

MMWRTM

*Recommendations
and
Reports*

MORBIDITY AND MORTALITY WEEKLY REPORT

Recommendations for Preventing the Spread of Vancomycin Resistance

Recommendations of the Hospital Infection Control Practices Advisory Committee (HICPAC)

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Centers for Disease Control
and Prevention (CDC)
Atlanta, Georgia 30333



The *MMWR* series of publications is published by the Epidemiology Program Office, Centers for Disease Control and Prevention (CDC), Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA 30333.

SUGGESTED CITATION

Centers for Disease Control and Prevention. Recommendations for preventing the spread of vancomycin resistance: recommendations of the Hospital Infection Control Practices Advisory Committee (HICPAC). *MMWR* 1995;44(No. RR-12): [inclusive page numbers].

Centers for Disease Control and Prevention..... David Satcher, M.D., Ph.D.
Director

The material in this report was prepared for publication by:

National Center for Infectious Diseases..... James M. Hughes, M.D.
Director

Hospital Infections Program..... William J. Martone, M.D.
Director

The production of this report as an *MMWR* serial publication was coordinated in:

Epidemiology Program Office..... Stephen B. Thacker, M.D., M.Sc.
Director

Richard A. Goodman, M.D., M.P.H.
Editor, MMWR Series

Scientific Information and Communications Program

Recommendations and Reports..... Suzanne M. Hewitt, M.P.A.
Managing Editor

Lanette B. Wolcott
Project Editor

Peter M. Jenkins
Visual Information Specialist

Use of trade names and commercial sources is for identification only and does not imply endorsement by the Public Health Service or the U.S. Department of Health and Human Services.

Copies can be purchased from Superintendent of Documents, U.S. Government Printing Office, Washington, DC 20402-9325. Telephone: (202) 783-3238.

Contents

Summary.....	1
Introduction.....	1
Recommendations.....	3
Prudent Vancomycin Use	3
Education Programs	5
Role of the Microbiology Laboratory in the Detection, Reporting, and Control of VRE.....	5
Identification of Enterococci.....	5
Tests for Antimicrobial Susceptibility.....	5
When VRE Are Isolated From a Clinical Specimen	6
Screening Procedures for Detecting VRE in Hospitals Where VRE Have Not Been Detected.....	6
Preventing and Controlling Nosocomial Transmission of VRE	7
Preventing and Controlling VRE Transmission in All Hospitals.....	7
Hospitals With Endemic VRE or Continued VRE Transmission	9
Detecting and Reporting VRSA and VRSE.....	9
References.....	10

**Hospital Infection Control Practices Advisory Committee
Membership List — November 1994**

CHAIRMAN

Walter J. Hierholzer, Jr., M.D.
Yale-New Haven Hospital
New Haven, CT

EXECUTIVE SECRETARY

Julia S. Garner, M.N., R.N.
Centers for Disease Control and
Prevention
Atlanta, GA

MEMBERS

Audrey B. Adams, R.N.
Montefiore Medical Center
Bronx, NY

Donald E. Craven, M.D.
Boston City Hospital
Boston, MA

David W. Fleming, M.D.
Oregon Health Division
Portland, OR

Susan W. Forlenza, M.D.
Nassau County Medical Center
East Meadow, NY

Mary J. Gilchrist, Ph.D.
Veterans Administration Medical
Center
Cincinnati, OH

Donald A. Goldmann, M.D.
Children's Hospital
Boston, MA

Elaine L. Larson, Ph.D.
Georgetown University
Washington, DC

C. Glen Mayhall, M.D.
University of Texas Medical Branch
Galveston, TX

Rita D. McCormick, R.N.
University of Wisconsin Hospital and
Clinics
Madison, WI

Ronald L. Nichols, M.D.
Tulane University School of Medicine
New Orleans, LA

**Subcommittee on Prevention and Control
of Antimicrobial-Resistant Microorganisms in Hospitals**

CHAIRMAN

Donald A. Goldmann, M.D.
Children's Hospital
Boston, MA

MEMBERS

Mary J. Gilchrist, Ph.D.
American Society for Microbiology
Veterans Administration
Medical Center
Cincinnati, OH

Rita D. McCormick, R.N.
University of Wisconsin
Hospital and Clinics
Madison, WI

C. Glen Mayhall, M.D.
University of Texas Medical Branch
Galveston, TX

CONSULTANTS

Charles E. Edmiston, Jr., M.D.
Surgical Infection Society

Barbara J. Russell, R.N.
Association for Professionals in
Infection Control and Epidemiology

Dennis G. Maki, M.D.
Infectious Diseases Society
of America

Robert A. Weinstein, M.D.
American Hospital Association

Gina Pugliese, R.N.
American Hospital Association

These guidelines were prepared for publication by the following CDC staff:

Ofelia C. Tablan, M.D.
Fred C. Tenover, Ph.D.
William J. Martone, M.D.
Robert P. Gaynes, M.D.
William R. Jarvis, M.D.
Martin S. Favero, Ph.D.
J Shaw

*Hospital Infections Program
National Center for Infectious Diseases*

in collaboration with the

Subcommittee on Prevention and Control
of Antimicrobial-Resistant Microorganisms in Hospitals

Recommendations for Preventing the Spread of Vancomycin Resistance

Recommendations of the Hospital Infection Control Practices Advisory Committee (HICPAC)

Summary

Since 1989, a rapid increase in the incidence of infection and colonization with vancomycin-resistant enterococci (VRE) has been reported by U.S. hospitals. This increase poses important problems, including a) the lack of available antimicrobial therapy for VRE infections, because most VRE are also resistant to drugs previously used to treat such infections (e.g., aminoglycosides and ampicillin), and b) the possibility that the vancomycin-resistant genes present in VRE can be transferred to other gram-positive microorganisms (e.g., *Staphylococcus aureus*).

An increased risk for VRE infection and colonization has been associated with previous vancomycin and/or multiantimicrobial therapy, severe underlying disease or immunosuppression, and intraabdominal surgery. Because enterococci can be found in the normal gastrointestinal and female genital tracts, most enterococcal infections have been attributed to endogenous sources within the individual patient. However, recent reports of outbreaks and endemic infections caused by enterococci, including VRE, have indicated that patient-to-patient transmission of the microorganisms can occur either through direct contact or through indirect contact via a) the hands of personnel or b) contaminated patient-care equipment or environmental surfaces.

This report presents recommendations of the Hospital Infection Control Practices Advisory Committee for preventing and controlling the spread of vancomycin resistance, with a special focus on VRE. Preventing and controlling the spread of vancomycin resistance will require coordinated, concerted efforts from all involved hospital departments and can be achieved only if each of the following elements is addressed: a) prudent vancomycin use by clinicians, b) education of hospital staff regarding the problem of vancomycin resistance, c) early detection and prompt reporting of vancomycin resistance in enterococci and other gram-positive microorganisms by the hospital microbiology laboratory, and d) immediate implementation of appropriate infection-control measures to prevent person-to-person transmission of VRE.

INTRODUCTION

From 1989 through 1993, the percentage of nosocomial enterococcal infections reported to CDC's National Nosocomial Infections Surveillance (NNIS) system that were caused by vancomycin-resistant enterococci (VRE) increased from 0.3% to 7.9% (1). This overall increase primarily reflected the 34-fold increase in the percentage of VRE infections in patients in intensive-care units (ICUs) (i.e., from 0.4% to 13.6%), although

a trend toward an increased percentage of VRE infections in non-ICU patients also was noted (1). The occurrence of VRE in NNIS hospitals was associated with larger hospital size (i.e., a hospital with ≥ 200 beds) and university affiliation (1). Other hospitals also have reported increased endemic rates and clusters of VRE infection and colonization (2-8). The actual increase in the incidence of VRE in U.S. hospitals might be greater than reported because the fully automated methods used in many clinical laboratories cannot consistently detect vancomycin resistance, especially moderate vancomycin resistance (as manifested in the VanB phenotype) (9-11).

Vancomycin resistance in enterococci has coincided with the increasing incidence of high-level enterococcal resistance to penicillin and aminoglycosides, thus presenting a challenge for physicians who treat patients who have infections caused by these microorganisms (1,4). Treatment options are often limited to combining antimicrobials or experimental compounds that have unproven efficacy (12-14).

The epidemiology of VRE has not been clarified; however, certain patient populations are at increased risk for VRE infection or colonization. These populations include critically ill patients or those with severe underlying disease or immunosuppression (e.g., patients in ICUs or in oncology or transplant wards); persons who have had an intraabdominal or cardio-thoracic surgical procedure or an indwelling urinary or central venous catheter; and persons who have had a prolonged hospital stay or received multiantimicrobial and/or vancomycin therapy (2-8). Because enterococci are part of the normal flora of the gastrointestinal and female genital tracts, most infections with these microorganisms have been attributed to the patient's endogenous flora (15). However, recent studies have indicated that VRE and other enterococci can be transmitted directly by patient-to-patient contact or indirectly by transient carriage on the hands of personnel (16) or by contaminated environmental surfaces and patient-care equipment (3,8,17).

The potential emergence of vancomycin resistance in clinical isolates of *Staphylococcus aureus* and *Staphylococcus epidermidis* also is a public health concern. The *vanA* gene, which is frequently plasmid-borne and confers high-level resistance to vancomycin, can be transferred in vitro from enterococci to a variety of gram-positive microorganisms (18,19), including *S. aureus* (20). Although vancomycin resistance in clinical strains of *S. epidermidis* or *S. aureus* has not been reported, vancomycin-resistant strains of *Staphylococcus haemolyticus* have been isolated (21,22).

In November 1993 and February 1994, the Subcommittee on the Prevention and Control of Antimicrobial-Resistant Microorganisms in Hospitals of CDC's Hospital Infection Control Practices Advisory Committee (HICPAC) responded to the increase in vancomycin resistance in enterococci by meeting with representatives from the American Hospital Association, the American Society for Microbiology, the Association for Professionals in Infection Control and Epidemiology, the Infectious Diseases Society of America, the Society for Healthcare Epidemiology of America, and the Surgical Infection Society. Meeting participants agreed with the need for prompt implementation of control measures; thus, recommendations to prevent the spread of VRE were developed. Public comments were solicited and incorporated into the draft recommendations. In November 1994, HICPAC ratified the following recommendations for preventing and controlling the spread of vancomycin resistance, with special focus on VRE.

HICPAC recognizes that a) data are limited and additional research will be required to clarify the epidemiology of VRE and determine cost-effective control strategies, and b) many U.S. hospitals have concurrent problems with other antimicrobial-resistant organisms (e.g., methicillin-resistant *S. aureus* [MRSA] and beta-lactam and aminoglycoside-resistant gram-negative bacilli) that might have different epidemiologic features and require different control measures.

RECOMMENDATIONS

Each hospital—through collaboration of its quality-improvement and infection-control programs; pharmacy and therapeutics committee; microbiology laboratory; clinical departments; and nursing, administrative, and housekeeping services—should develop a comprehensive, institution-specific, strategic plan to detect, prevent, and control infection and colonization with VRE. The following elements should be addressed in the plan.

Prudent Vancomycin Use

Vancomycin use has been reported consistently as a risk factor for infection and colonization with VRE (2,4,7,8,17) and may increase the possibility of the emergence of vancomycin-resistant *S. aureus* (VRSA) and/or vancomycin-resistant *S. epidermidis* (VRSE). Therefore, all hospitals and other health-care delivery services, even those at which VRE have never been detected, should a) develop a comprehensive, antimicrobial-utilization plan to provide education for their medical staff (including medical students who rotate their training in different departments of the health-care facility), b) oversee surgical prophylaxis, and c) develop guidelines for the proper use of vancomycin (as applicable to the institution).

Guideline development should be part of the hospital's quality-improvement program and should involve participation from the hospital's pharmacy and therapeutics committee; hospital epidemiologist; and infection-control, infectious-disease, medical, and surgical staffs. The guidelines should include the following considerations:

- Situations in which the use of vancomycin is appropriate or acceptable:
 - For treatment of serious infections caused by beta-lactam-resistant gram-positive microorganisms. Vancomycin may be less rapidly bactericidal than are beta-lactam agents for beta-lactam-susceptible staphylococci (23,24).
 - For treatment of infections caused by gram-positive microorganisms in patients who have serious allergies to beta-lactam antimicrobials.
 - When antibiotic-associated colitis fails to respond to metronidazole therapy or is severe and potentially life-threatening.
 - Prophylaxis, as recommended by the American Heart Association, for endocarditis following certain procedures in patients at high risk for endocarditis (25).
 - Prophylaxis for major surgical procedures involving implantation of prosthetic materials or devices (e.g., cardiac and vascular procedures [26] and total hip replacement) at institutions that have a high rate of infections caused by MRSA or methicillin-resistant *S. epidermidis*. A single dose of vancomycin administered immediately before surgery is sufficient unless the procedure lasts

>6 hours, in which case the dose should be repeated. Prophylaxis should be discontinued after a maximum of two doses (27–30).

- Situations in which the use of vancomycin should be discouraged:
 - Routine surgical prophylaxis other than in a patient who has a life-threatening allergy to beta-lactam antibiotics (28).
 - Empiric antimicrobial therapy for a febrile neutropenic patient, unless initial evidence indicates that the patient has an infection caused by gram-positive microorganisms (e.g., at an inflamed exit site of Hickman catheter) and the prevalence of infections caused by MRSA in the hospital is substantial (31–37).
 - Treatment in response to a single blood culture positive for coagulase-negative staphylococcus, if other blood cultures taken during the same time frame are negative (i.e., if contamination of the blood culture is likely). Because contamination of blood cultures with skin flora (e.g., *S. epidermidis*) could result in inappropriate administration of vancomycin, phlebotomists and other personnel who obtain blood cultures should be trained to minimize microbial contamination of specimens (38–40).
 - Continued empiric use for presumed infections in patients whose cultures are negative for beta-lactam-resistant gram-positive microorganisms (41).
 - Systemic or local (e.g., antibiotic lock) prophylaxis for infection or colonization of indwelling central or peripheral intravascular catheters (42–48).
 - Selective decontamination of the digestive tract.
 - Eradication of MRSA colonization (49,50).
 - Primary treatment of antibiotic-associated colitis (51).
 - Routine prophylaxis for very low-birthweight infants (i.e., infants who weigh <1,500 g [3 lbs 4 oz]) (52).
 - Routine prophylaxis for patients on continuous ambulatory peritoneal dialysis or hemodialysis (48,53).
 - Treatment (chosen for dosing convenience) of infections caused by beta-lactam-sensitive gram-positive microorganisms in patients who have renal failure (54–57).
 - Use of vancomycin solution for topical application or irrigation.
- Enhancing compliance with recommendations:
 - Although several techniques may be useful, further study is required to determine the most effective methods for influencing the prescribing practices of physicians (58–61).
 - Key parameters of vancomycin use can be monitored through the hospital's quality assurance/improvement process or as part of the drug-utilization review of the pharmacy and therapeutics committee and the medical staff.

Education Programs

Continuing education programs for hospital staff (including attending and consulting physicians, medical residents, and students; pharmacy, nursing, and laboratory personnel; and other direct patient-care providers) should include information concerning the epidemiology of VRE and the potential impact of this pathogen on the cost and outcome of patient care. Because detection and containment of VRE require an aggressive approach and high performance standards for hospital personnel, special awareness and educational sessions might be indicated.

Role of the Microbiology Laboratory in the Detection, Reporting, and Control of VRE

The microbiology laboratory is the first line of defense against the spread of VRE in the hospital. The laboratory's ability to promptly and accurately identify enterococci and detect vancomycin resistance is essential for recognizing VRE colonization and infection and avoiding complex, costly containment efforts that are required when recognition of the problem is delayed. In addition, cooperation and communication between the laboratory and the infection-control program will facilitate control efforts.

Identification of Enterococci

Presumptively identify colonies on primary isolation plates as enterococci by using colonial morphology, a Gram stain, and a pyrrolidonyl arylamidase (PYR) test. Although identifying enterococci to the species level can help predict certain resistance patterns (e.g., *Enterococcus faecium* is more resistant to penicillin than is *Enterococcus faecalis*) and may help determine the epidemiologic relatedness of enterococcal isolates, such identification is not routinely necessary if antimicrobial susceptibility testing is performed. However, under special circumstances or as laboratory resources permit, biochemical tests can be used to differentiate between various enterococcal species. Although most commercially available identification systems adequately differentiate *E. faecalis* from other species of enterococci, additional tests for motility and pigment production are required to distinguish *Enterococcus gallinarum* (motile and nonpigmented) and *Enterococcus casseliflavus* (motile and pigmented) from *E. faecium* (nonmotile and nonpigmented).

Tests for Antimicrobial Susceptibility

Determine vancomycin resistance and high-level resistance to penicillin (or ampicillin) and aminoglycosides (62) for enterococci isolated from blood, sterile body sites (with the possible exception of urine), and other sites as clinically indicated. Laboratories routinely may test wound and urine isolates for resistance to vancomycin and penicillin or ampicillin if resources permit (see Screening Procedures for Detecting VRE in Hospitals Where VRE Have Not Been Detected).

- Laboratories that use disk diffusion should incubate plates for 24 hours and read zones of inhibition by using transmitted light (62,63).
- Minimum inhibitory concentrations can be determined by agar dilution, agar gradient dilution, broth macrodilution, or manual broth microdilution (62–64). These test systems should be incubated for 24 hours.

- The fully automated methods of testing enterococci for resistance to vancomycin currently are unreliable (9–11).

When VRE Are Isolated From a Clinical Specimen

Confirm vancomycin resistance by repeating antimicrobial susceptibility testing using any of the recommended methods (see Tests for Antimicrobial Susceptibility), particularly if VRE isolates are unusual in the hospital, OR streak 1 μ L of standard inoculum (0.5 McFarland) from an isolated colony of enterococci onto brain heart infusion agar containing 6 μ g/mL of vancomycin, incubate the inoculated plate for 24 hours at 35 C (95 F), and consider any growth indicative of vancomycin resistance (62,63,65).

Immediately, while performing confirmatory susceptibility tests, notify the patient's primary caregiver, patient-care personnel, and infection-control personnel regarding the presumptive identification of VRE so that appropriate isolation precautions can be initiated promptly (see Preventing and Controlling VRE Transmission in All Hospitals). Follow this preliminary report with the (final) result of the confirmatory test. Additionally, highlight the report regarding the isolate to alert staff that isolation precautions are indicated.

Screening Procedures for Detecting VRE in Hospitals Where VRE Have Not Been Detected

In some hospital microbiology laboratories, antimicrobial susceptibility testing of enterococcal isolates from urine or nonsterile body sites (e.g., wounds) is not performed routinely; thus, identification of nosocomial VRE colonization and infection in hospitalized patients may be delayed. Therefore, in hospitals where VRE have not yet been detected, implementing special measures can promote earlier detection of VRE.

Antimicrobial susceptibility survey. Perform periodic susceptibility testing on an epidemiologic sample of enterococcal isolates recovered from all types of clinical specimens, especially from high-risk patients (e.g., those in an ICU or in an oncology or transplant ward). The optimal frequency of testing and number of isolates to be tested will vary among hospitals, depending on the patient population and number of cultures performed at the hospital. Hospitals that process large numbers of culture specimens need to test only a fraction (e.g., 10%) of enterococcal isolates every 1–2 months, whereas hospitals processing fewer specimens might need to test all enterococcal isolates during the survey period. The hospital epidemiologist can help design a suitable sampling strategy.

Culture survey of stools or rectal swabs. In tertiary medical centers and other hospitals that have many critically ill patients (e.g., ICU, oncology, and transplant patients) at high risk for VRE infection or colonization, periodic culture surveys of stools or rectal swabs of such patients can detect the presence of VRE. Because most patients colonized with VRE have intestinal colonization with this organism, fecal screening of patients is recommended even though VRE infections have not been identified clinically (2,4,16).

The frequency and intensity of surveillance should be based on the size of the population at risk and the specific hospital unit(s) involved. If VRE have been detected in other health-care facilities in a hospital's area and/or if a hospital's staff decides to

determine whether VRE are present in the hospital despite the absence of recognized clinical cases, stool or rectal-swab culture surveys are useful. The cost of screening can be reduced by inoculating specimens onto selective media containing vancomycin (2,17,66) and restricting screening to those patients who have been in the hospital long enough to have a substantial risk for colonization (e.g., 5–7 days) or who have been admitted from a facility (e.g., a tertiary-care hospital or a chronic-care facility) where VRE have been identified.

After colonization with VRE has been detected, all the enterococcal isolates (including those from urine and wounds) from patients in the hospital should be screened routinely for vancomycin resistance, and efforts to contain the spread of VRE should be intensified (i.e., by strict adherence to handwashing and compliance with isolation precautions) (see Preventing and Controlling VRE Transmission in All Hospitals). Intensified fecal screening for VRE might facilitate earlier identification of colonized patients, leading to more efficient containment of the microorganism.

Preventing and Controlling Nosocomial Transmission of VRE

Eradicating VRE from hospitals is most likely to succeed when VRE infection or colonization is confined to a few patients on a single ward. After VRE have become endemic on a ward or have spread to multiple wards or to the community, eradication becomes difficult and costly. Aggressive infection-control measures and strict compliance by hospital personnel are required to limit nosocomial spread of VRE.

Control of VRE requires a collaborative, institution-wide, multidisciplinary effort. Therefore, the hospital's quality-assurance/improvement department should be involved at the outset to identify specific problems in hospital operations and patient-care systems and to design, implement, and evaluate appropriate changes in these systems.

Preventing and Controlling VRE Transmission in All Hospitals

The following measures should be implemented by all hospitals, including those in which VRE have been isolated infrequently or not at all, to prevent and control transmission of VRE.

- Notify appropriate hospital staff promptly when VRE are detected (see When VRE Are Isolated From a Clinical Specimen).
- Inform clinical staff of the hospital's policies regarding VRE-infected or colonized patients. Because the slightest delay can lead to further spread of VRE and complicate control efforts, implement the required procedures as soon as VRE are detected. Clinical staff are essential to limiting the spread of VRE in patient-care areas; thus, continuing education regarding the appropriate response to the detection of VRE is critical (see Education Programs).
- Establish system(s) for monitoring appropriate process and outcome measures (e.g., cumulative incidence or incidence density of VRE colonization, rate of compliance with VRE isolation precautions and handwashing, interval between VRE identification in the laboratory and implementation of isolation precautions on the wards, and the percentage of previously colonized patients admitted to the ward who are identified promptly and placed on isolation precautions). Relay these data

to the clinical, administrative, laboratory, and support staff to reinforce ongoing education and control efforts (67).

- Initiate the following isolation precautions to prevent patient-to-patient transmission of VRE:
 - Place VRE-infected or colonized patients in private rooms or in the same room as other patients who have VRE (8).
 - Wear gloves (clean, nonsterile gloves are adequate) when entering the room of a VRE-infected or colonized patient because VRE can extensively contaminate such an environment (3,8,16,17). When caring for a patient, a change of gloves might be necessary after contact with material that could contain high concentrations of VRE (e.g., stool).
 - Wear a gown (a clean, nonsterile gown is adequate) when entering the room of a VRE-infected or colonized patient a) if substantial contact with the patient or with environmental surfaces in the patient's room is anticipated, b) if the patient is incontinent, or c) if the patient has had an ileostomy or colostomy, has diarrhea, or has a wound drainage not contained by a dressing (8).
 - Remove gloves and gown before leaving the patient's room and immediately wash hands with an antiseptic soap or a waterless antiseptic agent (68–71). Hands can be contaminated via glove leaks (72–76) or during glove removal, and bland soap does not always completely remove VRE from the hands (77).
 - Ensure that after glove and gown removal and handwashing, clothing and hands do not contact environmental surfaces in the patient's room that are potentially contaminated with VRE (e.g., a door knob or curtain) (3,8).
- Dedicate the use of noncritical items (e.g., a stethoscope, sphygmomanometer, or rectal thermometer) to a single patient or cohort of patients infected or colonized with VRE (17). If such devices are to be used on other patients, adequately clean and disinfect these devices first (78).
- Obtain a stool culture or rectal swab from roommates of patients newly found to be infected or colonized with VRE to determine their colonization status, and apply isolation precautions as necessary. Perform additional screening of patients on the ward at the discretion of the infection-control staff.
- Adopt a policy for deciding when patients infected or colonized with VRE can be removed from isolation precautions. The optimal requirements remain unknown; however, because VRE colonization can persist indefinitely (4), stringent criteria might be appropriate, such as VRE-negative results on at least three consecutive occasions (≥ 1 week apart) for all cultures from multiple body sites (including stool or rectal swab, perineal area, axilla or umbilicus, and wound, Foley catheter, and/or colostomy sites, if present).
- Because patients with VRE can remain colonized for long periods after discharge from the hospital, establish a system for highlighting the records of infected or colonized patients so they can be promptly identified and placed on isolation precautions upon readmission to the hospital. This information should be computerized so that placement of colonized patients on isolation precautions will not be delayed because the patients' medical records are unavailable.

- Local and state health departments should be consulted when developing a plan regarding the discharge of VRE-infected or colonized patients to nursing homes, other hospitals, or home-health care. This plan should be part of a larger strategy for handling patients who have resolving infections and patients colonized with antimicrobial-resistant microorganisms.

Hospitals With Endemic VRE or Continued VRE Transmission

The following measures should be taken to prevent and control transmission of VRE in hospitals that have endemic VRE or continued VRE transmission despite implementation of measures described in the preceding section (see Preventing and Controlling VRE Transmission in All Hospitals).

- Focus control efforts initially on ICUs and other areas where the VRE transmission rate is highest (4). Such areas can serve as reservoirs for VRE, allowing VRE to spread to other wards when patients are well enough to be transferred.
- Where feasible, cohort the staff who provide regular, ongoing care to patients to minimize the movement/contact of health-care providers between VRE-positive and VRE-negative patients (4,8).
- Hospital staff who are carriers of enterococci have been implicated rarely in the transmission of this organism (8). However, in conjunction with careful epidemiologic studies and upon the direction of the infection-control staff, examine personnel for chronic skin and nail problems and perform hand and rectal swab cultures of these workers. Remove from the care of VRE-negative patients those VRE-positive personnel linked epidemiologically to VRE transmission until their carrier state has been eradicated.
- Because the results of several enterococcal outbreak investigations suggest a potential role for the environment in the transmission of enterococci (3,8,16,17,79,80), institutions experiencing ongoing VRE transmission should verify that the hospital has adequate procedures for the routine care, cleaning, and disinfection of environmental surfaces (e.g., bed rails, bedside commodes, carts, charts, doorknobs, and faucet handles) and that these procedures are being followed by housekeeping personnel. To verify the efficacy of hospital policies and procedures, some hospitals might elect to perform focused environmental cultures before and after cleaning rooms that house patients who have VRE. All environmental culturing should be approved and supervised by the infection-control program in collaboration with the clinical laboratory (3,8,16,17,79,80).
- Consider sending representative VRE isolates to reference laboratories for strain typing by pulsed field gel electrophoresis or other suitable techniques to aid in defining reservoirs and patterns of transmission.

Detecting and Reporting VRSA and VRSE

The microbiology laboratory has the primary responsibility for detecting and reporting the occurrence of VRSA or VRSE in the hospital. All clinical isolates of *S. aureus* and *S. epidermidis* should be tested routinely, using standard methods, for susceptibility to vancomycin (62). If VRSA or VRSE is identified in a clinical specimen, confirm vancomycin resistance by repeating antimicrobial susceptibility testing using

standard methods (62). Restreak the colony to ensure that the culture is pure. The most common causes of false-positive VRSA reports are susceptibility testing on mixed cultures and misidentifying VRE, *Leuconostoc*, *S. haemolyticus*, or *Pediococcus* as VRSA (81,82).

Immediately (i.e., while performing confirmatory testing) notify the hospital's infection-control personnel, the patient's primary caregiver, and patient-care personnel on the ward on which the patient is hospitalized so that the patient can be placed promptly on isolation precautions (depending on the site[s] of infection or colonization) adapted from previous CDC guidelines (83) and those recommended for VRE infection or colonization in this report (see Preventing and Controlling Nosocomial Transmission of VRE). Furthermore, immediately notify the state health department and CDC, and send the isolate through the state health department to CDC (telephone [404] 639-6413) for confirmation of vancomycin resistance.

References

1. CDC. Nosocomial enterococci resistant to vancomycin—United States, 1989–1993. MMWR 1993;42:597–9.
2. Rubin LG, Tucci V, Cercenado E, Eliopoulos G, Isenberg HD. Vancomycin-resistant *Enterococcus faecium* in hospitalized children. Infect Control Hosp Epidemiol 1992;13:700–5.
3. Karanfil LV, Murphy M, Josephson A, et al. A cluster of vancomycin-resistant *Enterococcus faecium* in an intensive care unit. Infect Control Hosp Epidemiol 1992;13:195–200.
4. Handwerger S, Raucher B, Altarac D, et al. Nosocomial outbreak due to *Enterococcus faecium* highly resistant to vancomycin, penicillin, and gentamicin. Clin Infect Dis 1993;16:750–5.
5. Frieden TR, Munsiff SS, Low DE, et al. Emergence of vancomycin-resistant enterococci in New York City. Lancet 1993;342:76–9.
6. Boyle JF, Soumakis SA, Rendo A, et al. Epidemiologic analysis and genotypic characterization of a nosocomial outbreak of vancomycin-resistant enterococci. J Clin Microbiol 1993;31:1280–5.
7. Montecalvo MA, Horowitz H, Gedris C, et al. Outbreak of vancomycin-, ampicillin-, and aminoglycoside-resistant *Enterococcus faecium* bacteremia in an adult oncology unit. Antimicrob Agents Chemother 1994;38:1363–7.
8. Boyce JM, Opal SM, Chow JW, et al. Outbreak of multi-drug resistant *Enterococcus faecium* with transferable vanB class vancomycin resistance. J Clin Microbiol 1994;32:1148–53.
9. Tenover FC, Tokars J, Swenson J, Paul S, Spitalny K, Jarvis W. Ability of clinical laboratories to detect antimicrobial agent-resistant enterococci. J Clin Microbiol 1993;31:1695–9.
10. Sahm DF, Olsen L. In vitro detection of enterococcal vancomycin resistance. Antimicrob Agents Chemother 1990;34:1846–8.
11. Zabransky RJ, Dinuzzo AR, Huber MB, Woods GL. Detection of vancomycin resistance in enterococci by the Vitek AMS System. Diagn Microbiol Infect Dis 1994;20:113–6.
12. Moellering RC Jr. The Garrod lecture: the enterococcus—a classic example of the impact of antimicrobial resistance on therapeutic options. J Antimicrob Chemother 1991;28:1–12.
13. Hayden MK, Koenig GI, Trenholme GM. Bactericidal activities of antibiotics against vancomycin-resistant *Enterococcus faecium* blood isolates and synergistic activities of combinations. Antimicrob Agents Chemother 1994;38:1225–9.
14. Mobarakai N, Landman D, Quale JM. In-vitro activity of trospectomycin, a new aminocyclitol antibiotic against multidrug-resistant *Enterococcus faecium*. J Antimicrob Chemother 1994;33:319–21.
15. Murray BE. The life and times of the enterococcus. Clin Microbiol Rev 1990;3:46–65.
16. Rhinehart E, Smith N, Wennersten C, et al. Rapid dissemination of beta-lactamase-producing aminoglycoside-resistant *Enterococcus faecalis* among patients and staff on an infant and toddler surgical ward. N Engl J Med 1990;323:1814–8.
17. Livornese LL Jr, Dias S, Samel C, et al. Hospital-acquired infection with vancomycin-resistant *Enterococcus faecium* transmitted by electronic thermometers. Ann Intern Med 1992;117:112–6.

18. Uttley AH, George RC, Naidoo J, et al. High-level vancomycin-resistant enterococci causing hospital infections. *Epidemiol Infect* 1989;103:173–81.
19. Leclercq R, Derlot E, Weber M, Duval J, Courvalin P. Transferable vancomycin and teicoplanin resistance in *Enterococcus faecium*. *Antimicrob Agents Chemother* 1989;33:10–5.
20. Noble WC, Virani Z, Cree R. Co-transfer of vancomycin and other resistance genes from *Enterococcus faecalis* NCTC12201 to *Staphylococcus aureus*. *FEMS Microbiol Lett* 1992;72:195–8.
21. Veach LA, Pfaller MA, Barrett M, Koontz FP, Wenzel RP. Vancomycin resistance in *Staphylococcus haemolyticus* causing colonization and bloodstream infection. *J Clin Microbiol* 1990;28:2064–8.
22. Degener JE, Heck MEOC, Vanleeuwen WJ, et al. Nosocomial infection by *Staphylococcus haemolyticus* and typing methods for epidemiological study. *J Clin Microbiol* 1994;32:2260–5.
23. Small PM, Chambers HF. Vancomycin for *Staphylococcus aureus* endocarditis in intravenous drug users. *Antimicrob Agents Chemother* 1990;34:1227–31.
24. Cantoni L, Glauser MP, Bille J. Comparative efficacy of daptomycin, vancomycin, and cloxacillin for the treatment of *Staphylococcus aureus* endocarditis in rats and role of test conditions in this determination. *Antimicrob Agents Chemother* 1990;34:2348–53.
25. American Heart Association Committee on Rheumatic Fever and Infective Endocarditis. Prevention of bacterial endocarditis. *Circulation* 1984;70:1123–4.
26. Maki DG, Bohn MJ, Stolz SM, Kroncke GM, Acher CW, Myerowitz PD. Comparative study of cefazolin, cefamandole, and vancomycin for surgical prophylaxis in cardiac and vascular operations: a double-blind randomized trial. *J Thorac Cardiovasc Surg* 1992;104:1423–34.
27. Classen DC, Evans RS, Pestotnik SL, Horn SD, Menlove RL, Burke JP. The timing of prophylactic administration of antibiotics and the risk of surgical-wound infection. *N Engl J Med* 1992;326:281–6.
28. Conte JE Jr, Cohen SN, Roe BB, Elashoff RM. Antibiotic prophylaxis and cardiac surgery: a prospective double-blind comparison of single-dose versus multiple-dose regimens. *Ann Intern Med* 1972;76:943–9.
29. DiPiro JT, Cheung RP, Bowden TA Jr, Mansberger JA. Single-dose systemic antibiotic prophylaxis of surgical wound infections. *Am J Surg* 1986;152:552–9.
30. Heydemann JS, Nelson CL. Short-term preventive antibiotics. *Clin Orthop* 1986;205:184–7.
31. Rubin M, Hathorn JW, Marshall D, Gress J, Steinberg SM, Pizzo PA. Gram-positive infections and the use of vancomycin in 550 episodes of fever and neutropenia. *Ann Intern Med* 1988;108:30–5.
32. Shenep JL, Hughes WT, Roberson PK, et al. Vancomycin, ticarcillin, and amikacin compared with ticarcillin-clavulanate and amikacin in the empirical treatment of febrile neutropenic children with cancer. *N Engl J Med* 1988;319:1053–8.
33. Pizzo PA, Hathorn JW, Hiemenz J, et al. A randomized trial comparing ceftazidime alone with combination antibiotic therapy in cancer patients with fever and neutropenia. *N Engl J Med* 1986;315:552–8.
34. Karp JE, Dick JD, Angelopoulos C, et al. Empiric use of vancomycin during prolonged treatment-induced granulocytopenia: randomized, double-blind, placebo-controlled clinical trial in patients with acute leukemia. *Am J Med* 1986;81:237–42.
35. European Organization for Research and Treatment of Cancer (EORTC) International Antimicrobial Therapy Cooperative Group, National Cancer Institute of Canada Clinical Trials Group. Vancomycin added to empirical combination antibiotic therapy for fever in granulocytopenic cancer patients. *J Infect Dis* 1991;163:951–8.
36. Riikonen P. Imipenem compared with ceftazidime plus vancomycin as initial therapy for fever in neutropenic children with cancer. *Pediatr Infect Dis* 1991;10:918–23.
37. Lamy T, Michelet C, Dauriac C, Grulois I, Donio PY, Le Prise PY. Benefit of prophylaxis by intravenous systemic vancomycin in granulocytopenic patients: a prospective, randomized trial among 59 patients. *Acta Haematol* 1993;90:109–13.
38. Isaacman DJ, Karasic RB. Lack of effect of changing needles on contamination of blood cultures. *Pediatr Infect Dis J* 1990;9:274–8.
39. Krumholz HM, Cummings S, York M. Blood culture phlebotomy: switching needles does not prevent contamination. *Ann Intern Med* 1990;113:290–2.
40. Strand CL, Wajsbort RR, Sturmman K. Effect of iodophor vs iodine tincture skin preparation on blood culture contamination rate. *JAMA* 1993;269:1004–6.

41. Maki DG, Schuna AA. A study of antimicrobial misuse in a university hospital. *Am J Med Sci* 1978;275:271-82.
42. Ranson MR, Oppenheim BA, Jackson A, Kamthan AG, Scarffe JH. Double-blind placebo controlled study of vancomycin prophylaxis for central venous catheter insertion in cancer patients. *J Hosp Infect* 1990;15:95-102.
43. Henrickson KJ, Powell KR, Schwartz CL. A dilute solution of vancomycin and heparin retains antibacterial and anticoagulant activities. *J Infect Dis* 1988;157:600-1.
44. Schwartz C, Henrickson KJ, Roghmann K, Powell K. Prevention of bacteremia attributed to luminal colonization of tunneled central venous catheters with vancomycin-susceptible organism. *J Clin Oncol* 1990;8:1591-7.
45. Henrickson KJ, Dunne WM Jr. Modification of central venous catheter flush solution improves in vitro antimicrobial activity. *J Infect Dis* 1992;166:944-6.
46. Gaillard JL, Merlino R, Pajot N, et al. Conventional and nonconventional modes of vancomycin administration to decontaminate the internal surface of catheters colonized with coagulase-negative staphylococci. *J Parenter Enter Nutr* 1990;14:593-7.
47. Spafford PS, Sinkin RA, Cox C, Reubens L, Powell KR. Prevention of central venous catheter-related coagulase-negative staphylococcal sepsis in neonates. *J Pediatr* 1994;125:259-63.
48. Kaplan AH, Gilligan PH, Facklam RR. Recovery of resistant enterococci during vancomycin prophylaxis. *J Clin Microbiol* 1988;26:1216-8.
49. Graddon JD, Wu EH, Lutwick LI. Aerosolized vancomycin therapy facilitating nursing home placement. *Ann Pharmacother* 1992;26:209-10.
50. Weathers L, Riggs D, Santeiro M, Weibley RE. Aerosolized vancomycin for treatment of airway colonization by methicillin-resistant *Staphylococcus aureus*. *Pediatr Infect Dis* 1990;9:220-1.
51. Johnson S, Homann SR, Bettin KM, et al. Treatment of asymptomatic *Clostridium difficile* carriers (fecal excretors) with vancomycin or metronidazole. *Ann Intern Med* 1992;117:297-302.
52. Kacica MS, Horgan MJ, Ochoa L, Sandler R, Lepow ML, Venezia RA. Prevention of gram-positive sepsis in neonates weighing less than 1500 grams. *J Pediatr* 1994;125:253-8.
53. Lam TY, Vas SI, Oreopoulos DG. Long-term intraperitoneal vancomycin in the prevention of recurrent peritonitis during CAPD: preliminary results. *Perit Dial Int* 1991;11:281-2.
54. Bastani B, Freer K, Read D, et al. Treatment of gram-positive peritonitis with two intraperitoneal doses of vancomycin in continuous ambulatory peritoneal dialysis patients. *Nephron* 1987;45:283-5.
55. Newman LN, Tessman M, Hanslik T, Schulak J, Mayes J, Friedlander M. A retrospective view of factors that affect catheter healing: four years of experience. *Adv Perit Dial* 1993;9:217-22.
56. Capdevila JA, Segarra A, Planes AM, et al. Successful treatment of haemodialysis catheter-related sepsis without catheter removal. *Nephrol Dial Transplant* 1993;8:231-4.
57. Edell LS, Westby GR, Gould SR. An improved method of vancomycin administration to dialysis patients. *Clin Nephrol* 1988;29:86-7.
58. Soumerai SB, McLaughlin TJ, Avorn J. Quality assurance for drug prescribing. *Qual Assur Health Care* 1990;2:37-58.
59. Everitt DE, Soumerai SB, Avorn J, Klapholz H, Wessels M. Changing surgical antimicrobial prophylaxis practices through education targeted at senior department leaders. *Infect Control Hosp Epidemiol* 1990;11:578-83.
60. Soumerai SB, Avorn J, Taylor WC, Wessels M, Maher D, Hawley SL. Improving choice of prescribed antibiotics through concurrent reminders in an educational order form. *Med Care* 1993;31:552-8.
61. Soumerai SB, McLaughlin TJ, Avorn J. Improving drug prescribing in primary care: a critical analysis of the experimental literature. *Milbank Q* 1989;67:268-317.
62. National Committee for Clinical Laboratory Standards. Methods for dilution antimicrobial susceptibility tests for bacteria that grow aerobically. 3rd ed. Villanova, PA: National Committee for Clinical Laboratory Standards, 1993; publication M7-A3.
63. Swenson JM, Ferraro MJ, Sahn DF, Charache P, Tenover FC, National Committee for Clinical Laboratory Standards Working Group on Enterococci. New vancomycin disk diffusion breakpoints for enterococci. *J Clin Microbiol* 1992;30:2525-8.
64. CDC. Recommendations for prevention of HIV transmission in health-care settings. *MMWR* 1987;36(No. 2S).

65. Swenson JM, Clark NC, Ferraro MJ, et al. Development of a standardized screening method for detection of vancomycin-resistant *Enterococci*. *J Clin Microbiol* 1994;32:1700-4.
66. Edberg SC, Hardalo CJ, Kontrick C, Campbell S. Rapid detection of vancomycin-resistant enterococci. *J Clin Microbiol* 1994;32:2182-4.
67. Nettleman MD, Trilla A, Fredrickson M, Pfaller M. Assigning responsibility: using feedback to achieve sustained control of methicillin-resistant *Staphylococcus aureus*. *Am J Med* 1991;91(suppl 3B):228S-232S.
68. Doebbeling BN, Stanley GL, Sheetz CT, et al. Comparative efficacy of alternative hand-washing agents in reducing nosocomial infections in intensive care units. *N Engl J Med* 1992;327:88-93.
69. Jones MV, Rowe GB, Jackson B, Pritchard NJ. The use of alcohol paper wipes for routine hand cleansing: results of trials in two hospitals. *J Hosp Infect* 1986;8:268-74.
70. Nicoletti G, Boghossian V, Borland R. Hygienic hand disinfection: a comparative study with chlorhexidine detergents and soap. *J Hosp Infect* 1990;15:323-37.
71. Butz AM, Laughon BE, Gullette DL, Larson EL. Alcohol-impregnated wipes as an alternative in hand hygiene. *Am J Infect Control* 1990;18:70-6.
72. Korniewicz DM, Laughon BE, Butz A, Larson E. Integrity of vinyl and latex procedure gloves. *Nurs Res* 1989;38:144-6.
73. Korniewicz DM, Kirwin M, Cresci K, Markut C, Larson E. In-use comparison of latex gloves in two high-risk units: surgical intensive care and acquired immunodeficiency syndrome. *Heart Lung* 1992;21:81-4.
74. DeGroot-Kosolcharoen J, Jones JM. Permeability of latex and vinyl gloves to water and blood. *Am J Infect Control* 1989;17:196-201.
75. Paulssen J, Eidem T, Kristiansen R. Perforations in surgeons' gloves. *J Hosp Infect* 1988;11:82-5.
76. Korniewicz DM, Laughon BE, Cyr WH, Lytle CD, Larson E. Leakage of virus through used vinyl and latex examination gloves. *J Clin Microbiol* 1990;28:787-8.
77. Wade JJ, Desai N, Casewell MW. Hygienic hand disinfection for the removal of epidemic vancomycin-resistant *Enterococcus faecium* and gentamicin-resistant *Enterobacter cloacae*. *J Hosp Infect* 1991;18:211-8.
78. Favero MS, Bond WW. Sterilization, disinfection, and antisepsis in the hospital. Chapter 24. In: Balows A, Hausler WJ Jr, Herrman KL, Isenberg HD, Shadomy HJ, eds. *Manual of clinical microbiology*. 5th ed. Washington, DC: American Society for Microbiology, 1991:183-200.
79. Zervos MJ, Kauffman CA, Therasse PM, Bregman AG, Mikesell TS, Schaberg DR. Nosocomial infection by gentamicin-resistant *Streptococcus faecalis*: an epidemiologic study. *Ann Intern Med* 1987;106:687-91.
80. Wells VD, Wong ES, Murray BE, Coudron PE, Williams DS, Markowitz SM. Infections due to beta-lactamase-producing, high-level gentamicin-resistant *Enterococcus faecalis*. *Ann Intern Med* 1992;116:285-92.
81. Orberg PK, Sandine WE. Common occurrence of plasmid DNA and vancomycin resistance in *Leuconostoc* spp. *Appl Environ Microbiol* 1984;48:1129-33.
82. Schwalbe RS, Ritz WJ, Verma PR, Barranco EA, Gilligan PH. Selection for vancomycin resistance in clinical isolates of *Staphylococcus haemolyticus*. *J Infect Dis* 1990;161:45-51.
83. Garner JS, Simmons BP. Guideline for isolation precautions in hospitals. *Infect Control* 1983;4(suppl):245-325.

MMWR

The *Morbidity and Mortality Weekly Report (MMWR)* Series is prepared by the Centers for Disease Control and Prevention (CDC) and is available free of charge in electronic format and on a paid subscription basis for paper copy. To receive an electronic copy on Friday of each week, send an e-mail message to lists@list.cdc.gov. The body content should read *subscribe mmwr-toc*. Electronic copy also is available from CDC's World-Wide Web server at <http://www.cdc.gov/> or from CDC's file transfer protocol server at <ftp.cdc.gov>. To subscribe for paper copy, contact Superintendent of Documents, U.S. Government Printing Office, Washington, DC 20402; telephone (202) 512-1800.

Data in the weekly *MMWR* are provisional, based on weekly reports to CDC by state health departments. The reporting week concludes at close of business on Friday; compiled data on a national basis are officially released to the public on the following Friday. Address inquiries about the *MMWR* Series, including material to be considered for publication, to: Editor, *MMWR* Series, Mailstop C-08, CDC, 1600 Clifton Rd., N.E., Atlanta, GA 30333; telephone (404) 332-4555.

All material in the *MMWR* Series is in the public domain and may be used and reprinted without permission; citation as to source, however, is appreciated.