Regarding the concept of performing psychologic autopsies in all of these cases, although this methodology would certainly expand our understanding of the causality of suicide in each case, the retrospective nature of our study and use of a deidentified registry data source make such a task near impossible. We certainly agree with the comments regarding the need for oncologists and pulmonologists to promptly identify psychiatric symptoms, which may lead to timely and early referral to psychiatrists. Indeed, our motivation for conducting this research was to highlight the importance of this issue within the field of thoracic oncology, and we hope to encourage others to explore options of prospective screening for psychologic distress and indicators of suicidal ideation. Ultimately, we hope that such efforts may lead to a reduction in this preventable cause of death in patients diagnosed with lung cancer.

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Response

To the Editor:

We thank Drs Gnanavel and Robert for their interest in our recent article describing suicide in patients with lung cancer based on the Surveillance, Epidemiology, and End Results database.1 We agree with their comment that a significant proportion of those who committed suicide may have suffered from a comorbid (pre-existing) depressive disorder or other psychiatric illnesses. However, as discussed in our article, there are limitations to the data available in the Surveillance, Epidemiology, and End Results database. In particular, comorbid psychiatric diagnoses are not recorded.

Correspondence

To the Editor:

I read with interest a recent point editorial by Mohr and Doerschug1 published in CHEST (October 2013) on antipyretic therapy for febrile patients in septic shock. The authors stated that “the magnitude of fever has been associated with higher mortality in sepsis,”2 which is incorrect. In fact, results of the cited article2 indicated that the magnitude of fever is associated with higher mortality in patients without sepsis, not in those with sepsis. A correction of the abstract of the original article has been posted.3

Furthermore, as Drewry and Hotchkiss4 pointed out, external cooling for febrile patients with severe septic shock is associated with a trend toward decreased ICU mortality but not hospital mortality.5 Given the very poor quality of a patient’s life in the ICU, survival analyses for short-term outcomes have been considered not appropriate.6 Sepsis-associated mortality is shown to reach a plateau at 90 days after ICU admission.7 Therefore, I believe

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that without evidence of a longer-term benefit, external cooling should not be given routinely to febrile patients in septic shock.

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Response

To the Editor:

We appreciate the comments raised by Dr Shen regarding our editorial on fever control in septic shock.1 Dr Shen specifically highlights an issue with our interpretation of the data presented by Lee et al2 on mortality related to fever. Although the authors concluded that mortality was not significantly associated with maximum temperature, they included afebrile and hypothermic patients in their analysis. Among subjects with fever, there was a significant linear dose-response relationship (P = .02); that is, mortality was 16.9% for a maximum temperature of 37.5°C to 38.4°C and >30% for a temperature >39.5°C. Indeed, the magnitude of fever was associated with increased mortality in this report.

The report by Lee et al2 does not stand alone to illustrate this association. Hodgkin and Sanford1 reported that high fever (>39.4°C) in gram-negative rod bacteremia was associated with a mortality rate of 48%, whereas less extreme fever (37.2°C-38.3°C) was associated with mortality of only 33%. In a cohort of critically ill surgical patients, peak temperature was the most powerful predictor of mortality, and nonsurvivors were more likely to have infection (P = .02) and higher temperatures (P < .001).3 Among patients in the ICU without neurologic injury, the magnitude of fever was strongly associated with mortality (P < .001).4 In a very large study over 7 years (N = 20,466), Laupland et al5 found that high fever was associated with increased mortality.

Dr Shen also points out that the early survival advantage in the trial by Schortgen et al2 was attenuated over time. Although that is accurate, the intervention was time limited to 48 h. We agree that it would be prudent to power a larger trial to detect mortality differences later in the course of disease, which the trial by Schortgen et al2 was not powered to detect. Even so, no existing data support the notion that induced normothermia by external cooling results in the detrimental effects that fever proponents suggest it must. Despite excellent theory, cooling to normothermia has never been shown to increase mortality.

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