Infections and the Compromised Immune Status in the Chronically Critically Ill Patient: Prevention Strategies

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An estimated 2–3% of all hospitalized patients become critically ill. These patients are in a state of relative immune exhaustion, which cripples their response to infections. Patients are sicker, have many comorbidities, and undergo complex procedures. This clinical picture, combined with increasing technologies and improved survival, presents unique challenges and demands a high level of services and expertise over a prolonged period of time. Long-term acute care hospitals provide these services, and the migration of chronically critically ill patients to these institutions facilitates defining (and quantifying) the spectrum of disease and how to best manage them. The prevalence of multidrug-resistant organism colonization and infection upon arrival to long-term acute care hospitals is high. Admission screening, and appropriate isolation and infection control practices can prevent transmission of these organisms. The implementation of ventilator-associated pneumonia prevention protocols, blood stream infection prevention protocols, and minimizing Foley urinary catheter use can decrease hospital-acquired infection rates and keep them low. In addition, specific attention is required to environmental services and surface and equipment cleaning. A well organized infection control program and an antimicrobial stewardship program have become indispensable to achieve these goals. All of these key principles and recommendations are also relevant to the chronically ill patient in acute care hospital ICUs and step-down units. *Key words: multidrug-resistant organisms; prevalence; transmissions; infection rates; infection control.* [Respir Care 2012; 57(6):979–990. © 2012 Daedalus Enterprises]

Introduction

Our healthcare system is complex, and determining the ideal venue to provide care for a specific patient can be challenging. Most patients receive care at out-patient facilities. If a patient becomes acutely ill or decides to have an elective procedure, the patient is admitted to an acute care hospital, where the average stay is less than 6 days. In these settings, diagnoses are made and acute illnesses are treated; payments are based on diagnosis related groups (DRGs). Most patients are discharged home, with or without home healthcare. Some patients return to nursing homes, skilled nursing facilities, or acute in-patient rehabilitation units. However, older and sicker patients are receiving more aggressive care and more complex surgeries and procedures, and new technologies are implemented. Consequently, 3–4% of these patients require extended stays in critical care units.[1] These critical care unit survivors consume a large volume of financial resources, and require a team of specialist healthcare workers and the expertise of many ancillary services.

Long-term acute care (LTAC) hospitals specialize in providing care for these chronically critically ill (CCI) patients. LTAC hospitals are certified for Medicare payments by the Centers for Medicare and Medicaid Services as hospitals with an average stay of $>$ 25 days. Patients transferred to these facilities have multiple medical conditions requiring skilled, complex medical care that cannot be managed under a lower level of care; they may require an ICU. They have more severe illnesses, multisystem complications (such as ventilator dependence and complex wounds), and may still be suffering the consequences of sepsis or require total parenteral nutrition (or further nutritional support).

By focusing on LTAC hospitals, we have a window into the challenges and opportunities in servicing these patients. Infectious complications contribute largely to their morbidity and mortality. An LTAC hospital is only one of the settings where CCI patients receive care. Other settings include acute care hospital ICUs and step-down units. Independent of the site of care, CCI patients share the same risks, and benefit from the same strategies of good infection control practices and prevention protocols.

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Review of the Literature

Risk for Infection

CCI patients are particularly susceptible to infections, for many reasons. First, patients have multiple indwelling devices, such as intravenous lines, urinary catheters, nasogastric tubes, and tracheostomies, as well as other portals of infection, like skin and mucosal breakdown. Second, they are at risk for acquiring virulent nosocomial organisms because they are cared for in an environment where multidrug-resistant organisms thrive. Finally, these patients suffer from an immunologically deficient state commonly referred to as "immune exhaustion," in which diminished physiological reserves impair the patient's ability to fight infections.[2]

Immune Status

The specific immune deficit occurring during CCI is not completely understood. Following the acute or initial hyper-inflammatory response to sepsis, an immune system down-regulation can lead to prolonged immune dysfunction.[3,4] This period of "immune paralysis" has consequences: it limits the ability to fight infections and predisposes the patient to nosocomial infections and multi-organ dysfunction. Patients who survive this initial systemic inflammatory response syndrome enter a state of immune suppression and dysfunction.[2] This immune dysfunction includes loss of delayed hypersensitivity, inability to clear infections, impaired neutrophil function,[5] lymphocyte and dendritic cell apoptosis, an increase in T-regulatory cells (CD4+CD25), release of anti-inflammatory mediators,[6] lymphocyte anergy, and monocyte deactivation.

Additionally, these patients frequently have comorbidities that precede the acute event. Their defenses are already impaired at the beginning of the ICU admission by preexisting illnesses. The list includes: diabetes mellitus, renal insufficiency or failure, prior organ transplantation, chemotherapy, advanced age (with associated immune senescence),[7,8] and COPD (associated impaired humoral and cellular immune response).[9] Chronic renal failure is associated with defects in cellular immunity, neutrophil function, and complement activation.[10] Hyperglycemia may be related to prior diabetes or total parenteral nutrition, but may also be caused by insulin resistance related to ongoing sympathetic-like state associated with the acute illness.[11] Hyperglycemia also increases the already elevated risk of infection of CCI patients, as evidenced by the abnormal leukocyte function that both insulin-dependent and non-insulin-dependent diabetics experience during critical illness. Malnutrition further affects immunodeficiency by suppressing T cell production and function (eg, reducing intracellular killing)[9]; protein deficiency impairs antibody

production.[11] Patients requiring total parenteral nutrition have increased blood stream infection (BSI) rates, especially with *Candida* species. Malnutrition also reduces respiratory strength, thereby increasing the risk for atelectasis and pneumonia.[12] Corticosteroids, which are commonly used in ICU settings, cause further immunosuppression.

It is unclear how to best assess immunologic function in patients with CCI. Laboratory markers such as serum albumin, pre-albumin, or absolute lymphocyte counts have not been very useful. Cellular immune function can be assessed by in vitro lymphocyte stimulation assay to specific antigens; most noteworthy is the *Candida* lymphocyte stimulating assay. One study in a group of 24 patients showed that a low *Candida* assay response was correlated with hospital mortality, and supranormal *Candida* assay response was associated with weaning success.[2] Measuring quantitative immunoglobulins and antibody response to vaccine administration facilitates assessing the humoral immune response.

Multidrug-Resistant Organisms and Antibiotic Resistance

Multidrug-resistant organisms (MDROs) are common in this population because of extended hospital stays and exposures to critical care units.

Vancomycin-Resistant Enterococcus (VRE). Depending on the facility, VRE may account for 7–79% of enterococcus isolates (unpublished data). Treatment options for VRE include linezolid, daptomycin, and tigecycline. It is important to obtain sensitivities to these antimicrobial agents, because resistance has been reported.[13] Recently, we found (unpublished data), in a review of antibiograms for 43 LTAC hospitals, that linezolid sensitivity ranged from 63% to 100%, and daptomycin sensitivity ranged from 73% to 100%.

Methicillin-resistant *Staphylococcus aureus* (MRSA). MRSA break points to vancomycin were lowered by the Clinical and Laboratory Standards Institute (CLSI) in 2006.[14] Current vancomycin break points are: minimum inhibitory concentration (MIC) ≤ 2 is susceptible MRSA; MIC of 4–8 is vancomycin-intermediate *S. aureus*, and MIC ≥ 16 is vancomycin-resistant *S. aureus*. Vancomycin MIC can be obtained by broth dilution or by E-test diffusion.[15] E-test overestimates MIC values by 0.5–1.5 μg/mL. Recently, we compared both methods in 16 consecutive MRSA isolates from different patients at a local LTAC hospital. Using microdilution, no MRSA isolates had an MIC ≥ 2. Using E-test, 13 of the 16 MRSA isolates had an MIC of 2, and the other 3 isolates had an MIC of 1.5 (unpublished data). A vancomycin MIC of 2 may or may not correlate with vancomycin clinical failure.[16]

Vancomycin continues to be a cost-effective therapy for MRSA. Other antimicrobials, such as linezolid, daptomycin, tigecycline, minocycline, or telavancin, are frequently used in the setting of an MIC of ≥ 2. Ceftaroline is a new cephalosporin with MRSA activity. The use of newer agents can drive up treatment cost for MRSA infections. MRSA resistant to daptomycin[17] and linezolid[18] have been reported; therefore, susceptibility testing is warranted.

Ceftaroline is a cephalosporin that has been FDA approved for skin infections and community-acquired bacterial pneumonia. It is bactericidal against *S. aureus* (including MRSA) and other Gram-positive organisms. It also has activity against Gram-negative organisms: *Escherichia coli*, *K. pneumoniae* (non-extended-spectrum β-lactamases [ESBL] producing), *Haemophilus influenza*, and other enterobacteriaceae. It has no pseudomonal activity.

Telavancin has bactericidal activity against organisms resistant to methicillin, daptomycin, linezolid, vancomycin-intermediate *S. aureus*, and vancomycin-heterogeneous vancomycin-intermediate *S. aureus*.[19] Telavancin has been approved by the FDA only for complicated skin and skin structure infections. The FDA is currently reviewing the results from 2 pneumonia trials; the drug appears promising in this setting. Telavancin side effects include renal toxicity, altered taste, nausea, and vomiting, but there are less pruritus and infusion-related events, compared to vancomycin. Televancin does not interfere with coagulation but does interfere with certain tests used to monitor coagulation. Blood samples for these coagulation tests (such as prothrombin time, international normalized ratio, and activated partial thromboplastin) should be performed prior to dosing telavancin, to obtain accurate values. Most recently, there have been shortages of this antibiotic.

Daptomycin is a novel cyclic lipopeptide antibiotic that provides rapid bactericidal activity against Gram-positive pathogens.[20] It is FDA-approved for complicated skin and skin structure infections, as well as bacteremia and right-sided endocarditis. Daptomycin is not appropriate for the treatment of pneumonia. The main adverse effect is myopathy, which is associated with elevated creatinine phosphokinase levels.

Linezolid belongs to a class called oxazolidinones.[21] It is bacteriostatic against enterococci and staphylococci. It has been FDA-approved for VRE infections, nosocomial pneumonia, and community-acquired pneumonia caused by MRSA, *Streptococcus pneumoniae*, and complicated skin and skin structure infections. Its main adverse effects are diarrhea, nausea, vomiting, and headache. Myelosuppression (including anemia, leukopenia, thrombocytopenia, and pancytopenia), optic neuropathy, and peripheral neuropathy have been reported, especially with prolonged use. Serotonin syndrome with selective serotonin reuptake inhibitors is also a concern.

Tygecycline and minocin are both bacteriostatic agents with MRSA activity. Since they are also active against Gram-negative organisms, their main use is in the setting of polymicrobial infections.

Resistant Gram-Negative Organisms. Many Gram-negative bacteria have developed multidrug-resistance, creating treatment challenges. An important problem is a *K. pneumoniae,* which produces an ESBL or carbapenemase (KPC). Extended resistance is also associated with strains of *Acinetobacter* and *Pseudomonas aeruginosa.*

Carbapenem-resistant *K. pneumoniae* (CRKP) is the strain of Carbapenem-resistant Enterobacteriaceae most commonly encountered in the United States.[22] The most important CRKP resistance mechanism is the production of carbapenemase enzyme bla_{KPC}.[23] The gene that encodes the bla_{KPC} enzyme is carried on a transposon, a mobile piece of genetic material, which increases the risk for dissemination. CRKP infections have been associated with increased mortality, stay, and cost.[24] Carbapenem-resistant Enterobacteriaceae is difficult to detect in the microbiology laboratory, because some strains have MICs that are elevated but still within the susceptible range for carbapenems.[25] In January 2009, CLSI published a recommendation that, in the presence of elevated MICs or reduced disk diffusion zone sizes, the modified Hodge test should be used to detect the presence of carbapenemases.

There are few treatment options. Aminoglycosides, particularly amikacin, as well as tigecycline[26] and polymyxins,[27] may be the only effective therapies. Therapy should be guided by susceptibility testing. Important aminoglycoside toxicities include nephrotoxicity, ototoxicity, and neuromuscular blockade. Polymyxins (colistin or polymyxin B) can cause nephrotoxicity and neurotoxicity. Inhaled colistin is controversial for the risk of lung toxicity, poor drug distribution and alveolar penetration, and emergence of resistance. In 2007 an FDA health alert recommended that nebulized colistin be used immediately following preparation to prevent buildup of the active colistin form, which can be toxic to the lungs. Colistimethate is often mixed with sterile water to form a solution just prior to inhalation via nebulizer. After mixing with sterile water and a buffer, colistimethate undergoes spontaneous hydrolysis to the bioactive form colistin. A component of colistin, polymyxin E1, is toxic to lung tissue. Premixing colistimethate into an aqueous solution and storing it for longer than 24 hours results in increased concentrations of colistin in solution, increasing the potential for lung toxicity.[26,28]

A. baumannii is a non-lactose fermenting Gram-negative coccobacillus that is widespread in the environment and can survive for long periods of time in the hospital environment. Until recently, carbapenems such as imipenem/meropenem have been the drugs of choice for *Acinetobacter* infections. However, resistant *Acinetobacter*, defined as resistance to all agents in greater than 3 antimicrobial classes (including β-lactams, aminoglycosides, carbapenems, and fluoroquinolones),[29] is becoming more prevalent and virulent. There are few therapeutic options.[28,30] Of the β-lactam inhibitors, sulbactam possesses the greatest intrinsic bactericidal activity. Amikacin and tobramycin retain some activity, but resistance is increasing and susceptibility testing is required. Polymyxins, including colistin, polymyxin E and polymyxin B, have been used to treat highly drug-resistant Gram-negative bacteria. Colistin is most commonly used in the United States.

Tigecycline is the first member in the new glycylcycline antibiotic class.[26] The 90% minimum inhibiting concentrating (MIC_{90}) for carbapenem-resistant *Acinetobacter* isolates is 2 μg/mL; CLSI has not yet determined susceptibility break points. Favorable clinical responses have been reported, but the rapid movement of tigecycline into tissues following intravenous infusion is concerning in the setting of bloodstream infections. Patients with ventilator-associated pneumonia (VAP) who received tigecycline had lower cure rates, especially those with bacteremia. Nausea is an important adverse effect of tigecycline in over 25% of patients.

Minocycline and doxycycline are both available by intravenous infusion, and minocycline is FDA-approved to treat *Acinetobacter* and is the most active in vitro agent against this organism.[31] Susceptibility testing is required, and tetracycline cannot be used as a surrogate marker because tetracycline-resistant isolates may be sensitive to minocycline.[32] Although combination therapy remains controversial,[28,30] some reports describe the use of colistin with other agents.

Prolonged infusion of β-lactams is emerging as an alternative treatment for *Acinetobacter* infection. Extended β-lactam infusion achieves drug concentrations in excess of the MIC for longer times for less susceptible organisms (ie, those with MICs between 4 μg/mL and 16 μg/mL). Doripenem's longer half-life makes it more useful, compared to imipenem and meropenem, which have half-lives of < 4 hours.

Many outbreaks of MDROs have been reported in acute care hospitals, LTAC hospitals, and in communities where patients move between institutions.[24] How to best control and avoid transmission of MDROs remains a challenge.

Prevalence of Multidrug-Resistant Organisms on Admission

There have been some reports of prevalence of MDROs in CCI patients on admission to LTAC hospitals. One 4-year prevalence study assessed patients for colonization

using rectal swabs, wound cultures, nasal cultures, gastric tubes cultures, and tracheal aspirate cultures.[33] Upon examining all culture sites, 69% of patients were found to be colonized with one MDRO, 23% had 2 MDROs, and 8% had 3 or more MDROs. Among rectal swabs, at least one MDRO was present in 50% of samples. The most common rectal swab pathogens were VRE (38%) and ESBL-producing Gram-negative rods (9%). For wound cultures, 33% had an MDRO, with 18% of those positive for VRE, 7% for MRSA, and 7% for imipenem-resistant *Acinetobacter*. For nasal cultures, 15% had MDROs, and of those, 7% were Gram-negative rods, 6% were MRSA, and 4% were VRE. For gastric tube cultures, 21% had some type of MDROs: 8% of those were VRE and 4% were ESBL producing Gram-negative rods. This reflects the high colonization rates of CCI patients in short-term acute care hospitals.

Tran et al reported a 46% incidence of multidrug-resistant *Acinetobacter* cultures in LTAC hospitals in southern California in 2008.[34] The most common infectious manifestation was nosocomial pneumonia, and most patients were already colonized or infected with *Acinetobacter* at hospital admission. The University of Maryland reported a single day MRSA point prevalence of 28% and *Acinetobacter* point prevalence of 30% at a 180 bed LTAC hospital in December 2005.[35] Researchers at a Los Angeles LTAC hospital[36] showed a 12.9% prevalence of *Clostridium difficile* on admission, with 6.5% of those being asymptomatic. The conclusion was that *C. difficile* carriage and unsuspected clinical infection are important reservoirs in the LTAC hospital setting.

As part of an outbreak investigation in the Tampa Bay area, surveillance admission cultures have been done since 2009 on all patients admitted to 2 local LTAC hospitals. These surveillance cultures include: rectal, wound, gastric tube or jejunostomy tube site, sputum, urine cultures, and stool for *C. difficile* screening. In 2010, monthly admission prevalence of MDRO in one LTAC hospital was as follows: 26–61% VRE, 33–61% MRSA, 3–22% multidrug-resistant *Acinetobacter*, and 0–2% CRKP. *C. difficile* toxin was detected in 3–21% of stool specimens on admission (unpublished data).

The prevalence of MDROs in patients transferred from short-term acute care hospitals to LTAC hospitals is very high and a possible source of horizontal transmission within institutions. Which organisms are important varies according to the region. It appears important to do admission surveillance cultures for appropriate placement and isolation, especially if an outbreak is suspected or if there is a need to define what organisms are present on admission. Nasal screens may not be sufficient, and multiple sites increase the yield of finding MDRO.[37] However, each institution needs to decide what surveillance cultures are appropriate based on local prevalence and referral short-term acute care hospitals. Another approach that may be considered is placing all patients in contact isolation.

Transmission Rates for Multidrug-Resistant Organisms

There are limited published data on transmission rates of MDROs in the LTAC hospital patient population. Only a few publications address how transmission may be controlled.[38–40] In 2006 the Center for Disease Control and Prevention (CDC), in conjunction with the Healthcare Infection Control Practices Advisory Committee published guidelines for the management of MDRO in the healthcare setting.[47] An outbreak of carbapenase-producing *Klebsiella pneumoniae* (CPKP) was successfully controlled at an LTAC hospital in 2008.[41] The facility implemented a bundled intervention to include: chlorhexidine bath, enhanced environmental cleaning, admission surveillance and serial point prevalence surveillance, isolation precautions, and personnel training. The CRKP prevalence decreased from 21% to 0% over 5 months. The authors concluded that a bundled intervention was successful in preventing horizontal spread of these Gram-negative organisms, despite ongoing admission of patients who were colonized with CRKP.[41]

Control of an outbreak and the nosocomial transmission of multidrug-resistant *Acinetobacter* in a 54 bed LTAC hospital in Ohio[42] was achieved through environmental decontamination using vaporized hydrogen peroxide combined with comprehensive infection control measures (strict adherence to hand hygiene and transmission based precautions). Thirteen patients infected or colonized with MDRO *Acinetobacter* were identified from January 2008 through June 2008. Nosocomial acquisitions of the pathogen ceased after the vaporized hydrogen peroxide intervention. When patients colonized with multidrug-resistant *A. baumannii* reoccupied rooms, environmental contamination recurred.

In a local LTAC hospital, nosocomial horizontal MDRO transmission rates (both infection and colonization combined) during 2010 were 0.87 cases per 1,000 patient days for VRE, 0.81 cases per 1,000 patients for MRSA, 0.06 cases per 1,000 patient days for *C. difficile*, 1.25 cases per 1,000 patient days for multidrug-resistant *Acinetobacter*, and 0.51 cases per 1,000 patient days for KPC (unpublished data).

In early 2010, an increase in CRKP prevalence and transmission was noted at another local LTAC hospital. The point prevalence of CRKP was 34% (unpublished data). To control CPKP transmission, we implemented admission surveillance cultures of multiple sites (sputum or tracheal aspirates, urine, stool, rectal swabs, wound, and gastric tube and jejunostomy tube sites); ongoing surveillance cultures with rectal swabs every 2 weeks; plus contact isolation. For all colonized and infected patients, ad-

Table 1. National Benchmarks (Pooled Mean) for Utilization Ratios

	Long-Term Acute Care Hospital	Adult Step-Down (post-critical care)
Permanent central line	0.13	NA
Temporary central line	0.54	0.18
Urinary catheter	0.51	0.24
Ventilator	0.27	0.11

NA = not applicable
(From Reference 48, with permission.)

Table 2. National Benchmarks (Pooled Mean) for Infection Rates

	Long-Term Acute Care Hospital	Adult Step-Down (post-critical care)
Permanent BSI rate	0.9	NA
Temporary BSI rate	1.7	1.5
CAUTI	2.6	1.9
VAP	0.6	1.5

BSI = blood stream infection
NA = not applicable
CAUTI = catheter-associated urinary tract infection
VAP = ventilator-associated pneumonia
(From Reference 48, with permission.)

herence to all infection control measures (eg, hand hygiene, use of personal protective equipment) was monitored. Patients were placed in the same hall, and dedicated staff were assigned to the colonized or infected patients with CRKP.[43] Prevention guidelines for VAP,[44] BSI,[45] and catheter-associated urinary tract infection (CAUTI)[46] were reviewed, and environmental measures were emphasized. Extensive education was conducted at all levels of the organization. Environmental measures[47] that were implemented included: use of patient-dedicated non-critical equipment, monitoring of both daily room cleaning and terminal room cleaning, with a checklist for housekeepers. All equipment utilized in patient rooms (eg, x-ray machines, glucometers) were cleaned after each use. Housekeeping staff was increased and had more resources allocated. Post intervention, in June 2011, the transmission rate was 0.8 cases per 1,000 patient days, and in July 2011 it was 0 cases per 1,000 patient days. Low transmission rates were seen, independent of admission prevalence (unpublished data). The implementation of aggressive admission surveillance,[22,47] appropriate patient isolation, and strong infection control practices allowed control of CPKR transmission, which has been maintained for 3 months (unpublished data). The prevalence of all MDRO on admission remains high, but transmission of all MDRO has been controlled; therefore, efforts to control a CPKP outbreak had a beneficial impact on transmission on all MDRO.

$$\text{BSI rate} = \frac{\text{Number of BSIs}}{\text{Number of central line days}} \times 1{,}000$$

$$\text{Central line utilization ratio} = \frac{\text{Number of central line days}}{\text{Number of patient days}}$$

$$\text{VAP rate} = \frac{\text{Number of VAPs}}{\text{Number of ventilator days}} \times 1{,}000$$

$$\text{Ventilator utilization ratio} = \frac{\text{Number of ventilator days}}{\text{Number of patient days}}$$

$$\text{CAUTI rate} = \frac{\text{Number of catheter associated UTIs}}{\text{Number of urinary catheter days}} \times 1{,}000$$

$$\text{Urinary catheter utilization ratio} = \frac{\text{Number of urinary catheter days}}{\text{Number of patient days}}$$

$$\text{\% Prevalence on admission} = \frac{\text{Number of cases for specific MDRO}}{\text{Number of admissions}} \times 100\%$$

$$\text{\% Point prevalence} = \frac{\text{Number of cases for specific MDRO}}{\text{Patient census for specific day}} \times 100\%$$

$$\text{MDRO transmission rate} = \frac{\text{New positive cultures of specific MDRO}}{\text{Patient days}} \times 1{,}000$$

Fig. 1. Formulas to calculate device infection rates and utilization rates as well as multidrug-resistant organism (MDRO) transmission rates and prevalence. BSI = blood stream infection. VAP = ventilator-associated pneumonia. CAUTI = catheter-associated urinary tract infection.

Device-Associated Infections

National benchmarks are available for device-associated infection rates and device utilization ratios[48] (Tables 1 and 2, Fig. 1). The National Healthcare Safety Network (NHSN) was established in 2005 to integrate 3 prior surveillance systems at the CDC: the National Nosocomial Infections Surveillance System, the Dialysis Surveillance Network, and the National Surveillance System for Healthcare Workers. The most recent data (to 2009) were published in June 2011.[48] Rates for specific healthcare settings, including LTAC hospitals and step-down units, are available for comparison across different patient populations and in different settings. Rates are decreasing, most likely due to implementation of effective hospital acquired infection prevention strategies.

Ventilator-Associated Pneumonia Rates

VAP is the second most common nosocomial infection in the critical care setting. It is associated with both increased morbidity and use of healthcare resources.[49] The VAP rate in LTAC hospitals can be lower than VAP rates

reported in acute care hospitals. CDC guidelines are applicable to CCI patients regardless of setting.[44] VAP prevention strategies include: head of bed elevation ≥ 30°, daily sedation interruption, regular oral care, peptic ulcer disease prophylaxis, and deep venous thrombosis prophylaxis. Other strategies include: avoid gastric over-distention, avoid unplanned extubation and reintubation, use a cuffed endotracheal tube with in-line or subglottic suctioning, maintain an endotracheal cuff pressure of at least 20 cm H_2O. Orotracheal intubation is preferable to nasotracheal intubation, to decrease risk of sinusitis. Some unresolved issues that will require further studies include the use of antiseptic-impregnated endotracheal tubes and selective digestive tract decontamination. A vaccination program for *S. pneumoniae* and influenza is also important.[44]

A 207-bed LTAC[50] with a 42-bed mechanical ventilation capability implemented a bundle for VAP prevention. The VAP rate decreased from 3.8 cases per 1,000 ventilator days prior to implementation, to 1.67 cases per 1,000 following implementation.

We reported 10 years of declining nosocomial VAP rates following implementation of a pneumonia prevention protocol.[51] The protocol included: elevation of the head to 30°, twice weekly whole-body chlorhexidine baths, mupirocin ointment to the nares,[52] handwashing, nutrition, tracheostomy by day 7, monitoring of staff adherence, and an infection control campaign involving posters, handouts, and small group education. The initial VAP rate was 6.1 cases per 1,000 ventilator days in 1998; following implementation of this protocol the rate decreased to 1.3 cases per 1,000 ventilator days and has continued to improve over the past 13 years. The 2009 VAP rate was 0.30 cases per 1,000 ventilator days, for 2010 it was 0.44 cases per ventilator days, and there have been no VAPs through August 2011. Prevention is important because VAP often involves MDROs, polymicrobial infections, and high mortality rates.[50] Performing tracheostomies on all prolonged ventilation patients, along with the high level of adherence to prevention bundles, are the 2 most important reasons for the continued decline in incidence rates of VAP in the LTAC setting.[50]

The VAP rates in LTAC hospitals are not very different from the pneumonia reported in skilled nursing facilities, where patients do not require mechanical ventilation. In fact, it may be somewhat similar to the pneumonia rate for chronically ill patients who do not require mechanical ventilation.[50] Interestingly, the LTAC population with VAP was found to have an increased likelihood of neurological impairment as a primary etiology for prolonged mechanical ventilator support.[50]

In the most recent data from NHSN, the pooled mean VAP rate for LTAC hospitals was 0.6 cases per 1,000 ventilator days, and for adult step-down units (post-critical care), the rate was 1.5 cases per 1,000 ventilator days.[48]

Blood Stream Infection Rates

BSIs constitute one of the most common healthcare acquired infections in the United States, and are associated with an elevated mortality rate. Patients treated in LTAC facilities are at an increased risk for BSI because the majority have an indwelling vascular device.[53,54] In the past, the BSI rates have been reported as high as 4–9 cases per 1,000 line days; there is one report from 2 LTAC hospitals with a rate of 16.4 cases per 1,000 line days.[55–57] Increased BSI rates have been reported with the use of needleless mechanical valve devices.[57] When needleless systems are used, a split septum valve is preferred.[45] Conversely, chlorhexidine baths have been reported to decrease BSI rates from 9.5 cases per 1,000 line days during the pre-intervention period, to 3.8 cases per 1,000 line days during the intervention period, to 6.4 per 1,000 line days during the post-intervention period.[55]

Implementation of BSI prevention strategies keeps rates low. BSI prevention strategies include hand hygiene, maximal barrier precautions at insertion, chlorhexidine skin preparation, optimal catheter site selection, and daily review of line necessity.[45] In our institution the BSI rate was 1.33 cases per 1,000 line days in 2009, 1.89 cases per 1,000 line days in 2010, and 1.0 cases per 1,000 line days for the first 6 months of 2011 (unpublished data). The most recent NHSN data show a pooled mean BSI rate for temporary lines in LTAC hospitals of 1.7 cases per 1,000 line days, with a temporary utilization ratio of 0.54 (ratio of central line days per number of patient days).[48]

The most common organism associated with BSI in this population is coagulase-negative *Staphylococcus*, but *Enterococcus*, MRSA, and Gram-negative organisms are also frequently found.[56–58] *Candida* is responsible for 5–10% of BSIs. Since antifungal susceptibilities are not readily available for most institutions, we examined the difference (both species and susceptibility) between acute care hospitals isolates and LTAC hospitals isolates.[58] In the LTAC hospitals, 25% of isolates were *Candida albicans*, while 75% of the isolates were non-*C. albicans*. In contrast, 47.9% of the isolates were *C. albicans* and 52% were non-*C. albicans* in the acute care hospital. *C. albicans* isolates were universally susceptible to fluconazole at both acute and LTAC hospitals. Only 65% of the non-*C. albicans* isolates at LTAC hospitals were susceptible to fluconazole. This finding has implications for empiric therapy when a fungal infection is suspected. A positive germ tube test indicates *C. albicans* and allows the use of fluconazole. Otherwise, an echinocandin (eg, mycofungin, caspofungin, or anidulafungin) should be used.

Catheter-Associated Urinary Tract Infection Rates

Since the number of patients with Foley catheters in this population is so high, CAUTI are common. A 3-year Penn-

sylvania study showed CAUTI rates of 4.2 cases per 1,000 catheter days in 2002, 3.2 cases per 1,000 catheter days in 2003, and 2.1 cases per 1,000 catheter days in 2004.[54] The CAUTI pooled mean rate published by the NHSN for LTAC hospitals is 2.6 cases per 1,000 catheter days, and for adult step-down units (post-critical care) is 1.9 cases per 1,000 catheter days.[48] During 2010 our institution had a CAUTI rate of 4.88 cases per 1,000 catheter days, and a urinary catheter utilization ratio of 0.76 catheter days per number of patient days. A multidisciplinary group was charged with educating staff regarding basic infection control and decreasing the use of urinary catheters. For the first 8 months of 2011, the urinary catheter utilization rate has decreased to 0.58 catheter days per number of patient days; the June and July rates were 0.45 and 0.47 catheter days per number of patient days, respectively. As of August 2011, the year-to-date CAUTI rate is 3.93 cases per 1,000 catheter days, with a June rate of 1.76 cases per 1,000 catheter days, and a July rate of 1.6 cases per 1,000 catheter days (unpublished data). A barrier to decreased urinary catheter use in males is the onset of urinary retention, secondary to enlarged prostate. The use of silver-impregnated catheters to decrease CAUTI is controversial, and their benefit unclear.[59] Latex catheters should be avoided, and silicone catheters are preferred.[60] Stricture formation has been reported with the use of latex catheters. Silicone catheters cause less strictures and less bladder irritation; they are also less prone to obstruction by encrustation.[60] Whether there is an added benefit to using silver-coated silicone catheters is unclear, but it appears that their use could decrease CAUTI rates; this will require further studies.

Clostridium difficile Infection Rates

C. difficile infection presents with specific symptoms, usually diarrhea and abdominal pain, plus either a stool test positive for *C. difficile* toxins, polymerase chain reaction testing, or histopathologic findings consistent with pseudomembranous colitis.[61] Complications can include an ileus and even toxic megacolon. Enzyme immunoassay testing for *C. difficile* toxin A and B is rapid, but less sensitive. Polymerase chain reaction testing is rapid, sensitive, and specific. Testing stool for cure is not recommended.

C. difficile carriage rate on admission to a Los Angeles LTAC facility was found to be 12.9%. During 2010,[36] monthly admission prevalence in our local LTAC varied from 4% to 34%. During the same period, the horizontal transmission rate was 1.51 cases per 1,000 patient days. However, the horizontal transmission rate was down to 0.46 during the first 6 months of 2011, no doubt as a result of efforts related to CPK outbreak interventions (unpublished data).

Risk factors for *C. difficile* infection include exposure to antimicrobial agents, age > 64, and duration of hospitalization.[61] Oral metronidazole is used only for initial mild or moderate disease, where white-blood-cell counts are $< 15{,}000$, and serum creatinine is < 1.5 times the premorbid levels.[61] For initial severe episodes and recurrent *C. difficile* infection, oral vancomycin should be used.[61] There is some evidence that oral vancomycin is more effective than metronidazole in eliminating the carriage state following therapy.[61] In the presence of ileus or megacolon, oral vancomycin plus intravenous metronidazole is indicated. If a complete ileus is present,[61] vancomycin retention enemas can be added to the regimen.

For recurrent disease, tapered or pulsed oral vancomycin may be helpful. "Fecal transplant" has been successful in uncontrolled case series, but the donor should be screened for transmissible agents.[62] Other possible therapies include: rifaximin, nitazoxanide, and intravenous immunoglobulin. A new agent, fidaxomicin, has been FDA-approved for *C. difficile* infection; however, there is very limited information for CCI patients, and the drug is very expensive.

Infection Control Programs

Disparities between infection control programs of acute care hospitals and LTAC facilities have been a concern.[63] The LTAC hospitals require infection control programs similar to acute care hospitals,[43,64,65] but it could be argued that LTAC facilities require more aggressive infection control programs because prevalence of MDROs in CCI patients is so high, and prevention of transmission is critical.[47] With the implementation of well proven infection control practices to prevent VAPs,[44] BSIs,[45] and CAUTIs,[46] nosocomial infection rates can be reduced and maintained at low rates.[65,66]

An LTAC facility infection control program should have physician leadership, with expertise in infection control, and, depending on hospital size, a dedicated full time infection preventionist with adequate support from administration. Daily collaboration and communication between physician and infection preventionist is critical to the success of the infection control program. The infection control committee has to be functional and multidisciplinary, to include the above individuals, plus nursing leadership, pharmacy, housekeeping, respiratory therapy, physical therapy, and representatives from rehabilitation services and plant operation. The committee should meet at least every other month, and, depending on local issues, it may have to meet more frequently.

Patients who are admitted to LTAC facilities show a high prevalence of MDROs; therefore, admission screening should be considered. Individual facilities will need to consider local prevalence MDRO transmission rates, any ongoing outbreaks, and other local factors to decide the

extent of admission screening. While awaiting culture results, newly admitted patients should remain in a single room (if available) and isolated. Once the surveillance cultures return, if the patient tested negative for MDRO, then he or she can be removed from isolation. Of course, transmission-based precautions[67] must still be observed in addition to the obvious: thorough handwashing. On the other hand, any patient with an MDRO should be placed in contact isolation, with the use of handwashing, gloves, masks, and gowns. In contrast, the concept of universal glove use for all patient care activities has been advocated by some infection preventionists and maybe a cost-effective approach.[40,68] Cohorting of patients who are colonized with similar MDROs can help with bed allocation if single rooms are unavailable. Handwashing with alcohol-based products is acceptable, except in the presence of *C. difficile*, where soap and water are required. Daily chlorhexidine baths for these patients have been used as part of bundled interventions for outbreak control, and to decrease BSI rates[55] and VAP rates.[52]

Surveillance

If screening cultures on admission to a specific facility are to be implemented, these could include rectal swab, sputum or tracheal aspirate, wounds, gastric tube/jejunostomy tube sites, and urine. Depending on the institution, stool may need to be tested for *C. difficile*. MDRO transmission rates have to be monitored and reported to the infection control committee for organisms that are prevalent at that particular hospital. In order to prevent horizontal transmission and to maintain proper isolation during outbreaks, rectal screening surveillance may be required every 2–4 weeks. Device-associated infection rates (eg, BSIs, VAPs, CAUTIs) should be monitored and reported to the committee.

All institutions carry the obligation of instituting prevention protocols based on CDC guidelines, and adherence must be supervised. Because patients frequently move between institutions and maintenance of proper isolation is key to prevent horizontal transmission, inter-facility communication should be formalized by means of a transfer form or other agreed upon method.

Education

Infection control education and familiarity with all policies and procedures at all levels of the organization are imperative. A family education program should be formalized with brochures and face-to-face teaching.[69] Education on all facets of isolation and how to properly use personal protective equipment should be ongoing, while adherence is monitored. Handwashing campaigns and zero tolerance for non-adherence should be instituted. Understanding how to use equipment is very important; patient-dedicated equipment must be left in rooms, and there should be a standard cleaning process for shared equipment.

Housekeeping

Since housekeeping constitutes such a critical part in the prevention of horizontal transmission of MDRO, there is no choice but to dedicate ample resources to this endeavor. Proper cleaning and disinfecting of shared medical equipment (eg, ultrasound machines, x-rays, glucose meters) are of paramount importance.[70] There must be delineation of which healthcare worker or provider is responsible for cleaning high-touch surfaces and shared equipment each day. For example, nurses may need to be responsible for cleaning medication preparation areas, housekeeping for cleaning the rooms and bathrooms using a checklist, and respiratory therapists for cleaning ventilators. Environmental Protection Agency-registered hospital products have to be used for routine cleaning and disinfection. In rooms with patients with *C. difficile* infection, sporicidal agents (such as bleach) need to be added. Dispatch (Clorox, Oakland, California) is an Environmental Protection Agency-approved disinfectant that has a 1:10 stable sodium hypochlorite (bleach) solution; it can be used for all rooms in the hospital.

New technologies that effectively clean rooms and save time but have substantial associated expenses include hydrogen peroxide vapor[42,71] and ultraviolet C band-emitting devices.[72] Ultraviolet C devices inactivate all microorganisms (including spores), and appear to be more effective than manual terminal disinfection. A standard patient room may be disinfected in < 10 min with an ultraviolet C device. A challenge is that rooms must be emptied for patient safety, so double-occupancy rooms present a problem. Proper manual terminal cleaning of a room can take one hour for a single housekeeper. New technologies may provide some advantages.

Antimicrobial Stewardship

CCI patients are frequently colonized and eventually develop infections with MDROs. *C. difficile* infections are common. However, the overuse of antibiotics in this population can cause harm. Antibiotics must be used judiciously, and this is a challenge that can be met only by implementing an antimicrobial stewardship program. This should be a multidisciplinary group that includes physicians, pharmacists, and an infection preventionist, with input from the microbiology laboratory and nursing staff. Their objective: focusing on choosing the most appropriate and cost-effective therapies. The Infectious Disease Society of America and the Society for Healthcare Epidemiology of America have published guidelines for devel-

oping an institutional program to enhance antimicrobial stewardship. Leadership by an infectious disease physician is crucial.[73]

Colonization should not be treated. Treatment must be explicitly avoided in cases where screening surveillance cultures are used. When there is a change in the clinical status of the patient, appropriate wide-spectrum antibiotics should be started.[61,74,75] All catheters and devices should be evaluated and removed if possible. De-escalation of antimicrobials should occur as soon as cultures are available or the diagnosis established. Duration of therapy should be concomitant to the diagnosis, keeping in mind that extended antimicrobial use builds resistance. Yearly antibiograms, from both the LTACH and the referring short-term acute care hospital, compiled with the help of the microbiology laboratory, allow appropriate empiric antimicrobial therapy, and help monitor any changes of antibiotic resistance.

Microbiology Laboratory

The support of a microbiology laboratory that is capable of providing accurate and rapid information (especially for MDROs) is key to a successful antimicrobial stewardship program and to provide quality care to these complex patients. The microbiology laboratory should follow CLSI recommendations.[26]

Physicians need to become familiar with the methods used by the microbiology department for susceptibility testing (eg, broth dilution vs E-test). The modified Hodge test needs to be performed for all Gram-negative rods with elevated MICs to carbapenems.[22] Physicians should know which *C. difficile* testing method is used; polymerase chain reaction is the most accurate. At a minimum, the microbiology laboratory should offer germ tube tests to differentiate *C. albicans* from non-*C. albicans*.

Summary

CCI patients are highly susceptible to infections. Multiple devices, an environment with a high MDRO prevalence, an already impaired immune system, all place these patients at high risk for nosocomial infections. In order to protect patients from hospital acquired infections, the best strategy is a comprehensive approach meticulously executed at all levels and many fronts. Meeting this challenge requires admission surveillance strategies, sound infection control practices, special attention to environmental cleaning, and detailed prevention protocols to minimize device related infections.

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Discussion

MacIntyre: Infection control and infection management are essential with these long-term patients. Let me ask you about a specific strategy. Palmer et al,[1] in New York, have advocated aerosolized antibiotics as soon as the sputum of a ventilated patient starts to get green and ugly. They call it tracheobronchitis, before the full-fledged pneumonia infiltrate has occurred. He advocates Gram staining it: if it's Gram-positive, you give them aerosolized vancomycin, and if it's Gram-negative, you aerosolize amikacin.

Does that make sense to you? I've never seen anybody else do it, but the idea behind it sounds appealing, and he has some small studies that say it reduces the progression from tracheal bronchitis to VAP. Does it make some sense?

1. Palmer LB, Smaldone GC, Chen JJ, Baram D, Duan T, Monteforte M, et al. Aerosolized antibiotics and ventilator-associated tracheobronchitis in the intensive care unit. Cri Care Med 2008;36(7):2008-2013.

Cancio: The problem is that we have to be careful with the overuse of antibiotics in this population, since these patients are colonized with many MDROs. If aerosolized amikacin is used on every patient . . .

MacIntyre: No, not every patient: just the Gram-negatives.

Carson: Which is every patient.

Cancio: Right. So we have to decide when and whether aerosolized antibiotics are beneficial. The real question is how to differentiate between colonization and infection. Up to 60% of patients are colonized with some kind of MDRO. We can't change that. We have to focus on only treating infections. It's hard to figure out the difference between tracheobronchitis and pneumonia. Even for the purpose of doing surveillance to find VAP, there are 3 criteria to be met, and they include x-ray findings. I would not recommend indiscriminate use of aerosolized antibiotics, but let's look at a few issues and recommendations.

Aerosolized or systemic antimicrobials are not recommended as prophylaxis for VAP prevention.[1] As it re-

lates to therapy, a good review on this subject was published in February 2011.[2] The difficulty in differentiating between ventilator-associated tracheobronchitis and VAP is again highlighted. Some researchers believe a high concentration of inhaled antibiotics could suppress biofilm formation, inhibit bacterial growth, and even reduce emergence of MDROs. A consensus regarding treatment of ventilator-associated tracheobronchitis with any antibiotics remains elusive. The use of inhaled antibiotics alone requires more data to establish when this practice is appropriate.

In the setting of established VAP with multi-drug-resistant Gram negative bacilli, inhaled antibiotics, especially aminoglycosides and colistin, may have a role. In ventilated patients, inhaled antibiotics can clog respiratory filters and cause bronchospasm. It may be most reasonable to use inhaled antibiotics in targeted, time limited protocols. Further studies are needed to define their role in both ventilator-associated tracheobronchitis and VAP.

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Muldoon:* The National Health Care Safety Network includes a new LTAC column that is just a few years old and being populated more and more.[1] I think it's worthwhile to make sure there's no misunderstanding of the intent behind that separation, so there's no error in interpretation. The intent of that column was to acknowledge that the patients who end up in the LTAC are different from those in the other categories: ICU, neonatal ICU, respiratory care unit, and the like. Because there was not an LTAC category, it was not possible for an LTAC to ask, "How am I doing?" So we created that category under the assumption that an LTAC group is as different from the others categories as a surgical ICU is from a step-down unit. Consequently, when we see that the numbers in the LTAC column are different, we should say, "Well, that proves the point." They tend to have less VAP and more urinary tract infections. It would be a mistake to compare the LTAC column with any other column. The intent was that an individual LTAC hospital could compare itself to its peers, now created by the LTAC column.

1. Dudeck MA, Horan TC, Peterson KD, Allen-Bridson K, Morrell GC, Pollock DA, Edwards JR. National Healthcare Safety Network (NHSN) report, data summary for 2009, device-associated module. Am J Infect Control 2011;39(5):349-367.

Cancio: The ventilator utilization rate in LTAC hospitals was 27%, whereas the utilization rate in the step-down unit was only 11%. The patients going to LTAC hospitals seem to require more ventilators and have more devices than the patients in step-down units. Based on utilization rates, patients being cared for at each setting have some differences. It is good to have a comparison, because up to now we did not. We had internal benchmarks, and we improved on those by following prevention protocols.[1] Similar efforts, based on prevention protocols, have improved other device related rates. It is good to have outside benchmarks to compare hospital specific efforts, as long as the comparison includes similar patient populations.

1. Korah JM, Rumbak MJ, Solomon DS, Cancio MR. Ten year review of decline in nosocomial ventilator-associated pneumonia rates in a long-term ventilator care facility post implementation of a pneumonia prevention protocol. Am J Respir Crit Care Med 2009;179:A1743.

Carson: I think we also have to be careful with benchmarking data. To compare yourself to a benchmark can give you a goal to work towards within your institution. If people are held too tightly to pay-for-performance issues and the like, it's yet another artificial factor that can affect who you admit.

The biggest risk factor for VRE bacteremia is VRE colonization. The biggest risk factor for VRE transmission is the percentage of patients coming in with VRE colonization. You don't want these factors to start impacting who you're going to admit. Benchmarking is for use with your own internal quality control measures.

I agree that surveillance on admission is important, so you know what you're dealing with. We have a 50% MDRO colonization rate on admission to our acute ICU from patients on our floor coming into the emergency department. We now isolate everyone until they're proven negative. They come in, they're isolated, they get their swabs, and if they're VRE negative, we let them off isolation. Are you doing that? Does that make sense? Is that overkill?

Cancio: No, that's exactly right. Screen and place patients in isolation at admission. Half of the patients are going to be colonized with an MDRO and half will not. The half that are not colonized can go to a regular room. The other half remain isolated. A strong infection control program is a requirement. A critical area is environmental services, since proper cleaning of rooms is very important. We do have to recognize that private rooms may not be available, and cohorting of patients with similar MDROs is acceptable. We also have to consider local prevalence issues; therefore, admission screening may not be the best strategy for some hospitals. Universal contact isolation or universal glove use may be a different option for other

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institutions. Hand hygiene remains critical for the success of any strategy. The role of new technologies, such as ultraviolet C devices, will need to be defined.

Carson: Yes, we have one of those, and it may create some very interesting hallucinations for our delirious patients, with those robots going around.

Snyder:† I understand your theory on surveillance cultures, but I worry that not everybody in medical practice is really tuned into colonization versus infection. My fear is that if doctors see bacteria on a culture, they're going to treat it. Do you think that surveillance cultures increase treatment, when it's colonization versus infection?

Cancio: That's not been our experience. These strategies cannot be done in a vacuum. It has to be done in conjunction with a good antimicrobial stewardship program, which requires collaboration with pharmacy, infectious disease physicians, and the treating physicians and consultants. The answer is no: the surveillance cultures allow the implementation of appropriate isolation, and MDRO transmission is prevented.

For example, in the case of a KPC outbreak, this is usually an infection control issue, not an antibiotic overutilization problem. Case finding, isolation, and excellent infection control practices, including good environmental cleaning, are required for control of an outbreak. During our KPC outbreak, in addition to admission surveillance, we did rectal swabs every 2 weeks, to make sure transmission was not continuing. Physicians were aware of the outbreak and did not treat colonization.

There are also other ways of measuring overuse of antibiotics, such as antibiotic cost per patient day. Most institutions can compare themselves to prior years or to other comparable hospitals. The duration of therapy for specific diagnoses is getting shorter, so the number of patients on longer than a given number of days of antibiotics can be another surrogate marker. Prevention is better than treatment. Treating colonization must be discouraged.

Cheifetz: I completely agree with your comment about the importance of prevention. I read a report last week about nosocomial spread of infection by medical staff, not from their hands, but via clothes, uniforms, scrubs, white coats, ties, etc.[1] The report was scary: 63% of staff grew potentially pathogenic bacteria on their uniforms, and a portion of these organisms were antibiotic-resistant.

How do you realistically and completely prevent this potential type of nosocomial spread? Strict hand hygiene campaigns are obvious. Isolation gowns and gloves for those patients with known resistant organisms have become standard. But should isolation gowns be worn for all direct contact with in-patients? I am not necessarily advocating for such an approach, just raising the topic for discussion. How would you suggest dealing with this complex issue?

1. Wiener-Well Y, Galuty M, Rudensky B, Schlesinger Y, Attias D, Yinnon AM. Nursing and physician attire as possible source of nosocomial infections. Am J Infect Control 2011;39(7):555-559.

Cancio: Pediatrics is a tough one.

Cheifetz: This was not a pediatric study. It focused on medical staff providing care to all patients.

Cancio: So, you are making a case for universal contact precautions. We are not there yet. We must concentrate all efforts on hand hygiene and cleaning of equipment that is shared between patients. I am unaware of any outbreaks related to healthcare clothing. The exclusive use of hospital issued clean scrubs is becoming more common. In our acute care hospital, scrubs from home are no longer allowed. Daily bathing of patients with chlorhexidine is associated with lower BSI and VAP rates, decreased MDRO transmission between patients, and decreased carriage on healthcare workers.[1]

1. Munoz-Price LS, Hota B, Stemer A, Weinstein RA. Prevention of bloodstream infections by use of daily chlorhexidine baths for patients at a long-term acute care hospital. Infect Control Hospital Epidemiol 2009;30(11):1031-1035.

Cheifetz: I agree with your comments, but we need to remember that adherence to infection control strategies is often less than optimal.

Cancio: We are going to see more emphasis on universal glove use for every patient interaction. More institutions are using daily chlorhexidine baths for patients. Some of the new technologies for environmental cleaning are also promising.

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