

CORRESPONDENCE

received hyperbaric lidocaine, 5%, and 14 (58%) received hyperbaric bupivacaine, 0.5%.

The authors conclude that intrathecal administration of lidocaine to patients who do not describe pain or paresthesias during needle placement is associated with 75% of the neurologic injuries in that group. If one agrees with this conclusion, then logically one must also agree that administration of bupivacaine is associated with 92% of all neurologic injuries in patients who have pain or paresthesia during needle insertion. In addition to the authors' conclusions, these data suggest that lidocaine may be neuroprotective in patients who have paresthesia during needle insertion. Clearly, each of these conclusions is based on flawed assumptions and *post hoc* evaluation of an incomplete database.

In conclusion, we applaud the authors for the insights their study provides into the relative risk of serious complications associated with different techniques of regional anesthesia. However, the study was not designed prospectively to collect sufficient data to define risk factors for serious complications. In the absence of such data, specu-

lation that any single factor increases the risk for serious complications is not justified scientifically.

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In Reply:—The most important part of the letter by Drs. Price and Carpenter begins shortly before the paragraph that introduces their table 1. Before that, the letter reviews the caveats presented in the original article, or in the two editorials that accompanied it. These clearly stated and discussed interpretative limitations imposed by not knowing how many spinals were performed using lidocaine or how many were performed using bupivacaine. With respect to whether the study was prospective, we also note the "ideal" concern of R. L. Smith's editorial (1990) that data for the "relevant prognostic and outcome variables are collected from patients as they are treated."¹ Such was attempted in the study design, with the primary data being recorded as the patient was treated and the questionnaires being used to gather such data from those who treated the patients. As explained in the original article, all participants knew they were participating before the study took place, before regional anesthesia was administered, and before data were entered in the anesthetic record or on the questionnaires. Also, all physicians knew there would be follow-up inquiries about the cases. The questionnaire procedure was very different from a retrospective approach, in which unexpected inquiries are made to surprised individuals, asking them whether they recall various procedural events. Coincidentally, the thoughts cited by Drs. Price and Carpenter (1990) are addressed in a more recent editorial (1998) by Dr. P.G. Duncan² entitled, in part, "That was then, this is now!," in which practical limitations to large observational studies are acknowledged. Such limitations include logistics and cost, and the Duncan's² editorial offers the possibility that, in the 21st century, some readers will be able to find at least one technical flaw in every future trial or study conducted. The crucial issue, however, as stated by Duncan,² is, "when do data from an observational study achieve the standard necessary to become incorporated into one's evidence-based medical practice?"

Drs. Price and Carpenter state that our data "suggest a recall bias in reporting for patients with neurological deficits." They then construct their table 1 to argue this point. We are concerned that their table

reflects a superficial assessment. If paresthesia or pain were totally independent of the agent used, (*i.e.*, as in paresthesia or pain during needle insertion only), then statistically equal divisions of "paresthesia or pain" and "no paresthesia or pain" *might* be expected in the lidocaine and bupivacaine groups of their table 1. Because one starts with the group that had deficits, there is no *a priori* reason why the incidence of paresthesias and pain must be the same in both drug groups. The text of our article clearly refers to pain *during injection*, which might be agent dependent. It is possible that "no pain" during injection occurs less frequently during injections of a particular toxic substance than during injection of another less-toxic substance. Thus, the numbers in their table 1 do not form the basis for their subsequent reasoning that we have proven an obviously absurd hypotheses (*i.e.*, that paresthesia and pain during needle insertion before bupivacaine injection is more likely than paresthesia and pain during needle insertion before lidocaine injection or that pain during lidocaine use is neuroprotective). In the next to last paragraph of their article, Drs. Price and Carpenter need to change "paresthesia during needle inser-

Table 1. Characteristics of Patients with Permanent Neurologic Deficit after Spinal Anesthesia

	Lidocaine 5%	Bupivacaine 0.5%	Total
Paresthesia during needle insertion or pain during injection	1	0	1
No paresthesia during needle insertion and no pain during injection	3	0	3
Total	4	0	4

CORRESPONDENCE

tion" to "paresthesia during needle insertion or pain during injection." It is logical to conclude, after doing one arithmetic operation, that we found this to be associated with 92% of the neurologically injured patients who received bupivacaine.

After constructing table 1, Drs. Price and Carpenter ask, "are these data sufficient to support speculation regarding risk for nerve injury?" A more relevant version of their table 1 would be the following, which restates data presented in the "Neurologic Complications", section of our paper.³ The table herein suggests that yes, there is reason to speculate that 5% lidocaine causes nerve injury.

We are concerned about the intensity with which Drs. Price and Carpenter seek to counter this important message of our study: that lidocaine toxicity *might* exist. They state that "it is certainly possible that lidocaine was chosen more frequently . . . in a high risk patient population." However, this is entirely speculation on their part. Although it is mathematically possible, it has no factual basis.

Drs. Price and Carpenter essentially congratulate us for gathering enormous amounts of uninterpretable data. However, we believe it is important to note that both of these people represent Astra USA, which manufactures lidocaine. Dr. Carpenter also has protested previously the notion of lidocaine toxicity,⁴ even before our study was conducted. He wrote, criticizing an earlier study by others, that (1) "This study . . . fails to clearly identify lidocaine as the cause for . . ."; (2) "Astra has taken a proactive approach to this controversy"; and (3) "I plan to continue to use hyperbaric lidocaine." Because Drs. Price and Carpenter clearly believe that the available information is insufficient, a logical final question is whether Astra, which funds expensive studies of new molecules, is also willing to fund the extraordinarily expensive, "high-quality" clinical studies that they seem to believe are needed to address a problem with an "old molecule" such as lidocaine?

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Henry K. Beecher, M.D.: An Historical Perspective?

To the Editor:—I read with fascination the article by Gravenstein¹ about Henry K. Beecher. The author is to be commended for including so many interesting, personal antidotes about Dr. Beecher. This paper humanizes a historic person in a unique way, making Dr. Beecher's personality available to those who never had the chance to meet him.

However, the historic record does not support Dr. Gravenstein's thesis. Dr. Beecher did not, as Gravenstein wrote, make anesthesiology a university specialty. In fact, despite the author's comments to the contrary, Beecher's contributions to the academic practice of the specialty are modest when compared to his contemporaries. The man

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who did more to make anesthesiology an equal in the university setting was Ralph Milton Waters of the University of Wisconsin. His teaching and departmental organization are used in more than 60% of the academic departments in the United States.² Richard Kitz, Beecher's successor, is from this lineage, and no doubt, even at the Massachusetts General Hospital (MGH), the influence of Ralph Waters prevails.

It is interesting to compare the appointments of the chief of anesthesia at the MGH in 1936 with the same position at Bellevue Hospital in New York City a year previously. Beecher, after completing 3 yr of training as a surgical house officer and a year working with August Krogh, the Danish