)</td>
<td>1.67 (1.17–2.38)</td>
<td>1.38 (1.06–1.79)</td>
<td>1.68 (1.4–2.03)</td>
<td>1.46 (1.22–1.75)</td>
<td>1.53 (1.37–1.7)</td>
</tr>
<tr>
<td>AMK</td>
<td>0.43 (0.36–0.5)</td>
<td>1.33 (1.19–1.49)</td>
<td>2.1 (1.78–2.47)</td>
<td>1.45 (1.18–1.177)</td>
<td>2.4 (2.04–2.82)</td>
<td>2.34 (2.06–2.65)</td>
<td>2.25 (2.0–2.53)</td>
</tr>
</tbody>
</table>

Resistance rates with a *P* value ≤0.05 were considered similar (↔). In case of significantly higher (†) or lower (‡) resistance rate, the odds ratio and the corresponding 95% confidence interval are indicated.
and urine (31.3% versus 9.5%, \( P < 0.0001 \); OR: 2.07, 95% CI: 1.87–2.29), but not for blood culture isolates (20.7% versus 19.9%, \( P = 0.8 \); OR: 1.05, 95% CI: 0.81–1.36).

Resistance to other antimicrobial agents

The resistance rates of the isolates in each subgroup are shown in Table 2.

The resistance rates of blood-isolates were not throughout representative for RT (six of eight rates similar), wound- (7/8), or urine-isolates (5/8). Most importantly, RT- (\( P < 0.0001 \); OR: 1.66, 95% CI: 1.3–2.12), wound- (\( P < 0.0001 \); OR: 1.62, 95% CI: 1.29–2.02), and urine-isolates (\( P = 0.0006 \); OR: 1.55, 95% CI: 1.25–1.93) were more often resistant to ciprofloxacin. The resistance rates to fusidic acid were higher in blood-isolates than in isolates from the RT (\( P = 0.009 \); OR: 2.59, 95% CI: 1.24–5.44) and from urine (\( P = 0.03 \); OR: 1.9, 95% CI: 1.05–3.42). Blood culture isolates were significantly more often resistant to clindamycin than urine isolates (\( P = 0.01 \); OR: 1.33, 95% CI: 1.07–1.65).

The resistance rates of blood-isolates were more representative for isolates from inpatients (five of eight rates similar) than from ICU-patients or outpatients (each 3/8).

The comparison of the resistance rates of the first patient isolates to the resistance rates of the other subgroups is shown in Table 3.

Discussion

The aim of this study was to determine the impact of data stratification by type of specimen and by patient population on the resistance rates of \( S. aureus \) on the basis of 29 611 isolates recovered during a 7 year period. Nine antibiotics including oxacillin were analysed. Glycopeptide antibiotics were not considered, because no vancomycin intermediate susceptible or resistant \( S. aureus \) strains (as determined by disc diffusion test according to NCCLS guidelines) were detected during the study period.

The study especially focused on the question of whether blood culture-based resistance rates are representative for isolates from other specimens, which may also be considered clinically significant. For this study, samples from the respiratory tract (excluding throat swabs, which are occasionally taken for screening purposes), wounds (if the specimens were definitely designated as material from a wound and not just as ‘swab’), and urine (from patients with microbiologically significant bacteriuria) were chosen for comparison.

With regard to the MRSA-rate, blood culture isolates were representative for isolates from the respiratory tract, wounds and urine. The inclusion of all (i.e. first and repeat) isolates instead of the first isolates per patient gave a significantly higher MRSA-rate in all subgroups with the exception of blood culture isolates. A possible explanation for this finding is that the frequency of taking control cultures is probably higher in patients with MRSA than in patients with MSSA. In contrast, in patients with bacteraemia or sepsis due to \( S. aureus \), the number of control cultures is rather determined by the clinical course of the patient than by the \( in vitro \) resistance pattern of the pathogen. Resistance of \( S. aureus \) to oxacillin (or methicillin) is essentially determined by the mecA gene, which encodes an altered penicillin-binding protein (PBP 2a), a membrane-bound enzyme.\(^7\) As the mutations leading to the expression of PBP 2a are complex, acquisition of resistance to oxacillin due to antibiotic pressure is nearly impossible. Therefore, it is biologically plausible that the MRSA-rate in the specimens considered clinically significant is essentially similar. In contrast, inpatient and outpatient isolates (not regarding the specimen) showed significantly lower MRSA-rates than clinically significant isolates, whereas the MRSA-rate of isolates from the ICU patient population was similar to the MRSA-rate of the clinically significant isolates. Obviously, screening samples, which are occasionally obtained without clinical evidence of infection (from nose, skin, and throat) have decreased the MRSA-rate significantly. The fact that infections with MRSA (compared with infections with MSSA) are more likely to occur in seriously ill patients has been described previously.\(^7\) The accumulation of MRSA strains in clinically significant sites and ICU patients may be explained by a higher virulence of MRSA compared to MSSA.\(^3\)

With regard to compounds other than oxacillin, the resistance rates were higher in inpatients than in outpatients, and highest in ICU patients. This is predictable given the expected antibiotic exposure. The resistance rates of blood culture isolates were poorly representative for these groups of patients. The overall agreement between blood culture isolates and other clinically significant isolates was good, with the important exception of ciprofloxacin. Ciprofloxacin resistance in \( S. aureus \) is acquired more easily than resistance to most other (non-quinolone) compounds, due to a stepwise acquisition of chromosomal mutations.\(^7\) With regard to patients with chronic respiratory tract infections (e.g. COPD), chronic wounds (e.g. diabetic patients), or recurrent urinary tract infection, a higher resistance of such isolates to quinolones is biologically plausible.

In addition to ciprofloxacin, significant differences between blood culture isolates and other clinically significant isolates were observed for fusidic acid, which had lower resistance rates in isolates from the respiratory tract and from urine. Natural mutants resistant to fusidic acid are present in normal populations of \( S. aureus \) with a frequency of 1 in \( 10^6 \) to 1 in \( 10^8 \). Although it may not occur in a high frequency in clinical practice, selection of resistant mutants during therapy has been recognized \( in vivo.\(^{10} \) Fusidic acid is a second-line compound, which is notably used for treatment of patients with skin and soft tissue infections. Wounds are an important source of \( S. aureus \) bacteraemia.\(^1\) Therefore it may be speculated that the higher resistance of blood and wound isolates is directly attributable to the acquisition of secondary resistance. It should be noted that the significant differences in fusidic acid resistance between blood culture isolates (2.8%) and isolates from urine (1.5%) or respiratory tract (1.1%) are primarily of epidemiological interest, while they may have little clinical impact. The differences in clindamycin (blood versus urine: 21.8% versus 17.3%) and ciprofloxacin resistance (blood versus respiratory tract: 25.7% versus 36.4%) were more pronounced. However, it should be noted that ciprofloxacin is not a first-line compound for the treatment of \( S. aureus \) infections.

It is a limitation of this study that the presented resistance rates, notably for compounds which enable the acquisition of secondary resistance by \( S. aureus \), are influenced by local antibiotic policy. The number of isolates tested against the investigated antimicrobial agents varied, because for some classes of antibiotics (e.g. quinolones), different reference compounds were used, and because some second-line compounds (e.g. trimethoprim) were not tested routinely.
In recent years, increasing interest has focused on the occurrence of ‘community-acquired’ MRSA strains. These strains have been reported to cause infections in healthy community-dwelling persons without established risk factors for MRSA acquisition, and may be primarily identified by their unusual susceptibility to non-β-lactam antibiotics. It has been shown that ‘community-acquired’ MRSA strains are genotypically distinct from hospital-acquired MRSA strains, which can be confirmed by multi-locus sequence typing. The emergence of these strains will have to be considered in resistance surveillance in the future; however, it is difficult to assess the proportion of these strains in a retrospective format (i.e. on the basis of the *in vitro* susceptibility pattern) without the availability of molecular markers.12,13

In conclusion, the present data indicate that the resistance of blood culture isolates enables a good overall assessment of the resistance of other clinically significant isolates. This is notably true with regard to the MRSA-rate. Nevertheless, the definition of additional types of specimens for routine resistance surveillance may enable a more detailed assessment of the local resistance epidemiology. In addition, pooling of isolates from blood, respiratory tract (excluding throat swabs if they are taken for screening purposes), wounds and urine increases the number of samples available for analysis, which may be especially important for small hospitals with low numbers of blood culture isolates. However, resistance data derived from clinically significant isolates must not be equated with the overall resistance situation in the hospital.

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**References**