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Reply to Moehring et al

To the Editor—The work of Moehring et al1 (hereafter, Moehring) is a welcome addition to the discussion of postdischarge-detected (post-DD) hospital-onset (HO) methicillin-resistant Staphylococcus aureus (MRSA). Their work highlights a valuable surveillance resource that allows for the tracking of patients with MRSA infections. Although the analyses of Moehring tell a story similar to our work, it is important to note the differences in methodology. Moehring attributed post-DD HO-MRSA to hospitalizations that occurred up to 1 year prior to the detection of MRSA, whereas we limited the time frame for attribution to hospitalizations within 30 days prior to detection. In fact, 59% of hospitalizations in our study occurred within 2 weeks prior to the MRSA detection admission. This may help address Moehring’s concern that our study may not represent healthcare-associated exposure. The brief interval between hospital discharge and evidence of MRSA suggests that the MRSA was likely healthcare associated and attributable to the recent hospital stay. Similarly, the brief interval makes it unlikely that community sources of MRSA were important sources of MRSA acquisition. We fully agree that calculating MRSA acquisition rates using a 1-year window for prior hospital exposure, such as is reflected in the analysis performed by Moehring, may well represent substantial community and healthcare exposures. An analysis of Moehring’s data with a 30-day restriction would be interesting and would allow a more direct comparison between the 2 analyses.

In addition, Moehring mentions the fact that our population included substantial fractions of patients discharged to nursing homes and suggests that this may introduce additional important sources of MRSA acquisition. This is true. However, we note that our sensitivity analyses explicitly excluded individuals known to have had contact with a skilled nursing facility or acute inpatient stay between the MRSA acquisition admission (assigned by us) and the MRSA detection admission. This information was based on variables in the administrative data describing the discharge disposition and the source site prior to admission. We removed 1,237 (43%) post-DD MRSA cases on the basis of the discharge location and 86 (3%) on the basis of the next admission location. These results are described in our article.2

To address Moehring’s concerns about patient contact with hemodialysis centers, we preformed an additional sensitivity analysis in which we reanalyzed the data excluding an additional 168 (6%) post-DD cases occurring in people with renal disease on the basis of codes from the International Classification of Diseases, Ninth Revision. The results of this analysis showed that the inclusion of post-DD MRSA increased the median number of HO-MRSA cases per 10,000 at-risk admissions by a factor of 2.0 (12.2 to 24.4; P = .0003), compared with the 3.0-fold increase when all patients were included (12.2 to 35.7; P < .0001). Thus, even when patients with other healthcare facility exposures were removed from the analysis, we found that MRSA acquisition was double what would otherwise be found within a hospital stay. This is supported by the assessment conducted by Moehring.

Another important difference between the work by Moehring and our study is that Moehring was able to identify and assess MRSA infections. For our own analysis, we were limited to MRSA carriage due to the known imperfections of administrative data in identifying MRSA infection. While there is evidence that MRSA carriage increases the risk of future infection,3 the identification of MRSA infection is more clinically meaningful, and therefore it is particularly important that Moehring found that significant amounts of MRSA infection come to light only after discharge.

In an era of public reporting of healthcare-associated infections, there is strong pressure to engage in a blame game, as mentioned by Moehring. However, the purpose of our study was not to focus on blaming hospitals but rather to galvanize policy makers and members of our field of infection prevention to join together to tackle the larger issue of a contagion that crosses hospital boundaries and is broadly shared across facilities. Our intent was to highlight the striking
potential for collaborative regional strategies to improve both the detection of and, more importantly, the prevention of HO-MRSA.

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When an Infection Prompts Removal of an Unnecessary Device

In recent decades, there has been a worldwide increase in the number of implanted devices, including neurosurgical shunts. Device-related infection represents a worrisome complication, and the prevention of such infections is primarily based on the use of aseptic measures during device insertion, proper management of the device itself, and perioperative antibiotic prophylaxis when needed. Over the long term, however, the best preventive approach remains the removal of the device when it is no longer necessary.

Cerebrospinal fluid (CSF) shunts significantly improve the quality of life for patients with hydrocephalus, and the insertion rate has dramatically increased over the past 20 years.1 In the United States, 40,000 neurosurgical ventricular shunts are inserted annually.1 The rate of shunt-associated infections ranges from 1% to 18%, and such infections are associated with high morbidity and mortality.2 Assessment of the optimal functioning of a CSF shunt is subject to variation, and once a CSF shunt is inserted, it almost always remains for the life of the patient. However, CSF shunt independence (ie, a shunt that is no longer necessary), although uncommon, is not exceptional.4 We describe two cases in which a CSF shunt infection prompted the recognition of shunt independence and led to the removal of the shunt.

A 67-year-old woman with chronic kidney disease presented with an 8-month history of intermittent fever, neutrophilia (white blood cell [WBC] count, 16,000 cells/mm³), elevated C-reactive protein (CRP) level, and elevated erythrocyte sedimentation rate (ESR). Three years earlier, she had had a road traffic accident complicated by an intracerebral hemorrhage and subsequent hydrocephalus that was treated with insertion of a ventriculoatrial shunt. Methicillin-resistant Staphylococcus epidermidis was isolated from 3 consecutive blood cultures. A transesophageal echocardiogram revealed vegetations 4 and 8 cm in diameter that were adherent to the ventriculoperitoneal shunt and located on the tricuspid valve, respectively (Figure 1). Treatment with intravenous daptomycin (6 mg/kg/day) was commenced. After 3 days, defervescence was observed, and the patient’s WBC count and inflammatory markers decreased. On day 10 after presentation, the patient experienced tachypnea and fever. Computed tomography (CT) of the chest revealed septic emboli. Daptomycin therapy was continued, and on day 15, blood cultures showed no growth and chest radiograph findings were unremarkable. The shunt was externalized, and 10 mL was drained over a 24-hour period without radiological signs of hydrocephalus, which suggested shunt independence and led to the definitive device removal.

A 21-year-old man was admitted to the hospital with a 30-day history of intermittent fever and headache. Two years earlier, he had had a road traffic accident that resulted in the development of posthemorrhagic hydrocephalus that required placement of a ventriculoperitoneal shunt. At admission to the hospital, physical examination findings were normal. Investigations showed that the patient’s WBC count was 11,300 cells/mm³, his ESR and CRP level were elevated, and CT of the brain revealed hydrocephalus. The remaining laboratory test results were normal. On day 3 of hospitalization, the patient complained of abdominal pain. An abdominal ultrasound revealed the presence of ascites with peritoneal thickening, and fluid from paracentesis had a WBC count of 3,000 cells/mm³ (60% neutrophils). Candida albicans was isolated from the fluid sample. The ventriculoperitoneal shunt...