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In Reply:—We thank Drs. Xia and Irwin for their interest in our study on the role of β -adrenergic signaling in anesthetic postconditioning¹ and in the accompanying editorial view.² We agree with Drs. Xia and Irwin that, besides their energy-sparing effect, several alternative mechanisms of β blockers might be responsible for their infarct size-reducing capacity. Apart from their effect on the interaction between β receptor activation and reactive oxygen species production³ and scavenging,⁴ β blockers can inhibit calcium/calmodulin-dependent protein kinase II⁵ and phospholipase A,⁶ exert membrane stabilizing effects,⁷ and may even have direct effects on mitochondrial electron transport and reactive oxygen species production.⁸ The role of alternative mechanisms is certainly supported by the finding that infarct size reduction by β blockade is independent of heart rate, the main determinant of myocardial oxygen consumption.⁹ It is entirely conceivable that the combination of different cardioprotective principles at different time points during reperfusion might provide additive protective effects. In this context, it is of interest that calcium/calmodulin-dependent protein kinase II is necessary for desflurane-induced postconditioning, whereas prolonged postischemic calcium/calmodulin-dependent protein kinase II blockade might attenuate adverse effects of ischemia/reperfusion injury, including remodeling.¹⁰ Thus, it might be reasonable to apply anesthetic postconditioning at the onset of reperfusion and to initiate β blockade later during reperfusion. However, further basic research and clinical studies will be necessary to determine an optimized cardioprotective approach and to identify the possible clinical consequences of these experimental findings.

The rabbits used in this study were between 8 and 12 weeks of age and weighed between 2.5 and 3.0 kg. Although cardioprotection by ischemic¹¹ and pharmacological¹² preconditioning can be attenuated or lost in senescent hearts, there is some evidence of preserved ischemic postconditioning in the aged myocardium.¹³ Thus, the impact of aging on the cardioprotective effects of β blockade, anesthetic postconditioning and their interaction with reactive oxygen species needs to be determined in future studies.

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Goals Neither Validated Nor Met in Goal-directed Colloid versus Crystalloid Therapy

To the Editor:—Kimberger *et al.*¹ and the editors² are to be commended for attempting to shed light on an important topic: What is the optimal intraoperative fluid and resuscitation target?

Many experienced physicians, including us, who provide anesthesia for major intraabdominal surgery have evolved over time from crystal-

loid-only, “show me the proof” physicians to those being in philosophical agreement with both the author and the editorial writers—goal-directed therapy with colloid is best in intestinal cases. We believe this produces less gut edema without compromising gut or other critical organ perfusion (not to mention reducing the anesthesiologist’s aural discomfort from the oft repeated surgeon lament that the anesthesia team is “drowning” the patient). Indeed, Victor Hugo once said, “All the forces in the world are not so powerful as an idea whose time has come.”*

Unfortunately, despite our hope to the contrary, all the forces in the world may have to wait a little longer, because this study does not

The above letter was sent to the authors of the referenced Editorial. The authors did not feel that a response was required. —James C. Eisenach, M.D., Editor-in-Chief.

*Available at http://famouspoetsandpoems.com/poets/victor_hugo/quotes. Accessed May 29, 2009.