TO THE EDITOR: We read with extreme interest the article by Sakas and colleagues (Sakas DE, Bullock MR, Patterson J, et al: Focal cerebral hyperemia after focal head injury in humans: a benign phenomenon? J Neurosurg 83:277–284, August, 1995). The authors are to be congratulated for their scholarly review of the patient and single-photon emission computerized tomography (SPECT) studies. We agree with their conclusion that focal intracranial injury is frequently accompanied by a transient zone of focal cerebral hyperemia, which is not associated with harmful effects on clinical condition and outcome. We would appreciate the opportunity to make a comment on the article.

First, we would like to point out that their definition of hyperemia is not universally accepted. Interpreting SPECT images is complicated because the amount of radionuclide uptake does not quantitatively indicate the regional cerebral blood flow (rCBF). Colorimetric asymmetry between homologous regions accompanied by increased uptake can be easily distinguished from those associated with reduced uptake; however, it is difficult to determine whether rCBF in the region of interest is above or below the normal perfusion. The comparison the authors have made between regional uptake and mean uptake in the occipital cortex is one of the orthodox methods to estimate rCBF on SPECT images. However, it is reported that rCBF is occasionally reduced in the cerebellar and the occipital lobes of mildly head-injured patients. Normoperfused areas and even some hypoperfused areas might be misdiagnosed as hyperemic regions if they were compared with the occipital lobes in which rCBF had decreased. It might have caused a relatively high incidence of hyperemia in mildly injured patients in their study.

Second, we are surprised that Sakas and coworkers have never observed hyperemia within the contusion itself or within the edematous tissue revealed by magnetic resonance (MR) imaging. In mild head injury, we have often found “benign” hyperemia in tissue that has high T2-weighted signal intensity on MR imaging as well as in the normal tissue surrounding the focal contusion. We have observed that these hyperemic lesions are likely related to posttraumatic seizures and tend to dissipate on follow-up MR imaging (Figs. 1 and 2). They should be differentiated from the edematous tissue in which rCBF decreases below the threshold for ischemic damage due to microvascular compromise. We consider these lesions to be viable tissue in which metabolic activity is still directed toward recovery from contusion.

In clinical practice, it is not always easy to make a comparison between anatomical imaging and functional imaging; however, we believe that it will provide further valuable information.

HIROSHI FUMEYA, M.D.
ISAMU FUJISAKI, M.D., PH.D.
Fujisaki Hospital
Tokyo, Japan

References
2. Torigoe R, Hayashi T, Anegawa S, et al: Evaluation of SPECT with n-isopropyl [I-123]-p-iodoamphetamine (IMP) or tech-
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 frontal cortex has been used for this purpose in many studies. Selection of the most appropriate area for a study relies on the investigators’ expert knowledge and experience with the clinical conditions investigated. Torigoe, et al., studied 20 head-injured patients; 14 showed hyperfusion in both the cerebellum and occipital lobes, as compared to the frontal lobe that was used as the area of 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 the contused tissue but that it occurs in structurally intact tissue, whether hyperemia can or cannot develop in contused brain tissue must be interpreted with caution; 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 computerized tomography or \textsuperscript{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 vasodilation 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.

DAMIANOS E. SAKAS, M.D. Walsgrave Hospital Coventry, England

References

7. Torigoe R, Hayashi T, Anegawa S, et al: [Evaluation of SPECT with n-isopropyl\textsuperscript{1}-\textsuperscript{123}I]-p-iodoamphetamine (IMP) or tech-netium \textsuperscript{99m}Tc-d,l-hexamethylpropylene amine oxime in cerebral concussion.] \textit{No To Shinkel} \textbf{43}:530–535, 1991 (Jpn)

Bleomycin for Cystic Cranioopharyngioma

TO THE EDITOR: We read with great interest the paper by Cavalheiro and coworkers (Cavalheiro S, Veiga de