Response

To the Editor:

We thank Dr. Barie and his colleagues for their interest in our data.\(^1\) It is satisfying to note that other investigators also have had a similar experience regarding the influence of nonpulmonary organ dysfunction and failure on outcome in patients with respiratory failure. Contrary to our findings, they have reported\(^2\) that hepatic dysfunction had a significant influence of patient mortality in the patients they studied. The study appears to have been conducted at a primarily surgical ICU. Unfortunately, we do not have access to the detailed methodology and results of the abstract cited by Barie et al.\(^3\) in this regard. In another study,\(^4\) on patients with ARDS who were managed at a surgical ICU, they found that the dysfunction of all nonpulmonary organs contributed to an adverse outcome. The difference in the quantum of contribution of individual organ dysfunction to overall mortality is perhaps, therefore, related to the variability in case mix at their ICU and ours. Additionally, we used sequential organ function assessment (or SOFA) scores, and they used multiple organ dysfunction (or MOD) scores. Some differences in findings could therefore also be explained by variations in the definitions used to describe and stratify individual organ dysfunction. Despite these minor differences, we concur with them regarding the importance of recognizing and managing nonpulmonary organ dysfunction in patients with respiratory failure.

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REFERENCES


Patient-Focused Sedation and Analgesia

To the Editor:

The article by Sessler and Varney, \(^1\) “Patient-Focused Sedation and Analgesia in the ICU” (February 2008), was brilliant. For decades, the concerns of sedation and analgesia in the ICU have troubled me. During the past year, I have been extensively investigating patient risk specific to sedation and analgesia in multiple environments.

Beyond the long-term psychological outcomes, there is the matter of the pharmaceutical impact on respiratory depression. During mechanical ventilation, breathing is controlled. Aside from the weaning complexities that may result from sedation and analgesia, there remains the burden of managing postextubation hazards specific to respiratory depression. Once extubated, patients’ pain and anxiety remain and are therapeutically treated.

Certainly, patients are closely monitored in the ICU after mechanical ventilation. Pulse oximetry and ECG monitoring are ubiquitous to the critical care arena. Nevertheless, respiratory depression is a risk during sedation even in the ICU.

In a report presented at the Society for Technology in Anesthesia Annual Congress held in San Diego this January, researchers from the University of Alabama at Birmingham presented a metaanalysis\(^2\) of patient safety during sedation and analgesia. They discovered these patients are at 28-times greater risk for having unrecognized respiratory depression when not monitored using capnography. They added that pulse oximetry alone might be dangerous.

It is important to manage pain in the ICU. Our tools for assessing pain and thus reducing it are part of a strategy to improve the total health-care experience for the patient. Ventilation monitoring after extubation should incorporate the diagnostic systems available to the ICU care provider. As an early warning to respiratory depression, during sedation and analgesia, capnography is a faster indicator than pulse oximetry.\(^3,4\) Furthermore, relying on respiratory rate alone without a measure of the adequacy of ventilation, such as exhaled carbon dioxide levels, is of limited clinical value.\(^5\)

Capnography is indispensable as a rapid monitor of respiratory depression that can be pharmaceutically imposed.\(^6\) Many bedside monitors allow caregivers to use capnography in the ICU. During critical care stays, patients may benefit from early indicators of impending respiratory depression; capnography is the fastest method to alert caregivers of respiratory depression. So long as we provide sedation and analgesia in the ICU, certain patients will remain in jeopardy for respiratory depression, and strategies can be in place to reduce that risk. Thank you Drs. Sessler and Varney for your timely and thought-provoking article.

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Tako-Tsubo Cardiomyopathy and Drowning Syndrome

Is There a Link?

To the Editor:

We read with great interest the recent article in CHEST (September 2007) on tako-tsubo cardiomyopathy (TTC) by Kurowski et al and would like to congratulate the authors for their elegant study. In 35 patients with TTC, they reported the occurrence of emotional stress events in 15 patients (42.8%) including a case of near-drowning syndrome (NDS). In our clinical series of 52 patients (51 women; mean [± SD] age, 63.58 ± 10.55 years), an emotional stress event was documented in 43.2%. We also registered a 57-year-old woman presenting with TTC after the occurrence of NDS. A few hours after the event, ECG changes developed, and a typical echocardiographic pattern of apical ballooning with a mild increase in serum troponin level was seen. Left ventriculography confirmed the apical ballooning, and coronary angiography revealed normal coronary arteries. ECG changes and apical contraction abnormalities were completely reversed within 3 weeks. Drowning is an extremely stressful situation that leads, via the uncommon combinations of different pathophysiologic mechanisms, to sympathetic nerve activation (SNA). In patients with NDS, submersion in liquid causes hypoxemia due to fluid aspiration or reflexive laryngospasm. Once hypoxemia occurs, cerebral hypoxia, pulmonary reflexes, and concomitant panic and/or struggle induce SNA. Hypoxemia related to NDS could have induced transient myocardial dysfunction mediated by an SNA. Although the pathogenesis of TTC remains unclear, a common pathophysiologic pathway seems to be an exaggerated sympathetic activation. Thus, SNA could be considered the “real” link between NDS and TTC.

From a clinical point of view, it should be kept in mind that prolonged but reversible anterior-apical contraction abnormalities without significant coronary artery disease may also occur in critically ill patients who have been hospitalized because of a wide variety of primary noncardiac illnesses (ie, neurogenic myocardial stunning). NDS should therefore be added to the already long list of stressful conditions that can lead to the development of TTC. Patients surviving the drowning represent a very interesting and challenging population with potential severe cardiac complications, including malignant arrhythmias and myocardial dysfunction. Thus, in the critical care setting, careful monitoring is needed in order to implement the appropriate diagnostic and therapeutic interventions.

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Late Pulmonary Hypertension

Looking Beyond the Scene

To the Editor:

Although many noninvasive laboratories are introducing exercise Doppler echocardiography (Ex-Echo) as a standard technique to unmask latent or presymptomatic pulmonary hypertension (exercise-induced pulmonary hypertension [Ex-PHtn]), several important issues need redress.¹,²

1. A consensus should be reached on the optimal exercise protocol (treadmill vs supine/upright/semirrecumbent bicycle) to be implemented since each protocol is characterized by different loading conditions. At the present time, semirecumbent exercise echocardiography appears to be more suitable for reliable and reproducible echo-Doppler measurements considering that measures are made during exercise and recovery.
2. There is a need to define the full physiologic range of pulmonary pressure responses to both bicycle and treadmill exercise in relation to age, gender, body mass index, and level of physical training.³ In highly trained athletes, a high workload is associated with moderate increases in pulmonary artery systolic pressure as a direct consequence of increased stroke volume and left ventricular filling pressures.³,⁴
3. An emerging question is who to screen for Ex-PHtn, knowing the yield of a screening examinations depends not only...