Trend in Ventilator-Associated Pneumonia Rates Between 2005 and 2013

From 2006 to 2012, the incidence of ventilator-associated pneumonia (VAP) reported to the Centers for Disease Control and Prevention National Healthcare Safety Network (NHSN) decreased.1,2 In medical and surgical intensive care units, between 2006 and 2012, the reported incidence of VAP per 1000 ventilator-days decreased from 3.1 to 0.9 (71% decline) and 5.2 to 2.0 (62% decline), respectively. Whether the decrease was attributable to better care or stricter application of subjective surveillance criteria is unclear.3 The Medicare Patient Safety Monitoring System (MPSMS)4 has independently measured VAP rates since 2005, using a stable definition of VAP. Trends in MPSMS VAP rates from 2005 through 2013 were analyzed.

Methods | To track the national frequency of safety events in hospitalized patients, the MPSMS abstracted a random selection of acute-care hospital records from 2002-2013, except 2008 (because of a 1-year lapse in federal funding). Between 18,000 and 34,000 records were abstracted from between 730 and 4000 randomly selected hospitals across the nation each year. Detailed MPSMS methods have been previously reported.4 This analysis included MPSMS VAP rates during calendar years 2005 through 2013 among Medicare patients 65 years and older with principal diagnoses of acute myocardial infarction (AMI), heart failure, pneumonia (including a primary diagnosis of sepsis or

Table. Medicare Patient Safety Monitoring System Patient Characteristics and Observed VAP Rates

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<thead>
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</thead>
<tbody>
<tr>
<td>Hospitals, No.</td>
<td>222</td>
<td>249</td>
<td>490</td>
<td>369</td>
<td>1330</td>
</tr>
<tr>
<td>MPSMS patients ≥65 y, No.</td>
<td>11,752</td>
<td>15,246</td>
<td>33,307</td>
<td>23,730</td>
<td>86,035</td>
</tr>
<tr>
<td>Condition</td>
<td></td>
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<tr>
<td>AMI</td>
<td>1360 (11.6)</td>
<td>2223 (14.6)</td>
<td>7816 (22.1)</td>
<td>5999 (25.3)</td>
<td>17,092 (18.7)</td>
</tr>
<tr>
<td>Heart failure</td>
<td>2689 (22.9)</td>
<td>3268 (21.4)</td>
<td>9417 (26.7)</td>
<td>7353 (31.0)</td>
<td>25,622 (29.8)</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>2889 (24.6)</td>
<td>4900 (32.1)</td>
<td>10,480 (29.7)</td>
<td>7353 (31.0)</td>
<td>25,622 (29.8)</td>
</tr>
<tr>
<td>Major surgery</td>
<td>4814 (41.0)</td>
<td>4855 (31.8)</td>
<td>7594 (21.5)</td>
<td>5685 (24.0)</td>
<td>22,948 (26.7)</td>
</tr>
<tr>
<td>Age, mean (SD), y</td>
<td>77.7 (7.8)</td>
<td>78.5 (8.3)</td>
<td>78.9 (8.4)</td>
<td>78.6 (8.5)</td>
<td>78.6 (8.4)</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>5293 (45.0)</td>
<td>6825 (44.8)</td>
<td>15,998 (45.3)</td>
<td>10,677 (45.0)</td>
<td>38,793 (45.1)</td>
</tr>
<tr>
<td>Women</td>
<td>6459 (55.0)</td>
<td>8421 (55.2)</td>
<td>19,309 (54.7)</td>
<td>13,053 (55.1)</td>
<td>47,242 (54.9)</td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>White</td>
<td>10,372 (88.3)</td>
<td>13,400 (87.9)</td>
<td>30,740 (87.1)</td>
<td>20,583 (86.7)</td>
<td>75,095 (87.1)</td>
</tr>
<tr>
<td>Black</td>
<td>785 (6.7)</td>
<td>1049 (6.9)</td>
<td>2869 (8.1)</td>
<td>1957 (8.3)</td>
<td>6660 (7.7)</td>
</tr>
<tr>
<td>Other</td>
<td>595 (5.1)</td>
<td>797 (5.2)</td>
<td>1698 (4.8)</td>
<td>1190 (5.0)</td>
<td>4280 (5.0)</td>
</tr>
</tbody>
</table>

Abbreviations: AMI, acute myocardial infarction; MPSMS, Medicare Patient Safety Monitoring System; VAP, ventilator-associated pneumonia.

* Race obtained from chart abstraction and provided here as a routine component of demographic data.
Results | The VAP rate was studied among 1856 patients. Numbers and characteristics of patients included in the sample during each period are reported in the Table. MPSMS VAP rates were stable over time (Figure), with an observed rate of 10.8% (95% CI, 7.4% to 14.4%) during 2005-2006, 9.7% (95% CI, 5.1% to 14.9%) during 2012-2013, and an adjusted average annual change of 0.00 (95% CI, −0.05 to 0.07).

Discussion | From 2005 through 2013, MPSMS VAP rates remained stable and substantial, affecting approximately 10% of ventilated patients. Persistently high VAP rates bolster concerns that most interventions purported to reduce VAP are supported by limited evidence.5

The data have limitations. The VAP rates were not measured in all hospitalized patients, just the subset included in the MPSMS (patients ≥65 years with 4 specific conditions). The discordance between these findings and the significant declines in VAP rates reported by the NHSN1,2 could in part be due to differences in MPSMS and NHSN measure definitions, hospitals or patient groups, changes in characteristics of hospitals reporting to the NHSN over time, or preferential declines in VAP rates among hospitals reporting to the NHSN.

Nonetheless, the dichotomy between VAP rates reported to the NHSN and measured in the MPSMS supports the concern that surveillance using traditional definitions may be unreliable.3 The ongoing risk to patient safety represented by VAP supports the NHSN’s decision to explore more objective surveillance targets.6

Mark L. Metersky, MD
Yun Wang, PhD
Michael Klompas, MD
Sheila Eckenrode, RN
Anila Bakullari, BS
Noel Eldridge, MS

Author Affiliations: Division of Pulmonary and Critical Care Medicine, University of Connecticut School of Medicine, Farmington (Metersky); Department of Biostatistics, Harvard School of Public Health, Boston, Massachusetts (Wang); Department of Population Medicine, Harvard Medical School, Boston, Massachusetts (Klompas); Qualidigm, Wethersfield, Connecticut (Eckenrode, Bakullari); Agency for Healthcare Research and Quality, United States Department of Health and Human Services, Rockville, Maryland (Eldridge).

Corresponding Author: Mark L. Metersky, MD, Division of Pulmonary and Critical Care Medicine, University of Connecticut School of Medicine, 263 Farmington Ave, Farmington, CT 06030-1321 (Metersky@uchc.edu).

Published Online: November 11, 2016. doi:10.1001/jama.2016.16226

Conflict of Interest Disclosures: All authors have completed and submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Dr Metersky reported that he has worked on various quality improvement and patient safety projects with Qualidigm, the Centers for Medicare & Medicaid Services (CMS), and the Agency for Healthcare Research and Quality (AHRQ). His employer has received remuneration for this work. No other authors reported disclosures.

Funding/Support: This work was supported by contract HHSA290201200003C from the Agency for Healthcare Research and Quality, United States Department of Health and Human Services, Rockville, Maryland. Qualidigm was the contractor.

Role of the Funder/Sponsor: AHRQ employees were involved with the design and conduct of the study; analysis and interpretation of the data; preparation, review, and approval of the manuscript; and decision to submit the manuscript for publication.

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COMMENT & RESPONSE

Lack of Benefit for Liraglutide in Heart Failure

To the Editor

In the Functional Impact of GLP-1 for Heart Failure Treatment (FIGHT) trial, Dr Margulies and colleagues tested liraglutide in patients with advanced, recently decompensated heart failure with reduced left ventricular ejection fraction (LVEF) and found no improvement in clinical outcomes or functional capacity compared with placebo. The lack of benefit and nonsignificant increases in adverse heart failure outcomes in the subgroup of patients with diabetes are reasons for concern.

Liraglutide is clinically indicated to improve glycemic control in diabetes (at the dose used in the study) and for weight loss in patients with obesity (at higher doses). The significant reduction in glycated hemoglobin and reduction in body weight at 30 days and 90 days were therefore predicted effects. As such, the negative results of the study cannot be attributed to a wrong dose or regimen but rather suggest a more complex explanation.

In patients with diabetes and high cardiovascular risk, liraglutide vs placebo showed a significant reduction in the composite end point of cardiac death, myocardial infarction, or stroke, and nonsignificantly lower rehospitalizations for heart failure in the Liraglutide Effect and Action in Diabetes: Evaluation of Cardiovascular Outcome Results (LEADER) study. In patients with clinically stable heart failure, albiglutide, another glucagon-like peptide 1 (GLP-1) agonist, improved peak oxygen consumption measured by ventilatory expired gas but not 6-minute walk test distance.

Why are the results of these trials discordant? The most obvious difference is that the FIGHT study included patients with advanced heart failure (New York Heart Association [NYHA] class IV), whereas in the other studies, patients had no or moderate heart failure (NYHA class II-III), and patients with advanced heart failure were excluded. One possibility is that the effects of GLP-1 agonists may diverge on the basis of heart failure severity. Advanced heart failure is characterized by loss of weight and lean mass, portending an unfavorable prognosis; therefore, further weight loss may have influenced the FIGHT study outcomes. In the LEADER study, liraglutide had more favorable effects in the subgroups with a body mass index (BMI) greater than 30 and without heart failure.

For hypothesis-generating purposes, we would like the authors to provide results stratified by BMI, weight loss, and severity of heart failure (ie, LVEF). If available, a body composition assessment would help determine whether BMI or fat or lean mass at baseline and interval changes predicted any functional improvement. In the era of precision medicine, the assessment of both heart failure severity and body weight or composition is warranted to find the most effective therapy.

Salvatore Carbone, MS
Ross Arena, PhD

Additional Contributions: We thank all the previous and current MPSMS team members for their contributions to this work, with a special thank you to the abstractors and other team members at the CMS Clinical Data Abstraction Center.