to gravitate toward rate-based approaches, which have been subject to criticism by advocates of causal inference methods,[3] while clinicians interested in prognostic questions may be more likely to veer toward cumulative risk-based approaches. Because of our primary interest in developing strategies for the prevention of HAI, which exclude modifying length of hospital stay, our research has tended to focus on rate-based approaches.[4,5]

In certain circumstances, a comparison of each approach may certainly be useful, while in others, it may not be worth the additional analytic burden. What is important is to understand and interpret the insights derived from cumulative risk-based and rate-based approaches correctly; to conflate the 2 approaches is to muddy the epidemiologic waters.

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Surveillance for Ventilator-Associated Pneumonia: Can We Apply Centers for Disease Control and Prevention–National Healthcare Safety Network 2013 Definitions for All Settings?

To the Editor—In the present journal, we read the article by Greene et al[1] about the influence of Centers for Disease Control and Prevention–National Healthcare Safety Network (CDC-NHSN) 2015 definitions for catheter-associated urinary tract Infection surveillance with great interest. Here, we share our experience of using CDC-NHSN 2008 and 2013 criteria for ventilator-associated pneumonia (VAP) surveillance at different intensive care unit (ICU) settings.

VAP is one of the most important problems in ICUs. Adequate surveillance of VAP is critical in order to introduce effective control measures early. The surveillance criteria for VAP was released by CDC-NHSN in 2008 and 2013.[2,3] The 2013 criteria was based on the worsening pulmonary functions. The worsening oxygenation—as increase in positive end-expiratory pressure and fraction of inspired oxygen—was termed ventilator-associated condition. When an abnormal temperature or white blood cell count and new antibiotics are added to a ventilator-associated condition, this condition is described as infection-related ventilator-associated complication. Diagnosis of possible VAP requires detection of specific microbioogic etiology in addition to an infection-related ventilator-associated complication. From January 1, 2013, through March 30, 2015, we adapted CDC-NHSN 2013 definitions for VAP surveillance; however, we observed a huge difference between the number of patients with a clinical diagnosis of VAP versus the VAP rate detected by CDC-NHSN 2013 criteria, pariticularly in the surgical ICUs. Then, we decided to use CDC-NHSN 2008 criteria for VAP surveillance to understand the role of definitions in the rate of the rapidly changed VAP rate.

Our hospital is a 700-bed tertiary center. A patient-based infection control program has been set up more than 20 years. An infection control nurse daily visits all patients hospitalized in the ICU to detect ICU-acquired infections. All surveillance data are periodically discussed with the infection control doctor, who is an infectious diseases physician. There are 3 surgical ICUs with 42 beds (general surgery with 9 beds, cardiothoracic surgery with 22 beds, and neurosurgery with 11 beds), and a medical ICU (MICU) with 9 beds in our adult hospital. In surgical ICUs, there are no intensivists and all patients are followed by individual surgical teams who operate on the patients. Owing to lack of adequate number of staff, ventilator parameters are not recorded properly and even sometimes cannot be managed according to the actual clinical condition of the patients. Diagnostic tests such as complete blood count or cultures from respiratory tract and blood are delayed because of shortage of well-trained staff in surgical

TABLE 1. Rates of Ventilator-Associated Pneumonia (VAP) According to Type of ICU

Variable	Rate (95% CI)		
	1st period (January 1, 2013–December 31, 2013)	2nd period (January 1, 2014–March 31, 2015)	3rd Period (April 1, 2015–December 31, 2015)
Medical ICU			
- **VAP rate**	9.6 (5.12–16.44)	7.5 (4.03–12.95)	7.03 (2.83–14.5)
- **Ventilation utilization rate**	0.44 (0.43–0.47)	0.45 (0.44–0.47)	0.44 (0.42–0.46)
General surgery ICU			
- **VAP rate**	9.9 (3.99–20.43)	1.6 (0.41–9.06)	9.8 (3.21–23.06)
- **Ventilation utilization rate**	0.28 (0.27–0.30)	0.18 (0.17–0.20)	0.24 (0.22–0.26)
Cardiothoracic surgery ICU			
- **VAP rate**	19.2 (9.62–34.47)	1.7 (0.44–9.74)	22.5 (11.63–39.33)
- **Ventilation utilization rate**	0.29 (0.27–0.31)	0.25 (0.23–0.26)	0.33 (0.31–0.35)
Neurosurgery ICU			
- **VAP rate**	19.1 (11.16–30.69)	4.3 (1.58–9.36)	16.8 (8.70–29.4)
- **Ventilation utilization rate**	0.36 (0.34–0.38)	0.46 (0.44–0.48)	0.43 (0.40–0.45)

NOTE. VAP rate was calculated for 1,000 ventliation-days. ICU; ıntensive care unit.

ICUs. Intensivists are available for 24 hours in the MICU in contrast to the surgical ICUs. Mechanical ventilation support is managed according to the actual respiratory condition of the patient and all changes are recorded properly. Diagnostic tests are performed in a timely manner when there is a suspicion of ICU-acquired infection. Although there is a weaning algorithm in the MICU, it does not exist in surgical ICUs.

A total of 3,516 ventilation-days were followed from January 1, 2013, through December 31, 2013, in 4 ICUs. The VAP rates detected by CDC-NHSN 2008 criteria were as follows, per 1,000 ventilation-days: 9.6 in MICU, 9.9 in general surgery ICU, 19.2 in cardiothoracic ICU, and 19.1 in neurosurgical ICU.

During the second period, CDC-NHSN 2013 criteria was adapted for VAP surveillance and 4,298 ventilation-days were followed from January 1, 2014, through March 30, 2015. Diagnosis of possible VAP was confirmed by Ventilator-Associated Event Calculator, version 2.1 (CDC), during this period. The VAP rate was suddenly decreased in all surgical ICUs (from 9.9 to 1.6 in general surgery ICU, from 19.2 to 1.7 in cardiothoracic ICU, and from 19.1 to 4.3 in neurosurgical ICU) and a slight decrease was observed in MICU (from 9.6 to 7.5) (Table 1).

From April 1, 2015, through December 30, 2015, a total of 2,747 ventilation-days were followed. During this period all surgical ICUs had a sharp increase for VAP rates in surgical ICUs (from 1.6 to 9.8 in general surgery ICU, from 1.7 to 22.5 in cardiothoracic ICU, and from 4.3 to 16.8 in neurosurgical ICU) whereas the VAP rate in MICU was stable after reapplying the CDC-NHSN 2008 criteria for VAP surveillance. Ventilator utilization rates were similiar for all periods when intra-unit comparison was performed (Table 1).

Our study was not a head-to-head comparison of CDC-NHSN 2008 and 2013 criteria. However, a recent study reported that VAP rate showed a significant difference when different criteria was used. This multicenter study showed that 2 of the 4 ICUs achieved zero VAP rates using the CDC-NHSN 2013 algorithm.[4]

Subjective interpretation of radiologic findings of pneumonia is the major disadvantage of CDC-NHSN 2008 criteria of VAP surveillance. The primary aim of the CDC-NHSN 2013 criteria was to avoid the interpretation variability. However, it may not be so easy to follow the CDC-NSHN 2013 algorithm for VAP surveillance when the infrastructrure of the ICU has certain limitations. Our findings show the importance of addressing the limited usefulness of CDC-NHSN 2013 in settings where human resources are sparse. Potential failure to capture VAP can be an important problem for effective control of ICU-acquired infections.

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Relation of Diagnostic Accuracy of Viral Respiratory Polymerase Chain Reaction to Specimen Number and Source in Severe Adenovirus Pneumonia: Antimicrobial Stewardship Implications

To the Editor—I read with interest the article by Dr. Schleihauf and colleagues[1] on the number of nasopharyngeal (NP) specimens for diagnosis of respiratory virus by polymerase chain reaction (PCR). Their point is well taken and I agree that the diagnostic yield of respiratory viral PCR testing is not increased beyond 3. In intubated hospitalized adults with undiagnosed viral pneumonia, lower respiratory tract may be preferable to upper respiratory tract sampling. Recently, I had a patient who demonstrated the critical importance of specimen source in accurate diagnosis.

A 64-year-old woman presented with fever, chills, myalgias, dry cough, and shortness of breath. The patient had chronic obstructive pulmonary disease, atrial fibrillation, and recent contact with her sick grandson. She was in respiratory distress, her temperature was 38.7°C, her pulse was 127 beats/min (irregularly irregular), and her respiratory rate was 22 breaths/min. Physical examination was unremarkable except for bilateral conjunctival injection and expiratory wheezes. Laboratory studies included a white blood cell count of 11.5 K/mm^3 (neutrophils = 88%, lymphocytes = 4%, and monocytes = 7%), with a creatinine level of 1.1 mg/dL. Serum transaminases were unremarkable. Chest radiograph was clear with a questionable left lower lobe infiltrate. Procalcitonin (PCT) was 0.72 ng/mL. NP rapid influenza test was negative. NP PCR respiratory viral panel was negative for respiratory viruses. She was started on azithromycin, her respiratory status improved, but she remained febrile (temperature, 40.4°C). Her respiratory status deteriorated on hospital day (HD) 3, and she was placed on noninvasive positive pressure ventilation and transferred to the respiratory intensive care unit. Repeat NP respiratory viral panel was again negative. Repeat PCT was 2.36 ng/mL and on HD 2 ceftriaxone was added. Repeat chest radiograph showed increased interstitial markings, but no segmental/lobar infiltrates. Chest computed tomography showed diffuse lower lobe air space opacities. Ceftriaxone and azithromycin were discontinued and she was started on vancomycin, piperacillin/tazobactam, and doxycycline. Her creatinine level was now 4.55 mg/dL. She was intubated on HD 6, and bronchoscopy was performed. Bronchoalveolar lavage fluid showed 594 nucleated cells (many "smudge cells") and abundant red blood cells. Direct fluorescent antibody for *Pneumocystis* pneumonia was negative. Gram stain showed few polymorphonuclear leukocytes with no organisms and cultures were negative. Respiratory viral panel PCR performed on bronchoalveolar lavage fluid was positive for adenovirus and antibiotics were discontinued. Adenovirus antibody titer was elevated at 1:256 (normal <1:8) and serum quantitative adenovirus PCR was highly positive with 288,000 copies/mL. She defervesced on HD 6, but on HD 8, she developed loose, watery stools positive for *Clostridium difficile*. She was successfully treated for *C. difficile* diarrhea with metronidazole and vancomycin (Figure 1). Although there were no segmental/lobar infiltrates on chest radiograph to suggest bacterial pneumonia, her PCT was elevated and empirical antibiotics were given. Respiratory viral PCR performed on bronchoalveolar lavage fluid was positive for adenovirus and bronchoalveolar lavage fluid cytology showed adenovirus cytopathologic effects, which are large intranuclear basophilic inclusions resulting in a smudged appearance ("smudge cells ") pathognomic for adenovirus infection.[2,3]

The need to consider the *validity* of a sampling source has been reported previously. During the 2009-2010 influenza pandemic, a middle-aged immunocompetent man was