In Response:

There are obviously controversies about the possible effect of several anesthetics on platelet aggregation, perhaps due to the different technical approaches and interpretations of the results from study to study. Our investigation was in vitro. In no part of our paper do we extrapolate directly to in vivo conditions. Our results only describe a different behavior of propofol in vitro between two types of samples: isolated platelets and whole blood. In whole blood, erythrocytes and/or leukocytes play an important role in platelet function and in its inhibition by drugs. The in vitro effect of propofol is influenced by the nitric oxide-cGMP pathway interaction with platelet-leukocyte (1). For that reason, we think that whole blood aggregometry could demonstrate better than isolated platelets the inhibitory effect of propofol.

Even more, we obtained recent in vivo results in whole blood aggregometry in patients who received a bolus of propofol (1). In these patients, platelet function in isolated platelets was not modified statistically, but using whole blood aggregometry, we observed an inhibitory effect 5 min after the propofol bolus (50% effective dose for collagen 0.5 μg/mL before bolus, 1.9 μg/mL after bolus); moreover, plasma nitrites (as indicators of nitric oxide production) were increased after the propofol bolus.

In conclusion, our study is the first step of an investigation that requires other consecutive steps to obtain a definitive conclusion.

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References

Failure of a Noninvasive Automated Blood Pressure Monitor to Detect the Blood Pressure After Repositioning of the Patient's Arm

To the Editor:

We describe the failure of a noninvasive automated blood pressure (NIABP) monitor to detect the blood pressure after we repositioned a patient's arm. The patient sustained blunt trauma to the abdomen, presented in shock to the emergency center, was resuscitated, and was hemodynamically stable on transfer to the operating room. In the OR, the patient was placed on the table with his arms abducted at 90° on padded armboards. Vital signs, including blood pressure, were normal. Before induction, we repositioned the patient's arms to his sides, using elbow padding and the draw sheet. The NIABP monitor (Hevwlett Packard, Andover, MA) failed to measure the blood pressure and did not recycle automatically after this failure. The unwavering signal from the Spo2 monitor, applied to the arm on which we had placed the blood pressure cuff, alerted us to this failure. Trouble-shooting included examining the tubing for kinks, changing the NIABP electrical module, palpating the NIABP bladder during manual cycling of the device, and, finally, changing the blood pressure cuff. After removing the cuff, we noted a small hole at the junction of the nipple fitting and the bladder in the NIABP cuff (DURA-CUF™, Johnson & Johnson, Arlington, TX; Fig. 1). The leak was not present until the patient's arm was repositioned and shear forces partially detached the nipple from the cuff. The leak prevented an automatic determination of the blood pressure. The alarm for such a failure was not obvious in the noisy operating room. Had we placed the pulse oximeter on the side away from the NIABP, as is our usual practice, we would not have had a warning of the failure until after the induction of anesthesia. We have asked the manufacturer to modify the alarm for this type of malfunction.

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Full Disclosure in Study Design Is Essential

To the Editor:

Recent correspondence regarding the paper by Shore-Lesserson et al. (1) raised several issues that merit further comment. Although not mentioned in the Methods section (1), a one-tailed test was used to both estimate the sample size and analyze the data (2). One-tailed testing may be justified under specific circumstances; however, at the very least, one-tailed testing should be disclosed wherever it is used; for example, when the hypothesis of the study is defined, in the assumptions for the sample size estimation, and in the post hoc testing criteria. None of these occurred in this paper (1).

In addition, as sample size estimation becomes more commonplace in clinical research, it is important that all of the assumptions used are disclosed to the readers. In addition to specifying the α and β values, the effect size must be clearly defined (3). In their paper (1), the authors used a 33% decrease in “transfusions” between tranexamic acid and placebo as the minimal desired effect size. However, this criterion may be interpreted in at least two ways: 1) an arithmetic difference of 33% in the transfusion rate between the two groups (i.e., from 66% to 33%) or a fractional difference of 33% (i.e., a difference of 66% × 33%). Because sample size increases exponentially as the size of the larger proportion decreases (for a fixed percent decrement) (3), the rate of transfusion that they used could only be determined by back-calculating the rate based on a sample size of 20 patients per group. The authors should have reported the exact incidence that they used, as well as the source of their data. Failure to disclose these data limits our ability to judge whether their assumptions are clinically reasonable, makes it difficult to verify their sample size calculation, and limits the educational opportunity for trainees. Both the readers and the reviewers would benefit enormously if the authors clearly presented all of the assumptions in their study design.

Finally, what about one-tailed testing? Although it is limited by its inability to interpret a finding opposite to that defined in the hypothesis, it has the advantage that it decreases the sample size by 20%-25% compared with a two-tailed test. Thus, fewer patients would be enrolled, randomized, treated, and possibly harmed by...