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In Reply:

We reported that a "triple low" of mean arterial pressure, minimum alveolar concentration (MAC), and Bispectral Index (BIS) is associated with a fourfold increase in 30-day all-cause mortality compared with patients without low values.[1] Yu and Liu point out that an association between triple lows and mortality may not be causal. We emphasized this obvious point in our article: "as in all registry analyses, it is impossible to make causal conclusions from these observations."

Nonetheless, our results indicate that two "double low" combinations and a triple low of mean arterial pressure, BIS, and MAC strongly predict postoperative mortality. We agree that triple low events are probably mostly markers for underlying disease. But perhaps some mortality can be prevented by anesthetic management—which remains an intriguing possibility.

Yu and Liu also comment that we did not present baseline risk factors for various combinations of "single," double, and triple low combinations. We instead used a sophisticated multivariable model to adjust for baseline factors. This is a more useful approach than stratifying by risk because, to the extent that relevant potential confounders were included, results in each patient group can thus be directly compared. The more important question is whether all relevant confounders were included. Surely some are missing because even a dense registry such as ours does not include every potential predictor. Importantly, though, we included Risk Stratification Indexes; these powerful predictors of mortality and hospital length-of-stay are based on 240 and 1,096 International Classification of Disease version 9 codes and thus subsume considerable patient-level baseline and procedural detail.[2]

Yu and Liu state that we did not include intraoperative blood transfusion as a risk factor in our analysis. In fact, we did and our article specified that variables for each model were selected using forward conditional selection from a candidate pool containing "age, gender, race, body mass index, American Society of Anesthesiologists physical status, along with intraoperative factors including case-average estimates of blood concentration of propofol and fentanyl equivalents, estimated blood loss and administered erythrocyte volume, type of maintenance volatile anesthesia, whether nitrous oxide was used, and case duration." Transfused blood volume remained in our final model for mortality and was reported in table 3.

Postoperative troponin values are available only for a small fraction of nonrandom patients and thus could not be included. But even if they were available, troponin elevations are consequences of damage already done. And besides, postoperative laboratory values cannot be the basis for intraoperative management enhancements that might improve outcome—which is our real interest.

We agree that only a clinical trial can determine whether intraoperative intervention to prevent or ameliorate triple low events actually improves outcomes. One (NCT00998894) is already in progress.

Lotz and Kehl assert that we did not include cause of death, transfusion requirement, or American Society of Anesthesiologists physical status score in our analysis. As mentioned above, we did include transfusion requirement and American Society of Anesthesiologists physical status in the pool of candidate risk factors; details that were specified in the Methods section of our article. Physical status was retained in both the length-of-stay and mortality models as reported in tables 2 and 3. Lotz and Kehl also assert that we neglected case duration and complexity. As specified above, duration was directly included in our statistical model; and complexity is subsumed into the Risk Stratification Indexes. Cause of death is of interest, but date of death was obtained from the Social Security Death Index, which does not include mortality cause.

Lotz and Kehl warn "not to injudiciously confound low BIS values as a pure reflection of anesthetic depth in the critically ill." We fully agree and our article identified that there are "three potential causes of low BIS: (1) Low BIS is the normal response to generous doses of volatile anesthetics; (2) An alternative cause of low BIS is anesthetic sensitivity. This group is identified by the combination of low BIS and low MAC fraction. This is an atypical response because low MAC fraction should be associated with high BIS. That BIS was in fact low in some patients with low MAC fractions suggests an abnormal sensitivity to volatile anesthesia, potentially because of underlying illness; and (3) a third potential cause of low BIS is inadequate brain perfusion, resulting in ischemic suppression of brain metabolism. Brain hypoperfusion may especially occur in a fraction of patients who demonstrate low BIS combined with low mean arterial pressure." This last group is potentially the most interesting because brain hypoperfusion should be preventable with adequate hemodynamic control.

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Airway Anatomy of AirSim High-fidelity Simulator

To the Editor:

We read the article by Schebesta *et al.*[1] with great interest because it is the first study to compare airway simulators with normal human anatomy using objective anatomical measurements.

Previous studies have compared the effectiveness of four airway simulators, including Airway Management Trainer (Ambu, St Ives, United Kingdom), Airway Trainer (Laerdal, Stavanger, Norway), AirSim (Trucorp, Belfast, Northern Ireland), and Bill 1 (VBM, GmbH, Sulz, Germany) for demonstrating the LMA-Classic™ (LMA North America Inc., San Diego, CA), other supraglottic airway devices, difficult airway management procedures, and other advanced airway skills.[2–5] The results of these studies have rated AirSim as one of the better devices. Given the previous favorable ratings for the AirSim simulator, we were surprised that AirSim was not included in the study by Schebesta *et al.*[1]

At our institution, we are currently using the AirSim Bronchi simulator for training residents to place supraglottic airway devices, single lumen endotracheal tubes, double lumen endobronchial tubes, and bronchial blockers.[6]

Table 1. Airway Dimensions of 20 Patients from the Study by Schebesta *et al.*[1] Compared with High-fidelity AirSim Patient Simulator (Trucorp, Belfast, Northern Ireland)

	Patient		
	Mean (SD)	95% CI	AirSim
Palate			
Cross-sectional area, cm^2	2.7 (1.0)	2.3–3.2	2.7
Curved length, cm	3.7 (0.6)	3.4–4.0	2.4*
Height, cm	2.7 (0.7)	2.4–3.1	2.6
Center–pharynx, cm	1.5 (0.2)	1.4–1.6	1.6
Palate–epiglottis, cm	4.4 (0.9)	4.0–4.9	4.6
Palate–vallecula, cm	5.7 (1.0)	5.3–6.2	5.6
Epiglottis			
Anterior length, cm	1.6 (0.4)	1.4–1.7	1.4
Posterior length, cm	2.8 (0.7)	2.5–3.2	2.9
Tip–pharynx, cm	0.9 (0.4)	0.7–1.1	0.8
Vallecula–pharynx, cm	1.7 (0.6)	1.4–2.0	1.4
Total for palate and epiglottis (10 measures)			9/10
Tongue			
Cross-sectional area, cm^2	25.6 (3.7)	23.8–27.5	15.1*
Horizontal diameter, cm	6.3 (0.7)	5.9–6.6	5.9
Coronal diameter, cm	4.4 (0.5)	4.2–4.7	3.1*
Edge–pharynx, cm	1.6 (0.7)	1.2–1.9	2.2*
Overall distances			
Teeth–pharynx, cm	8.1 (0.6)	7.9–8.4	8.6*
Lip–pharynx, cm	9.5 (0.6)	9.2–9.8	11.0*
Total for tongue and overall distances (six measures)			1/6
Volumes			
Oral airspace, cm^3	4.3 (5.3)	1.9–6.8	93.3*
Retropalatal airspace, cm^3	5.1 (2.0)	4.2–6.1	5.8
Pharyngeal airspace, cm^3	13.5 (7.7)	9.9–17.1	26.2*
Total for volumes (three measures)			1/3
Total for all measures (19 measures)			11/19

* Values above or below 95% CI (level of clinical relevance).