Reply

We are naturally delighted that our recent article has been of interest both to discussants at the original presentation of the paper and now among readers of *Journal of Vascular Surgery.* I am pleased to respond to the questions posed by Drs Bush and Smith and Frawley.

There is a large literature in virtually all of the approaches to cerebral monitoring and cerebral protection during CEA, including the articles cited in Drs Bush and Smith's letter. We had cited the article by Coyle et al.[1] The article by Chaikof et al[2] was reviewed but not cited as part of an effort to keep the number of citations reasonable. I suspect some readers will think that the 80 citations retained are "too many." The articles by Illig et al[3] and Sternbach et al[4] were published after our article was presented and accepted for publication, but I believe most of the points raised are addressed in our article and postpresentation discussion.

We recognize that much of the recent literature discusses CEA with regional anesthesia. However, very little of this literature makes any effort to compare outcomes using different anesthetic approaches in contemporaneous patients from the same institution and none to my knowledge is randomized. We do not advocate general anesthesia and routine EEG as the only way to manage the carotid endarterectomy operation. On the contrary, we could not agree more with Dr Bush and Smith's final paragraph, and as we state in the final sentence of our article, this approach should be considered. Dr Bush cites results from the article by Coyle et al[1] that observed no stroke or mortality among patients with contralateral occlusion excluding those who underwent concomitant coronary surgery. These excellent outcomes are comparable with our experience as detailed in our article. Although the neurologic status of an awake patient is certainly a good method of assessing cerebral perfusion, I believe it is a leap to state "Local anesthesia- . . . provides the most accurate method . . . ." Even if one accepts that the neurologic status of an awake patient is the most sensitive indicator of cerebral perfusion at the time of carotid clamping, an alternative, and in our view at least as important question, is what method is most sensitive when the endpoint of interest is whether the patient emerges from anesthesia with a new neurologic deficit? As stated in our article, we chose EEG because the prior literature suggested EEG was nearly if not 100% sensitive using this standard, albeit at the price of less than perfect specificity, an impression corroborated by our observations. I would ask Drs Bush and Smith how they know from their own experience that "Local anesthesia . . . [is] . . . the most accurate method of monitoring the neurological status of the patient" if, as they state four sentences earlier, "Shunts are routinely used . . . "? Dr Bush and Smith state that they observed no complications related to shunt placement, but the literature, including some of the articles we cited in the first paragraph of the Discussion, clearly documents that this is not a universal experience. We state in the article that our current rate of selective shunting seems to provide an adequate familiarity, or using Dr Bush and Smith's terminology, allows us to " . . . maintain adequate skills . . . ," so that shunt placement is not unfamiliar when a shunt needs to be placed quickly.

With respect to the idea that local anesthesia is cerebroprotective, we would acknowledge that this is one possible conclusion suggested by Illig et al's[3] observation of a substantially lower overall rate of EEG changes in patients with regional anesthesia. This would seem at odds with the prior view that general anesthesia is cerebroprotective when compared with regional anesthesia, and there is at least one alternative explanation. All the difference between local and general anesthesia in Illig et al's[3] paper appeared to be due to global EEG changes in patients with general anesthesia, an observation not made in their patients with regional anesthesia. Global EEG changes may represent a general effect of the anesthetic agent used and may not indicate cerebral ischemia. Our approach requires a specific general anesthetic technique and is not appropriate if the anesthetic is given as in Illig et al's[3] work " . . . according to the anesthesiologist's preference . . . " since the EEG may be uninterpretable with some anesthetic agents. Indeed, Illig et al[3] acknowledges the use of nitrous oxide in general anesthesia patients, an agent known to cause alterations in the EEG and, therefore, among the anesthetic agents avoided in our practice as stated on page 1115 of our article in Methods. Were the changes observed by Illig et al[3] present before clamping or did they occur only after clamping? The former would suggest an explanation other than ischemia. Furthermore, the observed rate of EEG change in awake patients (7.4%) in Illig et al's[3] patients seems unusually low and may represent a sample size problem. For example, Dr Stoughton et al[5] appear to have observed EEG changes in 6.7% + 12.4% or approximately 19% of their awake patients, remarkably similar to our own (18%) and Illig et al's[3] (15%) observations in patients with general anesthesia. With respect to the use of the intensive care unit, we currently send fewer than 15% of our post–carotid endarterectomy patients to the intensive care unit and the remainder are sent to a standard surgical floor. Furthermore, we have reduced the direct costs associated with carotid endarterectomy by 53% at our hospital over the last 6 years despite the fact that we continue to employ routine EEG.

With respect to costs, also the subject of most interest to two of the four discussants after presentation of the paper, Dr Bush and Smith state " . . . at Emory University Hospital the EEG charges run close to $700 . . . ." We have made it clear in the article and subsequent discussion that we are discussing costs, not charges. In fact, as stated in the postpresentation discussion, our hospital charges something on the order of $1700 for this service, so perhaps it is Emory University that is " . . . on the low side . . . ." Our estimate includes total costs, not just technical costs. The costs we cite are very rough estimates since our hospital, like many others, has a very hard time providing indirect cost estimates for some component services, but it was very reassuring to hear that Dr Mackey's hospital estimated a cost of $355 for this service, similar to our own estimate. I would also respectfully offer that there is no reason to believe " . . . the time involved . . . must far outweigh the short time needed . . . ." On the contrary, as discussed in the postpresentation discussion, we suspect that the increased time to do the operation in the presence of the shunt, the lower level of satisfaction with the visualization and completion of the distal endpoint, and the costs of shunt-related complications may outweigh the costs associated with EEG.

We applaud Dr Frawley's excellent results with high-dose barbiturate cerebral protection. He and his associates have achieved these excellent results in a large sample (1000 cases at the time of their year 2000 publication). We think their results warrant a prospective controlled trial by other groups to try to confirm their results. I would be reluctant to employ this technique outside of such a trial until these excellent results have been duplicated by others.

We, too, would welcome a randomized trial comparing high-dose barbiturates with shunting, but I am surprised that Dr Frawley states in the same sentence that he feels ethically prevented from conducting such a trial. Who better to test this in a prospective randomized trial? As he correctly points out, results of CEA have improved " . . . around the world . . . " and this appears to be the case with multiple approaches to cerebral monitoring and cerebral protection. I would offer that had he continued with selective shunting, Dr Frawley's results with that approach might have improved along with those of the rest of the world and might not be distinguishable from his current results with high-dose barbiturates. I wonder whether the prolonged postoperative period of sedation due to the high-dose barbiturate increases utilization of other resources such as recovery room time, intensive care unit stays, ventilators, etc, and any randomized prospective trial should address these questions in addition to those of neurologic morbidity and mortality. If resource utilization is not increased, then our concerns about increased time and less satisfactory exposure of the endarterectomy endpoint would be obviated by routine use of high-dose barbiturates with no shunts with no apparent increase (or perhaps even a decrease) in total costs.

Once again, we are pleased that our work has stimulated so much discussion.

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REFERENCES

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