

The Baroreponse and Cardiovascular Depression by Halothane in Infants

To the Editor:—Differences in cardiovascular physiology exist between neonatal and adult mammals.¹ Many of these differences are well-understood, while others require further investigation. The recent article by Wear, Robinson, and Gregory,² reporting their study of the baroreponse of adult and baby rabbits, is an important contribution in this field.

One of the conclusions made by the authors, however, is not supported by their data. They concluded that, because of the marked depression of the baby's baroreponse by halothane, the baby's ability to compensate for hypotension would be limited. The authors studied the animals' heart rate response to *hypertension* (the depressor baroreponse), not the response to *hypotension* (the pressor baroreponse). While the two are undoubtedly related, the authors present no evidence to indicate that studying one response enables an investigator to draw conclusions about the other.

Their data do indicate that halothane depresses systolic blood pressure more in baby than in adult rabbits. Similar observations with humans and other mammals have been reported, as have possible contributing factors.³⁻⁶ An age-related difference in the baroreponse may indeed be an important contributing factor in the infant's cardiovascular depression by halothane. The data from this study, however, only suggest this to be a hypothesis and do not support it as a conclusion.

This point should be clarified, as it detracts from what was obviously a well-designed and executed experiment.

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In reply:—Dr. Friesen is correct; we did study the depressor response. We attempted to study the pressor response by administering nitroprusside, but the computer program would not handle the data without considerable revision. Hand calculation of data from a few animals plus the data of Abboud and associates¹ show that the pressor response is similar to the depressor response but is much flatter. In addition, we recently have shown that there is no change in heart rate in a group of preterm infants anesthetized with halothane who became hypotensive and had systolic blood pressure below 45 mmHg.² Both show that the ability of infants to respond to hypotension is less than their ability to respond to an increase in pressure. Therefore, we believe the

Perhaps Drs. Wear, Robinson, and Gregory will pursue their investigation further and examine age-related differences in halothane's effect on the response to hypotension.

ROBERT H. FRIESEN, M.D.
*Assistant Clinical Professor of Anesthesiology
and Pediatrics, University of Colorado,
Department of Anesthesiology
The Children's Hospital
Denver, Colorado 80218*

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statement we made that the baby's ability to compensate for hypotension is true based on the above data.

We appreciate Dr. Friesen's remarks. We agree that more information is required to better define the pressor response in young animals and humans.

R. E. WARE, M.D.

S. ROBINSON, M.D.

G. A. GREGORY, M.D.

*Department of Anesthesia
University of California,
School of Medicine
San Francisco, California 94143*

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Facial Pain Induced by Music

To the Editor:—The term “musicogenic epilepsy” first was used by Critchley¹ to describe seizures that were induced by certain types of music. Accordingly, my case might be called “musicogenic facial pain.” A 31-year-old man complained of a four-month history of right-sided facial pain combined with bilateral involuntary eye blinking induced by listening to rock music on a tape recorder. The pain was characterized as an electrical shock radiating to the right side in the distribution of the second branch of the trigeminal nerve and lasted 2–5 s. The facial pain with eye blinking occurred frequently at several second intervals during the music. Immediately upon cessation of the music, the facial pain with eye blinking disappeared completely. This type of pain with eye blinking was completely reproduced by rock music but could not be evoked by tactile or other sensory stimulation involving the face and mouth nor by other types of music. Following the administration of 200 mg carbamazepine a day, po, the facial pain with involuntary eye blinking during rock music disappeared promptly. This patient may have experienced this facial pain as a part of an episode of musicogenic epilepsy. There is no doubt that musicogenic epilepsy is a rare

form of epilepsy. Furthermore, partial sensory seizures induced by music are very rare. Among several theories concerned with the pathogenesis of trigeminal neuralgia, an epileptiform type of pain attack is worth considering. The character of the pain (sudden onset, short duration, trigger mechanisms) and the therapeutic effect of antiepileptic drugs appear to favor this hypothesis. This case report strongly suggests that the pathogenesis of atypical facial pain as well as true trigeminal neuralgia may be related to some type of epilepsy.

HISASHI YANAGIDA, M.D.

Chairman, Department of Pain Clinic and Anesthesia,
Tokyo Kosei Nenkin Hospital, 23, Tukudo-cho,
Shinjuku-ku, Tokyo, Japan
and

Visiting Professor of Erasmus University Rotterdam,
The Netherlands

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Some Corrections Concerning the Precipitation of Local Anesthetic Drugs in Cerebrospinal Fluid

To the Editor:—In a recent article, Moore¹ summarized our study² which appeared in *Der Anaesthetist* as follows:

Bupivacaine, etidocaine, mepivacaine, and tetracaine solutions have been stated to precipitate in CSF (cerebrospinal fluid). This conclusion was based on an *in vitro* aerobic study, which human CSF was frozen, then reconstituted at a later date, mixed with solutions of the local anesthetic drugs, titrated to the pH of CSF. The authors cautioned that injection of these drugs into the subarachnoid space might cause spinal cord damage.

I regret that none of these three statements represents an accurate interpretation of content, methods, or conclusions in our paper.

First, we have never concluded that “Bupivacaine, etidocaine, mepivacaine, and tetracaine solutions . . . precipitate in CSF.” It is true that we have investigated the solubility of local anesthetics in CSF *in vitro* and its dependence on the hydrogen ion concentration. We have shown that under control conditions of pH 7.371, P_{CO₂} 40 mmHg, and temperature 37° C, the solubilities of the local anesthetics in CSF are as follows: bupivacaine HCl: 0.83 ± 0.10 mg/ml CSF; carticaine HCl: 27.00 ± 2.80 mg/ml CSF; lidocaine HCl: 24.00 ± 1.30 mg/ml CSF; mepivacaine HCl: 14.80 ± 0.20 mg/ml CSF; and tetracaine HCl: 1.40 ± 0.12 mg/ml CSF.