of lesions associated with TBI is compounded by the Marshall classification of head injury based on CT scanning. These conditions cause coma when they affect brain perfusion and cortical or brain stem activity.

DAI is only one possible physiopathologic mechanism causing coma after TBI and typically occurs during high-speed impact trauma causing rotational acceleration. This causes axonal damage, as the axons are torn or stretched, thus, impairing axoplasmic transport and, ultimately, the electrical neural network.

It should be noted that, when dealing with DAI, CT scanning can offer some clues to diagnosis but it is often inaccurate in identifying injuries. MRI can clearly detect DAI deep lesions. Moreover, MRI is potentially useful as a clue to prognosis because it assesses the severity of DAI, which is classified as mild (as only corticospinal lesions are apparent), moderate (as corpus callosum is involved), or severe (as brain stem is involved).

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Response

To the Editor:

I thank Dr Gemma and colleagues for more fully explaining the pathologic manifestations of traumatic brain injury in their letter. I was unable to do so in my review of chronic disorders of consciousness following coma because of space limitations.

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Psychiatric Morbidity in Patients With Lung Cancer

To the Editor:

The article by Urban et al1 in CHEST (October 2013) made for an interesting read. We appreciate the authors’ earnest attempt to identify the subpopulation of patients with lung cancer at risk for committing suicide following diagnosis. The authors documented the highest standardized mortality ratios (SMRs) in male patients, older patients, patients with a higher-grade tumor and metastatic disease, and patients who did not receive, or refused, treatment. Despite the higher SMR among patients with metastatic disease, the finding that >50% of suicides occurred in those with locoregional and potentially curable disease and that a majority of suicides occurred within 3 months of diagnosis raises a great degree of concern.

It is possible that a significant proportion of the subset of patients with lung cancer who committed suicide despite locoregional or potentially curable illness also suffered from comorbid (preexisting) depressive disorder or other psychiatric illnesses. Various studies have reported the presence of suicidal illness in the majority of suicide completers in the general population, ranging from 81% to 100%.2-4 A few studies have documented depressive disorder as a risk factor for the development of lung cancer. For example, Chen et al5 found depressive disorder in 4.91% of subjects at risk for lung cancer. Hence, it is highly likely that not adjusting for psychiatric disorders, particularly depressive disorder, as a confounding variable in this population would have resulted in an inaccurate estimation of SMR.

It would have been ideal had the authors carried out psychologic autopsy in addition to their study of registry data. Although a cumbersome procedure, psychologic autopsy is a valuable research tool in completed suicides that retrospectively collects all available information on the deceased patient through structured interviews of family members, relatives, friends, and treating health-care personnel. In addition, information is collected from available health-care or psychiatric records, other documents, and forensic examination. Thus, a psychologic autopsy collaterally synthesizes information from various sources, providing clues regarding possible psychiatric morbidity prior to suicide apart from a systematic
documentation on the suicidal act.6 These data would have been helpful in addressing the issue of psychiatric morbidity as a potential and significant confounder in the estimation of SMR in this group of patients.

Prompt identification of psychiatric symptoms, particularly self-harm ideation and depressive cognitions like ideas of hopelessness and hopelessness, are likely to lead to early referral to psychiatrists. Sensitization of pulmonologists or oncologists is required to avoid this preventable cause of death in patients 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.

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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**REFERENCES**


**Fever Control and Sepsis Mortality**

**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,” 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 Drevry 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.2 Given the very poor quality of a patient’s life in the ICU, survival analyses for short-term outcomes have been considered not appropriate.5 Sepsis-associated mortality is shown to reach a plateau at 90 days after ICU admission.2 Therefore, I believe...