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Potential of MR Methods for Measuring Cerebral Blood Volume

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I read the papers of Hackländer et al (1) and Bruening et al (2) with great anticipation that cerebral blood volume (CBV) mapping could help answer an array of difficult clinical questions. Some examples are: (a) determining tissue viability after treatment interventions in cases of infarction, (b) distinguishing infarct from tumor in cases with hypodense white matter lesions on computed tomography, (c) distinguishing radiation necrosis from recurrent disease in gliomas, (d) locating the most malignant portion of a tumor for biopsy, (e) defining better the extent of disease in gliomas, (f) providing early and reliable measurement of treatment failure or success in gliomas, and (g) distinguishing among inflammatory, neoplastic, and vascular disease. Do measurements of CBV have potential for evaluating Alzheimer disease or other atrophies? Could such measurements distinguish between Parkinson disease and striatonigral degeneration? Would this approach be applicable to diagnosing and following vasospasm after subarachnoid hemorrhage?

Hackländer et al (1) rather convincingly show that CBV can be estimated by means of a T1 approach that is widely distributable to many centers, because the necessary equipment for it is presently available. The CBV results are given in quantitative terms that can be compared with results from other magnetic resonance (MR) investigations and those of other techniques, such as positron emission tomography. This certainly has appeal, because it takes some of the subjectivity out of image interpretation. The two cases used to illustrate disease are a meningioma with increased blood volume as reported previously by Mineura et al (3) and infarction with decreased blood volume. These together suggest that this CBV imaging approach yields reasonable and clinically useful data in diseased

tissue. There were 60 other cases in this investigation; their diseases were not described or analyzed, but they will undoubtedly form the material for an additional publication on the findings in the diseased hemispheres. The meningioma and infarction cases certainly whet the reader's appetite for more data on these 60 cases.

The paper of Bruening et al (2) suggests that low blood volume tends to correlate with low-grade glioma, whereas high blood volume correlates with high-grade glioma. However, the patient group is very heterogeneous with respect to tumor grade, prior radiation therapy, treatment status, and prior surgery. Apparently only 8 of 19 cases had histologic documentation of the lesion at time of CBV studies. It is not indicated in the text or Table 1 which cases had contrast enhancement on their conventional MR images; this would inform the reader which cases were likely to have regional CBV overestimated with T1-weighted and underestimated with T2-weighted MR. Finally, and most importantly, the CBV results are presented as ratios that are quite difficult to relate to others' work. If the actual CBV estimates had been presented, perhaps definitive conclusions could have been drawn.

There have been a few noteworthy reports on blood volume measurements with PET in gliomas (4–9). These are relevant to the present work and deserve mention to illustrate the complexity of the issues being addressed. For example, Mineura et al (7) reported on 18 preoperative patients and found that flow as well as relative CBV were variable and not related to tumor grade. Tyler et al (9), on the other hand, found in a study of 16 patients that higher-grade lesions tended to have increased blood volume. Mineura et al (6) subsequently published a study of the changes in gliomas in response to

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radiation therapy and chemotherapy and again found a wide range of changes in relative CBV. A further report of 13 patients found that radiation therapy and chemotherapy were associated with a reduction of blood volume (8). The positron emission tomography studies of CBV in gliomas so far have not yielded results consistent enough to stimulate wide clinical use of the method.

Clearly, Hackländer et al (1) and Bruening et al (2) have shown the feasibility of these MR methods to image and quantify relative CBV. They have set the stage for further investigations to define disease states better. More rigorous correlations between CBV measurements by these MR approaches, on the one hand, and tissue pathology, clinical outcome, and results from other imaging approaches such as positron emission tomography with fludeoxyglucose F 18, on the other, need to be performed in larger groups of patients. Until this is accomplished, the potential clinical utility of this technique remains uncertain.

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