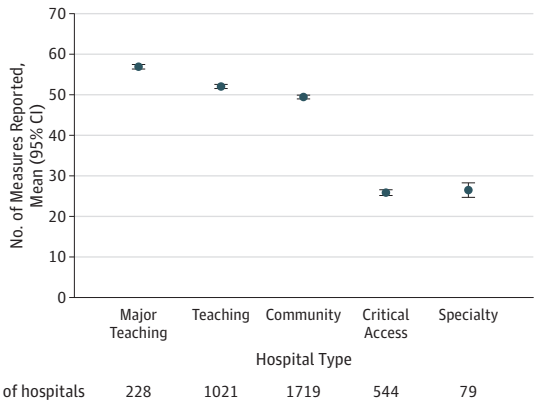
Figure. Mean No. of Hospital Quality Measures Reported (of 62 Total Possible Measures) by Hospital Type[a]



[a] Error bars represent 95% CIs. Includes hospitals that met the reporting threshold of at least 3 measures within at least 1 Outcome group (mortality, readmissions, safety). Using the Kruskal-Wallis test, $P<.001$ for all pairwise comparisons except between critical access and specialty hospitals ($P = .96$).

given to 46.9% of hospitals reporting the minimum threshold of 1 Outcome measure group, 31.2% of hospitals reporting 2 Outcome measure groups, and 25.5% of hospitals reporting all 3 Outcome measure groups ($P < .001$) (Table). Star ratings were based on the minimum eligibility threshold of only 3 of 7 measure groups for 49.4% of specialty and 33.3% of critical access hospitals; whereas, 94.3% of major teaching, 80.8% of other teaching, and 96% of community hospitals reported all 7 measure groups.

Discussion | Hospitals less frequently received a high star rating if they were larger, academic hospitals or cared for a higher proportion of disproportionate share patients. Specialty and critical access hospitals more frequently earned high star ratings compared with acute care hospitals. Critical access hospitals and some specialty hospitals (ie, certain cancer centers) are exempt from reporting-based payment incentives through the CMS Inpatient Quality Reporting system and may not collect many measures used in the star ratings.[4] Consequently, specialty and critical access hospitals reported systematically fewer measures. Although hospital type influenced the number and type of measures reported, the study was limited by the inability to determine whether differences in individual measure reporting by hospital type explained the differences in star ratings.

Because the measures used as the basis for calculating the star ratings differed by hospital type, failure to account for these differences may limit the utility of the star ratings, particularly when comparing different hospital types.

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Acquisition, analysis, or interpretation of data: All authors.
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COMMENT & RESPONSE

Global Burden of Disease Attributable to Hypertension

To the Editor The Global Burden of Disease (GBD) project has made important contributions to the field of public health surveillance. However, when providing cause-specific data, the limitations inherent in global estimates become apparent. In the article on the global burden of hypertension, an anomaly is the large number of disability-adjusted life-years from coronary heart disease assigned to sub-Saharan Africa and many parts of Asia.[1] It is well established that coronary heart disease remains infrequent to rare in sub-Saharan Africa and much of Asia.[2-5] In the supplemental material, the authors stated that the process of risk estimation was "standardized to enhance the comparability of results across risks, outcomes, populations and time," which implies that the same risk coefficients were used for calculations in all geographic regions. This

method will provide biased estimates of cardiovascular outcomes; although the relative risks may be similar across populations, the background of other risk factors, such as smoking and hyperlipidemia, vary and will influence the event rate and the number of deaths from coronary heart disease.[5] In populations like those in sub-Saharan Africa and much of Asia, serum lipids remain low and, in Africa at least, smoking is uncommon.[2-5] The large population-attributable fractions for these regions are therefore likely to be overestimates.

In addition, we question the reliability of this approach to surveillance given the absence of empirical data in many of the countries included. The calculation of events for the cardiovascular end points requires age- and sex-specific blood pressures, an appropriate risk coefficient for each disease category, and knowledge of the population size. These data do not exist for countries in sub-Saharan Africa. Regional estimates were used to impute missing data, but we would argue that there must be some limits to how far that process can be extended. The supplemental tables, for example, provide specific blood pressure values for many countries from which data for either 1990 and 2015 were nil or nonexistent (eg, Myanmar, North Korea, Syria, Kiribati) and for other countries that did not exist at the baseline date (eg, Montenegro, South Sudan). The questionable validity of this approach is apparent in the implausible results obtained for sub-Saharan Africa. We believe it is more informative to restrict disease burden estimates to countries from which adequate data are available.

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In Reply Dr Cooper and colleagues suggest that coronary heart disease remains infrequent to rare in sub-Saharan Africa and Asia. We disagree. The empirical evidence behind their claim is unclear as they only cite a few older studies.

In contrast, the GBD study relied on a larger and more recent set of primary sources regarding mortality due to ischemic heart disease (IHD) in these regions.[1] For sub-Saharan Africa, we used vital registration and verbal autopsy data from 11 countries, which showed the fraction of deaths due to IHD in sub-Saharan Africa was between 2% and 10% of deaths, among the lowest in the world but hardly rare. For Asia, the GBD study added 5 million deaths reported annually through the China Center for Disease Control Cause of Death reporting system from 2004 through 2014, helping to show that the fraction of deaths due to IHD varied widely in Asia based on location, ranging from 5% to 47%.

Cooper and colleagues question whether these effects are modified by levels of tobacco smoking or serum cholesterol in a population. We agree that tobacco smoking and cholesterol levels can modify the effect of elevated systolic blood pressure (SBP) on IHD, although this effect appears to be small.[3,4] We used estimates of the independent relative risks of elevated SBP, after adjustment for other risks, to address this issue.

The GBD study estimated that levels of tobacco smoking and cholesterol are lower in most parts of sub-Saharan Africa compared with other regions of the world, which may partially explain the relatively lower levels of IHD in that region.

In contrast, health surveys performed throughout sub-Saharan Africa in recent years have not shown significantly lower levels of SBP compared with other regions. There are numerous health surveys reporting blood pressure levels in Africa and Asia, including for many of the countries where Cooper and colleagues assumed data were unavailable, for example surveys from Myanmar in 2014 and North Korea in 2008.

Our findings suggest that elevated SBP is an important health hazard in many areas of sub-Saharan Africa and Asia. We disagree that the absence of data from some of these countries prevents us from producing country-specific estimates because geospatial modeling has a strong empirical foundation. Cooper and colleagues appear to suggest that only primary data should be used for global health estimation, and data should only be used just as reported by a country. Unfortunately, such an approach does not address some of the most pressing challenges in global health policy, whereas a model-based approach allows us to evaluate regional trends and identify diseases, such as cardiovascular disease, for which surveillance needs to be intensified. Models of global health burden can include data on risk exposures such as body weight that are both causal for and predict elevated SBP among populations. We believe that with rigorous and transparent evaluation of all sources of information and quantitative estimates of both predictive validity and uncertainty, model-derived estimates like those from the GBD study provide an important platform for integrating all available knowledge on population health. Ongoing efforts to improve these models will be seen in the annual results produced by the GBD collaboration.

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Conflict of Interest Disclosures: Both authors have completed and submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest and none were reported.

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Intubation During In-Hospital Cardiac Arrest

To the Editor In a large retrospective evaluation of intubation during cardiac arrest, Dr Andersen and colleagues suggested that intubation during the first 15 minutes of in-hospital cardiac arrest was associated with decreased survival.[1] Although the authors tried to balance the distribution of groups with statistical analysis, the original groups were significantly different, preventing like groups from being analyzed.

There were twice as many patients in the intubation group (n = 71 615) than in the no-intubation group (n = 36 464). As an example of group difference, the percentage of patients in the no-intubation group who received noninvasive assisted ventilation was 22%, compared with only 4% of intubated patients. In addition, patients who were intubated had a lower percentage of shockable rhythms (15%) compared with patients who were not intubated (24%). Intuitively, patients who have shockable rhythms are more likely to convert to a pulsed rhythm with a higher survival rate and therefore do not need intubation. Also, no analysis (*P* values) was presented to identify significant differences between the 2 groups. We do not believe the authors achieved their goal of minimizing selection bias.

Andersen and colleagues attempted to reduce this bias by propensity score matching[2] and presented 2 matched cohorts in Table 2 in the article. Paradoxically, the number in the matched no-intubation group (n = 43 314) was higher than the total number of patients in the original no-intubation group. Some of the patients initially in the intubation group appear to have been included in the matched no-intubation group in Table 2, which would introduce bias into the analysis.

As Dr Angus alluded to in his Editorial,[3] even data sets of this size are not sufficient to exclude residual confounding. Based on the data presented, we think that there are sufficient differences between the 2 original groups to doubt the authors' conclusions.

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Conflict of Interest Disclosures: Both authors have completed and submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest and none were reported.

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In Reply Dr Wilt and Ms Lee question the validity of our results due to between-group differences in baseline characteristics before propensity matching, specifically mentioning differences in noninvasive ventilation and the initial rhythm. As noted in Table 2 in the article, these characteristics were well matched in the propensity score–matched cohort. Therefore, these variables cannot confound the adjusted results. Furthermore, in our subgroup analysis according to initial rhythm, intubation was associated with poor outcomes in both those with initial shockable and nonshockable rhythms. As noted in the article, there might be unmeasured confounders we were unable to adjust for.

Wilt and Lee are also concerned about the lack of *P* values in Table 1. However, confounding is not related to statistical significance, and *P* values can be misleading with large (or small) sample sizes.[1] We therefore provided standardized differences in Table 2 indicating a well-balanced cohort.[1,2]

In addition, Wilt and Lee write, "Paradoxically, the number in the matched no-intubation group (n = 43 314) was higher than the total number of patients in the original no-intubation group." This is not paradoxical because our analysis used risk-set matching. As stated in the article, "At-risk patients included those who were still undergoing resuscitation and were not intubated before or within the same minute. At-risk patients therefore also included patients who were intubated later, as the matching should not be dependent on future events. As such, the matched group with no intubation includes patients who subsequently were intubated (although later than their matched counterpart)."[3] In the **Figure**, 5 hypothetical patients are schematically outlined. As an example, patient A who was intubated during minute 1 could be matched with any patient at risk of being intubated during minute 1, which includes patients B, C, D, and E. Patient C, who was intubated during minute 3, could be matched only with patient B because none of the other patients were at risk of intubation at this time point. Additional details and the rationale for this approach are provided in the supplemental material in the article as well as in a previous study from our group.[4] In brief, this approach is needed to avoid bias because patients with longer cardiac arrests are more likely to be intubated. As such, a traditional