Our study was limited by the lack of multilocus sequence typing analysis, which would contribute to the knowledge of the molecular epidemiology of CRAB isolates. Although many distinct sequence types of CRAB, including some international clones, have been identified in Brazil,[10,11] there still are no data from this Brazilian region.

In summary, we demonstrated the persistence of a few clones responsible for endemic levels of CRAB isolates in hospitals in a Brazilian city. Notably, 3 of 7 clones remained as the major strains at least 5 years after an initial outbreak in this city. These findings challenged the effectiveness of infection control measures to control the dissemination of CRAB after an initial large outbreak.

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 0899-823X/2015/3607-0027. DOI: 10.1017/ice.2015.52

REFERENCES

1. Peleg AY, Seifert H, Paterson DL. *Acinetobacter baumannii*: emergence of a successful pathogen. *Clin Microbiol Rev* 2008;21: 538–582.
2. Martins AF, Kuchenbecker R, Sukiennik T, et al. Carbapenem-resistant *Acinetobacter baumannii* producing the OXA-23 enzyme: dissemination in southern Brazil. *Infection* 2009;37:474–476.
3. Martins AF, Kuchenbecker RS, Pilger KO, et al. High endemic levels of multidrug-resistant *Acinetobacter baumannii* among hospitals in southern Brazil. *Am J Infect Control* 2012;40: 108–112.
4. Schimith Bier KE, Luiz SO, Scheffer MC, et al. Temporal evolution of carbapenem-resistant *Acinetobacter baumannii* in Curitiba, southern Brazil. *Am J Infect Control* 2010;38:308–314.
5. Higgins PG, Lehmann M, Seifert H. Inclusion of OXA-143 primers in a multiplex polymerase chain reaction (PCR) for genes encoding prevalent OXA carbapenemases in *Acinetobacter* spp. *Int J Antimicrob Agents* 2010;35:305.
6. Clinical and Laboratory Standards Institute (CLSI). *Performance standards for antimicrobial susceptibilitiy testing*. Wayne, PA: CLSI document M100-S21; 2011.
7. Seifert H, Dolzani L, Bressan R, et al. Standardization and interlaboratory reproducibility assessment of pulsed-field gel electrophoresis-generated fingerprints of *Acinetobacter baumannii*. *J Clin Microbiol* 2005;43:4328–4335.
8. Go ES, Urban C, Burns J, et al. Clinical and molecular epidemiology of *Acinetobacter* infections sensitive only to polymyxin B and sulbactam. *Lancet* 1994;344:1329–1332.
9. Mostachio AK, Levin AS, Rizek C, et al. High prevalence of OXA-143 and alteration of outer membrane proteins in carbapenem-resistant *Acinetobacter* spp. isolates in Brazil. *Int J Antimicrob Agents* 2012;39:396–401.
10. Martins N, Dalla-Costa L, Uehara AA, et al. Emergence of *Acinetobacter baumannii* international clone II in Brazil: reflection of a global expansion. *Infect Genet Evol* 2013;20: 378–380.
11. Chagas TP, Carvalho KR, de Oliveira Santos IC, et al. Characterization of carbapenem-resistant *Acinetobacter baumannii* in Brazil (2008-2011): countrywide spread of OXA-23-producing clones (CC15 and CC79). *Diagn Microbiol Infect Dis* 2014;79: 468–472.

Primary and Secondary Literature Should Be Distinguished When Searching for Data Used in Systematic Reviews of Nosocomial Outbreaks

To the Editor—In a recently published letter the editor,[1] Zorrilla-Vaca and Vaca-Gonzalez questioned the methodology and the results of our systematic review on nosocomial outbreaks due to contaminated drugs, especially on outbreaks due to contaminated propofol.[2] In their opinion, important articles had not been included in our review because of a poor search strategy and/or insufficient bibliographic sources, resulting in an incorrect mortality rate. Herewith, we would like to respond to their questions and remarks.

The main concern of Zorrilla-Vaca and Vaca-Gonzalez addresses our omission of an article by Bennett et al[3] in 1995, which summarizes 7 nosocomial outbreaks that could be traced to contaminated propofol. Although we were well aware of this publication at the time of our review, we decided not to include it because all of these outbreaks had previously been published by the Centers for Disease Control and Prevention (CDC)[4] and this primary publication had already been included in our work, cited as reference 113. Thus, including the

article by Bennett et al would have resulted in bias due to double publication.

Secondly, Zorrilla-Vaca and Vaca-Gonzalez criticize that an editorial by Trépanier et al[5] in 2003 on nosocomial infections caused by propofol had not been adequately acknowledged in our review. Once again, this omitted publication is not a primary description of a nosocomial outbreak but rather is a summary of events published previously. It refers to the aforementioned article by Bennett et al[3] and to 3 additional outbreaks reports: Kuehnert et al[6] in 1997, McNeil et al[7] in 1999, and Henry et al[8] in 2001. Two of those articles are also included our review, cited as references 35 and 87, respectively.[6,8] The article by McNeil et al.[7] was not included because it only reports 1 of the 7 outbreaks that had been published by the CDC previously.[4]

However, we cannot deny the likelihood that some reports of nosocomial outbreaks caused by contaminated propofol or other contaminated substances were not included in our review. No matter how complex the search algorithm for a literature search, the possibility always remains that some relevant data are lacking. In addition, the focus of our review was infections due to contaminated drugs in general (original title: "Hospital acquired infections related to contaminated substances") rather than infections caused by propofol in particular. Thus, the key words described in the methods section of our publication did not take a specific type of substance into account. If we had specified particular substances, the number of possible substances that we would have had to investigate individually would have been too great to handle: solutions of sodium chloride, potassium, or glucose, propofol, heparin, insulin, erythrocyte concentrates, plasma albumin, all other kinds of formulas for an intravenously application, ultrasound gels, disinfection fluids, drugs used for inhalation, all kinds of substances for external use only, and many more.

Finally, Zorrilla-Vaca and Vaca-Gonzalez suggest the use of additional bibliographic sources for a more robust data search in systematic reviews on nosocomial outbreaks. Our work was based on searches of PubMed (one of the databases they recommend), the Outbreak Database,[9] and reference lists of all retrieved articles. To date, 3,200 outbreak reports have been filed in the Outbreak Database. To our knowledge, the Outbreak Database represents by far the largest collection on nosocomial outbreaks available worldwide, and it has often been used for research on various topics related to nosocomial outbreaks such as general epidemiological research, risk factor analysis, and infection control guideline preparation.[10–12]

Nevertheless, we do agree with Zorrilla-Vaca and Vaca-Gonzalez that reviews on specific high-risk substances such as propofol should be carried out more regularly because several new outbreaks caused by contaminated propofol[13,14] have been published since our own systematic review in 2007. More up-to-date reviews will keep staff on the wards and infection control personnel better informed regarding the risk and the epidemiology of nosocomial infections.

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0899-823X/2015/3607-0028. DOI: 10.1017/ice.2015.58

REFERENCES

1. Zorrilla-Vaca A, Vaca-Gonzalez PA. Inconsistencies regarding the number of outbreaks and mortality rate of hospital-acquired infections caused by contaminated propofol. *Infect Control Hosp Epidemiol* 2015;36:489–490.
2. Vonberg RP, Gastmeier P. Hospital acquired infections related to contaminated substances. *J Hosp Infect* 2007;65:15–23.
3. Bennett SN, McNeil MM, Bland LA, et al. Postoperative infections traced to contamination of an intravenous anesthetic, propofol. *New Eng J Med* 1995;333:147–154.
4. Centers for Disease Control and Prevention. Postsurgical infections associated with an extrinsically contaminated intravenous agent – California, Illinois, Maine, and Michigan, 1990. *MMWR Morb Mortal Wkly Rep* 1990;39:426–427, 433.
5. Trépanier CA, Lessard MR. Propofol and the risk of transmission of infection. *Can J Anesth* 2003;50:533–537.
6. Kuehnert MH, Webb RM, Jochimsen EM, et al. *Staphylococcus aureus* bloodstream infections among patients undergoing electroconvulsive therapy traced to breaks in infection control and possible extrinsic contamination by propofol. *Anesth Analg* 1997;85:420–425.
7. McNeil MM, Lasker BA, Lott TJ, Jarvis WR. Postsurgical *Candida albicans* infections associated with an extrinsically contaminated intravenous anesthetic agent. *J Clin Microbiol* 1999;37:1398–1403.
8. Henry B, Plante-Jenkins C, Ostrowska K. An outbreak of *Serratia marcescens* associated with the anesthetic agent propofol. *Am J Infect Control* 2001;29:312–315.
9. Vonberg RP, Weitzel-Kage D, Behnke M, Gastmeier P. Worldwide Outbreak Database: the largest collection of nosocomial outbreaks. *Infection* 2011;39:29–34.
10. Vonberg RP, Gastmeier P. Quality of outbreak descriptions in medical literature. *Lancet Infect Dis* 2007;7:699–700.
11. Vonberg RP, Kuijper EJ, Wilcox MH, et al. Infection control measures to limit the spread of *Clostridium difficile*. *Clin Microbiol Infect* 2008;14(Suppl 5):2–20.
12. Lanini S1, Puro V, Lauria FN, Fusco FM, Nisii C, Ippolito G. Patient to patient transmission of hepatitis B virus: a systematic review of reports on outbreaks between 1992 and 2007. *BMC Med* 2009;7:15.
13. Gutelius B, Perz JF, Parker MM, et al. Multiple clusters of hepatitis virus infections associated with anesthesia for

outpatient endoscopy procedures. *Gastroenterology* 2010;139: 163–170.
14. Klein J, Huisman I, Menon AG, et al. Postoperative infection due to contaminated propofol. *Ned Tijdschr Geneeskd* 2010;154:A767.

Availability of Automatic Water Tap in Hospitals in Bangkok, Thailand

To the Editor—Pathogens can contaminate the environment and cause infections. In hospitals, contamination of the environment is frequent and expected. Toilets in hospitals are an area of concern. The high contamination rates of toilet tap handles or levers for manual flushing are reported in many publications.[1,2] Toilet seats and handles are commonly found to be contaminated.[1] A good "toilet design" is proposed that could help control the spread of nosocomial infection.[3] To reduce the problem of contamination, toilets with hands-free automatic flushing mechanisms or water taps have been available for a few years. Here, the authors report a field survey of 180 toilets from 25 hospitals in Bangkok, Thailand. According to the survey, automatic hands-free flushing mechanisms were available in 65 toilets (36.1%). Most of the toilets studied lacked automatic water taps and classic toilet tap handles are still in use. The findings are potentially important and not just of local interest. Numerous hospitals in many countries in the world may still use manual flushing mechanisms. Promotion of the automatic water tap in hospitals will help improve hand hygiene in healthcare workers, visitors, and patients and may help reduce the problem of possible pathogen transmission.

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REFERENCES

1. Casey AL, Adams D, Karpanen TJ, et al. Role of copper in reducing hospital environment contamination. *J Hosp Infect* 2010;74:72–77.
2. Bellamy K, Laban KL, Barrett KE, Talbot DC. Detection of viruses and body fluids which may contain viruses in the domestic environment. *Epidemiol Infect* 1998;121:673–680.
3. Breathnach AS, Cubbon MD, Karunaharan RN, Pope CF, Planche TD. Multidrug-resistant *Pseudomonas aeruginosa* outbreaks in two hospitals: association with contaminated hospital wastewater systems. *J Hosp Infect* 2012;82:19–24.

Epidemiology of Antimicrobial Resistance in an Oncology Center in Eastern India

The epidemiology of multidrug-resistant organisms has local, national, and global significance.[1,2] In this study we describe the epidemiology of antimicrobial resistance from a new oncology and bone marrow transplantation center in eastern India. The method of antimicrobial susceptibility testing was per the Clinical Laboratory Standards Institute guidelines. Stool surveillance culture was performed according to the method described by Landman et al.[3] An automated system (Vitek2; bioMérieux) and disc diffusion (Bio-Rad) were employed for antibiotic susceptibility tests. The data refer to the period from April 1, 2012, through March 31, 2013. The data came from 4,723 samples, 1,474 patients (inpatients and outpatients), and 1,965 bacterial and yeast isolates. Gram-positive bacteria were detected in 25% of isolates, gram-negative bacilli in 68%, and yeasts in 7%. Positivity rates for different sample types were blood culture, 15.2%; urine, 33.3%; respiratory samples, 57.9%; pus, 65.8%; and body fluids, 37.0%. Stool samples for surveillance culture of multidrug-resistant organisms were positive in 35.1%. In total 30.6% were positive by culture.

Among patients with various infections antibiotic susceptibility of coliform bacteria (Enterobacteriaceae family) showed a high level of resistance with extended-spectrum beta-lactamase prevalence of 72%, carbapenem resistance in 23%, and resistance to amikacin, gentamicin, piperacillin-tazobactam, and ciprofloxacin to be 26%, 49%, 48%, and 71%, respectively. Nonfermentative gram-negative bacilli (eg, *Pseudomonas*, *Acinetobacter*) showed 36% resistance to carbapenems, 35% to piperacillin-tazobactam, 35% to amikacin, 38% to gentamicin, and 43% to ciprofloxacin. Resistance to meropenem and resistance to third-generation cephalosporins such as ceftazidime (marker of extended-spectrum beta-lactamase production) were detected respectively in cultures of 30.6% and 64.2% of blood isolates, 27% and 66.2% of urine samples, 25.7% and 47.8% of respiratory isolates, and 18.1% and 50.0% of pus isolates. Antibiotic resistance in gram-positive bacteria was noted in only 12% of the isolates (eg, methicillin-resistant *Staphylococcus aureus*), and inducible clindamycin resistance was noted in 23% of isolates. Antifungal susceptibility testing of *Candida* species (n = 123) showed 14% resistance to fluconazole. Among 356 patients with bloodstream infections, 55% were due to