required to attend postcertification courses in the infectious diseases field.

In conclusion, Belgium is often considered as an example for the reduction of antibiotic consumption in the community. However, this reduction is only apparent when expressed in PID, whereas a significant increase is objectified when expressed in DIDs. In our daily practice, we continue to observe numerous cases of overuse and misuse of antibiotics in the community, as is illustrated with fluoroquinolones, and there are currently no data available about the quality of prescriptions in our country. We very strongly agree with the WAAAR statement regarding antibiotics as “special drugs and a treasure to protect,” not only in the hospital but also in the community, as outpatient antibiotic consumption represents >90% of the global amount of antibiotics used daily [1, 7].

Note
Potential conflicts of interest. All authors: No reported conflicts. All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

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References

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Significant Reduction of External Ventricular Drainage–Associated Meningoventriculitis by Chlorhexidine-Containing Dressings: A Before-After Trial

To the Editor—Meningoventriculitis (MV) associated with external ventricular drainage (EVD) is a serious complication in neurosurgical patients, and infections are mainly due to skin-derived bacteria [1, 2]. Currently, data on rates of EVD-associated MV are scarce, with estimate ranging from 2 to 20 cases per 1000 EVD days [3, 4]. Interestingly, for central line–associated sepsis, the positive impact of using chlorhexidine-containing dressings to reduce sepsis rates has been demonstrated in several trials [5, 6]. Owing to the analogy in pathogenesis and thus causative microorganisms of the 2 device-associated infections, we initiated a before-after trial aimed at decreasing MV infection rates by using chlorhexidine-containing dressings in each patient with EVD, after completing a pilot investigation for safety issues [7]. This intervention should not substantially increase daily workload or hospital running costs.

The intervention consisted of using a chlorhexidine-containing dressing (3M Tegaderm CHG; 3M) instead of a gauze dressing beginning on day 2 for each new EVD, from 1 October 2012 to 31 March 2015. All adults needing EVD at the neurosurgical intensive care unit, University Hospital Aachen, were consecutively enrolled during the 30-month intervention period. Continuous evaluation and calculation of the MV rates according to national and international surveillance protocols were performed as in previous years by an interdisciplinary and interprofessional health team twice weekly during infectious disease rounds (http://www.cdc.gov/HAI/surveillance/index.html; www.nrz-hygiene.de). The local ethics committee approved this investigation. Accumulated yearly rates were calculated followed by determining confidence intervals for individual rates based on Ulm’s method [8]. A Poisson regression model to the rates accounting methods was fitted to the data using proc GENMOD from SAS® version 9.1 software under windows XP for computations. Effects were assessed as significant in cases for which the P-value falls below the significance margin of 5% (.05).

During the 6 years before the intervention period, 42 months of discontinuous surveillance was performed. A total of 5383 EVD days were documented, and a total of 43 EVD-associated MV cases occurred. During the 30-months “after” study period, continuous surveillance was performed, documenting >10 000 patient-days, including a total of 2512 EVD days during which 5 cases of EVD-associated MV occurred. Statistical analysis revealed a significantly lower EVD-associated MV rate during the intervention period: 1.70 cases (standard deviation [SD], 3.88) versus 6.98 (SD, 5.67) per 1000 EVD days in the control phase (P = .005). This decrease was driven mainly by a reduction in MV cases caused by typical skin commensals, a finding in line with the working hypothesis concerning the antiseptic dressing (Table 1). Dressings were changed only every sixth day. No adverse events (eg, skin reactions) occurred.

This significant reduction in MV rates of >300-fold was observed in an already highly trained healthcare setting in which several previous interventions to prevent MV infections had been in place since 2005 and in which high compliance with hand hygiene (>80% compliance), written standard operation procedures for EVD placement and maintenance, and participation in surveillance were standard. In the context of the current national benchmark, this rate is much lower than the mean of 4.0 MV cases per 1000 EVD days and approaches the 25th percentile of 1.67 per 1000 EVD days (www.nrz-hygiene.de). Thus, our intervention significantly reduced rates of EVD-associated MV without increasing costs or workloads [9].
Table 1. Shift in Microorganisms Causing External Ventricular Drainage–Associated Meningoventriculitis

<table>
<thead>
<tr>
<th>Causative Agent</th>
<th>Meningoventriculitis Cases, No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2005–2009</td>
</tr>
<tr>
<td></td>
<td>2012–2015</td>
</tr>
<tr>
<td>Coagulase-negative staphylococci</td>
<td>29 (73)</td>
</tr>
<tr>
<td>Staphylococcus aureus</td>
<td>7 (18)</td>
</tr>
<tr>
<td>Enterococci</td>
<td>1 (2.5)</td>
</tr>
<tr>
<td>Enterobacteria</td>
<td>0</td>
</tr>
<tr>
<td>Pseudomonas aeruginosa</td>
<td>1 (2.5)</td>
</tr>
<tr>
<td>Acinetobacter baumannii</td>
<td>1 (2.5)</td>
</tr>
<tr>
<td>Candida albicans</td>
<td>1 (2.5)</td>
</tr>
</tbody>
</table>

Notes

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Multidrug–Resistant Mycoplasma genitalium Is Increasing

To The Editor—We read with great interest the article of Lau et al [1] on the declining efficacy of a single–dose regimen of 1 g of azithromycin for the treatment of Mycoplasma genitalium infections. The microbiological cure rate in studies conducted prior to 2009 was 85.3% (95% confidence interval [CI], 82.3%–88.3%), whereas that in studies conducted since the beginning of 2009 was 67.0% (95% CI, 57.0%–76.9%). A recent increase of M. genitalium with azithromycin resistance was suggested.

Macrolide–resistant clinical strains of M. genitalium were isolated from azithromycin–treatment failure cases of nongonococcal urethritis (NGU) [2]. Mutations in these strains were found in residues corresponding to A–2058 and A–2059 in the 23S ribosomal RNA (rRNA) gene of Escherichia coli. Mycoplasma genitalium with such mutations was resistant to the single–dose regimen of 1 g of azithromycin. In Japan, a single–dose regimen of 2 g of extended–release azithromycin is available that provides pharmacokinetics similar to those of a 3–day regimen of 500 mg of azithromycin. However, we observed treatment failures with this regimen in eradicating M. genitalium with mutant 23S rRNA [3].

For M. genitalium infections unsuccessfully treated with azithromycin, moxifloxacin regimens were effective [4], but moxifloxacin treatment failures were also reported [5]. Since we first detected fluoroquinolone resistance–associated amino acid changes of Ser83→Asn, Asp87→Tyr, and Asp87→Val in ParC, corresponding to those at Ser80 and Gln84 in ParC of Escherichia coli, in M. genitalium in urine specimens of patients with NGU [6], identical or analogous alterations in ParC have been found in M. genitalium in clinical specimens [5, 7]. Such alterations in ParC were associated with treatment failures with moxifloxacin [5]. In Japan, we have been prescribing a 7–day regimen of 100–mg sitafloxacin twice daily for treatment of NGU [8]. During in vitro susceptibility tests, sitafloxacin was more active than moxifloxacin against ciprofloxacin–selected laboratory mutants of M. genitalium with decreased susceptibility to fluoroquinolones [9]. The sitafloxacin regimen succeeded in eradicating M. genitalium with altered ParC in all 11 cases of NGU treated in our previous study [3]. The regimen appeared to overcome the fluoroquinolone resistance conferred to M. genitalium by single amino acid changes in ParC. However, the acquisition of a single amino acid change in GyrA or ParC might be the first step in the development of clinically significant resistance to fluoroquinolones. Therefore, the emergence of M. genitalium with such altered ParC is a matter of concern.