inconsistent.[3] Finally, even if mediators play a role in propagating lung injury, the most important clinical aspect is that simply reducing tidal volume has resulted in a marked reduction in mortality from adult respiratory distress syndrome, whereas to date all clinical trials of antimediator therapies in critically ill patients have been negative.

Competing Interests

The authors declare no competing interests.

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In Reply:

We thank Drs. Dreyfuss and Saumon for their comments on our recent work on one-hit models to study the biotrauma hypothesis.[1] I agree that our study supports some of his earlier concerns and that my view has been too simplistic. However, it is important to note that our recent work does not discredit the biotrauma hypothesis itself. What our work suggests is that the biotrauma hypothesis is difficult to study in one-hit models using ventilation as the only hit, because in such models, there is either mild inflammation without lung injury or severe mechanical injury followed by secondary inflammation. One-hit models, therefore, do not well recapitulate the clinical situation where injured and inflamed lungs are exposed to a second proinflammatory stimulus, namely ventilation.

To me, the biotrauma hypothesis still offers a relevant explanation for the findings of the low tidal volume Acute Respiratory Distress Syndrome Network (ARDSnet) trial.[2] In that study, neither barotrauma, oxygenation, nor hypercapnia correlated with mortality—only inflammation did.[2,3] Similar correlations were found in a second, independent trial.[4,5] For obvious reasons, such studies cannot be repeated, and we will need complex and more realistic experimental animal models mimicking intensive care unit–like conditions to understand the complex interplay between ventilation and inflammation in patients with adult respiratory distress syndrome. In contrast to Dr. Dreyfuss, I believe that such studies are possible.

Competing Interests

The author declares no competing interests.

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Anesthesia, Consciousness, and Language

To the Editor:

I was fascinated to read the recent paper and editorial concerning anesthesia and consciousness, and I wondered whether we might learn more about the effects of anesthesia if we consider one of the brain's most impressive faculties—that of human language.[1,2] There have been a number of reports of patients fixating on a second language while under the effects of anesthesia, either during sedation or sometimes for hours postoperatively.[3–8] In all cases, the switching of the production of speech to exclusively the patient's second language appears to be a direct and involuntary effect of anesthesia, one that spontaneously

resolves without sequelae once recovery is complete. Patients often report not being able to remember speaking in their second language after the fixation event, and more intriguingly, even deny an ability to speak their second language at all (when not having spoken it voluntarily for many years).

It is well known that the production of a first language is associated with Broca's area, while production of a second language that is acquired after approximately the age of seven can involve a number of cortical areas in both hemispheres.[9] I previously have hypothesized that this fact might explain why it is that language switching under the effects of anesthesia appears to occur in only one direction, that is, from first to second language.[9] The idea being that if there are a number of scattered brain areas associated with a second language, but only one area for a first language, there may be a greater chance that an anesthetic agent that differentially affects brain structures could impair the first language while functionally sparing some of the more numerous areas associated with the second language.

Hashmi *et al.* provide quantitative evidence suggesting that something like this may, in fact, be occurring.[2] If loss of consciousness is due to the blocking of certain information "hubs" in the brain, reducing the efficiency of global information transfer, even in the face of remaining local brain activity, then Broca's area is likely to be such a hub for the production of the patient's first language. With a blocked first-language hub, local networks that remain active may contain some of the more numerous areas capable of producing the patient's second language. This also could explain why patients often do not remember speaking in their second language, given the impairment of global information networks and conscious awareness. This language switching phenomena may be underreported. If the patient's second language is not recognized by those attending the patient at the time, it is likely that the fixation event would simply be put down to postoperative confusion (in three of the known cases only a single staff member spoke the patient's second language). If a way could be devised to study this rare phenomenon more systematically, the large number of anesthetic procedures conducted throughout the world every day would present a valuable opportunity for a natural experiment with the potential to tell us much about language and consciousness.

Research Support

Support was provided solely from institutional and/or departmental sources.

Competing Interests

The author owns a small number of shares in Safer Sleep Ltd. (London, United Kingdom), a company that aims to improve safety during anesthesia, but this is unrelated to the topic of this letter.

This letter was sent to the author of the editorial view referenced above (Mashour), who declined to respond. —Evan D. Kharasch, M.D., Ph.D., Editor-in-Chief

This letter was sent to the author of the original article referenced above (Hashmi). The authors declined to reply, explaining that the subject matter referenced is beyond the scope of their expertise. —Evan D. Kharasch, M.D., Ph.D., Editor-in-Chief

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Ideal Body Weight Is Not Really Ideal

To the Editor:

As Moreault *et al.*[1] recently discussed in this journal, some anesthetic management guidelines as well as many drug dosing regimens[2] are based on a patient's ideal body weight (IBW). Despite being an important measure in clinical practice, there is no consensus as to what IBW really represents or how to calculate it.[3] IBW has no physiologic basis and there is no single weight that is ideal for any patient of a given height.[4] For both men and women, IBW often is described as a body mass index (BMI; BMI = kg/m^2) between 20 and 25 kg/m^2. BMI is not a measure of adiposity because it considers weight irrespective of the source, and excess amounts of fluid, muscle, and bone can each increase BMI.

Given that adipose is poorly perfused and contributes minimally to metabolism, fat-free mass or lean body weight (LBW) would be a better measure for clinical purposes. LBW can be obtained using dual-energy x-ray absorptiometry, but measurements are cumbersome and difficult to apply in clinical medicine.[5] The many formulae currently used to estimate IBW give widely different values for the same patient.