especially when used in the context of the clinical suspicion of myocardial ischemia, and negative troponin results are still valuable in excluding myocardial damage.

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9 Kollef MH, Ladenson JH, Eisenberg PR. Clinically recognized cardiac dysfunction: an independent determinant of mortality among critically ill patients; is there a role for serial measurement of cardiac troponin I? Chest 1997; 111:1340–1347

To the Editor:

We appreciate the interest in and comments on our recent article in CHEST (May 2004)1 by van Bockel et al. Unfortunately, the reports that they cited2,3 had not yet been published at the time of our review and thus were not included. However, Ammann et al4 recently reported higher levels of tumor necrosis factor-α, its soluble receptor, and interleukin-6 in troponin-positive critically ill patients compared to those who were negative for troponin. Therefore, we believe that controversy still exists, at the present time, regarding the role of cytokines in troponin release in this subset of patients.

We also maintain our opinion on the prognostic value of elevated troponin levels in sepsis and/or critically ill patients. In addition to the several reports cited in our review, two recent studies by Ammann et al5 and Wu et al6 found troponin to be an important risk factor for mortality in these patients. As stated by Dr. van Bockel and colleagues, a study by Kollef et al7 found clinically recognized cardiac dysfunction but not elevation of troponin levels to be an independent predictor of mortality in their patients. However, Kollef et al8 defined clinically recognized cardiac dysfunction as the presence of cardiac arrest, congestive heart failure, unstable angina, or myocardial infarction. We believe that their study design may have underestimated the prognostic value of troponin levels.

Finally, we agree that further studies are needed, and we laud van Bockel and colleagues for their effort at elucidating the mechanism and importance of the elevation of troponin levels in septic and critically ill patients.

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ICU Transport

Interhospital Transport of Critically Ill Patient With Dedicated Intensive Care Ventilator

To the Editor:

The use of inhaled prostacyclin to enable the safe interhospital helicopter transport of a patient with ARDS complicated by life-threatening hypoxemia was described by Reilly et al (April 2004). Their efforts to optimize the patient for transport to provide optimal care should be acknowledged. However, their choice for an unconventional ARDS therapy like inhaled prostacycline, not initially administered during treatment in their department, could be debated. Instead of a pharmacologic

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therapy with potential side effects despite their clinical experience, a dedicated intensive care (IC) ventilator mounted on an IC transport trolley (a mobile ICU) could have been used to facilitate this high-risk air medical transport. Mechanical ventilation was possible in the ICU before departure but not with the transport ventilator, which points to an (transport) equipment limitation rather than adverse effects of transport itself.

The inability of transport ventilators compared to ICU ventilators to adequately ventilate ARDS patients has been recognized and is probably due to pneumatic characteristics. Gas compression in the ventilatory circuit, technical limits of the Venturi flow delivery system with pressurization gas source with cylinders, or insufficient delivery of positive end-expiratory pressure could cause the failure to adequately ventilate an ARDS patient with transport ventilators. Therefore, IC transfers should be executed with mobile ICUs equipped with IC ventilators for the continuation of critical care on the move.

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Amsterdam, the Netherlands

To the Editor:

Thank you for the opportunity to respond to the letter you received from Drs. Lieshout and Vroom about our report published in CHEST (July 2004). We appreciate the critique of Drs. Lieshout and Vroom, who suggest that the use of a mobile ICU equipped with a more sophisticated ICU ventilator might be preferable to our use of inhaled prostacyclin to enable the ICU equipped with a more sophisticated ICU ventilator might be an advantage of not only minimizing out-of-hospital time, but also transport provides a much more rapid rescue system, and has the advantage rather than adverse effects of transport itself.

The inability of transport ventilators compared to ICU ventilators to adequately ventilate ARDS patients has been recognized and is probably due to pneumatic characteristics. Gas compression in the ventilatory circuit, technical limits of the Venturi flow delivery system with pressurization gas source with cylinders, or insufficient delivery of positive end-expiratory pressure could cause the failure to adequately ventilate an ARDS patient with transport ventilators. Therefore, IC transfers should be executed with mobile ICUs equipped with IC ventilators for the continuation of critical care on the move.

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Thank you for the opportunity to respond to the letter you received from Drs. Lieshout and Vroom about our report published in CHEST (July 2004). We appreciate the critique of Drs. Lieshout and Vroom, who suggest that the use of a mobile ICU equipped with a more sophisticated ICU ventilator might be preferable to our use of inhaled prostacyclin to enable the interhospital helicopter transport of a very hypoxic patient. We feel that our use of prostacyclin is a superior approach in our health system for multiple reasons.

Several years ago our health system invested in a helicopter transport system, rather than a mobile intensive care ground transport ambulance, to support our level I trauma program, which is also utilized to transport appropriate critically ill nontrauma patients. Air transport provides a much more rapid rescue system, and has the advantage of not only minimizing out-of-hospital time, but also allows us to extend our tertiary care services to a much larger geographic community of patients. In the example of the patient described in our report, the estimated travel time from Princeton, NJ, to Philadelphia, PA, was cut by 1.5 to 2 h.

Since the frequency that our flight crews encounter a critically ill hypoxic patient that cannot be supported safely with our transport ventilator is approximately 1% of all transfers (approximately two patients per year), there is no justification for our health system to also invest in the additional equipment and staffing to support a mobile intensive care ground transport unit (at an additional estimated cost of $300,000 to $400,000 dollars per year). The benefit that this additional resource would provide us, as Drs. Lieshout and Vroom point out, is that it would enable our transport team to prescribe for the patient the same level of ventilatory support that they were receiving while at the ICU of the transferring hospital; however, as mentioned, we estimate that this would be necessary for only approximately two patients per year. In contrast, at a fractional cost of approximately $300/yr, for prostacyclin and disposable equipment (no additional staffing required), we can potentially improve oxygenation during transport for these few patients, even above the levels achieved at the referring hospital, despite the use of a less sophisticated ventilator. We realize, based on data from ICU patients treated with prostacyclin, that the magnitude of response in oxygenation seen in our patient will not be realized in all patients; however, given the simplicity of a prostacyclin trial, the rapidity of the response and the opportunity cost, we believe it is the most appropriate intervention to employ in this circumstance.

For hospitals that only use a mobile intensive care ground transport unit to transfer critically ill patients our demonstration of inhaled prostacyclin use during transport remains relevant, since acute arterial desaturation may still occur during ground travel even though support is being provided with an ICU ventilator. In summary, inhaled prostacyclin provides a quick, simple, inexpensive, and very safe way to treat life-threatening hypoxic events in the small minority of patients where this may occur during transport outside the ICU, analogous to its use as a salvage treatment for this problem within the ICU.

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Airway Hyperresponsiveness and β2-Adrenoceptor Genotypes and Diplotypes at Positions 16 and 27

To the Editor:

Litonjua and colleagues1 (July 2004) examined the association between airway hyperresponsiveness and β2-adrenoceptor polymorphism at positions 16 and 27 in >500 white men from Boston, MA. However, it is curious that no subgroup analysis was performed to examine the distribution of β2-adrenoceptor genotypes and diplotypes in the asthmatic vs the nonasthmatic population. Although we agree that airway hyperresponsiveness is not synonymous with asthma, it would have been of great interest to have known the genetic distribution especially of the arginine 16 genotype and diplotype in the asthmatic vs the nonasthmatic population. We realize, based on data from ICU patients treated with prostacyclin, that the magnitude of response in oxygenation seen in our patient will not be realized in all patients; however, given the simplicity of a prostacyclin trial, the rapidity of the response and the opportunity cost, we believe it is the most appropriate intervention to employ in this circumstance.

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