hence we do not mentioned the role of patient positioning and its impact on VAP.1 Second, we disagree with the opinion that the orientation of the endotracheal tube below horizontal will result in reduced incidence of VAP for the reason that it is based on insufficient clinical data: three experimental animal (sheep) studies,2–4 a randomized controlled trial with 60 ventilated infants that compared the tracheal colonization rate and not the VAP incidence in supine versus lateral position,5 and unpublished observations in adult patients.

The possible body positions for orientation of the endotracheal tube below horizontal are head-down (Trendelenburg) position and lateral head-down positions. In our opinion, these positions are uncomfortable, unsafe for patients with raised intracranial pressure, and inappropriate for patients in the weaning process. Furthermore, there is evidence that the semirecumbent position is the optimal body position for VAP prevention in critically ill patients.6

Dr. Sathishkumar and Fassl report the advantages of the LoTrach™ tube (Hi-Lo Evac; Mallinckrodt, Athlone, Ireland) and the cuff pressure controller regarding the prevention of pulmonary aspiration during mechanical ventilation. In fact, the LoTrach tube and the cuff pressure controller are designed to offer triple protection against pulmonary aspiration: The low-volume, low-pressure cuff without folds offering effective tracheal seal at permanent tracheal wall pressure between 20 and 30 cm H2O; the triple subglottic ports for intermittent suctioning of secretions and retrograde cleansing of the entire upper airway by irrigation with normal saline; and the nonstick inner lumen designed for reduction of adhesion of biologic material and biofilm formation.

We believe that the LoTrach™ tube and the cuff pressure controller will contribute substantially to VAP prevention. However, there are still limited data about the clinical impact of the use of the LoTrach™ tube on the incidence of VAP, and further clinical research is required.7

References

1. Bause GS. Boyle, a most skeptical chemist. Anesthesiology 2009; 110:610–611


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 nebulus 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.

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 β1-adrenergic blocker esmolol or the β2-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 β1-blockade with esmolol during the entire period of reperfusion not only failed to abolish desflurane-induced postconditioned 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).5–8 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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References


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