Staphylococcus aureus bacteremia (SAB) is common and increasing worldwide. A retrospective review was undertaken to quantify the number of cases, their place of acquisition, and the proportions caused by methicillin-resistant *S. aureus* (MRSA) in 17 hospitals in Australia. Of 3,192 episodes, 1,571 (49%) were community onset. MRSA caused 40% of hospital-onset episodes and 12% of community-onset episodes. The median rate of SAB was 1.48/1,000 admissions (range 0.61–3.24; median rate for hospital-onset SAB was 0.7/1,000 and for community onset 0.8/1,000 admissions). Using these rates, we estimate that ≈6,900 episodes of SAB occur annually in Australia (35/100,000 population). SAB is common, and a substantial proportion of cases may be preventable. The epidemiology is evolving, with >10% of community-onset SAB now caused by MRSA. This is an emerging infectious disease concern and is likely to impact on empiric antimicrobial drug prescribing in suspected cases of SAB.

Bacteremia caused by *Staphylococcus aureus* continues to be a common problem worldwide. In the preantibiotic era, most cases occurred in young patients without underlying disease. The associated death rate was 82% (1). Even with antimicrobial drug treatment, death rates remain high; in a recent meta-analysis of 31 studies, estimates of death rates for methicillin-resistant strains (MRSA) varied from 0.0% to 83.3% (median 34.2%), while those for methicillin-sensitive strains (MSSA) varied from 3.6% to 51.7% (median 25.0%) (2). Many of these infections are healthcare associated and thus are potentially preventable.

Antimicrobial drug resistance in *S. aureus* arose early after the development of antimicrobial agents and continues to evolve. In Australia, hospital strains are frequently methicillin resistant and resistant to several other antimicrobial drugs (3). This resistance limits the choice of potentially efficacious agents and results in frequent use of glycopeptides, such as vancomycin. The reliance on vancomycin causes difficulties because vancomycin has been shown to be less effective than isoxazolyl penicillins (e.g., flucloxacillin) in treating severe infections caused by *S. aureus* (4,5). This may be an explanation for the higher death rate associated with bacteremia caused by MRSA, compared with that caused by MSSA (2,6). Although MRSA tends to be the bacterium discussed most often in relation to healthcare-associated infections, MSSA strains are responsible for the largest proportion of hospital-acquired infections (3).

*S. aureus* remains a common cause of bloodstream infections of community onset. Increasing numbers of these community-onset infections are being caused by MRSA. Some of these infections may be caused by hospital strains carried into the community by patients or health-care workers, but others are caused by true community strains in patients who have had no recent healthcare contact (7–9). These strains have emerged in many countries, including Australia, New Zealand, the United States, Canada, France, Switzerland, Greece, Denmark, Finland, Scotland, and the Netherlands. They are susceptible to most or all non–β-lactam antimicrobial drugs, are highly pyogenic, and are often associated with indigenous populations (10,11).

Although *S. aureus* is a well-known major cause of bacteremia, population-based estimates of its incidence are lacking. This study used hospital data to estimate the incidence of *S. aureus* bacteremia in Australia. In addition, we...
classified episodes on the basis of community or hospital onset and on the basis of methicillin susceptibility.

Methods

*S. aureus* bacteremia data were obtained from microbiology departments that prospectively collected information for >12 months on episodes of laboratory-confirmed bacteremia for the hospitals they serviced from January 1, 1999, to December 31, 2002. Information retrieved from existing databases included the total number of episodes of community- and hospital-onset bacteremia, the number of episodes of community- and hospital-onset MRSA and MSSA bacteremia, the total number of hospital separations (defined as completed hospital admissions), and the mean length of stay. Multiple positive blood cultures in the same patient within 14 days were considered a single episode. Episodes were considered to have a hospital onset when the first positive blood culture was collected >48 hours after admission to hospital. All other infections were designated community onset (for example, day-only dialysis related episodes were defined as community onset, as were infections with their onset in nursing homes). Organism identification and susceptibility testing were by standard methods. All these laboratories participate in external quality assurance programs as well as AGAR national surveys (3,12), which have quality control procedures to ensure these laboratories accurately detect methicillin resistance. Published data were used for the details on the number of hospital beds and separations for Australia and for the classification of different types of hospitals (13). The term separations, rather than admissions, is used in the published data because hospital abstracts for inpatient care are based on information gathered at the time of discharge. We have used the more commonly applied term of admissions, however, for these episodes.

In Australia, most healthcare-associated MRSA is caused by 1 clone defined by multilocus sequence type (ST) 239; this clone is characteristically resistant to multiple antimicrobial agents, including gentamicin (3,12). Most of the remaining healthcare-associated infections are caused by a recently introduced strain, ST22, which is indistinguishable from epidemic MRSA-15 in the United Kingdom. It is invariably resistant to ciprofloxacin (12). Thus, in Australia, MRSA that is acquired in the community and is sensitive to both ciprofloxacin and gentamicin is not likely to be associated with healthcare facility acquisition. We used this pattern as a surrogate marker for community acquisition of MRSA without healthcare-associated risk factors.

Results

We detected 12,771 bloodstream infections in the 17 hospitals participating in this study (12 principal referral metropolitan, 3 large metropolitan, 1 private hospital, and 1 medium-sized public hospital, and 1 private hospital with 2,013,534 total separations; Table 1). There were 3,192 episodes of *S. aureus* bacteremia identified (i.e., 25% of the total true bloodstream infections). The median rate of *S. aureus* bacteremia was higher in the principal referral metropolitan hospitals (1.59/1000 admissions) than in large metropolitan hospitals (1.3) or the private hospital (0.6). The range varied from 0.60 to 3.24 (Table 2). The median rate of community-onset bacteremia episodes was 0.80/1000 admissions (range 0.11–0.99). The median rate of hospital-onset bacteremia was 0.72 episodes/1,000 admissions (range 0.13–1.30). The median rate of hospital-onset MRSA episodes was 0.22/1,000 admissions (range 0–0.89). When expressed as MRSA episodes per 1,000 occupied bed days (OBDs), the rates varied from 0 to 0.30 with a median rate of 0.08. If day-only cases are removed from the denominator then the median rate was 0.10 per 1,000 OBDs (range 0–0.39).

Of these 3,192 SAB episodes, 1,621 (51%) were of hospital onset, and 1,571 (49%) had their onset in the community. Of those with a hospital onset, 40% were MRSA in comparison to 12% with a community onset. Of all MRSA bacteremia episodes, 23% had a community onset, and 77% had hospital onset. Of the 193 community-onset episodes of MRSA that occurred, only 47 (24%) had a sensitivity pattern (sensitive to gentamicin and ciprofloxacin) that suggests that they were community acquired.

When both MRSA and MSSA were considered, data were available for 560 community-onset SAB infections (but only from 4 hospitals). The proportions of these episodes that were noninpatient, healthcare-associated were 35%, 42%, 18% and 16%, respectively (from hospitals A, D, E, and N). In those hospitals, the percentage of *S. aureus* episodes that were healthcare associated overall (i.e., all hospital-onset cases and those community-onset cases associated with healthcare exposure) were 75%, 69%, 64%, and 36%, respectively.

Mortality data were available for 526 patients from 2 hospitals. At hospital E, the mortality rate at day 7 was 10% (27 of 267 patients). When a subgroup of these patients at hospital E (52 patients) was followed for a longer period (2001–2002), the mortality rate was 23% at 30 days and 35% at 6 months. For those 24 patients with a community-onset episode of bacteremia that was not healthcare associated, mortality rates were 6% at day 7, 17% at 1 month, and 21% at 6 months, respectively. At hospital H (259 patients), the mortality rate at 30 days was 19%. At hospital H, the mean length of stay for those with SAB was 25.6 days compared to 6.2 days in matched controls. The mean length of stay was longer for MRSA infections (39.2 days) than for MSSA infections (23.3 days).
The rates of *S. aureus* bacteremia in different hospital populations were used to estimate the incidence for Australia. Using our median bacteremia rate for *S. aureus* bacteremia in different types of public hospitals (1.27/1,000 admissions, range 0.68–3.24) and in private hospitals (0.6/1,000 admissions), we estimated ≈6,900 episodes per year nationally (range 3,826–20,658) or 35/100,000 per year (Tables 3 and 4). Some data are available from other countries for comparison; the lowest annual rates are in Northern Ireland (23/100,000) and the highest in the United States (56/100,000; Table 4). However only 2 countries, Denmark and England, appeared to have comprehensive collection systems, and their rates were 29/100,000 and 37/100,000, respectively (17,20,22).

### Discussion

*S. aureus* bacteremia is very common. Approximately one fourth (26%) of all *S. aureus* bacteremia episodes were caused by MRSA, and, as expected, the onset of most of...
these episodes was in hospitals (77%). Notably, however, 12% of all community-onset S. aureus infections were MRSA, which was 23% of all MRSA bloodstream infection episodes. A recent study from the United States similarly showed that 15% of community-onset SAB episodes were MRSA (14). Most of the community-onset strains in our study were multiresistant or phenotypically consistent with UK EMRSA-15 (15) and thus most likely to have been acquired by patients who had previous hospital contact, with nursing home contact a major factor in at least 1 of the hospitals in this study (hospital G). However, approximately one fourth of these community-onset MRSA infections were caused by other phenotypes of non–multiresistant MRSA and thus more likely to be true community-acquired episodes of MRSA bacteremia. Severe cases of MRSA bacteremia not associated with prior healthcare contact have been reported previously in Australia (7, 9, 16).

Use of the >48 hours postadmission definition of hospital onset underestimates the number of episodes of bacteremia that are healthcare associated. Many patients with chronic conditions are now treated in the community or on a day-only basis. Vascular lines are increasingly used in the community and outpatient settings, providing a potential source of bacteremia. The collection of data on the true association of episodes of bacteremia to healthcare is time-consuming and was not done by most institutions participating in this study. However, 3 principal referral hospitals (hospitals A, D, and E) did collect these data for 971 episodes, and 64%–75% of their total S. aureus bacteremia episodes were healthcare associated. Only 46%–61% of the episodes were acquired while the patient was an inpatient (i.e., >48 h in hospital). This finding means that in these larger hospitals approximately one third of healthcare-associated episodes were acquired by either outpatients or short-stay patients. These episodes are better defined as “nonpatient, healthcare-associated.” In a recent study in the United States, 62% of their community-onset SAB infections were healthcare related (with intravenous [IV] catheters the most common clinically apparent site of infection) (14). On the basis of our data, we conclude that in Australia approximately two thirds of all SAB episodes were associated with healthcare or medical procedures (i.e., all hospital-onset and approximately one third of community-onset episodes). A similar situation is evident in Denmark (17), where in 2002, at least 59% of all S. aureus infections were associated with healthcare procedures. Clearly, substantial scope exists internationally for interventions in healthcare settings to decrease the numbers of these episodes (especially those related to IV catheters). Interventions to reduce S. aureus bacteremia need to target healthcare-associated infections in the broadest sense and include those following non–inpatient-related medical procedures.

Community-onset infections that have no healthcare association are also common and associated with a high death rate (17% and 19% at hospitals E and H at 1 month,
respectively). How best to intervene to decrease these infections is difficult to determine. Vaccination is a possibility for the future; a recent trial of a conjugated capsular polysaccharide vaccine in renal dialysis patients estimated efficacy at ≈60% (18). However, vaccination for the general population is unlikely to be available soon. We should therefore concentrate on reducing the number of deaths from established infections. Because the mortality rate associated with community-acquired bacteremia increases with inadequate empiric therapy (19), all efforts should be made to promote compliance with published guidelines for treatment of severe staphylococcal sepsis, including adequate duration of therapy.

Available data suggest that staphylococcal bacteremia is a major global health problem. The median death rate for MSSA infections is 25%, and for MRSA infections, 34% (20). Thus, >1,700 deaths in Australia are likely associated with *S. aureus* bacteremia per year (assuming 6,900 episodes or a bacteremia rate of 35/100,000/year). This estimate of the rate of SAB is similar to England (20,22) but much lower than in the United States on the basis of the rate derived from the figures available in the only comparative study (55/100,000) (14). Our estimated rate in Australia is higher than that in Denmark (17,21). It is also higher that those reported from Wales (22) and Ireland (23) (Table 4); however, all episodes from these last 2 countries likely were not reported in their voluntary reporting schemes. England changed recently from a similar voluntary reporting scheme to a compulsory scheme, and the numbers of reported episodes increased by almost 50% (24).

The rate of MRSA bacteremia in England was higher per 1,000 OBDs than in our figures from Australia (0.17 compared to 0.10 episodes per 1,000 OBDs, respectively). MRSA was a substantial cause of episodes of SAB in this study (26%). However, this percentage was lower than that seen in most other countries (e.g., Wales, 47%; Table 4) with the notable exception of Denmark (0.6% in 2002) (17).

We may have overestimated the number of cases of bacteremia occurring in Australia because of the overrepresentation of larger hospitals in our survey. However, these hospitals participated because they had in place surveillance systems for measuring all episodes of bacteremia. The rates of SAB may have been relatively lower in these hospitals because they were also more likely than were hospitals without surveillance systems to have infection control programs in place to try to decrease the numbers of these episodes. If systems were in place that better captured and reported on all bacteremia episodes in well-defined populations (e.g., all of Australia or a state), then this would give a more accurate rate. Such systems appear only to be in place in Denmark and England (17,21,24). Currently, no such systems are operating in Australia. Limited data are available from a voluntary surveillance

| Published data for Australia 2001–2002 (13) | Total hospitals† | Acute public‡ | Private | Australia-wide |
|--------------------------------------------|------------------|---------------|---------|----------------|
| No. hospitals                              |                  | 724           | 537§    | 1,306          |
| No. beds                                   |                  | 49,004        | 27,407  | 75,516         |
| Total admissions (x1,000)                  |                  | 3,950         | 2,426   | 6,376          |
| Same day separations (x1,000)              |                  | 1,886         | 1,453   | –              |
| Average length of stay                     |                  | 4.1           | 2.9     | 3.5            |
| *S. aureus* BSI episodes; (calculated rates from data in this study) | | | | |
| *S. aureus* BSI rate/1,000 admissions     |                  | 0.68–3.24     | 0.6     | 0.6–3.24       |
| Estimated episodes/y                       |                  | 2,370–12,798  | 1,456   | 3,826–20,658   |
| Median rate/1,000 admissions               |                  | 1.37          | 0.6     | NA             |
| Estimated episodes/y (based on median)     |                  | 5,412         | 1,456   | 6,867          |
| Hospital-onset MSSA                         |                  |               |         |                |
| Rate/1000 admissions                       |                  | 0.08–0.74     | 0.44    | 0.10–0.97      |
| Estimated episodes/y                       |                  | 316–2,923     | 1,067   | 638–4,718      |
| Median rate/1,000 admissions               |                  | 0.47          | 0.44    | NA             |
| Estimated episodes/y (based on median)     |                  | 1,769         | 1,067   | 2,836          |
| Hospital-onset MRSA                        |                  |               |         |                |
| Rate/1,000 admissions                      |                  | 0.05–0.89     | 0.05    | 0.05–0.89      |
| Estimated episodes/y                       |                  | 198–3,516     | 121     | 255–5,675      |
| Median rate/1,000 admissions               |                  | 0.25          | 0.05    | NA             |
| Estimate episodes/y (based on median)      |                  | 868           | 121     | 1,015          |

*BSI, bloodstream infection; MSSA, methicillin-sensitive *S. aureus*; MRSA, methicillin-resistant *S. aureus*; NA, not applicable.

†For full details for individual hospitals in this study and hospital grouping, see Table 3 at http://www.cdc.gov/ncidod/eid/vol11no04/04-0772.htm#table3
‡Acute public hospitals exclude psychiatric hospitals.
§Of private hospitals, 246 were day only, 314 others had admissions for >24 h.
A number of reports have already highlighted the clinical impact of infection due to these strains (9,28–30). Surveillance data show that their prevalence is increasing in our capital cities, but the situation in rural Australia is not well documented (3). This increase will inevitably affect guidelines for empirical antimicrobial drug prescribing for staphylococcal infections and for patients in the community with suspected SAB. Further surveillance of staphylococcal infections, including bacteremia, is warranted to guide recommendations for empirical therapy and infection control interventions.

Acknowledgments

We greatly appreciate the assistance of the many laboratory staff members at each of the participating hospitals and as well as many infection control practitioners who assisted in the collection of the data.

The Australian Group for Antimicrobial Resistance is currently funded by a grant from the Department of Health and Aging of the Australian Government with funding in the past from Eli Lilly (no funds received for 3 years).

The Australian Group on Antimicrobial Resistance (AGAR) is a group that represents 21 teaching hospital microbiology laboratories and 5 private laboratories. AGAR meets every 6 months. At these meetings, Drs. Gottlieb and Collignon made the initial proposal for this project. All members of AGAR were able to participate in the discussion of the project and

Table 4. International rates and numbers of Staphylococcus aureus bacteremia (SAB)*

| Country | Y       | Population | SAB/y | SAB/10^6/y | % MRSA |
|---------|---------|------------|-------|------------|--------|
| Australia | Present report | 1998–2002 | 19,500,000 | 6,900 | 35 | 27 |
|         | Victoria (25)† | 1990–1999 | 4,502,000 | 804 | 27 | 28 |
| Denmark | Northern Jutland (21) | 1996–1998 | 493,000 | 155 | 31 | ND |
|         | Whole of Denmark (17)‡ | 2002 | 5,350,000 | 1,488 | 28 | 0.6 |
|         | Ireland (23)§ | 1999 | 3,700,000 | ND* | 25 | 36 |
| United Kingdom | England (20,22)¶ | 2002–2003 | 49,200,000 | 18,403 | 37 | 40 |
|         | 2003 | | 19,244 | 39 | 41 |
|         | Northern Ireland (22,24)# | 2002 | 1,697,000 | 397 | 23 | 38 |
|         | 2003 | | 569 | 34 | 44 |
| Wales (22)# | 2003 | 2,920,000 | 742 | 25 | 47 |
| USA | Connecticut (14)** | 1998 | 1,124,337 | 634 | 56 | ND* |

*MRSA, methicillin-resistant Staphylococcus aureus; ND, no data given.
†In Victoria, 8,036 SAB episodes were reported, resulting in a rate of 17.8/100,000. The final rate (27.0) for the entire state was extrapolated from this figure. The Victorian scheme is estimated to capture about two thirds of all bacteremia episodes that occur in that state per year.
‡System in place in Denmark since 1960, with numbers of episodes continually rising (e.g., in 1966, 400 per year and total population 4.8 million or 8/100,000). Collection data based on reviewing all discharge summaries and laboratory samples (15 of 16 counties). Associated 23% mortality rate in 2002, and 22% of these deaths were directly related to sepsis.
§Rates in different regions varied from 8.9 to 37.1 per 100,000. Likely underreporting (22).
¶Compulsory reporting system. Unclear if all community onset episodes were included. In England, underreporting occurred when a voluntary system was in place (only 13,770 episodes reported for 2003; thus, a 50% increase with compulsory system) (22).
#This rate is based on voluntary reporting system. Real rate might be 50% higher (22,24).
**Retrospective case analysis. Rate increased with age, urban areas, and African American ethnicity. 15% of community-onset SAB episodes were MRSA.
suggest modifications of the project design. Only 10 hospital laboratories had collected details on all their *S. aureus* bacteremia data prospectively, and these formed the AGAR participants able to participate in this study. Archie Darbar and Denise Daley were involved in the collection of data at their hospitals. Jan Roberts was involved in the collection of data at her hospital and also in the spreadsheet analysis of the data of all the participating hospitals.

Drs. Collignon, Nimmo, Gottlieb, and Gosbell were involved in the writing of the manuscript. They made substantial contributions to the conception and design of the study, as well as to the acquisition, analysis, and interpretation of data. They also drafted the article and revised it critically for intellectual content. Additionally, all of the other participants in this AGAR project provided comment and feedback on numerous drafts over a 6-month period. All authors have reviewed this version and given final approval for publication.

Dr. Collignon is an infectious diseases physician as well as a pathologist in clinical microbiology. He is a professor at the Canberra Clinical School of the Australian National University. His major research interests include antimicrobial resistance from medical use and in food animals and hospital-acquired infections, particularly bloodstream infections resulting from use of intravascular catheters.

**References**

1. Waldvogel FA. *Staphylococcus aureus* (including staphylococcal toxic shock). In: Mandell GL, Bennett JE, Dolin R, editors. Principles and practice of infectious diseases. Philadelphia: Churchill Livingstone; 2000. p. 2069–100.

2. Cosgrove SE, Sakoulas G, Perencevich EN, Schwaber MJ, Karchmer AW, Carmeli Y. Comparison of mortality associated with methicillin-resistant and methicillin-susceptible *Staphylococcus aureus* bacteremia: a meta-analysis. Clin Infect Dis. 2003;36:53–9.

3. Nimmo GR, Bell JM, Mitchell D, Gosbell IB, Pearman JW, Turnidge JD. Antimicrobial resistance in *Staphylococcus aureus* in Australian teaching hospitals 1989–1999. Microb Drug Resist. 2003;9:155–60.

4. Johnson LB, Almoujahed MO, Ilg K, Maolood L, Khatib R. *Staphylococcus aureus* bacteremia: compliance with standard treatment, long-term outcome and predictors of relapse. Scand J Infect Dis. 2003;35:782–9.

5. Chang FY, Peacock JEJ, Mushier DM, Triplett P, MacDonald BB, Mylotte JM, et al. *Staphylococcus aureus* bacteremia: recurrence and the impact of antibiotic treatment in a prospective multicenter study. Medicine (Baltimore). 2003;82:333–9.

6. Whithy M, McMaws ML, Berry G. Risk of death from methicillin-resistant *Staphylococcus aureus* bacteremia: a meta-analysis. Med J Aust. 2001;175:264–7.

7. Collignon P, Gosbell I, Vickery A, Nimmo G, Stylianopoulos T, Gottlieb T. Community-acquired methicillin-resistant *Staphylococcus aureus* in Australia. Lancet. 1998;352:146–7.

8. Cookson BD. Methicillin-resistant *Staphylococcus aureus* in the community: new battlefronts, or are the battles lost? Infect Control Hosp Epidemiol. 2000;21:398–403.

9. Nimmo GR, Playford EG. Community-acquired MRSA bacteremia: four additional cases including one associated with severe pneumonia. Med J Aust. 2003;178:245.

10. Vandenesch F, Naimi T, Enright MC, Lina G, Nimmo GR, Heffernan H, et al. Community-acquired methicillin-resistant *Staphylococcus aureus* carrying Panton-Valentine leukocidin genes: worldwide emergence. Emerg Infect Dis. 2003;9:978–84.

11. Faria N, Oliveira DC, Westh H, Monnet DL, Larsen AR, Skov R, et al. A new community-acquired methicillin-resistant *Staphylococcus aureus* clone circulating in Denmark. In: 43rd Interscience Conference on Antimicrobial Agents and Chemotherapy, Chicago, Illinois, Sept 14–17, 2003. Washington: American Society for Microbiology; 2003. p.150.

12. Coombs GW, Nimmo GR, Bell JM, Huygens F, O’Brien FG, Malkowski MJ, et al. Genetic diversity among community methicillin-resistant *Staphylococcus aureus* strains causing outpatient infections in Australia. J Clin Microbiol. 2004;42:4735–43.

13. Australian Hospital Statistics 2001–02. In: Health services series. Canberra: Australian Institute of Health and Welfare; 2003 [cited July 26, 2004]. Available from http://www.aihw.gov.au/publications/lhe/ahs01-02/index.html

14. Morin CA, Hadler JL. Population-based incidence and characteristics of community-onset *Staphylococcus aureus* infections with bacteremia in 4 metropolitan Connecticut areas, 1998. J Infect Dis. 2001;184:1029–34.

15. Johnson AP, Aucken HM, Cavendish S, Ganner M, Wale MC, Warner M, et al. Dominance of EMRSA-15 and -16 among MRSA causing nosocomial bacteraemia in the UK: analysis of isolates from the European Antimicrobial Resistance Surveillance System (EARRS). J Antimicrob Chemother. 2001;48:142–4.

16. Collins N, Gosbell IB, Wilson SF. Community-acquired MRSA bacteremia. Med J Aust. 2002;177:55–6.

17. Danish *Staphylococcus aureus* bacteremia group. Annual report on *Staphylococcus aureus* bacteremia in Denmark, 2002. Statens Serum Institut. Published 8/06/2004 [cited July 26, 2004]. Available from http://www.ssi.dk/graphics/dk/overvagning/Annual02.pdf

18. Shinefield H, Black S, Fatton A, Horwith G, Rasgon S, Ordonez J, et al. Use of a *Staphylococcus aureus* conjugate vaccine in patients receiving hemodialysis. N Engl J Med. 2002;346:491–6.

19. Valles J, Rello J, Ochagavia A, Garnacho A, Alcalia MA. Community-acquired bloodstream infection in critically ill adult patients: impact of shock and inappropriate antibiotic therapy on survival. Chest. 2003;123:1615–24.

20. The second year of the Department of Health’s mandatory MRSA bacteremia surveillance scheme in acute Trusts in England: April 2002–March 2003 [cited July 26, 2004]. CDR Weekly. 2003;13:1–9. Available from http://www.hpa.org.uk/cdr/PDFfiles/2003/cdr2503.pdf

21. Schonheyder HC. Two thousand seven hundred and thirty nine episodes of bacteremia in the county of Northern Jutland 1996–1998. Presentation of a regional clinical database. Ugeskr Laeger. 2000;162:2886–91.

22. *Staphylococcus aureus* bacteremia: England, Wales and Northern Ireland, January to December 2003. CDR Wkly. 2004;14:1–5. [cited February 28, 2005]. Available from http://www.hpa.org.uk/cdr/PDFfiles/2004/staph_ann_1604.pdf

23. McDonald P, Mitchell E, Johnson H, Rossney A. Epidemiology of *Staphylococcus aureus* bacteremia surveillance scheme in acute Trusts in England: April 2000–March 2003 [cited July 26, 2004]. CDR Weekly. 2003;13:1–9. Available from http://www.hpa.org.uk/cdr/PDFfiles/2004/staph_ann_1604.pdf

24. McDonald P, Mitchell E, Johnson H, Rossney A. Epidemiology of MRSA: the North/South study of MRSA in Ireland 1999. J Hosp Infect. 2003;54:130–4.

25. Vandenesch F, Naimi T, Enright MC, Lina G, Nimmo GR, Heffernan H, et al. Community-acquired methicillin-resistant *Staphylococcus aureus* carrying Panton-Valentine leukocidin genes: worldwide emergence. Emerg Infect Dis. 2003;9:978–84.
26. An Expert Working Group of the Australian Infection Control Association. National surveillance of healthcare associated infection in Australia. 2001 [cited July 26, 2004]. Available from http://www.health.gov.au/pubhlth/strateg/jetacar/pdf/scope.pdf

27. Okuma K, Iwakawa K, Turnidge JD, Grubb WB, Bell JM, O’Brien FG, et al. Dissemination of new methicillin-resistant Staphylococcus aureus clones in the community. J Clin Microbiol. 2002;40:4289–94.

28. Munckhof WJ, Schooneveldt J, Coombs GW, Hoare J, Nimmo GR. Emergence of community-acquired methicillin-resistant Staphylococcus aureus (MRSA) infection in Queensland, Australia. Int J Infect Dis. 2003;7:259–67.

29. Gosbell IB, Mercer JL, Neville SA, Crone SA, Chant KG, Jalaludin BB, et al. Non-multiresistant and multiresistant methicillin-resistant Staphylococcus aureus in community-acquired infections. Med J Aust. 2001;174:627–30.

30. Nimmo GR, Schooneveldt J, O’Kane G, McCall B, Vickery A. Community acquisition of gentamicin-sensitive MRSA in south-east Queensland. J Clin Microbiol. 2000;38:3926–31.

Address for correspondence: Peter Collignon, Infectious Diseases Unit and Microbiology Department, The Canberra Hospital, PO Box 11, Woden, Australian Capital Territory 2602, Australia; fax: 61-2-6281-0349; email: peter.collignon@act.gov.au