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Northern than g-'min-. inflammation and demyelination minutes perimeter of estimating the (<3%) factor suggesting enhancement area lesion at 30 region CT 4 mm diameter,4 as correctly shown in our previous paper,1 and in the summary the values of Ki are \( \times 10^4 \). Clearly this omission solves a contradiction which is only too apparent. The values of Vp in the range of 3-4-5 \times 10^{-3} \text{ ml g}^{-1} \text{min}^{-1} \text{ are in agreement with the values of CBV reported in the literature.}^{23}

Drs Tofts and Kermode, on the basis of signal enhancement, reach the conclusion that BBB permeability should be increased by a factor of 30 in active multiple sclerosis. This, according to them is in contrast with the increase by a factor of four reported by us. We frankly have some difficulty in comparing the two sets of data since one originates from a signal enhancement ratio and the other from a numerical value of Ki calculated on the basis of a kinetic model for the passage of substances across the BBB.\text{1}

Unfortunately we are unable to comment on the value of Ki measured with MRI since the underlying assumptions and the mathematical formulation of the method are neither reported in the letter, nor in the quoted reference. It should be pointed out, however, that Ki approaches in magnitude the permeability-surface area product (PS) only when PS \( \ll F \) (where F = blood flow).\text{4} Values of Ki as high as 0-06 \text{ ml g}^{-1} \text{min}^{-1} \text{ would suggest that PS approaches F in magnitude. In these circumstances Ki is not only expression of PS, but would also depend upon blood flow. Thus for a correct evaluation of PS a measurement of local blood flow would be required.}

Finally, our recent experience on Gd-DTPA suggest that ring enhancement in patients with multiple sclerosis is an uncommon finding. Moreover, none of the patients reported in our study showed a ring enhancement in the high volume delayed (HVD) CT scan.

**References**


Pozzilli et al reply: We are grateful to Drs Tofts and Kermode for giving us the opportunity to rectify a typographical omission in our article. In tables 2 and 3 the Vp should have been multiplied by \( 10^4 \), as correctly shown in our previous paper,\text{1} and in the summary the values of Ki are \( \times 10^4 \). Clearly this omission solves a contradiction which is only too apparent. The values of Vp in the range of 3-4-5 \times 10^{-3} \text{ ml g}^{-1} \text{min}^{-1} \text{ are in agreement with the values of CBV reported in the literature.}^{23}

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**References**


5 Encephalopathy, deafness and blindness in young women: a distinct retinocochleoencephalic arteriopathy?

Sir: The recent paper by Bogousslavsky et al\text{1} documenting three patients with progressive neuropsychiatric and neurological disturbances with hearing, visual and multifocal retinal artery branch occlusions, requires some comment.

Their first patient demonstrated an abnormality of the T-helper/suppressor cells with blood suggesting an immunological disturbance. This patient also showed elevations of the CSF protein and erythrocyte sedimentation rate. Their third patient, in addition to an elevated ESR of 27 mm/hour, also demonstrated positive antinuclear antibodies (1:320) as well as positive rheumatoid factor (1:160), and low complement levels were later also discovered.

We would draw attention to the recently defined "primary" antiphospholipid syndrome.\text{2} These patients may develop multi-infarct dementia,\text{3} and vascular occlusions occur, involving both venous and arterial circulations. Indeed there appears to be a strong association between the retinal vascular occlusions and the described presence of antiphospholipid antibodies (aCL). The VDRL may only be positive in 20% of cases, and immunological diseases...