

CORRESPONDENCE

11. Bhalotra R: Baclofen therapy for intractable hiccoughs. *J Clin Gastroenterol* 1990; 12:122
12. Krahn A, Penner SB: Use of baclofen for intractable hiccups in uremia. *Am J Med* 1994; 96:391
13. Guelaud C, Similowski T, Bizec JL, Cabane J, Whitelaw WA, Derenne JP: Baclofen therapy for chronic hiccup. *Eur Respir J* 1995; 8:235-7
14. Ramirez FC, Graham DY: Treatment of intractable hiccup with Baclofen: Results of a double-blind randomized, controlled, cross-over study. *Am J Gastroenterol* 1992; 87:1789-91

15. Petroianu G, Hein G, Petroianu A, Bergler W, Rüfer R: Idiopathic chronic hiccup: Combination therapy with Cisapride, Omeprazole and Baclofen. *Clin Ther* 1997; 19:1031-8
16. DeJong RH: Neurontin: Pie in the sky o pie on the plate? *Pain Digest* 1996; 6:143-4
17. Petroianu G, Hein G, Petroianu A, Bergler W, Rüfer R: ETICS-Studie: Empirische Therapie des Idiopathischen Chronischen Singultus. *Z Gastroenterol* 1998; 36 (in press)

(Accepted for publication July 7, 1998.)

Anesthesiology
1998; 89:1285
© 1998 American Society of Anesthesiologists, Inc.
Lippincott Williams & Wilkins

In Reply:—We did not claim that “the failure of simple hiccup-terminating maneuvers warrants proceeding directly to potentially damaging ‘last-resort’ maneuvers such as phrenic nerve interruptions.” Instead, we fully agree with Dr. Petroianu’s claim that “the logical next step (after failure of simple maneuvers) is the use of appropriate drugs.” Our statement, “block of the phrenic nerve has been suggested as ‘the last resort,’”¹ means that the use of this block should be considered if other less-invasive methods, including the use of potentially effective drugs, have failed. Most patients with intractable hiccups are referred from other departments in which a variety of methods, including potentially effective drugs such as baclofen, have been used in vain, although we did not described these explicitly.

The main point we made in our report¹ was not advocating the phrenic nerve block but that the use of electric nerve stimulation enables one to avoid unnecessary attempts at repeated blocks. In fact, in one of our patients,¹ we judged that a successful block of the phrenic nerve would not decrease hiccups and abandoned this method. There is no “holy grail” for intractable hiccups: neither baclofen nor phrenic nerve block is always effective. The importance—we believe—is to judge whether each treatment method is effective in each patient and to stop ineffective methods at an early stage.

Anesthesiology
1998; 89:1285-6
© 1998 American Society of Anesthesiologists, Inc.
Lippincott Williams & Wilkins

Yasuhisa Okuda, M.D.

Associate Professor of Anesthesiology
y-okuda@dokkyomed.ac.jp
Toshimitsu Kitajima, M.D.
Professor
First Department of Anesthesiology
Dokkyo University School of Medicine
Mibu, Tochigi, Japan
Takashi Asai, M.D., Ph.D.
Research Associate
Department of Anesthesiology
Kansai Medical University
Osaka, Japan

Reference

1. Okuda Y, Kitajima T, Asai T: Use of a nerve stimulator for phrenic nerve block in treatment of hiccups. *ANESTHESIOLOGY* 1998; 88:525-7

(Accepted for publication July 7, 1998.)

Hypoxic Apnea, Epidural Anesthesia, and Infants

To the Editor:—In reviewing the recent report by Hogan *et al.*¹ regarding hypoxic apnea in rabbits receiving epidural anesthesia, I was struck by the similarity of their report to a phenomenon seen in hypoxic newborn and premature infants: to wit, a brief hyperventilatory response followed by hypoventilation and apnea.² In many ways, the newborn or premature infant is similar to the authors’¹ rabbit-with-epidural-anesthesia model. Obviously, the blood pressure of the newborn or premature infant is much lower than that in an older child or adult. At the same time, infants

exhibit primarily parasympathetic tone; this is seen in their exaggerated bradycardic responses to laryngoscopy, anesthetics, and succinylcholine, and in the absence of hypotension after pharmacologic sympathectomy with spinal or epidural anesthesia. These similarities between the rabbit-with-epidural-anesthesia model and the newborn or premature infant led me to wonder whether there might be a common mechanism for hypoxic apnea and a role for blood pressure augmentation, adrenergic agonists, or vagolytics in the prevention or treatment of infant apnea.

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 authors¹ 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.

Anesthesiology

1998; 89:1286

© 1998 American Society of Anesthesiologists, Inc.

Lippincott Williams & Wilkins

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.

Anesthesiology

1998; 89:1286-7

© 1998 American Society of Anesthesiologists, Inc.

Lippincott Williams & Wilkins

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-

Joel B. Gunter, M.D.
Associate Professor
Clinical Anesthesia and Pediatrics
Department of Anesthesiology
University of Cincinnati
Cincinnati, Ohio
gunterjb@email.uc.edu

References

1. Hogan QH, Amuzu J, Clifford PS, Bosnjak ZJ, Kampine JP: Hypoxia causes apnea during epidural anesthesia in rabbits. *ANESTHESIOLOGY* 1998; 88:761-7
2. Todres ID, Gore R: Growth and development, *A Practice of Anesthesia for Infants and Children*. 2nd edition. Edited by Cote CJ, Ryan JF, Todres ID, Goudsouzian NG. Philadelphia, W. B. Saunders, 1993, pp 17-8

(Accepted for publication July 7, 1998.)

Quinn H. Hogan, M.D.
Department of
Anesthesiology
Medical College of Wisconsin
Milwaukee, Wisconsin
ghogan@post.its.mcw.edu

References

1. Eccles RM, Loring Y, Oshima T: Effects of hypoxia on the mono-synaptic reflex pathway in the cat spinal cord. *J Neurophysiol* 1966; 29:315-32
2. Hossman KA, Sato K: The effect of ischemia on sensorimotor cortex in the cat. *Z Neurol* 1970; 198:33-45
3. Hogan QH, Amuzu J, Clifford PS, Bosnjak ZJ, Kampine JP: Hypoxia causes apnea during epidural anesthesia in rabbits. *ANESTHESIOLOGY* 1998; 88:761-7

(Accepted for publication July 7, 1998.)

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.