Editor—We would like to thank Drs Mausser and Schwarz for their interest in our article.  

We would agree that as with all laboratory investigations, extrapolation of findings to clinical practice must be carefully guarded and is certainly not appropriate to small infants, neonates, or obese patients. However, our aim was to add to knowledge of the basic physical processes involved during HFJV and not to promote a preferred way of administration of HFJV. The particular advantage of our model is its simplicity which allows us to study different configurations under controllable and reproducible steady-state conditions. We recognize the limitations of the model and problems of direct extrapolation of the results, but would note that high pressures or hyperventilation have been previously reported in animal and human studies using HFJV. These points are clearly stated in the discussion.

Our main conclusion was that air entrainment is likely to be responsible for the higher airway pressures observed during ASV and a lack of entrainment during BSV results in lower airway pressures. The degree of stenosis also has a significant effect on entrainment, delivered volumes, and pressures distal to the stenosis. We would congratulate Drs Mausser and Schwarz on their clinical series using SHFJV but feel it is impossible to directly compare SHFJV with HFJV: SHFJV steady state is difficult if not impossible to define, the jet is placed laterally in relation to the airway inlet, and the distance from the jet orifice to the airway inlet is longer. Furthermore, the jet laryngoscope used during SHFJV described by Rezaie-Majd and colleagues encroaches on the supralaryngeal area; air entrainment (or obstruction to outflow of gases) cannot be assumed to be similar to ASV using a cannula. Although these authors stated that no barotrauma has been detected in supraglottic SHFJV, we note that recordings of airway pressure were presented for only 13 patients (out of 1515) and would suggest that it is impossible to draw any conclusions regarding airway pressures on such low numbers. In addition, the results of that paper and our own are not directly comparable because of differences in ventilator settings, degree of stenoses (which are not stated), and other clinical variables.

In contrast to Rezaie-Majd’s assumptions that: “The pressure below the stenosis cannot be higher than the pressure above the stenosis with any supraglottic technology. Stenosis will reduce the inflow of jet gas, and the resulting distal airway pressure behind the stenosis will be reduced as well”, our data show that the converse is true using ASV. We agree that further work is required before our results could be safely applied to clinical practice. However, when choosing a method of ventilation in cases of upper airway stenosis,
we would strongly recommend caution and attention to the details of configuration of the interface between the ventilator and the respiratory spaces of the patient’s lung.

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Changes in S100B levels rather than absolute values may be a better marker of severity of septic encephalopathy

Editor—We read with interest the article by Piazza and colleagues, where they report that elevated S100B levels did not correlate with the severity of encephalopathy during sepsis. We would like to ask the authors that even though the S100B levels did not correlate with the severity of encephalopathy on ICU admission, whether the rate of increase in serum S100B levels could be indicative of severity of encephalopathy. The time of ICU admission is a variable in itself, in terms of when the duration of sepsis began. In fact, a third of the patients, three with GCS scores ≤8, had normal range S100B levels at the time of admission. We assume that these values did not remain at normal levels but proceeded to increase. Investigating the rate of change of serum S100B levels at an individual level would allow the removal of inherent individual variation due to sex or to age—which in this study spans 47–84 yr.

S100B expression is not restricted to neuronal tissues and serum S100B levels have been shown to be increased after bone fractures or after ischaemia of the liver, gut, or kidney. Therefore, S100B may be elevated during sepsis alone, and it may be that the rate of release of serum S100B is higher if there is an underlying encephalopathy, in addition to sepsis. This rate of increase in serum S100B levels may be a more sensitive readout of ongoing pathology, with high rate of increase indicating an underlying brain encephalopathy. In the present study, the average S100B levels seem to be increased at day 7 compared with levels at ICU admission, with a larger spread indicating some patients have lower levels whereas other patients have much higher levels than the average. It would be interesting if this rate of release of S100B was an indicator of prognosis, where if S100B levels are still increasing by day 7, prognosis may be poor and a slight increase or decrease would indicate a better prognosis.

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Editor—Drs Panni suggest that S100B changes rather than absolute values may be a better marker of severity of sepsis-associated encephalopathy. They assume that comatose patients who have normal S100B values on ICU admission may proceed to higher values later. As previously described, our sepsis patients showed alteration of consciousness at admission; they did not develop it during their ICU stay. In our experience, S100B was measured at ICU admission and after 3 and 7 days: there was not any correlation with neurological outcome at any time point. Any prognostic marker should be precocious and specific: we respectfully disagree with Drs Panni when they suggest that the rate of S100B increase after 7 days of coma may add useful information in prognosis formulation but we support the hypothesis that S100B serum levels measured by commercial ELISA kit do not allow the observers to distinguish brain S100B release from peripheral tissues production.

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