hospice patients were not significantly different. Finally, as has been noted in acute care hospitals, the rates of device-related infections are decreasing.

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Use of Peripherally Inserted Central Catheters to Prevent Catheter-Associated Bloodstream Infection in Children

Bloodstream infection (BSI) is the most common healthcare-associated infection in pediatric intensive care units (ICUs).[1,2] Risk factors for central venous catheter (CVC)–associated BSI are poorly understood in middle-income developing countries. We used a prospective cohort study design to evaluate the infection rate and risk factors for BSI associated with short-term use (duration, less than 30 days) of a CVC in a pediatric hospital in Rio de Janeiro.

The Instituto de Puericultura e Pediatria Martagão Gesteira (IPPMG) is a tertiary care pediatric hospital with 60 beds in 6 wards. This is a reference center for patients requiring specialists in multiple diseases and admits patients aged 0–18 years. Because there is no pediatric ICU, patients with critical conditions are admitted to the wards.

All patients admitted to the wards from March 2003 through March 2006 who had a short-term CVC inserted at the IPPMG were monitored daily by the infection control team for development of CVC-associated BSI. A structured questionnaire based on National Nosocomial Infection Surveillance System criteria was used for active surveillance.[3] If a patient had multiple CVCs in place simultaneously, 1 catheter-day was assigned for each day of multiple CVC use, and the first catheter placed was used for surveillance purposes. CVCs inserted at another hospital, CVCs in place before admission to the wards, and CVCs placed for less than 1 day were excluded from analysis in this study.

Peripherally inserted central catheters (PICCs) were inserted by a nurse, and other CVCs for short-term use (ie, those placed in the subclavian, intrajugular, and femoral veins) were inserted by surgeons. Before catheter insertion, 4% chlorhexidine-germicide solution and 0.5% chlorhexidine-alcohol solution were used to prepare skin. After insertion, 5% chlorhexidine-alcohol solution was applied, and dressings were monitored by nursing staff. Conventional gauze dressings were applied to CVCs and were replaced with new dressings every 48 hours. Transparent dressings were applied to PICCs and were changed if bleeding and soiling occurred or if they did not stay in place.

CVCs (PICCs or conventional catheters) were removed immediately if patients showed signs of local infection or, for febrile patients, if their fever had no explanation other than local infection. The 5-cm portion of the tip of each CVC removed because of suspected infection was sent to the microbiology laboratory for culture; 2 samples of peripheral blood from the corresponding patient were also sent for culture.[4] Patients were observed for up to 48 hours after CVC removal.

Data analyses were performed using Stata, version 9.0

(Stata). Bivariate analysis was performed using a Wilcoxon 2-sample test (also known as the Mann-Whitney *U* test). The Fisher exact test was used for analysis of categorical variables. Variables with a *P* value of .20 or less were included in the multivariate analysis. A main-effects logistic regression model was fitted using the stepwise maximum likelihood estimation technique. The level of significance for removal of a variable in backward regression was .10. Interactions were assessed using the −2 log likelihood ratio test. The Pearson χ^2 goodness-of-fit test and the Hosmer-Lemeshow test were used to evaluate the fitness of the model. This study was reviewed and approved by the IPPMG ethical research committee.

During the study, 166 patients required 313 CVCs. Eighty-two CVCs did not satisfy inclusion criteria, and 27 CVCs were not evaluated because they were associated with infection at the catheter-exit site rather than with BSI. Thus, 204 CVCs were analyzed. Among the 166 patients in the study, the hospitalization duration was 12,370 patient-days, and the duration of CVC use was 2,197 CVC-days. The patients were aged 24 days to 14 years (median, 31.89 months), and 106 (64%) were male. Of the 204 CVCs studied, 38 (16%) were from patients with BSI, yielding an incidence of 17.30 infections per 1,000 CVC-days. Ten BSIs were classified as CVC-related BSI, and 28 were classified as CVC-associated BSI. Eighteen CVC-associated BSIs were laboratory-confirmed infections, and 10 were classified as clinical sepsis. Among 204 CVCs followed up, 53% were inserted by the Seldinger technique, 28% required surgical cut down, and 19% were placed percutaneously. Thirty-six percent of CVCs were inserted in brachiocephalic veins, 30% in internal jugular veins, 17% in femoral veins, 15% in subclavian veins, and 2% at another anatomic site. Ninety percent of CVCs were inserted using full-barrier precautions.

Findings of bivariate analysis are shown in the Table. Multivariate analysis revealed that total parenteral nutrition use (odds ratio [OR], 2.68 [95% confidence interval {CI}, 1.13–6.37]) and the number of CVC-days (OR, 1.06 [95% CI, 1.01–1.12]) were independently associated with CVC-associated BSI. PICC use was protective against CVC-associated BSI (OR, 0.17 [95% CI, 0.03–0.96]).

In this study, the incidence of CVC-associated BSI was 17.3 infections per 1,000 catheter-days, which is greater than the incidence observed in most pediatric ICUs in industrialized countries.[1,2] It is important to note that this study involved patients admitted to hospital wards, some of whom were in critical condition, and that 90% of CVCs were inserted using full-barrier precautions. An important risk factor for CVC-associated BSI is the duration of CVC use.[1,2] By use of multivariate analysis, we demonstrated that the odds of CVC-associated BSI increased a mean of 6% per CVC-day, although this rate is not linear and probably increases with each CVC-day.

We compared the risk of CVC-associated BSI among patients who received PICCs with that among patients who received other CVCs. Of interest, PICC use was independently protective against CVC-associated BSI. Several advantages of PICC use have been described elsewhere,[5-7] but the protection against CVC-associated BSI conferred by PICC use has not been well established among children.[1] To our knowledge, our study is the first to report such protection.

Of final note, our hospital does not have a pediatric ICU. Therefore, factors associated with patients in critical condi-

TABLE. Bivariate Analysis of Risk Factors for Central Venous Catheter (CVC)–Associated Bloodstream Infection (BSI) among Patients at a Brazilian Pediatric Hospital, 2003–2006

Risk factor	CVC-associated BSI	Non-BSI	Relative risk (95% CI)	*P*
Age, months, mean	54.36	48.63	…	.38
Male sex	10/38 (26)	54/166 (33)	0.82 (0.4 –1.67)	.45
Immunosuppression	20/38 (52)	87/164 (53)	0.98 (0.5–1.75)	.96
Length of stay, patient-days, mean	11.18	4.17	…	<.01
Length of CVC use, CVC-days, mean	12.58	9.03	…	<.01[a]
Disease at baseline	33/37 (89)	134/166 (81)	1.46 (0.61–3.49)	.38
PICC use	2/38 (5)	29/163 (18)	0.29 (0.07–1.22)	.04[a]
Subclavian vein insertion site	8/36 (22)	25/163 (15)	1.47 (0.70–2.78)	.36
Full-barrier precautions not used	2/37 (5)	20/166 (12)	0.39 (0.12–1.82)	.23
Antibiotic use	34/38 (89)	156/166 (94)	0.95 (0.2–1.5)	.30
TPN use	12/38 (32)	21/166 (13)	2.46 (1.34–4.24)	.004[a]
Neutropenia[b]	3/38 (8)	16/165 (10)	0.83 (0.3–2.4)	.73
MDRO colonization[c]	15/37 (41)	51/166 (31)	1.32 (0.83–2.60)	.18[a]
Remote-site infection	13/38 (34)	35/164 (21)	1.62 (1.28–4.00)	.007[a]

NOTE. Data are proportion (%) of patients, unless otherwise indicated. CI, confidence interval; MDRO, multidrug-resistant organism; PICC, peripherally inserted central catheter; TPN, total parenteral nutrition.

[a] Included in the multivariate analysis.

[b] Defined as a granulocyte count of $\leqslant 500$ cells/mm^3.

[c] Methicillin-resistant *Staphylococcus aureus* and gram-negative extended-spectrum β-lactamase producers.

tion, such as mechanical ventilation, arterial catheter use, and urinary catheter use, were not studied because of sample size limitations.

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Nosocomial Transmission of Undetected, Imported Measles in Taiwan, 2008

Measles remains a leading cause of death among young children.[1] Measles is highly contagious, and nosocomial transmission that has generated clusters of secondary cases has been described.[2] Healthcare workers (HCWs) have a nearly 19-fold higher risk of infection with measles compared with the general population.[3] This report describes nosocomial measles in 7 children and 1 HCW in Taiwan. The isolation of measles virus genotype H1 and the recent arrival from China of the source patient who sparked the epidemic indicate that the disease was imported from China.

In Taiwan, the transmission of indigenous measles has been successfully interrupted by massive measles vaccination programs, which were introduced in 1978. High vaccination coverage (90% at first dose and 95% at second dose) has been achieved since 1996.[4-11] Fewer than 10 confirmed cases of measles have occurred annually since 2003.[4-12]

This investigation was begun when a 39-year-old pediatric HCW and a 9-month-old pediatric patient (index patient) developed measles. The index patient was admitted to Kaohsiung Veterans General Hospital on December 8, 2008, with acute bronchitis and was discharged on December 17 but was readmitted on December 25 because of 2 days' fever and rash. The measles virus H1 genotype was isolated from throat swab samples obtained from the index patient (patient 1) and the infected HCW (patient 2). The source patient (patient 3) who transmitted the virus to index patient 1 was a 29-month-old child with fever and rash attributed to Kawasaki disease who stayed in the same hospital room as index patient 1 from December 13 through 17, 2008. Investigation revealed that source patient 3 had had contact with a cousin aged 28 months (patient 4) who had measles after being hospitalized in another hospital (hospital B) in early December 2008. The source patient who sparked the epidemic (patient 5) turned out to be a 20-month-old child who had recently arrived from China. On November 1, 2008, source patient 5 had received the mumps, measles, and rubella (MMR) vaccine. On November 5, 2008, he was admitted to hospital B with a fever and rash that had lasted 2 days. Subsequently, 4 pediatric inpatients in hospital B (patients 4, 6, 7, and 8) developed measles, including the cousin (patient 4) of source patient 5 (Table). The H1 genotype of the measles virus isolated from a throat swab sample obtained from source patient 5 had 100% homology in the 456 nucleotides that encode the COOH terminus of the nucleoprotein (N) with the viral genotype found in the 2 patients in our hospital and 1 patient in hospital B. All patients recovered uneventfully. None of the other children had received measles vaccination prior to becoming ill. Given the possibility that the outbreak in the pediatric ward of Kaohsiung Veterans General Hospital might spread within the hospital and the community, we initiated active contact tracing, investigating exposed HCWs and children hospitalized in the pediatric department.

All pediatric patients admitted during the contagious period were traced by telephone to ascertain their history of vaccination, measles infection, and symptoms of fever and rash after discharge. Serological tests for measles were performed on children who had fever and rash when they were admitted, on pediatric nurses, and on symptomatic HCWs. HCWs who were exposed to the virus in the emergency and outpatient departments were observed for fever and rash.