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In Reply:—I thank Professor Jorge Dagnino, M.D., for his chronology of the founding of the Royal Society. With my six-sentence limitation on caption space for *Anesthesiology Reflections*, I thought that I had dealt reasonably with the Royal Society's nebulous origins by writing that Wren, Boyle, and others had met "by" (not "first met") in November of 1660.

Just as I acknowledged 21 yr ago, Wren was the "brains" behind the intravenous goose quill experiment of 1656.¹ So I concur with Professor Dagnino on these facts. In "Boyle, a Most Skeptical Chemist," the 1659 date in the caption was my typographical error.²

My thanks to Professor Dagnino for his thoughtful feedback on my telegraphic captions.

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Esmolol May Abolish Volatile Anesthetic-induced Postconditioning by Scavenging Reactive Oxygen Species

To the Editor:—We read with great interest the article recently published by Lange *et al.*¹ In an *in vivo* rabbit model of myocardial ischemia-reperfusion induced by 30 min of coronary occlusion and 180 min reperfusion, the authors observed that β -adrenergic receptor blockade during early reperfusion with either the β 1-adrenergic blocker esmolol or the β 2-adrenergic blocker ICI 118,551 abolished desflurane-induced postconditioning cardioprotection manifested as reduced myocardial infarct size. However, neither esmolol nor ICI 118,551 had a significant effect on postischemic myocardial infarct size when used alone during the first 30 min of early reperfusion, in the absence of desflurane. This is a very interesting finding. However, the more interesting point of the study, as commented on by Dr. Riess in an editorial² accompanying this article, is that sustained β 1-blockade with esmolol during the entire period of reperfusion not only failed to abolish desflurane-induced postconditioning cardioprotection, but instead actually conferred a similar degree of cardioprotection. We want to join Dr. Riess² in congratulating the authors for this comprehensive study detailing the role of β -blockers in volatile anesthetic postconditioning. However, we do not entirely agree that the energy-sparing effect of β -blockade, mainly heart rate reduction, may have been the principal reason for the infarct size reduction. We propose that β -blockers may have conferred cardioprotection primarily by reducing the production of reactive oxygen species (ROS)^{3,4} during reperfusion, and that esmolol may have abolished desflurane-induced postconditioning by scavenging ROS.

ROS has been shown to play an essential role in β -adrenergic signaling in cardiac myocytes.⁵ Volatile anesthetic-induced generation of small amounts of ROS plays a critical role in anesthetic preconditioning,^{6,7} and likely in anesthetic postconditioning as well, since they share similar mechanisms. Esmolol has been shown to increase antioxidant activity and reduce ROS-induced lipid peroxidation in patients with acute myocardial infarction.⁸ Therefore, it is reasonable to postulate that esmolol abolished desflurane-induced postconditioning *via* its antioxidant action in the study of Lange *et al.*¹ If this is the case, it could be possible that the cardioprotection conferred by a combination of desflurane postconditioning and delayed β -adrenergic blockade

during reperfusion could be superior to desflurane postconditioning or β -adrenergic blockade alone. We are interested in the authors' opinion on this possibility, and the clinical relevance of their findings.

It should be noted that the volatile anesthetic isoflurane-induced ROS production and anesthetic preconditioning cardioprotection is attenuated in senescent hearts,⁹ likely because ROS production is already increased in the senescent. Information regarding the age or body weight of the study animal (New Zealand White rabbits) is not provided in the study of Lange *et al.*¹ Presumably, the study was conducted in young animals. It would be also interesting if the authors could provide this information and comment on the potential effect of aging on the effectiveness of anesthetic postconditioning.

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