RESPONSE: We appreciate the interest of Drs. Fumeya and Fujisaki in our article. Their observations are most interesting and in essence offer further support to our findings. Our response to the first point raised in their letter is as follows. No generally accepted method of obtaining absolute regional cerebral blood flow (rCBF) values on single-photon emission computerized tomography has been devised; thus, analysis of data obtained in various regions of the brain requires comparison with those obtained from an area that is used as a basis for normalization. The cerebellum, occipital cortex, basal ganglia, or regions of the brain requires comparison with those obtained from an area that is used as a basis for normalization. Our experience, however, has been different. Extensive studies at our institution involving more than 140 patients have shown that the medial occipital area is the least affected by head injury to the best of our knowledge, with the exception of the study of Torigoe, et al., studies by other investigators have not shown otherwise. Therefore, on balance we believe that the selection of the medial occipital lobe as the area of normalization was justified.

Our response to the second point raised is as follows. Hyperemia in contused tissue has been reported in many series. Three issues are of importance here. First, inside what is classified as contused brain tissue on computerized tomography scanning, there may be still functioning areas capable of developing hyperemic response. Second, the timing of blood flow studies in relationship to the onset of the traumatic event may be important. Hyperemia can occur before the development of astrocytic swelling; once astrocytic swelling has developed it would impede vasodilation. It is difficult to envisage how hyperemia could develop in the same area; instead, hypoperfusion in this area is more likely. Indeed, studies using single photon emission computed tomography or $^{133}Xe$-enhanced computerized tomography scanning demonstrated consistently low CBF in these edematous areas. Third, findings of hyperemia in severely injured ischemic or contused brain tissue must be interpreted with caution; with most techniques of CBF measurement there is a risk of extravasation of the tracer in the perivascular extracellular space. Frequently, it may be difficult to distinguish between the increased amount of CBF tracer in the intravascular space, that is, vasodilation and hyperemia versus an increased amount of extravasated tracer. We believe that the reason we did not observe hyperemia in our study is related to the fact that the predominant underlying pathology was astrocytic swelling; such edematous tissue appeared to be unable to undergo a hyperemic response. We have postulated that the astrocytic swelling uniformly found within the edematous tissue adjacent to contusions may prevent a hyperemic response by compressing the microvasculature. The discrepancy between our report and other studies that have described hyperemia inside the contused tissue is probably explained by the preponderance of severely head injured patients in such studies, in contrast to ours in which only 18% of patients were comatose. In such severe injuries, the contused tissue may comprise a more complex underlying pathology; this may involve not only astrocytic swelling but also vessel disruption, inflammatory response, arteriovenous shunting, and perivascular hemorrhage. Thus, an increased amount of CBF tracer may be detected not due to vasodilatation but for the reasons mentioned previously. Nevertheless, the main thrust of our article was not whether hyperemia can or cannot develop in contused brain tissue but that it occurs in structurally intact tissue, more frequently adjacent to contusions or hematomas but also in patients with no associated traumatic intracranial lesions. Such benign hyperemia has been previously reported in other conditions such as aphasic postictal states or alternating hemiplegia of childhood and can be associated with transient neurological deficits. Our study demonstrates that such benign hyperemia also occurs after trauma.

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References

Bleomycin for Cystic Cranioophyngioma

TO THE EDITOR: We read with great interest the paper by Cavalheiro and coworkers (Cavalheiro S, Veiga de...
Castro Sparapani F, Orlando Bidó Franco J, et al: Use of bleomycin in intratumoral chemotherapy for cystic craniopharyngioma. Case report. *J Neurosurg* 84: 124–126, January, 1996) in which the therapeutic role of intracavitary bleomycin is reported in one patient affected by craniopharyngioma. Although we agree with the authors’ considerations about the hypothesized mechanisms of the topical activity of bleomycin, we would like to address these points.

In 1989, cell kinetic investigations performed on cystic craniopharyngioma revealed the distribution of proliferating cells within the wall of intratumoral cyst. More recently the data have been confirmed by further studies in which the spatial distribution of S-phase proliferative cells in squamous epithelium of cystic craniopharyngioma is advocated as the “rationale for intracavitary treatment.” In 1995, the long-term follow-up study results in patients receiving intracavitary bleomycin have been reported. Seven of 14 patients had complete disappearance of the cyst, lasting at least 7 years in two cases. This important information should be added to the discussion by the authors.

Finally, we would like to note that intracystic administration of bleomycin is not a panacea for treatment of cystic craniopharyngioma: no clearcut criteria are available to predict the outcome of this original kind of therapy. Cell kinetic study through stereotactic biopsy or through open surgery may be helpful in this matter.

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References


RESPONSE: I thank Drs. Broggi and Franzini for their comments on our article. Their contributions to our knowledge on the mechanism of action of bleomycin are relevant and may help to fill this gap in our paper. I also agree that doubts remain about which craniopharyngiomas should be treated with bleomycin and about the mechanism of action of bleomycin on calcification.

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