Extrusion of Venticuloperitoneal Shunt Catheter Through the Rectum and Retrograde Meningitis

To the Editors:

Ventriculoperitoneal (VP) shunt systems are affected by complications such as infection and functional and mechanical failure. Most VP shunt complications are related to the proximal component. Abdominal complications include: inguinal hernia; cerebrospinal fluid pseudocyst; intestinal perforation; shunt migration to distant locations, such as intrathoracic or subphrenic areas or scrotum; hollow viscous perforation; subphrenic abscess; small bowel perforation with secondary formation of cerebrospinal-enteric fistula; intractable ascites; and protrusion of the shunt from anus, vagina, umbilicus and urethra.1–4

We report a case of total extrusion of the distal component of a VP shunt with defecation.

The patient, a 7-year-old boy delivered by cesarean section with a cervicothoracic myelomeningocele, had undergone surgical repair when he was 7 days old. He gradually developed hydrocephalus, and a VP shunt was placed at the age of 20 months (a Phoenix system, medium pressure). He had motor delay and did not start to walk until the age of 3 years.

At the age of 7 years, the patient developed fever and abdominal pain. Six days later, frontal headache, drowsiness and loss of appetite were also noted. The patient had neck stiffness; the abdomen was soft and diffusely tender without masses. Bowel sounds were present. A lumbar puncture revealed acute bacte rial meningitis (glucose, 8 mg/dL; white blood cells, 1600/mm3; polymorphonuclear cells, 70%), but cerebrospinal fluid culture showed meningitis with Klebsiella pneumoniae (glucose, 27 mg/dL; white blood cells, 250/mm3; polymorphonuclear cells, 85%). The reservoir and proximal catheter were removed, and an external ventricular drain was inserted for 5 days. He was uneventfully discharged after 3 weeks of systemic antibiotic therapy. Six months later, he was clinically active and healthy without a VP shunt and had no intracranial hypertension symptoms.

This case highlights an unusual complication of a VP shunt that caused much distress for the patient and his family with catheter extrusion through the rectum. The catheter had disconnected itself at its junction on the skull and migrated into the bowel, passing out per rectum with defecation.

The mechanism of bowel perforation by peritoneal catheter is uncertain. Irritation of the external bowel wall by a shunt tube may cause inflammation, leading to wall weakness and finally perforation. After this event, the shunt is usually propelled distally by peristalsis, which may dislodge the tube from its proximal point of fixation and excrete along with the bowel waste product that requires quite a sustained force, given that there would be a lot of resistance.3,5

Our case was referred with late shunt infection and abdominal pain 2 months before peritoneal catheter extrusion. The abdominal symptoms might have been evaluated more specifically with dye injection into the reservoir to show a probable bowel perforation. It could have prevented our patient’s second meningitis with early diagnosis of bowel perforation and shunt removal.

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Source of Infection in Household Transmission of Culture-Confirmed Pertussis in Brazil

To the Editors:

The World Health Organization estimates that pertussis causes 300,000 deaths a year worldwide. A better understanding of the role of adults and adolescents in the spread of the disease is one of the priorities for control.1

In the State of Pernambuco, Brazil there has been complete (100%) vaccine coverage for 3 doses of pertussis vaccine in infants since 1998.2 In this study, we identified household members of children with confirmed pertussis presenting to a medical school hospital. We followed the definitions of the Centers for Diseases Control and Prevention to confirm pertussis cases and to classify them as primary (source of infection) or secondary cases.3

Cases were tabulated by age of primary and secondary cases. The similarity of proportions by age was tested by $\chi^2$ test. $P$ was considered significant when $p \leq 0.05$.

During 2003, we identified pertussis cases in 57 households of patients referred to the Pediatric Infectious Disease Service at the Oswaldo Cruz Hospital in Recife, Brazil. Pertussis was confirmed in 158 of the 349 household members, of whom 65

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were primary cases and 93 were secondary cases; 52 of all cases were confirmed by culture and 106 by epidemiologic link. Of the remaining, 169 did not have pertussis and 22 were excluded because of insufficient information to confirm or exclude pertussis. Seventy-five percent (28 of 37) of the cases younger than 6 months of age were secondary cases. The primary case was older than 11 and 6 months years for 79% (66 of 84) of all secondary cases and in 76% (19 of 25) if the secondary case was younger than 6 months of age.

These findings were similar to those of Bisgard et al4 in United States. They found a family member or relatives as the suspect source of pertussis for 75% of the infants from zero to 3 months of age and 73% of the infants from 4 to 11 months of age. Children younger than 6 months of age are at highest risk of complications and death from pertussis.

The findings that subjects older than 11 years and 6 months were the sources for the majority of secondary cases, including these younger than 1 year of age, indicate that actions to prevent pertussis should be targeted at the age group older than 11 years and 6 months.

Préziosi and Halloran5 have observed that pertussis vaccination decreases the transmission of a vaccinated subject with pertussis. Given that a pertussis vaccine for adults is available, the potential impact (and cost and benefit) of vaccination of older children and adults on interrupting transmission should be considered.

This study was supported by the Pan-American Health Organization/World Health Organization.

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Positive Coombs Test in Postpneumococcal Hemolytic-Uremic Syndrome

To the Editors:

H emolytic-uremic syndrome can occur as a complication of invasive Streptococcus pneumoniae infection.1 In a recent survey, S. pneumoniae accounted for nearly 40% of cases of nonidiopathic hemolytic-uremic syndrome.2

Pneumococcal neuraminidase exposes the cryptic Thomsen-Friedenreich antigen present on red blood cells, platelets and glomerular endothelial cells. Antibodies directed against the Thomsen-Friedenreich antigen, normally present in human plasma, bind to the antigen, exposed through the effect of pneumococcal neuraminidase, on the surface of the above mentioned cells (called Thomsen-Friedenreich activation), finally resulting in hemolytic-uremic syndrome.1 The direct Coombs test, detecting the coating of red blood cells with antibodies or components of complement, has been reported to be positive in postpneumococcal hemolytic-uremic syndrome, as recently reviewed.3 However, the usefulness of this test in the context of postpneumococcal hemolytic-uremic syndrome has not been fully elucidated.

During the period 2000–2004, 2 children with pneumococcal infection, a 20-month old girl with meningitis and a 14-month old boy with pleuropneumonia, were treated at our institutions because of hemolytic-uremic syndrome. In both children, the direct Coombs test, in which polyspecific antihuman globulins were used, was strongly positive. During the same period, this test was negative in 6 randomly chosen children with invasive pneumococcal infection without associated hemolytic-uremic syndrome (4 boys and 2 girls ages 9 to 45 months; median, 24 months; meningitis, N = 1; bacterial pneumonia, N = 3; bacteremia, N = 2). Furthermore the test was also negative in 5 randomly chosen children (3 girls and 2 boys, ages 5 to 56 months; median, 10 months) with postdiarrheal hemolytic-uremic syndrome.

In patients with postpneumococcal hemolytic-uremic syndrome and Thomsen-Friedenreich activation, the use of washed blood products is often advocated.4 Therefore early diagnosis of this condition is mandatory. A simple and reliable test detecting Thomsen-Friedenreich activation would improve the management of patients with possible postpneumococcal hemolytic-uremic syndrome. It is concluded that in hemolytic-uremic syndrome a positive direct Coombs test might indicate a pneumococcal origin. Further studies including larger cohorts are needed to evaluate the accuracy of the direct Coombs test in the diagnosis of postpneumococcal hemolytic-uremic syndrome.

We acknowledge the support of the Associazione Bambino Nefropatico.

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4. Cochran JB, Panzarino VM, Maes LY, Teck-

Antifungal and Antibacterial Resistance Profiles Between Cambodia and Kenyan Children With Human Immunodeficiency Virus Infections Receiving Trimethoprim-Sulfamethoxazole Prophylaxis

To the Editors:

There is a fear that antibacterial prophylaxis with trimethoprim-sulfamethoxazole (TMP-SMX) in children with acquired immunodeficiency syndrome (AIDS) might lead to increase of antimicrobial resistance in bacteria. We compared culture results and resistance profiles in children receiving TMP-SMX prophylaxis 3 times weekly orally in Phnom Penh, Cambodia and, in Nairobi, Kenya, after 12–18 months of highly active antiretroviral therapy (HAART) therapy. Thirty-three children from Phnom Penh (group A) and 42 patients from Nairobi (group B) were compared.

CD4 count varied from 10 to 220 at admission. Oropharyngeal swab cultures were transported to the reference laboratory of antimicrobial resistance of the Slovak Ministry of Health. Tables 1 and 2 show comparison of culture results and resistance patterns in human immunodeficiency virus-positive children in Phnom Penh and Nairobi, at the initiation of HAART and after 12–18 months of therapy. In Phnom Penh, the occurrence of multiresistant Gram-negative bacteria after 12–18 months of HAART decreased ($P < 0.01$), and oxacillin resistance in methicillin-resistant Staphylococcus aureus increased. In Nairobi no difference in frequency of isolation of selected organisms in culture results was observed. However after 12 months of HAART, the percentage of penicillin-resistant (PEN-R) pneumococci increased from 12% to 75%, and ampicillin-resistant organisms (AMP-R) in Haemophilus influenzae increased from 0% to 50%. Table 3 compares resistance patterns after TMP-SMX prophylaxis in children in Nairobi and Phnom Penh.

The proportion of methicillin-resistant S. aureus in Phnom Penh was signifi-

**TABLE 1.** Results of Positive Cultures and Resistance of Respiratory Isolates in Children With AIDS After 12–18 Months of HAART and TMP-SMX Prophylaxis in Phnom Penh

<table>
<thead>
<tr>
<th>Resistance Patterns (%)</th>
<th>Baseline (%)</th>
<th>12–18 mo (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Resistance</td>
<td>Etiology</td>
</tr>
<tr>
<td>ERY-R Streptococcus pyogenes</td>
<td>48</td>
<td>7.5</td>
</tr>
<tr>
<td>OXA-R Staphylococcus aureus</td>
<td>48</td>
<td>10</td>
</tr>
<tr>
<td>PEN-R Streptococcus pneumonia</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>AMP-R Haemophilus influenzae</td>
<td>33</td>
<td>7.5</td>
</tr>
<tr>
<td>CTAZ-R Klebsiella/Enterobacter</td>
<td>100</td>
<td>50</td>
</tr>
<tr>
<td>FLU-R Candida spp.</td>
<td>10</td>
<td>25</td>
</tr>
</tbody>
</table>

ERY-R indicates erythromycin-resistant; OXA-R, oxacillin-resistant; CTAZ-R, ceftazidine-resistant; FLU-R, fluconazole-resistant; NS, not significant.

**TABLE 2.** Results of Positive Cultures and Resistance of Respiratory Isolates in Children With AIDS After 12–18 Months of HAART and TMP-SMX Prophylaxis in Nairobi

<table>
<thead>
<tr>
<th>Resistance Patterns (%)</th>
<th>Baseline (%)</th>
<th>12–18 mo (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Resistance</td>
<td>Etiology</td>
</tr>
<tr>
<td>ERY-R Streptococcus pyogenes</td>
<td>14</td>
<td>19</td>
</tr>
<tr>
<td>PEN-R Streptococcus pneumonia</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>AMP-R Haemophilus influenzae</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>MR Staphylococcus aureus</td>
<td>40</td>
<td>26</td>
</tr>
<tr>
<td>AMP-R Moraxella catarrhalis</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>CTAZ-R Klebsiella/Enterobacter</td>
<td>39</td>
<td>26</td>
</tr>
<tr>
<td>FLU-R Candida spp.</td>
<td>0</td>
<td>21</td>
</tr>
</tbody>
</table>

ERY-R indicates erythromycin-resistant; MR, methicillin-resistant; CTAZ-R, ceftazidine-resistant; FLU-R, fluconazole-resistant; NS, not significant.

**TABLE 3.** Comparison of Culture-Positive Results and Antimicrobial Resistance in Respiratory Isolates of Kenyan and Cambodian Children With AIDS After 12–18 Months of TMP-SMX Prophylaxis

<table>
<thead>
<tr>
<th>Resistance Patterns (%)</th>
<th>Nairobi</th>
<th>Phnom Penh</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>PEN-R Streptococcus pneumonia</td>
<td>75</td>
<td>0</td>
<td>0.01</td>
</tr>
<tr>
<td>AMP-R Haemophilus influenzae</td>
<td>50</td>
<td>0</td>
<td>0.01</td>
</tr>
<tr>
<td>OXA-R Staphylococcus aureus</td>
<td>0</td>
<td>100</td>
<td>0.01</td>
</tr>
<tr>
<td>CTAZ-R Klebsiella/Enterobacter</td>
<td>67</td>
<td>50</td>
<td>NS</td>
</tr>
<tr>
<td>FLU-R Candida spp.</td>
<td>33</td>
<td>33</td>
<td>NS</td>
</tr>
<tr>
<td>AMP-R Moraxella catarrhalis</td>
<td>16</td>
<td>26</td>
<td>NS</td>
</tr>
<tr>
<td>ERY-R Streptococcus pyogenes</td>
<td>0</td>
<td>100</td>
<td>0.01</td>
</tr>
</tbody>
</table>

OXA-R indicates oxacillin-resistant; CTAZ-R, ceftazidine-resistant; FLU-R, fluconazole-resistant; ERY-R, erythromycin-resistant; NS, not significant.
icanently higher than in Nairobi ($P = 0.01%$), and the proportion of PEN-R pneumococci and AMP-R $H. influenzae$ was significantly higher in Nairobi ($P = 0.01%$). High baseline resistance in community isolates of $S. aureus$ and Strep
tococcus pyogenes (47.5%) in Phnom Penh before initiation of prophylaxis might explain the difference between resistance patterns from Nairobi and Phnom Penh because prophylactic strategy with TMP-SMX, antimicrobial treat-
ment strategies and HAART regimen are same in both centers.

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To the Editors:

Esposito et al$^1$ recently reported a beneficial effect of azithromycin treatment of recurrent respiratory tract infections in children. Their study addresses an important issue in pediatrics, and the results, if valid, would be clinically significant. We therefore discussed the study with pediatric residents in our hospital.

Participants were concerned about lack of blinding of the study but decided that this would be unlikely to explain the apparent benefit solely to the subgroup of children with evidence of atypical bacterial infection at enrollment. A more seri-
ous concern that threatens the validity of the study is evidence of baseline dif-
f erences between the azithromycin and placebo groups. Although the study is a randomized, controlled trial comparing outcomes among patients who received azithromycin with those who did not receive any antibiotic, the authors do not present a comparison of baseline demo-
graphic and clinical characteristics of the 2 groups, an unusual omission in a random-
ized trial. This is concerning, because Ta-
ble 4 in Ref. 1 shows that the proportion of patients with atypical bacterial infections at baseline was markedly different between the 2 groups: 76 of 177 (43%) in the group treated with azithromycin versus
114 of 175 (65%) in the group who did not receive antibiotic ($P = 1.5 \times 10^{-5}$). This discrepancy suggests that there was a problem with the randomization, causing baseline imbalance in the study. The com-
bination of this statistically significant baseline maldistribution and the failure of the authors to mention it undermines our confidence in the results.

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REFERENCE
1. Esposito S, Bosis S, Faelli N, et al. Role of atypical bacteria and azithromycin therapy for children with recurrent respiratory tract infec-

Reply:

On the basis of the careful com-
ments of Benerjee et al on our paper recently published in the Jour-
nal, we have provided a table (Table 1) with the baseline characteristics of pa-
ients with recurrent respiratory tract infec-
tions (RRTIs) treated with azithromy-
 cin plus symptomatic therapy compared with those treated with symptomatic therapy only. As can be seen from the table, there were no significant differ-
ences between the 2 groups. The differ-
ence in the proportion of patients with atypical bacterial infections for the 2 groups seems to be casual, although ge-
netic characteristics may explain this finding. On the other hand, the main aims of this study were to evaluate whether atypical bacteria play a role in causing acute respiratory episodes in children with RRTIs and whether spe-
cific antibiotic treatment of the infec-
tions caused by these bacteria can im-
prove the course of the acute episodes and reduce recurrences. On the basis of patients' characteristics at the time of randomization, no imbalances were ob-
erved.

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<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Therapy with Azithromycin + Symptomatics ($N = 177$)</th>
<th>Therapy with Symptomatics Only ($N = 175$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median age (yr)</td>
<td>3.5 [1–14]$^a$</td>
<td>3.6 [1–14]</td>
</tr>
<tr>
<td>Male</td>
<td>99 (55.9)$^b$</td>
<td>102 (58.3)</td>
</tr>
<tr>
<td>Breast-feeding &gt;3 mo</td>
<td>103 (58.2)</td>
<td>106 (60.6)</td>
</tr>
<tr>
<td>Attending day care or school</td>
<td>147 (83.1)</td>
<td>152 (86.9)</td>
</tr>
<tr>
<td>Exposed to passive smoking</td>
<td>59 (33.3)</td>
<td>69 (39.4)</td>
</tr>
<tr>
<td>No. of family members living together (median)</td>
<td>3 [2–10]</td>
<td>4 [5–9]</td>
</tr>
<tr>
<td>No. of respiratory tract infections in previous 6 mo (median)</td>
<td>6 [4–10]</td>
<td>5 [3–12]</td>
</tr>
<tr>
<td>No. of antibiotic courses in previous 6 mo (median)</td>
<td>3 [0–9]</td>
<td>3 [1–10]</td>
</tr>
<tr>
<td>No. of hospitalizations in previous 6 mo (median)</td>
<td>0 [0–2]</td>
<td>0 [0–1]</td>
</tr>
</tbody>
</table>

$^a$No difference was significant.

$^b$Numbers in brackets, range.

$^c$Numbers in parentheses, percent.