Remote Cerebellar Hemorrhage after Supratentorial Surgery

To the Editor:

We read with great interest the recent article by Friedman et al. (3). In their study, the authors very eloquently described their analysis of the cases of 42 patients who developed cerebellar hemorrhage after supratentorial surgical procedures with special emphasis on identifying the risk factors associated with this phenomenon. Preoperative aspirin use and moderately elevated intraoperative systolic blood pressure emerged as potentially modifiable risk factors after exhaustive statistical analysis of all possible variables.

In 1990, we set out to examine the hypothesis that abrupt increases in blood pressure, particularly during the last stages of the intracranial procedure and during prompt awakening, are causally related to the development of postoperative intracranial hematoma. If this hypothesis is true, then the abolition of these increases in blood pressure by appropriate anesthesiological techniques should diminish the incidence of this complication to negligible levels. The protocol of anesthesia was designed to achieve suppression of the stress response to surgery and to steady the blood pressure level without abrupt changes during the whole procedure until awakening, and it was based on deep opioid analgesia (fentanyl, ≤30 μg/kg). During the period from January 1990 to April 1998, 526 consecutive patients underwent intracranial procedures in which the aforementioned anesthetic protocol was used. The results of the study were published in 1999 (10). In summary, no postoperative hematomas were observed in this series of patients; that is, no patients had neurological impairments due to postoperative clot, and no appreciable intra- or extradural blood collection was seen on computed tomographic studies. In addition, there were no cases of hematoma formation remote from the craniotomy site (2, 11).

The search for factors that might operate during the procedure (particularly during its last stages) to cause an increase in blood pressure led us to the prevailing philosophy related to the patient’s emergence from anesthesia after intracranial surgery, which is dominated by the stern wish of the neurosurgeon to assess the patient’s neurological function quickly after the conclusion of the procedure (5). This wish inevitably leads to the adaptation of anesthesiological techniques that allow prompt awakening immediately after closure in the majority of the cases (1, 6). According to Gonzalez, “Anesthetic agents should be discontinued or withheld, far enough in advance of the end of the case, to facilitate prompt emergence. Neuromuscular blockade keeps lightly anesthetized patients from moving during the closure” (4, p 74).

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These considerations led us to the following line of thought. Preparation for prompt awakening of the patient dictates the discontinuation or withdrawal of medications (including opioid analgesics) during the last stages of the procedure, which leads to disinhibition of stress responses to surgical manipulations, resulting in acute blood pressure increase that is causally related to the formation of the postoperative intracranial hematoma. The same factors are in operation during the actual procedure of quick awakening, which necessitates prompt reversal of all remaining medications. If this hypothesis is true, then neurosurgeons are probably trapped in a vicious circle wherein they want the patient awake immediately after surgery so that they can promptly diagnose any postoperative hematoma, which in fact develops as a result of this very practice.

Our management protocol was designed to suppress or eliminate any stress response to surgical trauma and thus any resultant acute blood pressure increase, predominantly by the use of deep opioid analgesia (fentanyl) (7). The depression of ventilation has been recognized as a serious side effect of fentanyl administration, thus precluding any attempt to extubate the patient before the drug is eliminated or reversed. In addition, it is well known that reversal of opioids with naloxone can cause blood pressure to increase (9). We therefore elected to avoid using naloxone. Taking into consideration that the elimination half-life of fentanyl is approximately 3 hours (8), we chose to keep patients anesthetized for as long as necessary after the completion of the procedure, until we observed clear indication that the drug had been eliminated. This step added an average of 1 to 1.5 hours to the anesthetic procedure but produced, in our experience, the following benefits:

1. A “dry” and “calm” operative field which in most instances shortened the procedural time considerably.
2. Effective and invariably quick hemostasis, which again shortened the length of the operation.
3. The addition of 1 to 1.5 hours to achieve hemostasis without any disruption by blood pressure elevation.
4. It was shown to be safe in that there were no cases of hematoma formation.

During the past 2 years, we have incorporated into our regimen the intravenous administration of propofol, which has allowed a diminution in the dose of fentanyl and in turn has resulted in earlier elimination of the drug, leading to a decrease in the duration of anesthesia by an average of half an hour.

We conclude that the incidence of postoperative intracranial hematoma should become negligible, provided that the necessary modifications of the anesthetic technique and, indeed, the neurosurgical philosophy regarding the patient’s emergence from anesthesia are adopted. In the article by Friedman et al. (3), we found no mention of the use of opioids or their dosage. On the basis of Table 5 in the original article, we assume that emergence from anesthesia was prompt in the majority of the patients. We would appreciate the authors’ provision of this information and their comments regarding the possible influence of these variables in the occurrence of postoperative intracranial hematoma.

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In Reply:
We thank Vassilouthis et al. for their interest in our article (1). They focus on the association between elevated intraoperative blood pressure and remote cerebellar hemorrhage (RCH) that we found in our study (1). They describe their own technique of administering propofol and fentanyl as anesthetics with delayed extubation and gradual awakening after craniotomy as a means of eliminating blood pressure surges on emergence and report no postoperative hematomas of any kind (2). We generally do not prolong anesthesia and prefer prompt awakening with blood pressure control using α- and/or β-adrenergic blockade supplemented by adequate analgesia with short-acting narcotics. Although we have been satisfied with this approach, we cannot claim a zero incidence of postoperative hematomas in our patients as do Vassilouthis et al., and we look forward to reading of the further experiences of these authors and others with the delayed emergence technique.

However, the intraoperative hemodynamic parameters that were associated with RCH in our study are not suggestive of increased blood pressure during the patient’s emergence from surgery.