References

RESPONSE: We thank Dr. Haines for his letter and would agree that the article by Young and Lawner mentioned by him is important and makes a significant contribution to the literature. We would like to make several observations concerning that paper.

1. The article was a “single-blind” study, and in reference to the comments made by Dr. Haines we clearly stated that “after several decades of controversy, there are still no completed double-blind placebo-controlled published trials regarding the use of prophylactic antibiotics in clean neurosurgical cases.” This is still the case. The conclusions reached by the authors agree with the conclusions of our review that “the use of perioperative antibiotics for prophylactic use in clean neurosurgical cases is justified.”

2. The drugs used by Young and Lawner in their study included both cefazolin and gentamicin. No case of Gram-negative infection occurred either in the control group or in the active drug group. All isolates from patients with infections were Gram-positive and were limited to Staphylococcus aureus, coagulase-negative Staphylococcus (S. epidermidis), and the Enterococcus (gamma-Streptococcus). Therefore (as stated by Young and Lawner), the need to add an aminoglycoside antibiotic to the prophylactic regimen is indeed questionable.

3. Both groups of patients in their study received varying amounts of an irrigating solution containing bacitracin (50 U/ml in normal saline solution). Although this probably did not affect the results of the study, it certainly is a confounding factor and needs to be addressed in the interpretation of the results.

4. If an aminoglycoside antibiotic such as gentamicin, tobramycin, or amikacin is to be used, based on pharmacokinetic principles, the use of a loading dose of at least 1.5 to 2 mg/kg of body weight is necessary to achieve therapeutic levels in serum and/or tissue. When loading doses are not used in only a one- or two-dose regimen, therapeutic levels would only be reached in the urine.

We did include the other two papers mentioned by Dr. Haines. This includes the fine work from Cork, Ireland, by Geraghty and Feely, and the prematurely interrupted trial by Shapiro, et al. Neither of these studies qualifies as a double-blind placebo-controlled trial.

If one really wanted to be a “therapeutic purist” regarding trials of antibiotic prophylaxis, each individual class of surgical procedures should be studied separately. In most of the studies in clean neurosurgery cases, all types of procedures including craniotomy, laminectomy, and the placement of shunts are included. We do not believe this invalidates our conclusions and recommendations. Of course, from a practical point of view such specialized studies would be virtually impossible to accomplish in a reasonable length of time. Therefore, we believe that conclusions are best arrived at by careful review of all available literature.

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References

EKG Artifacts During Intraoperative Evoked Potential Monitoring

TO THE EDITOR: During intraoperative monitoring of somatosensory evoked potentials (SEP’s) at our institution, the electrocardiogram (EKG) signal displayed on the screen of the anesthesiologist’s Datascope 2000 monitor* was often obscured by large stimulus artifacts. The same artifacts appeared on “delayed” hardcopy output produced by the Datascope (Fig. 1A) but not on “diagnostic” hardcopy output (Fig. 1B). The SEPs were recorded by a Nicolet Pathfinder I signal averager† using constant-current stimulators and stimulus isolation units. The square-pulse electrical stimuli were 200 μsec in duration and delivered at a rate of 6.1/sec to

† Signal averager manufactured by Nicolet Instruments, Madison, Wisconsin.
paired stimulating electrodes over the median or posterior tibial nerves; stimulus intensities ranged from 15 to 30 mA.

Rigorous testing of the Pathfinder failed to demonstrate any malfunction. Another evoked averager, the Lifescan, produced similar artifacts. We then discovered that the large artifacts were produced by a “pacer enhancement circuit” in the Datascope 2000, which modifies data sent to the screen display and “delayed” hardcopy but not to the “diagnostic” hardcopy. This circuit increases the visibility of small pacemaker spikes by incorporating a high-amplitude square pulse in the EKG data when a pacemaker spike is detected. While the electrical artifacts from the somatosensory stimuli were not large enough to obscure the EKG signal by themselves, their steep slopes led to their identification as pacemaker spikes.

The “pacer enhancement circuit” of the Datascope 2000 can be disabled, eliminating this problem. Other EKG monitors designed for intraoperative use may also incorporate such circuitry. Thus, we are reporting our findings for others who may have encountered similar difficulties.

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Treatment of Carotid Sinus Hypersensitivity

TO THE EDITOR: We have read with great interest the article by Simpson, et al., in which they reported three cases of carotid sinus hypersensitivity (Simpson RK, Pool JL, Grossman RG, et al: Neurosurgical management of carotid sinus hypersensitivity. Report of three cases. J Neurosurg 67:757–760, November, 1987). All three patients harbored a neck tumor, which was treated by intracranial section of the glossoaryngeal nerve and the upper rootlets of the vagus nerve.

At the 34th Congress of the Italian Society of Neurosurgery in 1985, one of us (A.B.) reported with his colleagues three cases of carotid sinus syndrome (unpublished data). Only one of the patients harbored a neck tumor; the other two had a vasodepressive type of carotid sinus syndrome. At the 8th European Congress of Neurosurgery held in Barcelona, Spain, in 1987, the same author and coworkers presented six cases of carotid sinus syndrome in which only two of the patients had a neck tumor (unpublished data).

As in the cases of Simpson, et al., our results were very good. We agree with the authors with regard to their selection criteria, choice of surgical technique, the absence of mortality, and the acceptable level of morbidity. The authors call the disease “carotid sinus hypersensitivity.” We prefer to classify it as “carotid sinus syndrome” for two reasons. First, hypersensitivity occurs frequently in older individuals, but usually without clinical manifestations. Second, the disease may be due to compression of the glossoaryngeal nerve terminations by neck tumors or a mass in the parapharyngeal space, without involvement of the carotid sinus. For these latter cases, one of us (R.C.) is proposing a new nosographic classification — parapharyngeal space-lesion syncope syndrome — a form quite distinct from carotid sinus syndrome.

Also, in our opinion, the two syndromes are different from a clinical point of view because the latter manifests itself with short episodes of syncope and is triggered by factors such as head movements, whereas the former has no triggering factors and the episodes of syncope are more frequent and protracted. In four cases with idiopathic etiology that we treated, the results were as good as with the symptomatic form.

The length of follow-up evaluation in our patients with unknown etiology ranged from 1 to 6 years, and all are symptom-free. Only one patient had an episode suspicious of orthostatic hypotension 4 months after the operation.

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Reference

RESPONSE: We would like to thank Drs. Bollati and Cicogna for their interest in our recent article concerning the operative treatment of carotid sinus hypersensitivity. We certainly agree with the desirability of identifying patients with this syndrome by etiology and/or location of carotid sinus reflex arc irritation. Likewise, we agree that the mechanisms for carotid sinus hypersensitivity in elderly patients with atherosclerotic dis-