

Unfortunately, the methods chosen by the authors do not allow us to determine whether the critical factor in hypoxic apnea is the level of sympathetic tone or simply the absolute blood pressure. It would be interesting to see whether hypoxic apnea occurs in rabbits in which hypotension is induced by a nonsympatholytic mechanism (hypovolemia, sodium nitroprusside) as well as mechanisms involving blockade of the sympathetic system (trimethaphan, epidural anesthesia). Similarly, it would be interesting to see whether hypoxic apnea could be avoided in rabbits undergoing epidural anesthesia using nonsympathomimetic interventions (volume loading, vagolytics) rather than administration of adrenergic agonists. Although it is certainly possible that the critical factor is oxygen delivery to the brain, as mediated by arterial oxygen content and arterial blood pressure, the authors' methods do not rule out the possibility of specific, sympathetic modulation of respiratory drive mediated through the thoracic sympathetic system.

The authors¹ are to be commended for shedding light on the issue of unexpected cardiorespiratory arrest in patients undergoing spinal or epidural anesthesia. At the same time, their work raises provocative questions about apnea in infants and control of respiratory drive.

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In Reply:—We have no data regarding the age dependence of the ventilatory response to hypoxia during epidural anesthesia. The rabbits (approximately 2 kg) we studied are not mature, heightening the relevance of Dr. Gunter's comparison to human infants. Although we did not typically see the hyperventilatory phase before arrest as described by Dr. Gunter, that mirrors the pattern of neural activity identified in the central nervous system.^{1,2} Regarding the mechanism of apnea, we noted a similar response to hypoxia in the absence of sympathetic blockade in animals with hypotension caused by problems with surgical preparation, such as excessive blood loss. This suggests that circulatory compromise, not sympathetic block *per se*, is the critical component that sets the stage for hypoxic arrest. Findings from our study³ that support (but do not prove) the key role of circulatory compromise include the lack of a change in ventilation after sympathetic block alone and the effectiveness of hemodynamic resuscitation in preventing arrest.

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Predicting Long-term Postoperative Cardiovascular Outcomes

To the Editor:—Referring to the article of Badner *et al.*,¹ Dr. Mangano² encourages assessment of the value of perioperative observations and interventions for predicting quality of life, event-free survival, and cost. Badner *et al.*¹ furthers this goal by relating postoperative signs and symp-

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toms to longer-term risk of myocardial infarction (MI) and associated mortality. This valuable data set could provide even further insight into the risk profile for MI and MI-related mortality among surgical patients by a more complete analysis of the available information.